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Semantic dementia and persisting Wernicke's aphasia: Linguistic and anatomical profiles

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ABSTRACT

Few studies have directly compared the clinical and anatomical characteristics of patients with progressive aphasia to those of patients with aphasia caused by stroke. In the current study we examined fluent forms of aphasia in these two groups, specifically semantic dementia (SD) and persisting Wernicke's aphasia (WA) due to stroke. We compared 10 patients with SD to 10 age- and education-matched patients with WA in three language domains: language comprehension (single words and sentences), spontaneous speech and visual semantics. Neuroanatomical involvement was analyzed using disease-specific image analysis techniques: voxel-based morphometry (VBM) for patients with SD and overlays of lesion digitized lesion reconstructions in patients with WA. Patients with SD and WA were both impaired on tasks that involved visual semantics, but patients with SD were less impaired in spontaneous speech and sentence comprehension. The anatomical findings showed that different regions were most affected in the two disorders: the left anterior temporal lobe in SD and the left posterior middle temporal gyrus in chronic WA. This study highlights that the two syndromes classically associated with language comprehension deficits in aphasia due to stroke and neurodegenerative disease are clinically distinct, most likely due to distinct distributions of damage in the temporal lobe.

1. Introduction

Modern descriptions of language disorders date back to the mid-1800s/early 1900s, when physicians began systematic correlations of behavioral deficits with brain damage (Broca, 1861; Pick, 1901, 1904; Wernicke, 1874). Much of this literature has focused on language deficits caused by sudden brain damage, such as stroke, but within the last few decades, there has been an increasing interest in progressive language disorders resulting from focal neurodegenerative diseases (Mesulam, 1982; Warrington, 1975).

One such syndrome, semantic dementia (SD), a variant of primary progressive aphasia, is characterized by a progressive degradation of semantic memory and presents with fluent, but anomic, spontaneous speech, with relatively spared syntax, prosody, articulation and phonology (Hodges, Patterson, Oxbury, & Funnell, 1992; Neary et al., 1998; Snowden, Goulding, & Neary, 1989). Descriptions of SD have noted severe anomia as a core, initial feature (Gorno-Tempini et al., 2004; Hodges et al., 1992; Mesulam et al., 2009; Snowden et al., 1989). Single-word comprehension is also severely impaired in SD, especially for low-frequency items (e.g., 'peacock' vs. the more familiar/frequent 'dog'). Poor naming and comprehension of single words are manifestations of a general semantic memory deficit that also causes impairments in object and face recognition in the visual, auditory, tactile and olfactory modalities (Bozeat, Lambon-Ralph, Patterson, Garrard, & Hodges, 2000; Coccia, Bartolinin, Luzzi, Provinciali, & Lambon-Ralph, 2004; Hodges et al., 1992; Luzzi et al., 2007; Snowden et al., 1989). Another symptom of SD that has recently been ascribed to semantic memory breakdown is surface dyslexia, which refers to difficulty in reading words with exceptional orthographic-to-phonological correspondence (e.g., regularizing irregularly spelled words, so that 'colonel' is read as /kolonel/; Wilson et al., 2009; Woollams, Ralph, Plaut, & Patterson, 2007). Anatomically, SD is associated with atrophy in the ventral and lateral portions of the anterior temporal lobes bilaterally, although atrophy is typically greater on the left side (Chan et al., 2001; Galton et al., 2001; Gorno-Tempini et al., 2004; Mummery et al., 2000; Rosen et al., 2002). Pathologically, SD has been consistently associated with a TDP-43 proteinopathy (Davies et al., 2005; Snowden, Neary, & Mann, 2007).

