

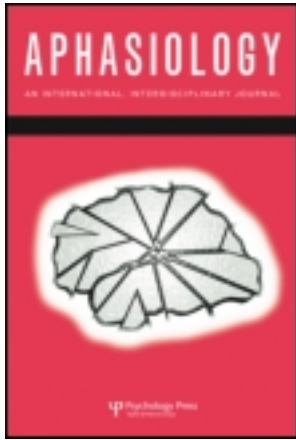
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Publisher: Psychology Press

Informa Ltd Registered in England and Wales Registered Number: 1072954

Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



Aphasiology

Publication details, including instructions for authors and subscription information:

<http://www.tandfonline.com/loi/paph20>

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Jamie Reilly^a, Joshua Troche^a, Alison Paris^a, Hyejin Park^a,
Micheline Kalinyak-Fliszar^b, Sharon M. Antonucci^c & Nadine
Martin^b

^a Department of Speech, Language, and Hearing Sciences,
University of Florida, Gainesville, FL, USA

^b Department of Communication Sciences and Disorders,
Temple University, Philadelphia, PA, USA

^c Department of Communicative Sciences and Disorders, New
York University, New York, NY, USA

Available online: 10 Nov 2011

To cite this article: Jamie Reilly, Joshua Troche, Alison Paris, Hyejin Park, Micheline Kalinyak-Fliszar, Sharon M. Antonucci & Nadine Martin (2012): Lexicality effects in word and nonword recall of semantic dementia and progressive nonfluent aphasia, *Aphasiology*, 26:3-4, 404-427

To link to this article: <http://dx.doi.org/10.1080/02687038.2011.616926>

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Lexicality effects in word and nonword recall of semantic dementia and progressive nonfluent aphasia

Jamie Reilly¹, Joshua Troche¹, Alison Paris¹, Hyejin Park¹,
Micheline Kalinyak-Fliszar², Sharon M. Antonucci³,
and Nadine Martin²

¹Department of Speech, Language, and Hearing Sciences, University of Florida, Gainesville, FL, USA

²Department of Communication Sciences and Disorders, Temple University, Philadelphia, PA, USA

³Department of Communicative Sciences and Disorders, New York University, New York, NY, USA

Background: Verbal working memory is an essential component of many language functions, including sentence comprehension and word learning. As such, working memory has emerged as a domain of intense research interest both in aphasiology and in the broader field of cognitive neuroscience. The integrity of verbal working memory encoding relies on a fluid interaction between semantic and phonological processes. That is, we encode verbal detail using many cues related to both the sound and meaning of words. Lesion models can provide an effective means of parsing the contributions of phonological or semantic impairment to recall performance.

Methods & Procedures: We employed the lesion model approach here by contrasting the nature of lexicality errors incurred during recall of word and nonword sequences by three individuals with progressive nonfluent aphasia (a phonological dominant impairment) compared to that of two individuals with semantic dementia (a semantic dominant impairment). We focused on psycholinguistic attributes of correctly recalled stimuli relative to those that elicited a lexicality error (i.e., nonword → word OR word → nonword).

Outcomes & Results: Patients with semantic dementia showed greater sensitivity to phonological attributes (e.g., phoneme length, wordlikeness) of the target items relative to semantic attributes (e.g., familiarity). Patients with PNFA showed the opposite pattern, marked by sensitivity to word frequency, age of acquisition, familiarity, and imageability.

Conclusions: We interpret these results in favour of a processing strategy such that in the context of a focal phonological impairment patients revert to an over-reliance on preserved semantic processing abilities. In contrast, a focal semantic impairment forces both reliance on and hypersensitivity to phonological attributes of target words. We relate this interpretation to previous hypotheses about the nature of verbal short-term memory in progressive aphasia.

Keywords: Working memory; Recall; Semantic dementia; Aphasia; Progressive nonfluent aphasia.

Address correspondence to: Jamie Reilly PhD, University of Florida, PO Box 100174, Gainesville, Florida 32610, USA. E-mail: jjreilly@phhp.ufl.edu

Research support: US Public Health Service grants R01 DC0101977 (NM) and K23 DC010197 (JR).

