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Treatment for Anomia in Semantic Dementia

Maya L. Henry, M.S.¹, Pélégie M. Beeson, Ph.D.^{1,2}, and Steven Z. Rapcsak, M.D.^{2,3}

¹ Department of Speech, Language, and Hearing Sciences, University of Arizona, Tucson, Arizona

² Department of Neurology, University of Arizona, Tucson, Arizona

³ Neurology Section, Southern Arizona VA Health Care System, Tucson, Arizona

Abstract

Anomia is a striking and consistent clinical feature of semantic dementia (SD), a progressive aphasia syndrome associated with focal cortical atrophy of the anterior temporal lobes. Word retrieval deficits in patients with SD have been attributed to the loss of conceptual knowledge, resulting in an impairment referred to as semantic anomia. Whereas an abundance of research has been dedicated to treatment for anomia in individuals with focal brain damage due to stroke, considerably less work has been done regarding treatment for patients with progressive language decline. The purpose of this article is to review the available literature concerning the nature and treatment of anomia in individuals with SD. Several studies have shown that new lexical learning remains possible in these patients. However, newly learned information is likely to be constrained by the learning context, and increased reliance on perceptual and autobiographical contextual information may be necessary to provide critical support for new vocabulary acquisition. There is also evidence suggesting that treatment may slow the progression of anomia over time, even affording some protective benefit to lexical items that are not yet lost. However, treatment efforts are likely to be most beneficial at early stages of the disease, when residual semantic knowledge as well as relatively spared episodic memory may support new learning.

Keywords

Naming; rehabilitation; lexical retrieval; memory; primary progressive aphasia

Cognitive models of language hypothesize that spoken naming involves both semantic and phonologic levels of processing. In general, abstract conceptual information, or semantic knowledge, is accessed first, followed by the activation of a corresponding phonologic word form in the lexicon.^{1,2} Subsequently, peripheral procedures are enacted, including the planning and execution of motor movements for spoken word production. Lexical retrieval impairment, or anomia, may result from damage to semantic or phonologic representations, or it may reflect impaired transmission of information between these cognitive components. Anomia resulting from loss of conceptual knowledge is referred to as *semantic anomia*, which can be distinguished from lexical retrieval impairment caused by postsemantic deficits involving impaired access or damage to phonologic word forms.³ This paper will focus on semantic

Address for correspondence and reprint requests: Maya L. Henry Department of Speech, Language, and Hearing Sciences, University of Arizona, Tucson, P.O. Box 210071, 1131 E. 2nd Street, Tucson, AZ 85721-0071 (e-mail: E-mail: maya@email.arizona.edu).

Learning Outcomes: As a result of this activity, the reader will be able to (1) list characteristic features of the language impairment observed in semantic dementia, (2) discuss the manner in which different types of memory may contribute to learning in individuals with semantic dementia, and (3) enumerate general principles for assessment and treatment in individuals with semantic dementia.

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333 Seventh Avenue, New York, NY 10001, USA. Tel: + 1(212) 584-4662.

anomia in individuals with the progressive aphasia subtype known as *semantic dementia* (SD), a fluent aphasia syndrome characterized by a gradual deterioration of conceptual knowledge.

Converging evidence from studies of aphasic patients with focal brain lesions and functional imaging studies in normal individuals suggest that semantic processing is performed by a network of extrasylvian cortical regions (see Antonucci and Reilly, this issue). Components of this distributed neural system include left inferior prefrontal cortex (BA 47), bilateral anterior and inferolateral temporal cortex (BA 38, 20/21), and the left posterior temporoparietal region (BA 37/39).^{4–6} The specific contribution of each of these regions is yet to be determined conclusively. Jeffries and Lambon-Ralph⁷ posited that frontal areas contribute to executive processes that allow semantic information to be accessed and manipulated in a task-specific manner, collectively referred to as *semantic control*. With regard to the role of the temporal lobes, left posterior inferior temporal regions (BA 37) may be important for the semantically based retrieval of phonologic word forms.⁶ Finally, a dominant but not undisputed view of semantic organization holds that amodal conceptual representations that comprise the semantic memory store are supported by the anterior and inferolateral temporal lobes bilaterally^{8,9} (see Antonucci and Reilly, this issue, for a more comprehensive review concerning the organization of the semantic system and its neural substrates). Because of the nature of the vascular supply of the brain, the anterior temporal lobes are rarely damaged in isolation in patients with stroke.¹⁰ However, damage to this region is a consistent finding in SD and thus is widely considered to be the neural substrate of the pervasive semantic memory impairment that is the hallmark of this condition.¹¹ Furthermore, although a variety of neurodegenerative disorders including Alzheimer's disease (AD) may be associated with anomia,¹² research exploring the underlying factors contributing to poor performance on semantic tasks in these disorders¹³ suggests that SD represents the most pure degradation of semantic knowledge.