Within the stroke-induced aphasia syndromes, Wernicke's aphasia (WA) (Brookshire, 1997; Goodglass & Kaplan, 1983; Kertesz, 1979) has also been associated with fluent speech, severe

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comprehension difficulties and damage to the temporal lobe. This WA syndrome was first described by Carl Wernicke, a German physician who reported two patients with a pattern of fluent speech, "containing meaningless and garbled words," and significant comprehension impairments (Wernicke, 1874). WA is characterized by impaired auditory comprehension and fluent speech spoken at a normal or rapid rate. Meaningless words or jargon are sometimes used, with some sparing of syntactic structures (Goodglass & Kaplan, 1983; Kertesz, 1979). Patients with WA typically have great difficulty participating in day-to-day conversation, due to impaired comprehension of single words and an inability to monitor their verbal output. Patients are often unaware of the degree to which their speech is empty and difficult to understand. Anatomically, WA was originally associated with damage to Wernicke's area (posterior Brodmann area 22), but after the advent of structural imaging, it became apparent that a larger lesion within the left middle and superior temporal and inferior parietal regions and underlying white matter is typically associated with WA syndrome (Dronkers & Baldo, 2009; Kertesz, Harlock, & Coates, 1979; Mazzocchi & Vignolo, 1979; Poeck, de Bleser, & von Keyserlingk, 1984). Furthermore, not all patients with WA have lesions to Wernicke's area (Basso, Lecours, Moraschini, & Vanier, 1985; Dronkers, Redfern, & Knight, 2000).

Although SD and WA are both associated with significant language comprehension deficits and temporal lobe damage, direct comparisons of the clinical profiles of these patient groups are still lacking. Three recent studies compared stroke patients to patients with SD, however, the stroke populations included patients with different types and severity of aphasia (Corbett, Jefferies, Ehsan, & Lambon-Ralph, 2009; Jefferies & Lambon-Ralph, 2006; Jefferies, Rogers, Hopper, & Lambon-Ralph, 2010).

In the current study, we compared the performance of patients with SD and chronic WA on measures of language comprehension, fluency and visual semantics. Based on clinical observations and the small extant literature, we hypothesized that patients with SD and WA would show different patterns of language deficits, with relative preservation of sentence comprehension and spontaneous speech in SD but greater visual semantic deficits. These behavioral profiles were also expected to correspond with anatomical differences in temporal lobe involvement, with more anterior damage in SD and more posterior involvement in persisting WA.

2. Methods

2.1. Participants

Participants in this retrospective study included 10 patients with SD and 10 age- and education-matched patients with persisting WA. Inclusion criteria for both SD and WA groups included native English proficiency, right-handedness, and no prior history of neurologic injury, psychiatric disability, or substance abuse.

The patients with SD consisted of seven men and three women who were assessed at the University of California San Francisco (UCSF) Memory and Aging Center. Their mean age was 63 years (SD = 6.8; range 57–79), when they were an average of 3.7 years into the disease. Clinical history, neurological examination, and general neuropsychological and language testing were used to obtain the SD diagnosis based on current clinical criteria (Neary et al., 1998). The Clinical Dementia Rating (CDR; Morris et al., 1997) was used to assess functional impairment in the patients with SD. They received an average rating of 0.5 (range 0.5–1.0).

The WA group consisted of one woman and nine men, with a mean age of 68 years (SD = 6.8; range 56–80) at testing (see Table 1 for demographics). Average time post-stroke for the WA group was 2 years (SD = 1.3; range 1–5). Inclusion criteria for patients with WA included a single, left hemisphere cerebrovascular accident (CVA) (e.g., no tumor, progressive neurologic disease). Diagnosis was made based on clinical history, neurological examination and results from the Western Aphasia Battery (WAB; Kertesz, 1982). Patients with WA were evaluated at the Center for Aphasia and Related Disorders (CARD) at the Veterans Affairs Northern California Health Care System (VANCHCS) in Martinez, CA. Patients with WA were evaluated and scanned in the chronic stage of their aphasia, defined as being at least 1 year post onset.

All patients read and signed written informed consent prior to participating in research. Testing was approved by Institutional Review Boards at the VA and UCSF.

3. Materials and procedures

All patients were administered speech and language tests used to assess language comprehension, fluency and visual semantics. Tests included the WAB, a 15-item version of the Boston Naming Test (BNT; Kaplan, Goodglass, & Weintraub, 1983), the Pyramid and Palm Trees Test-Pictures (PPT-P; Howard & Patterson, 1992), subtests of the Curtiss-Yamada Comprehensive Language Evaluation-Receptive (CYCLE-R; Curtiss & Yamada, 1988) and a 60-s category (animals) fluency test (a verbal semantic task).