Verbal short-term memory (vSTM) is an essential component of many language domains, including word learning, sentence comprehension, narrative production, and appreciation of metaphor and non-literal language (Caplan & Waters, 1999; Gathercole, 2006; Gathercole, Hitch, Service, & Martin, 1997; Kempler, Almor, Tyler, Andersen, & MacDonald, 1998; Monetta & Pell, 2007). Within the realm of development, Gathercole, Hitch, Service, Adams, and Martin (1999) demonstrated that the immediate span of serial recall for pseudowords in young children is an exceptionally strong predictor ($R = .72$) of their later vocabulary size. Moreover, similarly robust correlations have been demonstrated in both typical and brain-injured adults with respect to functions such as lexical acquisition (Gathercole, 2006; Gupta & MacWhinney, 1997), but also even more fundamental tasks such as picture naming and auditory sentence comprehension (Miller, Finney, Meador, & Loring, 2010; Reilly, Peelle, Antonucci, & Grossman, 2011; Saito, Yoshimura, Itakura, & Lambon Ralph, 2003).

Verbal working memory (vWM) is grossly differentiated from vSTM by virtue of its role in both the passive storage and active manipulation of information during memory retrieval and encoding (e.g., Baddeley & Hitch, 1974). For example, a common task such as serial recall of a list of numbers (i.e., forward digit span) is typically regarded as loading more heavily on vSTM than vWM, although digit recall does include an active processing component that serves to retain and reproduce serial order of items in temporary storage. The “working” component of this most minimal working memory task can be altered by varying the content of items to be recalled (e.g., abstract words, nonwords) or by varying the task itself (e.g., backward digit span or mental summation of the same list of numbers). These task manipulations and stimuli modifications that combine with storage requirements to comprise working memory entail cognitive effort, and indeed many of the cognitive processes that serve to offset the rapid decay of memory rely on active functions (e.g., subvocal articulatory rehearsal, visuospatial imagery) and executive resources (e.g., vigilance, selective attention, inhibitory control) (Baddeley, 2003; Jonides et al., 1998; Stuss & Knight, 2002). In practice, vSTM and vWM are cognitive constructs that show a high degree of overlapping variance and are not always easily dissociable.

Impairment in vSTM (and also vWM) has emerged as a potential latent factor underlying many language disorders, including specific language impairment (Gathercole & Baddeley, 1990), Alzheimer’s disease (Almor, Kempler, MacDonald, Andersen, & Tyler, 1999; Collette, Van der Linden, Bechet, Belleville, & Salmon, 1998; MacDonald, Almor, Henderson, Kempler, & Andersen, 2001; Rochon, Waters, & Caplan, 2000) and stroke aphasia (Harris Wright & Shisler, 2005). As such, WM has emerged as an intense domain of focus in both aphasiology and the broader field of neuroscience (for review of cross-species investigations, see Jonides, Lacey, & Nee, 2005). Interest in WM from an aphasiology standpoint has seen cyclical popularity. For example, in the 1990s a burst of research articles followed Miyake and colleagues’ (Haarmann, Just, & Carpenter, 1997; Miyake, Carpenter, & Just, 1994, 1995) contentious claim that much of the language disturbance in aphasia is largely attributable to WM impairment. Today the field sees a steady, somewhat even, progression of research on the effects of WM on language functioning in aphasia, often complemented by a maturing body of parallel functional neuroimaging research.

Baddeley and Hitch (1974) offered the seminal model of WM that today remains a reference point for many other cognitive models with varying degrees of compatibility and modularity. For example, some have argued that WM represents a complex,

modality-independent form of attention (Cowan, 1988, 1995, 1999), whereas others have argued that attention and executive control constitute just one part of a multi-component “slave” memory system (Baddeley, 2003; see Caplan et al., 2011 this issue). Although the many extant WM models have dissimilarities, a number of stable findings (e.g., word length effects, within-modality dual task interference effects) have also emerged across studies. The working memory model of Baddeley and Hitch (1974) served as a framework for a number of neuropsychological studies in the 1980s and 1990s that focused on what appeared to be isolated impairments of phonological STM (e.g., Shallice, 1988; Vallar & Shallice, 1990). The model and its well-known components, a phonological store and articulatory loop that support rehearsal of stored phonological representations, fit well as an account of patients who demonstrate impaired phonological STM in the context of otherwise preserved ability to learn new verbal information. However, the classic working memory model has proved somewhat limited in its ability to account for a host of linguistic influences on performance of vSTM tasks by normal participants, as well as patterns of verbal STM impairment observed in aphasia and semantic dementia that implicate both semantic and phonological short-term stores.

In the 1990s several versions of a multi-store model of verbal STM were proposed (e.g., Martin & Saffran, 1990; Martin, Saffran & Dell, 1996; Martin, Dell, Saffran & Schwartz, 1994; Martin, Shelton & Yaffee, 1994). Such multi-store models offer the advantage of a fluid and often highly interactive division of labour between semantic and phonological processes during memory encoding that is not typically afforded by vWM models dominated by phonology.

vSTM AND LANGUAGE PROCESSING: CALLING ALL CUES . . .