SD: SPARED AND IMPAIRED COGNITIVE PROCESSES

In SD, the progressive deterioration of conceptual knowledge will affect performance on all tasks that rely on semantic memory. The pattern of deficits, however, can be expected to evolve over time as the semantic loss becomes more pervasive. Early in the disorder, the language profile may resemble that of anomic aphasia resulting from stroke, with word-finding difficulties in conversation manifesting as circumlocutions (e.g., “we went to the place where you buy things” for *grocery store*).¹¹ The anomia will be especially pronounced, however, on category fluency and confrontation naming tasks. Errors on confrontation naming tasks include omissions, coordinate errors (e.g., “cat” for “dog”), and superordinate errors (e.g., “animal” for “fox”).¹⁴ Phonemic cueing is unlikely to confer any benefit to these patients.¹¹ As the disease progresses, the naming impairment becomes more apparent during conversation, with patients exhibiting “empty” language, semantic paraphasias, and frequent pauses.^{11,15}

Although anomia is the most pronounced feature early in the illness, the deterioration of conceptual knowledge ultimately manifests as deficits in comprehension of language. Both naming and comprehension impairment are likely to be frequency-dependent, with more pronounced deficits on lexical items that have a lower frequency of occurrence.¹⁶ Tasks tapping nonverbal semantic processing, including picture sorting by category and tests of semantic association, such as the Pyramids and Palm Trees test,¹⁷ will ultimately reveal impairments as well. At some point in the disease progression, individuals may also show face and object recognition deficits (prosopagnosia and associative agnosia, respectively).¹⁵ Language abilities and other cognitive skills that do not rely on semantic knowledge are typically spared, including phonologic and syntactic processes, visuospatial skills, and, to some extent, episodic memory¹⁸ (i.e. the ability to remember personally experienced events).¹⁹

As noted earlier, the underlying pathology in SD affects the anterior and inferolateral temporal lobes.^{6,12,20–23} Atrophy may involve both temporal lobes; however, it is frequently asymmetric, with earlier and greater loss of tissue in the left hemisphere.^{11,24,25} Correlations have been observed between left (and in some cases, right) anterior and inferolateral temporal lobe atrophy and performance on semantic measures^{22,23,26} and on naming tests specifically,^{21,23} providing confirmatory evidence that this region is the critical neural substrate of the language impairment in SD (but see Grossman et al¹²). Much of the early-work examining patterns of cortical atrophy indicated a sparing of medial temporal cortex (i.e., the hippocampus and related structures) in SD (e.g., Mummery et al^{6,22}), which may account for the relative preservation of episodic memory in semantic dementia.¹¹ Thus, theories of new learning in SD have suggested that spared medial temporal lobe structures might support the acquisition of new information in these patients.

Recent attempts to discover the exact location and extent of cortical atrophy in SD indicate that it is prudent to take a more guarded view regarding sparing of the hippocampus,^{23,27} as many patients demonstrate atrophy in this structure at some point in disease progression. Furthermore, it appears that episodic memory tests do not reliably distinguish SD from AD patients.²⁸ Recent work by Nestor and colleagues,²⁹ however, indicated that, whereas medial temporal lobe atrophy is present in both SD and AD populations, other limbic-diencephalic structures (e.g., the mammillary bodies, thalamus, and posterior cingulate) remain intact in SD, and these structures could be capable of supporting new episodic learning.

Theoretical models have varied in terms of how they view the role of potentially dissociable semantic and episodic memory systems in new learning.^{30,31} Nonetheless, there is evidence that new episodic learning is possible in individuals with impaired semantic memory.³² There is also evidence for dissociation in the opposite direction, namely, that new semantic learning is possible in the absence of episodic memory, as in the case of amnesia resulting from damage to the medial temporal lobes.³³ Although it appears that new learning can be achieved in both SD and amnesia, some research suggests that without the normal interaction between complementary learning systems in neocortex and the hippocampus, flexible use of newly acquired memory representations is difficult if not impossible.³⁴ In fact, new semantic learning in amnesia has been described as “hyperspecific,”³⁵ and likewise, there is evidence that new learning in SD is also extremely rigid.³¹

Graham and colleagues³² found that recognition memory is largely preserved in SD patients, as long as perceptual qualities of stimuli are held constant from learning to test. The authors suggested that both perceptual and conceptual inputs support new episodic learning in normal individuals. In the case of absent or reduced input from the conceptual store, perceptual information becomes increasingly critical (hence the poor performance of SD patients on recognition memory tasks where perceptually different exemplars are presented at learning vs. test). Thus, new learning in SD is considered by Graham and colleagues to be bound by the perceptual qualities of the initial learning condition, because degradation of the neocortical systems that support semantic memory does not allow generalized semantic information to contribute in the normal way.³²

EARLY EVIDENCE FOR SUCCESSFUL LEXICAL LEARNING IN SD

The value of behavioral intervention for the naming impairments associated with SD has been examined in only a few single-subject studies. In the earliest, Graham and colleagues^{36,37} capitalized on the attempts of one of their SD patients (D.M.) to remediate his own naming impairment through intensive, self-directed practice of forgotten vocabulary. These researchers examined changes in naming (on verbal fluency tasks) and semantic knowledge longitudinally as his disease progressed. D.M.’s home practice included picture naming and naming from

verbal descriptions (using the *Oxford English Picture Dictionary* as well as self-generated notebooks containing pictured objects and faces). His response to this work was compared with that of another, albeit more severe, SD patient (A.M.) who also attempted to remediate word-finding deficits on his own.