The WAB is a standardized language test that assesses spontaneous speech, fluency, auditory comprehension, naming and repetition. For this study, we analyzed results from four subtests in particular: Information Content, Fluency, Auditory Word Recognition and Sequential Commands.

Sentence comprehension was further evaluated by 11 subtests from the CYCLE-R. Each subtest included five sentences and assessed comprehension for a range of syntactic structures that differed in complexity. After listening to a sentence, patients were asked to match that sentence to a corresponding black-and-white line drawing from an array of three or four pictures. Subtests were grouped according to complexity as follows: simple constructions (simple declaratives and possession), moderately complex constructions (active voice, passive voice, and double embedding), and complex constructions (object clefting, subject relative clauses, negative passives, object relative clauses, object relative clauses with relativized object) (see Dronkers, Wilkins, Van Valin, Redfern, & Jaeger, 2004; Gorno-Tempini et al., 2004).

As a test of visual semantics, we used the PPT-Pictures in which patients decided which of two items was most associated with a target (e.g., matching a pyramid to a palm tree vs. a pine tree). In the category fluency test, patients produced as many exemplars of animals as possible in 1 min. To assess object naming, we used a modified 15-item version of the BNT that assessed naming and word recognition. A three-alternative, multiple-choice task generated at the VA in Martinez was administered for missed items (e.g., if 'bed' was missed, three auditorily-presented options were presented, 'is it bed, bell or bear'?). The target and two distracters all began with the same phoneme.

Testing was administered to patients with SD at the time of their initial visit. Patients with WA were all assessed at least 1 year after their stroke, when the deficits were stable.

3.1. Imaging

For patients with SD, structural images were acquired on a 1.5T Siemens Magnetom VISION system (Siemens, Iselin, NJ) equipped with a standard quadrature head coil, using a magnetization prepared rapid gradient echo (MPRAGE) sequence (164 coronal slices; slice thickness = 1.5 mm; FOV = 256 mm; matrix 256 × 256; voxel size 1.0 × 1.5 × 1.0 mm; TR = 10 ms; TE = 4 ms; flip angle = 15°).

Table 1
Demographic and language data for patients with WA and SD.

	Wernicke's aphasia mean (sd) (N = 10)	Semantic dementia mean (sd) (N = 10)
Demographic information		
Age at testing	68.0 (6.8)	63.3 (6.8)
Education	16.2 (3.3)	16.3 (1.8)
Months post onset	24.1 (15.5)	40.8 (17.2)
Clinical dementia rating (CDR)	NA	.5 (.27)
Language testing (total possible)		
WAB information content (10)	3.7 (2.3)	8.4 (.70)[a]
WAB spontaneous speech fluency (10)	7.4 (.97)	8.9 (1.0)[a]
WAB sequential commands (80)	22.2 (16.2)	72.1 (8.6)[a]
CYCLE simple (%)	61.1 (14.5)	97.5 (3.7)[a]
Moderate (%)	34.4 (12.9)	98.1 (3.7)[a]
Difficult (%)	25.3 (8.5)	92.0 (5.2)[a]
WAB single-word comprehension (total = 60)	24.8 (11.7)	46.6 (10.6)[a]
Real objects (6)	4.2 (1.6)	4.4 (1.6)
Drawn objects (6)	3.9 (2.0)	5.2 (1.6)
Shapes (6)	2.1 (1.7)	2.9 (2.3)
Letters (6)	2.7 (2.2)	5.8 (.63)[a]
Numbers (6)	4.2 (1.8)	5.8 (.63)[a]
Colors (6)	2.8 (2.0)	5.8 (.42)[a]
Furniture (6)	2.0 (2.1)	5.2 (1.3)[a]
Body parts (6)	1.9 (2.1)	4.3 (1.6)[a]
Fingers (6)	1.0 (1.2)	4.0 (1.6)[a]
Left and right (6)	0.0 (0.0)	3.2 (2.0)[a]
Boston naming test (BNT) (15-item)	1.5 (2.6)	2.9 (3.0)
BNT multi-choice (% improvement)	69.3 (34.0)	56.3 (20.1)
BNT phonemic cue (add'l correct)	.25 (.46)	.75 (1.2)
Pyramid and palm trees-pictures (52)	42.3 (5.1)	35.2 (7.4)
Semantic fluency (animals) (60 s)	2.6 (3.2)	4.4 (2.2)

[a] $p < .05$, SD > WA.