There is an emerging consensus that effective memory encoding makes active use of many cues related to both form and meaning of words. That is, we employ a fluid division of labour between phonological and semantic processes. Perhaps the most readily apparent evidence for a semantic contribution to vWM is derived from the fact that we tend to show superior recall for words relative to nonwords (i.e., a lexicality advantage) (Gathercole, Pickering, Hall, & Peaker, 2001; Hulme, Maughan, & Brown, 1991). Yet one must exercise caution in attributing the lexicality advantage exclusively to word meaning. That is, recall accuracy for real words is also augmented by the fact that we construct lexical-phonological representations for real words based on repeated exposure to form. In contrast, the inherent novelty of a nonword hypothetically thwarts the benefit of a lexical-phonological contribution to recall (but see Gathercole, 1995). Although phonological frequency does clearly contribute to the lexicality effect, there also exists a compelling argument for a semantic contribution to recall based on empirical findings from a number of other experimental manipulations. For example, people tend to recall more concrete than abstract words (Walker & Hulme, 1999) and also show a significant recall advantage for semantically related lists of words (e.g., farm animals) relative to unrelated lists (Brooks & Watkins, 1990; Poirier & Saint Aubin, 1995; Shulman, 1971). In addition we tend to recall more verbal detail when we relate information to ourselves (i.e., self-reference effect) (Bellezza, 1984; Symons & Johnson, 1997).

Many early models of vWM focused intensely (sometimes exclusively) on acoustic factors that moderate efficiency of articulatory rehearsal and phonological storage.

Variables that negatively affect span include phonological similarity (Acheson, Postle, & MacDonald, 2010; Conrad & Hull, 1964) and word length (Baddeley, Thomson, & Buchanan, 1975; Tehan, Hendry, & Kocinski, 2001). Concurrent articulation demands such as uttering a redundant nonsense syllable (i.e., articulatory suppression) also impacts recall by blocking the covert rehearsal necessary for offsetting rapid decay of an unstable memory trace (Cowan, Cartwright, Winterowd, & Sher, 1987).

EFFECTS OF A FOCAL IMPAIRMENT OF PHONOLOGY OR SEMANTICS ON RECALL

In the presence of an otherwise intact encoding system there are two possibilities with respect to the effects of a focal impairment of either phonology or semantics. The first is that an individual compensates for degraded function in one domain by attempting to tap residual attributes of that particular domain. For example, a patient with a semantic impairment might show hypersensitivity to specific aspects of word meaning (e.g., familiarity, imageability). A second possibility is that impairment in one domain forces over-reliance on an alternative, preserved domain. Returning to the semantic dementia example, a patient who employs this strategy might encode almost exclusively via phonology.

Research in the domains of acquired alexia and repetition disorders supports the idea that patients often compensate for loss in one domain by reverting to another. In reading this pattern is evident in surface dyslexia, a common diagnostic marker for SD that is characterised by successful rote grapheme-phoneme conversion with a marked inability to read aloud orthographically irregular words (e.g., *yacht*) (Shallice, Warrington, & McCarthy, 1983; Woollams, Lambon Ralph, Plaut, & Patterson, 2007). A complementary reading impairment (i.e., deep dyslexia) has been associated with degraded phonology such that patients rely on semantics, are consequently unable to read aloud nonwords and often have disproportionate difficulties reading abstract relative to concrete words (Coltheart, Patterson, & Marshall, 1987; Glosser & Freedman, 1990).

Acquired neurological disorders of reading also have striking analogues in word repetition disorders. Consider the syndrome of transcortical sensory aphasia, a form of stroke aphasia associated with semantic access impairment that affects comprehension and production (Berthier, 1999). Although repetition of single words and nonwords is preserved, the impaired access to semantics leads to a reduction in imageability effects (typically associated with semantic processing) in repetition and lexical decision. In past work we have argued that transcortical sensory aphasia forces an over-reliance on phonological processing to repeat (Martin & Saffran, 1990). This limitation becomes apparent when taxing the span of immediate memory beyond two to three target items. For example, Martin and Saffran (1990) reported a case study of a person with transcortical sensory aphasia who was able to repeat two-word strings accurately, but when presented with strings of three or more words she consistently repeated the last two items first (in serial order or sometimes not) and then produced mostly nonword errors that were phonologically similar to the earlier items in the string. Martin and Saffran (1990) attributed this error pattern in repetition to an extreme reliance on activated phonological representations of the words, which is strongest for items in the most recent position of the word string. They further contended that in the absence of feedback from semantic representations of words