The authors asked D.M. to practice naming items from two sets of semantic categories (with six categories in each set) for a period of 2 weeks per set. After the period of home drill, verbal fluency performance was assessed for practiced as well as nonpracticed categories and was compared with that of a group of control participants. Results indicated that, through extensive training, D.M. was able to improve category fluency performance in an item-specific manner. He demonstrated significant improvement on practiced categories, with performance actually surpassing that of an age-matched normal control group, whereas matched, nonpracticed categories revealed little change. D.M.'s performance on practiced categories deteriorated after a period of no practice, however not to baseline levels³⁶

After a 2-year period during which D.M. continued his home practice with lists of words and pictures, another evaluation was conducted. At that point, Graham and colleagues³⁷ found that, despite D.M.'s continued efforts, category fluency performance had dropped substantially, returning to baseline levels. Further, at the follow-up assessment, D.M. produced items that did not belong to target semantic categories and was unable to provide semantic information for several items that he named during fluency tasks. These findings suggest that performance on these tasks was not semantically driven. The authors also documented a steady decline in performance on a nonverbal semantic measure (the Pyramids and Palm Trees test¹⁷) over the 4-year period when testing took place. The authors noted that D.M.'s decline in category fluency performance was not likely due to a decrease in time spent on vocabulary practice. In fact, at the 2-year follow-up, he was reported to be spending 7 to 8 hours per day studying his materials, having become increasingly dedicated to his efforts.

The work of Graham and colleagues demonstrated that an individual with SD was able to improve naming performance from baseline levels and to maintain this improvement for several years into his illness, despite a relentless deterioration of semantic knowledge. The authors suggested several factors that might have contributed to D.M.'s success, particularly relative to the performance of A.M., who did not show improved naming, despite extensive practice. D.M.'s practice regime included both phonologic and semantic stimulation, whereas A.M.'s rehearsal of vocabulary involved studying lists of words organized by first letter. Indeed, treatment research with individuals with stroke-induced aphasia suggests that activation of both semantic and phonologic representations during naming treatment may be of greater benefit than phonologic or semantic stimulation alone.^{37–41} In addition, D.M.'s anomia and his underlying semantic impairment were less severe than A.M.'s, suggesting that new learning may be possible early in the course of semantic memory loss but become increasingly difficult as the semantic system is eroded. Finally, Graham and colleagues³⁷ suggested that the nature of D.M.'s practice may have been inherently "errorless," which could have been a contributing factor in his ability to relearn lost vocabulary.

The authors offered several caveats to the encouraging results of D.M.'s practice regimen. First of all, they concluded that his improved verbal fluency was not a result of a restoration of semantic knowledge. Rather, they attributed his gains to rote memorization, maintained through continued and intensive practice. Second, the authors noted anecdotally that D.M.'s attempts at self-rehabilitation ultimately became obsessive and that they had negative effects on his psychological well-being.

The work of Graham and colleagues set the stage for further exploration of the capacity for patients with SD to relearn "lost" lexical items. They also raised the question of whether such

learning constitutes a rebuilding of the semantic store or some other compensatory learning processes that are reliant on spared cognitive and neural mechanisms.

RESEARCH EXAMINING MECHANISMS FOR NEW LEXICAL LEARNING IN SD

Snowden and Neary⁴² were intrigued by observations of new learning in SD patients' daily lives (such as learning new peoples' names), despite their poor performance on traditional verbal or pictorial memory tests in the laboratory.³¹ They proposed that relearning of verbal labels in SD requires some substrate in temporal neocortical regions. This "tag" on which to hang new phonologic word forms could be residual semantic knowledge or personalized contextual information (temporal and/or spatial) regarding a particular target. To explore this hypothesis, the authors examined the capacity for verbal learning in two individuals with SD characterized by severe anomia and comprehension deficits.