VBM was performed with SPM2 using an optimized method for the spatial normalization of gray matter (Good et al., 2001), as described previously (Brambati et al., 2006). Modulated gray matter images were then spatially smoothed with a 12-mm full-width half-maximum isotropic Gaussian kernel. Gray matter volume in SD was compared to 50 age- and gender-matched normal controls (age = 63.5 ± 5.6 years, F/M = 15/35). We plotted regions where patients with SD exhibited at least 15% volume loss relative to normal controls.

Images were available for 9 of the 10 patients with WA. Lesion were reconstructed from T1-weighted 3D MRI scans (N = 8) or a CT scan (N = 1) obtained at least 6 weeks after their stroke. MRI scans were obtained from a 1.5T Phillips Eclipse scanner. T1-weighted images were acquired with a Spoiled Gradient Recall (SPGR) sequence (TR/TE = 15/4.47 ms, FOV = 240 mm, 256 × 256 imaging matrix, flip angle = 35°, 0.94 × 1.3 × 0.94 mm^3 voxels, 212 coronal slices). The one CT scan was obtained with a Picker 3D CT scanner. The lesion reconstructions were drawn using MRIcro software and were approved by a board-certified neurologist who was blind to the aim of the study. This method has been shown to be reliable in previous studies (Friedrich, Egly, Rafal, & Beck, 1998; Knight, Scabini, Woods, & Clayworth, 1988). The patient's brain was then registered to MNI space using the standard nonlinear spatial normalization procedure from SPM2, with cost function masking to avoid distortions due to the lesion itself.

To identify regions that were lesioned in the majority of patients with WA, we overlaid the lesions using VLSM software (Bates et al., 2003), and plotted voxels that were included in the lesions of at least six of the nine patients.

4. Results

Patients with SD and chronic WA were compared on measures of language comprehension, spontaneous speech and semantics using Mann–Whitney U tests for independent samples. It should be noted that language measures were designed for patients, and thus, controls perform at ceiling. All statistical test results are shown in Table 1.

The pattern of comprehension deficits differed between the patients with SD and WA. Patients with SD showed overall better performance on single-word comprehension. However, when considering the categories of items tested by the WAB, patients with SD had clear difficulty comprehending objects, especially for low-frequency items such as 'matches'. They also had more difficulty with words and objects with a more abstract meaning, such as symbols. However, they had no difficulty pointing to high-frequency, concrete items such as body parts, single letters, numbers and common objects in the room.

In terms of sentence comprehension, patients with SD performed significantly better than those with persisting WA. This pattern was apparent on the WAB sequential commands subtest, as well as on the CYCLE sentence comprehension subtests. Here, patients with SD showed performance that was significantly better than the WA group for each of the three levels of sentence complexity (simple, moderate and difficult).

The content of the speech produced by patients with SD was significantly more relevant than that of patients with WA (see Table 2 for speech samples). Patients with SD also presented with speech that was significantly more fluent that that of patients with WA (see Table 2). Neologisms and phonological paraphasias were common in patients with WA, but not in patients with SD.

Naming performance on the BNT, a measure of verbal semantics, was comparable for patients with SD and WA, as both groups named very few items spontaneously. The SD and WA groups were also indistinguishable in terms of the effect of BNT phonemic

Table 2
Samples of spontaneous speech in patients with WA and SD when asked to describe the WAB picnic scene.