Snowden and Neary's first patient was described as having a "severe semantic disorder," showing impaired performance across a range of semantic measures. The study examined the effects of learning trials wherein the patient was presented with pictures of targets along with the corresponding written word. The patient was instructed to read the name aloud and to examine the picture; subsequently, the item's name was repeated by the clinician. In selecting items for treatment, the authors identified a set of items that the patient could not name, and the degree of residual semantic knowledge held by the patient for those items was assessed. Items were designated as "partially known" if the patient could provide defining information for the spoken word or pictured item or correctly match the word to its picture; otherwise, the item was designated as "unknown." Three successive learning trials were conducted, followed by a 2-week period without practice and then another three learning trials. Naming assessments were conducted before and after each learning trial. Mild improvement in naming was observed, which was maintained when retested at 2 weeks but not at 4 months. The researchers observed that relearning lexical items was mediated by the degree of residual semantic knowledge available for specific items. In fact, during probes, only one item was named for which the patient demonstrated no residual conceptual knowledge prior to treatment.

With their second patient, who also demonstrated a profound impairment of semantic knowledge, Snowden and colleagues added meaningful, personalized contextual cues, referred to as "experiential links," to the learning trials. In addition, the treatment was expanded to include a 3-week period of self-study with the pictured objects and their written names. During this self-study period, the personalized experiential links were provided by the patient's husband only when requested. After the 3-week period of self-study, the treatment materials were withdrawn; however, it was noted that the patient resumed self-study with her own materials in the days prior to subsequent evaluations. The authors observed an improvement in naming performance that was maintained for more than 1 month, with some decline in performance after 6 more months. It was noteworthy that some cues proved to be more meaningful to the patient than others, and the items associated with the more personally meaningful cues showed more durable treatment effects over time. In addition, the patient's performance appeared to be reliant on contextual information from training sessions. Specifically, naming accuracy suffered when items were presented in an unfamiliar context (e.g., on different-colored paper and in an unfamiliar order).

The performance of these two patients suggests that new learning in SD is possible, but that learned information may differ qualitatively from the rich semantic representations of unimpaired individuals. Relearned verbal labels in the first patient appeared to be supported by residual semantic knowledge and in the second patient by spatiotemporal and autobiographical information from recent personal experience. In the latter, this contextual and autobiographical support enabled her to relearn lexical targets for which she had seemingly no

semantic knowledge at the start of treatment. The authors were careful to note, however, that these newly acquired conceptual representations did not conform to the traditional definition of context-free semantic knowledge. They also predicted that the newly learned information would probably be lost without continued rehearsal, which is consistent with the notion that hippocampus-based, episodic learning is likely to decay more rapidly or to be “overwritten”.^{36,42} This study suggests that personalized autobiographical or spatiotemporal cues may be a means to support lexical learning, even though they may not facilitate true relearning of rich, elaborated, and lasting semantic representations.

Jokel and colleagues^{43,44} implemented a treatment program with an individual with SD characterized by a marked anomia and single-word comprehension impairment. They examined the relationship between word comprehension and the ability to relearn specific lexical items. Jokel and colleagues provided their patient with study materials consisting of a picture, a written word, and a brief description of the item. Three sets of 30 pictures were studied at home for 1 week each (30 minutes per day). The items included a set that was named and comprehended prior to treatment, one that was not named but was comprehended, and one that was neither named nor comprehended. All items selected for treatment were identified as being important to the patient, and definitions used in the treatment were based on personally relevant information. Results indicated a significant change in performance for items that were not named pre-treatment, with greater and longer-lasting effects for items that were comprehended prior to treatment. These findings support the notion that the degree of residual semantic knowledge, as indicated by performance on comprehension tasks, is a critical factor in relearning lexical items. There was also evidence that practice with items that could be named prior to treatment may have slowed the progression of semantic loss and anomia for those items. The authors proposed that the use of individually tailored and personally relevant items in treatment was an essential factor in this patient’s success, again suggesting that relevance of treatment targets and training materials to a patient’s recent personal experience can be a critical element in retraining vocabulary in SD.

ERRORLESS LEARNING IN SD

The above two studies explored the role of residual memory systems (both semantic and episodic) in new lexical learning, and additional research has examined the utility of a specific learning approach, referred to as *errorless learning*, in treatment for anomia in SD. Errorless learning paradigms are those that are designed to avoid incorrect or unwanted patterns of behavior, in hopes of forestalling stimulus-induced errors inadvertently reinforced during training. Graham and colleagues³⁷ suggested that the errorless nature of D.M.’s training regime might have played a role in his successful vocabulary learning. Exploring this approach more directly, Frattali⁴⁵ developed an errorless naming treatment, which was implemented with one individual presenting with SD characterized by pronounced anomia accompanied by milder comprehension deficits. The treatment protocol involved the experimenter instructing the patient *not* to name a pictured item but to engage in conversation about the item. The clinician and patient discussed perceptual and functional attributes of the target, as well as categorical, analogic, and associative properties. The clinician guided the conversational interchange so that errorful productions and even struggle toward production were avoided by the patient. Naming the target was neither required or even acknowledged at this stage. Subsequently, the patient was shown target pictures again and asked to attempt to name items only if she thought she could do so successfully. These responses were recorded as the measure of treatment outcome. The patient showed posttreatment improvement in naming of both nouns and verbs. There was no generalization to untrained items or word classes, and all gains were lost at a 3-month follow-up. Thus, the errorless approach proved beneficial, but changes in behavior were transitory and item-specific.