Patient with WA	"*It's um, quite grand, grandstand. And has, um… I'm trying to have words and I can't get them. Um, I have two words, two words here. They're gone. And the two dogs, three, three dogs. And the /kwer ledi/ or leather here it's /leow/ fire, farm, fur, something feet and um, dime farm here. And the farm any the farm here.*"
Patient with SD	"*Looks like there's a husband and wife. She's reading and she's drinking and then there's another young man who has a… I want to say 'flag', but it's not a flag, but he's holding something up in the air (referring to kite)…*"

Table 3
Naming samples from patients with WA and SD from the BNT.

Patient with WA	*Error types*: neologistic, e.g., */hiwork/* for 'helicopter' phonemic paraphasias, e.g., */hanner/* for 'hanger' associative semantic paraphasias, e.g., "*boat*" for 'house'
Patient with SD	*Error types*: supra-ordinate semantic paraphasia, e.g. "*animal*" for 'octopus' ordinate semantic paraphasias, e.g., "*dog*" for 'camel' circumlocutions (e.g., "*It's a plant, but I don't know what brand it is*" for 'flower')

cueing, which did little to help either group. Interestingly, the types of naming errors were qualitatively distinct for the two groups. Patients with SD made predominantly supra-ordinate or ordinate semantic errors and circumlocutions, while errors in the WA group were predominantly neologistic and phonemic, with occasional associative semantic paraphasias (see Table 3 for a comparison). On the three-alternative, forced choice test of BNT items not named spontaneously, both SD and WA patients showed a benefit.

On the PPT-P the SD and WA groups did not differ significantly in their scores, though the WA patients obtained higher scores on this visual semantics task. Similarly, on a verbal semantic fluency task, the number of animal names generated in 60-s did not differ between patients with SD and WA.

4.1. Post-hoc analysis of longitudinal performance on specific language tasks in SD

In order to evaluate the effect of disease progression on language measures of interest (sentence and single-word comprehension, spontaneous speech and visual semantics), we performed an analysis of results from follow-up testing 1 year later with the SD group. This analysis revealed that, as the disease progressed, patients with SD continued to show better sentence comprehension than those with WA for moderate and complex syntactic structures (CYCLE, moderate, time 2 in SD: 70.0 (20.2) $p < 0.05$ vs. WA 34.4 (12.9); CYCLE, difficult, time 2 in SD: 54.3 (19.4), $p < 0.05$ vs. WA: 25.3 (8.5). Overall single-word comprehension was equally impaired (at time 2: SD: 28.7 (13.8) $p < 0.05$ vs. WA: 24.8 (11.7), and at time 2, SD showed significantly worse performance on visual semantics (PPT-P in SD: 28.2 (2.0) $p < 0.05$ vs. WA: 42.3 (5.1).

4.2. Imaging

The VBM analysis of the 10 patients with SD showed predominantly left anterior temporal lobe atrophy, at the time of initial testing (Fig. 1b). Specifically, gray matter atrophy was found in the anterior portion of the left temporal lobe, including the pole, the superior, middle, inferior and fusiform gyri. Gray matter loss also extended to the medial portion of the left temporal lobe, including the hippocampus/amygdala. Less extensive gray matter atrophy was observed in the same regions of the right temporal lobe. Beyond the temporal lobes, gray matter atrophy was observed in the left insula and the caudate bilaterally.

An overlay of the nine patients with chronic WA showed the left posterior middle temporal gyrus as the common area of lesion overlap (Fig. 1a). A few patients' lesions extended into superior temporal, inferior parietal, anterior temporal and frontal areas, as well as subcortical white matter. Thus, patients with SD had a pattern of atrophy that was largely restricted to the anterior and ventral temporal lobes bilaterally, whereas the WA group had larger lesions encompassing most of the left middle temporal gyrus.

5. Discussion

In the current study we directly compared performance of patients with semantic dementia (SD) and stroke-induced persisting Wernicke's aphasia (WA) using measures of language comprehension, spontaneous speech and visual semantics. Overall, our results showed that SD and WA patients showed a different pattern of language comprehension, with strikingly preserved sentence comprehension in SD, despite impaired single-word comprehension and visual semantics.