In a second study examining errorless naming treatment in a patient with SD, Jokel and colleagues⁴⁶ explored the utility of a computer program, “MossTalk,”⁴⁷ wherein pictures and spoken descriptions of items were provided and the patient was instructed to attempt to name the target only if he was certain of his response. Three lists of nouns comprising both unknown and known items were addressed in treatment, with each list trained either to an 80% correct criterion or for 12 sessions. Treatment lasted for 4 months. Naming accuracy was maintained immediately posttreatment and at a 1-month follow-up. Known treated items were all named correctly posttreatment; however, known untreated items showed a decline in performance, suggesting that training with intact vocabulary may have contributed to its preservation. There was also generalization to untreated items on a picture-naming test. This study provides additional evidence in favor of the application of errorless learning principles in rehabilitation of anomia in SD, with preliminary evidence that generalization and maintenance of behaviors may be possible. However, because errorless techniques were not directly compared with those that allow the commission of errors, it is not possible to determine with certainty whether this was the critical factor in the positive treatment outcomes in these studies. In addition, the initial studies of lexical learning in SD showed that positive and even lasting benefits were achieved using paradigms that were not characterized as “errorless”.

ONE TREATMENT DOES NOT FIT ALL

The aforementioned studies suggest that rehabilitation of anomia in SD requires special consideration and that the nature of lexical learning in these individuals occurs through mechanisms that are different from the ones that support reacquisition of vocabulary in cases where semantic knowledge is not compromised. Thus, lexical retrieval treatment in SD should not necessarily be approached in the same manner as rehabilitation in cases of stroke-induced aphasia, or even other progressive aphasia syndromes. In a recent study,⁴⁸ we explored the relative benefits of a single-treatment paradigm in three individuals with highly similar spoken language profiles but differing etiologies and levels of cognitive deficit. We implemented a brief but intensive (16 day) semantically based treatment intended to promote strategic retrieval of lexical items within certain semantic categories, with verbal fluency as the outcome measure. Participants included one individual with SD, one with borderline-fluent primary progressive aphasia (PPA) with characteristics indicating a likely progression toward nonfluency, and one individual with anomic aphasia resulting from stroke. The latter two participants showed a relative sparing of semantic processing and thus were considered to have largely postsemantic deficits in naming.

The treatment included a variety of semantic tasks intended to stimulate lexical retrieval within trained categories (e.g., picture sorting by subcategory, generating semantic attributes of exemplars, and comparing and contrasting among exemplars). It also involved a strategic element intended to organize and facilitate the generation of exemplars (e.g., exhausting one subcategory before switching to another). This approach proved beneficial to the individual with borderline-fluent PPA and the focal lesion patient but not to the individual with SD. This individual exhibited considerable difficulty with the treatment tasks themselves. Further, he demonstrated a greater degree of impairment to episodic memory and had difficulty remembering lexical retrieval strategies from one treatment session to the next. The differential response to treatment in this study underscores the notion that treatment for anomia should take into account the nature of the deficit (e.g., semantic vs. postsemantic) as well as the presence of concomitant cognitive impairments that may limit the utility of the approach. Despite the fact that semantic tasks (e.g., picture sorting) and strategies (e.g., subcategorizing) are logical approaches to strengthening semantic representations, our findings show that their value is limited in patients with a marked deterioration of semantic knowledge. In fact, Reilly⁴⁹ recommended capitalizing on relatively spared phonologic (rather than semantic) processing abilities when addressing naming deficits in SD, as these skills tend to be better-

preserved longer into the illness. There should, of course, continue to be an emphasis on word meaning, however, as phonologic word forms must be linked with underlying concepts if vocabulary is to be used for meaningful communication.

CONSIDERATIONS FOR EVALUATION AND TREATMENT IN SD

The above-outlined research examining the rehabilitation of anomia in SD, though limited, has revealed some factors and considerations that may contribute to greater success in this endeavor. First, the issue of residual semantic knowledge is of critical importance. The differential response to treatment in Graham and colleagues' two patients was attributed partially to this factor, as well as the successful learning in patients in other studies.^{42–44,48} The available research indicates that acquisition of new lexical knowledge will be dependent on the availability of semantic support for those items. This can be conceived in a general way, such that individuals with greater deterioration of the semantic store are less likely to be responsive to naming treatment, and in a more specific fashion, with the capacity for relearning specific items being linked directly to the residual knowledge of those items in particular.

The findings from several studies support the idea that rehearsal of known lexical items may delay the progression of their loss.^{43,44,46} Reilly and colleagues⁵⁰ suggested that successful rehabilitation in SD should involve practice with a finite set of preserved, relevant lexical items, rather than attempts at restitution of lost vocabulary. This type of training would be inherently "errorless," as individuals would rehearse only items that they were still able to name with some success. Indeed, there is growing evidence suggesting that errorless learning may be a fruitful approach in individuals with SD.^{37,45,46}

An additional issue concerns the relative roles of temporal neocortex and the medial temporal lobes in mediating new learning in SD. It appears increasingly probable that individuals with some degree of sparing of the hippocampus and related structures may be able to achieve new lexical-semantic learning. This type of learning is likely, however, to be item-specific and constrained by autobiographical and spatiotemporal factors. Concepts relearned in this fashion can be expected to be less robust and also more susceptible to decay as the hippocampus, which is thought to have limited capacity, is forced to "overwrite" newly learned information,³⁶ and as the neocortical substrate critical for long-term maintenance of information in the temporal lobes becomes increasingly atrophic over time.

Nonetheless, the potential for SD patients to learn episodically highlights the importance of drawing upon personal, current experiences during treatment. The implications for therapy include use of personally relevant targets as well as cues that capitalize on recent experience. In addition, continued rehearsal of targets is probably necessary to maintain lexical items that are supported by more fragile and less elaborated conceptual representations. In fact, it is likely that targeted vocabulary will require frequent, ongoing rehearsal to prevent its loss in the face of inevitable and persistent erosion of the semantic store.

In addition to a comprehensive language evaluation, measures of episodic memory should be administered prior to implementation of a treatment plan. Both formal and informal assessment measures should be included, as patients may perform poorly on formal measures while retaining some functional skills for everyday life.³¹ The notion that new learning can be mediated by hippocampal structures, as temporal neocortex undergoes progressive deterioration, mandates that medial temporal lobe structures and the functional abilities they support (i.e., acquisition of new information) are reasonably intact. Given that atrophy is likely to spread to medial temporal structures at some point in disease progression,^{23,27} it is important that evaluation of memory systems be viewed as an ongoing endeavor.

It can be expected that treatment goals and strategies will shift over time with the changing needs and abilities of patients with degenerative disease.^{51,52} In SD, it has been proposed that the language impairment may evolve from primarily semantic in nature to involve phonologic processing later in the disease,⁴⁹ as cortical atrophy spreads beyond anterior and inferolateral temporal regions into the perisylvian language zone. Thus, a clear understanding of the status of semantic as well as phonologic processing abilities should be established prior to the initiation of treatment, as these will indicate what type of cueing and practice strategies may be most effective.

Treatment studies in SD thus far have highlighted the importance of the learning setting and the nature of treatment stimuli, suggesting that learning is likely to be constrained by the context in which it is undertaken. Newly acquired targets can be expected to be limited in their generalizability⁴² by both spatiotemporal context as well as perceptual qualities of items used in treatment. For this reason, items used in naming treatment should be actual items owned or used by the patient (or photos thereof), as verbal labels may not generalize to other exemplars. Treatment stimuli should also be carefully selected with regard to word frequency, as individuals are more likely to retain higher-frequency targets as their disease progresses.^{16,44}

CONCLUSION

The available research, albeit limited, examining treatment for anomia in individuals with progressive semantic memory loss suggests that a degree of optimism is warranted. Despite the inevitable decline in semantic knowledge in patients with SD, there is evidence indicating that spared neural and cognitive mechanisms can support new learning in these patients. It is also clear, however, that newly acquired lexical and conceptual information is not likely to be qualitatively as rich or as robust as that of a healthy individual. Furthermore, treatment for anomia may only be a realistic consideration at early stages of the disease, when lexical retrieval impairment is the primary complaint and residual semantic and episodic memory stores are still available to support new learning. Once comprehension deficits become pronounced and episodic memory significantly affected, restitutive treatment becomes less feasible.

Nonetheless, there is hope that some of the effects of the debilitating anomia suffered by these patients may be offset by an appropriate training protocol, at least as long as targets undergo active rehearsal. There is still much to learn regarding the design and implementation of efficacious treatment approaches for individuals with progressive semantic loss. Future research should attempt to confirm the typical progression and time course of linguistic and cognitive impairments in SD and to examine what types of treatment are most beneficial at each stage of the disorder.

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References

1. Shelton JR, Caramazza A. Deficits in lexical and semantic processing: implications for models of normal language. *Psychon Bull Rev* 1999;6:5–27. [PubMed: 12199314]
2. Raymer, AM.; Rothi, LJG. Cognitive approaches to impairments of word comprehension and production. In: Chapay, R., editor. *Language Intervention Strategies in Aphasia and Related Neurogenic Communication Disorders*. Vol. 4. Philadelphia, PA: Lippincott Williams & Wilkins; 2001. p. 524-550.
3. Benson DF. Anomia in aphasia. *Aphasiology* 1988;2:229–235.