Early reports of what has come to be called SD described patients with core semantic deficits that manifested as fluent progressive aphasia (Snowden et al., 1989; Warrington, 1975). The core, multimodal semantic deficit can actually be difficult to detect at the beginning of the disease and has been described as "fluent progressive aphasia with comprehension deficits" (Mesulam, 2001). Our study highlights the clinical differences between SD, a disorder of semantic memory that manifests as a fluent aphasia with language comprehension deficit, and WA, a core language impairment with fluent speech and comprehension deficits. Though both syndromes have been described as fluent forms of aphasia with severe comprehension difficulties and "semantic" deficits, we showed that their clinical presentations are strikingly different.

The most relevant difference in the pattern of language comprehension deficits between the two syndromes is the relative sparing of sentence comprehension in SD, even as the disease progresses. In contrast, WA patients showed severe impairment in both single-word and sentence comprehension. SD patients showed an overall milder deficit in single-word comprehension at year one, but still had clear difficulties with items that were sensitive to semantic impairment, such as objects and shapes. This is consistent with previous studies (Corbett et al., 2009; Jefferies & Lambon-Ralph, 2006) that compared semantic impairments in SD to stroke patients with comprehension deficits (not necessarily WA) and found that while performance on a battery of semantic tests was largely equivalent, the nature of errors between the two groups was different. Specifically, SD performance on semantic tasks was affected by word frequency and object familiarity, whereas performance in stroke patients was not. In our study, the pattern of performance in the single-word and sentence comprehension tasks of SD patients at follow-up also demonstrates that the observed dissociation with sentence comprehension is not simply due to a milder deficit in SD. In fact, despite overall comparable single-word comprehension deficits between WA and SD at year two, patients with SD still showed better

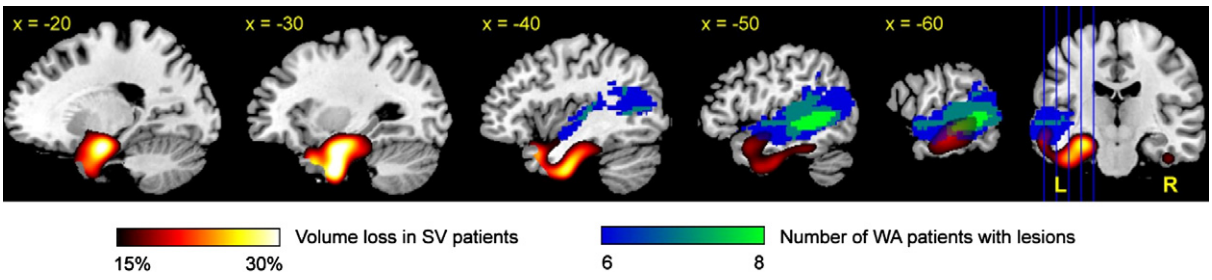

Fig. 1. Anatomical features of SD and WA. Atrophy in patients with SD (hot) was predominantly anterior temporal, whereas the regions commonly lesioned in patients with WA (blue–green) were predominantly posterior temporal.

performance on sentence comprehension tasks. It is also of note that patients with SD were able to comprehend difficult syntactic structures at year one and two, despite confrontation naming scores that were almost at floor, even in the recognition task. This dissociation between single word and sentence tasks is not commonly seen in vascular aphasia.

Contrary to our predictions, performance on a standard, non-verbal semantic association task (the PPT-P) was surprisingly comparable between WA and SD, at least at year one. At follow-up, patients with SD showed greater impairment than patients with WA on the PPT-P. We expected that WA patients would be relatively spared on such a generally non-linguistic task, however they showed a decrement in performance. It may be that patients with WA had difficulty understanding task instructions or they may have had problems with executive aspects of the task as has been shown before in patients with middle temporal lobe lesions (Baldo et al., 2005; Baldo, Bunge, Wilson, & Dronkers, 2010). This would be consistent with previous studies that compared vascular aphasia patients with semantic deficits, also called 'semantic aphasia', to patients with SD on a detailed semantic battery (Jefferies & Lambon-Ralph, 2006; Jefferies et al., 2010). Another study found that while both SD and semantic aphasia groups showed impaired performance on a semantic battery, semantic aphasia patients were more likely to fail on tasks requiring executive control of semantic knowledge (e.g., matching objects to a common function) (Corbett et al., 2009). However, these studies did not include patients with a Wernicke's type of language profile, but instead, stroke cases with semantic impairment and a transcortical sensory aphasia pattern. Therefore, results from these studies are difficult to compare.