4. Martin RC. Language processing: functional organization and neuroanatomical basis. *Annu Rev Psychol* 2003;54:55–89. [PubMed: 12359917]
5. Vandenberghe R, Price C, Wise R, Josephs O, Frackowiak RS. Functional anatomy of a common semantic system for words and pictures. *Nature* 1996;383:254–256. [PubMed: 8805700]
6. Mummery CJ, Patterson K, Wise RJS, Vandenberg R, Price CJ, Hodges JR. Disrupted temporal lobe connections in semantic dementia. *Brain* 1999;122:61–73. [PubMed: 10050895]
7. Jefferies E, Lambon Ralph MA. Semantic impairment in stroke aphasia versus semantic dementia: a case-series comparison. *Brain* 2006;129:2132–2147. [PubMed: 16815878]
8. Adlam AL, Patterson K, Rogers TT, et al. Semantic dementia and fluent primary progressive aphasia: two sides of the same coin? *Brain* 2006;129:3066–3080. [PubMed: 17071925]
9. Rogers TT, Lambon Ralph MA, Garrard P, et al. Structure and deterioration of semantic memory: a neuropsychological and computational investigation. *Psychol Rev* 2004;111:205–235. [PubMed: 14756594]
10. Caviness VS, Makris N, Montinaro E, et al. Anatomy of stroke, part I: an MRI-based topographic and volumetric system of analysis. *Stroke* 2002;33:2549–2556. [PubMed: 12411641]
11. Hodges JR, Patterson K, Oxbury S, Funnell E. Semantic dementia, progressive fluent aphasia with temporal lobe atrophy. *Brain* 1992;115:1783–1806. [PubMed: 1486461]
12. Grossman M, McMillan C, Moore P, et al. What's in a name: voxel-based morphometric analyses of MRI and naming difficulty in Alzheimer's disease, frontotemporal dementia and corticobasal degeneration. *Brain* 2004;127:628–649. [PubMed: 14761903]
13. Rogers TT, Ivanoiu A, Patterson K, Hodges JR. Semantic memory in Alzheimer's disease and the frontotemporal dementias: a longitudinal study of 236 patients. *Neuropsychology* 2006;20:319–335. [PubMed: 16719625]
14. Hodges JR, Graham N, Patterson K. Charting the progression of semantic dementia: implications for the organisation of semantic memory. *Memory* 1995;3:463–495. [PubMed: 8574874]
15. Neary D, Snowden JS, Gustafson L, et al. Frontotemporal lobar degeneration: a consensus on clinical diagnostic criteria. *Neurology* 1998;51:1546–1554. [PubMed: 9855500]
16. Bird H, Lambon Ralph MA, Patterson K, Hodges JR. The rise and fall of frequency and imageability: noun and verb production in semantic dementia. *Brain Lang* 2000;73:17–49. [PubMed: 10872636]
17. Howard, D.; Patterson, KE. Bury St Edmunds. UK: Thames Valley Test Co; 1992. The Pyramids and Palm Trees Test; p. 16
18. Hodges JR, Patterson K, Tyler LK. Loss of semantic memory: implications for the modularity of mind. *Cogn Neuropsychol* 1994;11:505–542.
19. Tulving E. Episodic memory: from mind to brain. *Annu Rev Psychol* 2002;53:1–25. [PubMed: 11752477]
20. Gorno-Tempini ML, Dronkers NF, Rankin KP, et al. Cognition and anatomy in three variants of primary progressive aphasia. *Ann Neurol* 2004;55:335–346. [PubMed: 14991811]
21. Rosen HJ, Gorno-Tempini ML, Goldman WP, et al. Patterns of brain atrophy in frontotemporal dementia and semantic dementia. *Neurology* 2002;58:198–208. [PubMed: 11805245]
22. Mummery CJ, Patterson K, Price CJ, Ashburner J, Frackowiak RS, Hodges JR. A voxel-based morphometry study of semantic dementia: relationship between temporal lobe atrophy and semantic memory. *Ann Neurol* 2000;47:36–45. [PubMed: 10632099]
23. Galton CJ, Patterson K, Graham K, et al. Differing patterns of temporal atrophy in Alzheimer's disease and semantic dementia. *Neurology* 2001;57:216–225. [PubMed: 11468305]
24. Lambon Ralph MA, McClelland J, Patterson K, Galton C, Hodges J. No right to speak? The relationship between object naming and semantic impairment: neuropsychological evidence and a computational model. *J Cogn Neurosci* 2001;13:341–356. [PubMed: 11371312]
25. Grossman M, Ash S. Primary progressive aphasia: a review. *Neurocase* 2004;10:3–18. [PubMed: 15849155]
26. Williams GB, Nestor PJ, Hodges JR. Neural correlates of semantic and behavioural deficits in frontotemporal dementia. *Neuroimage* 2005;24:1042–1051. [PubMed: 15670681]
27. Chan D, Fox NC, Scahill RI, et al. Patterns of temporal lobe atrophy in semantic dementia and Alzheimer's disease. *Ann Neurol* 2001;49:433–442. [PubMed: 11310620]

28. Scahill VL, Hodges JR, Graham KS. Can episodic memory tasks differentiate semantic dementia from Alzheimer's disease? *Neurocase* 2005;11:441–451. [PubMed: 16393758]
29. Nestor PJ, Fryer TD, Hodges JR. Declarative memory impairments in Alzheimer's disease and semantic dementia. *Neuroimage* 2006;30:1010–1020. [PubMed: 16300967]
30. Murre JMJ, Graham KS, Hodges JR. Semantic dementia: relevance to connectionist models of long-term memory. *Brain* 2001;124:647–675. [PubMed: 11287367]
31. Snowden JS, Griffiths HL, Neary D. Semantic-episodic memory interactions in semantic dementia: implications for retrograde memory function. *Cogn Neuropsychol* 1996;13:1101–1137.
32. Graham KS, Simons JS, Pratt KH, Patterson K, Hodges JR. Insights from semantic dementia on the relationship between episodic and semantic memory. *Neuropsychologia* 2000;38:313–324. [PubMed: 10678697]
33. Kitchener EG, Hodges JR, McCarthy R. Acquisition of post-morbid vocabulary and semantic facts in the absence of episodic memory. *Brain* 1998;121:1313–1327. [PubMed: 9679783]
34. McClelland JL, McNaughton BL, O'Reilly RC. Why there are complementary learning systems in the hippocampus and neocortex: insights from the successes and failures of connectionist models of learning and memory. *Psychol Rev* 1995;102:419–457. [PubMed: 7624455]
35. Schacter, DL. *Memory Systems of the Brain: Animal and Human Cognitive Processes*. New York, NY: Guilford Press; 1985. *Multiple Forms of Memory in Humans and Animals*.
36. Graham KS, Patterson K, Pratt KH, Hodges JR. Relearning and subsequent forgetting of semantic category exemplars in a cast of semantic dementia. *Neuropsychology* 1999;13:359–380. [PubMed: 10447298]
37. Graham KS, Patterson K, Pratt KH, Hodges JR. Can repeated exposure to “forgotten” vocabulary help alleviate word-finding difficulties in semantic dementia? An illustrative case study. *Neuropsychol Rehabil* 2001;11:429–454.
38. Howard D, Patterson K, Franklin S, Orchard-Lisle V, Morton J. Treatment of word retrieval deficits in aphasia. A comparison of two therapy methods. *Brain* 1985;108:817–829. [PubMed: 4075074]
39. Drew RL, Thompson CK. Model-based semantic treatment for naming deficits in aphasia. *J Speech Lang Hear Res* 1999;42:972–989. [PubMed: 10450915]
40. LeDorze G, Boulay N, Gadreau J, Brassard C. The contrasting effects of a semantic versus a formal-semantic technique for the facilitation of naming in a case of anomia. *Aphasiology* 1994;8:127–141.
41. Howard D, Patterson K, Franklin S, Orchard-lisle V, Morton J. The facilitation of picture naming in aphasia. *Cogn Neuropsychol* 1985;2:49–80.
42. Snowden JS, Neary D. Relearning of verbal labels in semantic dementia. *Neuropsychologia* 2002;40:1715–1728. [PubMed: 11992659]
43. Jokel R, Rochon E, Leonard C. Therapy for anomia in semantic dementia. *Brain Cogn* 2002;49:241–244. [PubMed: 15259401]
44. Jokel R, Rochon E, Leonard C. Treating anomia in semantic dementia: improvement, maintenance, or both? *Neuropsychol Rehabil* 2006;16:241–256. [PubMed: 16835150]
45. Frattali C. An errorless learning approach to treating dysnomia in frontotemporal dementia. *J Med Speech-Lang Pathol* 2004;12:11–24.
46. Jokel R, Cupit J, Rochon EA, Graham NL. Errorless re-training in semantic dementia using MossTalk words. *Brain Lang* 2007;103:205–206.
47. Fink, RB.; Brecher, A.; Montgomery, M.; Schwartz, MF. *Moss Talk Words* [computer software manual]. Philadelphia, PA: Albert Einstein Healthcare Network; 2001.
48. Henry ML, Beeson PM, Rapcsak SZ. Treatment for lexical retrieval in progressive aphasia. *Aphasiology*. 2008In press
49. Reilly J, Cross K, Troiani V, Grossman M. Single-word semantic judgements in semantic dementia: do phonology and grammatical class count? *Aphasiology* 2007;21:558–569.
50. Reilly J, Martin N, Grossman M. Verbal learning in semantic dementia: is repetition priming a useful strategy? *Aphasiology* 2005;19:329–339.
51. Murray LL. Longitudinal treatment of primary progressive aphasia: a case study. *Aphasiology* 1998;12:651–672.

52. Rogers MA, Alarcon NB. Characteristics and management of primary progressive aphasia. *Neurophysiol Neurogenic Speech Language Disord Newsletter* 1999;9:12–26.