With respect to spontaneous speech, the content and quality of speech produced by patients with SD and WA differed greatly. Patients with SD, despite severe anomia, used mostly complete, complex and relevant sentences with occasional paraphasias, circumlocutions, and word-finding pauses, whereas patients with WA used sentences that were sometimes complete, but irrelevant and often interspersed with jargon or neologisms. Patients with SD produced speech that was free of the jargon that is heard in patients with WA, making those with SD much more understandable to a conversational partner (see Table 2 for spontaneous speech examples). Clinically, this is important because, while the patient with WA struggles with basic communication, the patient with SD is able to express more thoughts and ideas, albeit while contending with worsening anomia.

The results of our analyses of anatomical damage in the two syndromes showed, as predicted by previous literature, that patients with SD present with more anterior patterns of temporal lobe damage. Although a direct comparison of the extent and site of the lesion is problematic because of the different nature of the biological and pathological processes involved in the two diseases, it is evident that the majority of damage is in bilateral inferior and anterior temporal areas in SD and in the left posterior middle and superior temporal regions in WA. This difference in the site of the most typical anatomical damage is the basis for the different patterns of comprehension difficulties in SD and WA. It should also be noted that poorer comprehension in WA cannot be explained by the sheer size of the lesion alone, as patients with SD also suffer from deterioration to large regions of the temporal lobe, with most cases showing atrophy in both hemispheres.

The relative sparing of sentence comprehension and fluency in SD is likely due to their largely preserved posterior temporal, parietal and frontal phonological and syntactic systems (Agosta et al., 2010; Hodges & Patterson, 1996). Sentence comprehension is a complex task, subserved by a network of brain regions that involve left frontal, parietal and temporal areas, which are probably involved in different aspects of the task (Caplan & Hildebrandt, 1998; Dronkers et al., 2004; Grossman & Moore, 2005). In SD, the relative sparing of left fronto-parietal working memory and linguistic systems probably provides enough resources to understand complex sentences, as long as the task does not heavily tap lexical-semantic processes (Agosta et al., 2010; Amici et al., 2007; Wilson et al., 2009). On the other hand, posterior middle temporal cortex, which is always damaged in persisting WA, is a core linguistic region, which may even play a crucial role in syntactic processing, possibly in the integration of syntactic information with lexical-semantic information (Friederici, Makuuchi, & Bahlmann, 2009).

The current study showed that patients with WA presented with core language deficits that affected all aspects of language, while patients with SD demonstrated a semantic deficit, but some comparatively surprising strengths, such as relative sparing of sentence comprehension. These distinctions have implications for the day-to-day lives of patients with these disorders and their treatment options. For example, while patients with WA may struggle with even basic communication, those with SD are able to carry on fairly complex conversations, their deficits, at least initially, being sometimes difficult to detect to the casual observer.

The main limitation of this study is its retrospective nature and thus the tasks used were not experimentally designed specifically for our patient groups. It became apparent in our analyses that some of the measures considered, which were designed for vascular aphasia, might not be particularly sensitive for symptoms of progressive aphasia. The Auditory Word Recognition subtest of the WAB is such an example. Finally, imaging results were not directly comparable because lesions resulting from strokes are relatively discrete, whereas neurodegenerative patients show regionally-specific atrophy on a continuum.

Further research is needed to clarify the relationship between neuroanatomical and cognitive differences in aphasia caused by stroke and neurodegenerative disorders. Better characterization of the disorders will help delineate the core speech and language impairments in each population and will inform the still nascent field of behavioral treatment for patients with primary progressive aphasia.

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