(due to the semantic access deficit) phonological activation of earlier items is not maintained, leading to the production of phonologically related nonwords. In contrast, phonologically based aphasias (conduction aphasia, phonological dysphasia) tend to manifest amplified imageability and frequency effects in repetition, thus demonstrating reliance on intact activation of semantic representations in order to repeat single words or recall word strings (see also Martin & Saffran, 1997). Related to this pattern is a syndrome known as deep dysphasia (parallel to the reading disorder, deep dyslexia), characterised by imageability effects and semantic errors in repetition of single words. This pattern has been attributed to a primarily phonological impairment coupled with some difficulty maintaining activation of semantic representations (e.g., Howard & Franklin, 1988; Martin, Dell, et al., 1994; Michel & Andreewsky, 1983). Other cases with phonological processing impairments have been reported to produce semantic errors in repetition of word sequences (Trojano & Grossi, 1995), semantic descriptions of words when repeating word sequences (Martin, Lesch & Bartha, 1999) and paraphrases when repeating sentences (Saffran & Marin, 1975).

In summary, in the context of impaired access to semantic and/or phonological representations, reading and repetition abilities often reflect graded reliance on accessibility to a single domain. Lesion models offer a powerful means for parsing the relative contributions of phonology and semantics to vWM. We employed this approach here by contrasting lexicality errors of two clinical populations with relatively focal impairments of either semantic memory (i.e., semantic dementia) or phonological processing (i.e., progressive nonfluent aphasia). Importantly, both populations tend to show relative preservation of medial temporal lobe structures that are dedicated to essential aspects of binding and retrieval of memory.

SEMANTIC DEMENTIA AS A LESION MODEL FOR A SELECTIVE IMPAIRMENT OF SEMANTIC MEMORY

There is perhaps no better naturally occurring lesion model for impairment of semantic memory than semantic dementia (hereafter SD). SD is a variant of frontotemporal dementia described by Warrington (1975) as a *selective impairment of semantic memory*. Decades of work have solidified these claims by demonstrating the stability and consistency of a multi-modal conceptual loss that underlies SD (Hodges, Salmon, & Butters, 1992; Hodges, Graham, & Patterson, 1995; Lambon Ralph, Graham, Patterson, & Hodges, 1999; Rogers et al., 2004). That is, unlike in stroke aphasia, patients with SD tend to show comparable impairment across many representational modalities as a result of degradation to conceptual knowledge (Jefferies & Lambon Ralph, 2006; Jefferies, Patterson, & Lambon Ralph, 2008). However, these deficits do tend to occur in the presence of often remarkably preserved function in non-semantic domains, including phonological perception and production, number knowledge, and complex visuospatial abilities (Green & Patterson, 2009; Jefferies, Patterson, Jones, Bateman, & Lambon Ralph, 2004; Jefferies, Patterson, & Lambon Ralph, 2006). Of note, patients with SD are often considered to show preserved AVSTM as evident by excellent single word repetition and essentially normal digit span (but see Reilly, Martin, & Grossman, 2005). Importantly, phonological difficulties tend to occur very late (if ever) during the course of the disease (Jefferies, Jones, Bateman, & Lambon Ralph, 2005; Jefferies et al., 2006; Kwok, Reilly, & Grossman, 2005; see also Reilly & Peelle, 2008).

The constellation of preserved versus degraded cognitive functions in SD has a neuroanatomical basis in the circumscribed cerebral atrophy that is a hallmark of frontotemporal dementia. The early to moderate stages of SD are characterised by relatively focal atrophy of grey matter within the inferolateral and anterior temporal lobes, with relative sparing of the hippocampal formation, primary auditory cortex, and frontal lobe structures that are critical for phonological production and perception (Brambati et al., 2009; Mummery et al., 2000; Pereira et al., 2009; Rohrer et al., 2008; Snowden, Goulding, & Neary, 1989; Snowden, Neary, & Mann, 2002).

PROGRESSIVE NONFLUENT APHASIA (PNFA) AS A LESION MODEL FOR A SELECTIVE IMPAIRMENT OF PHONOLOGICAL PROCESSING

PNFA is a progressive neurodegenerative disorder that is characterised by the degradation of phonological and grammatical production, localised primarily to the asymmetric atrophy of left inferior frontal and anterior perisylvian regions that are critical for speech production (Gorno-Tempini et al., 2006; Nestor et al., 2003; but see Patterson, Graham, Lambon Ralph, & Hodges, 2006). Early reports describing PNFA note the presence of phonological errors as potentially distinct from speech production errors (e.g., Croot, Patterson, & Hodges, 1998; Neary et al., 1998). Controversy continues relative to distinguishing production errors as characteristic of phonological processing versus motor speech impairment (Grossman, 2010; Grossman & Ash, 2004; Josephs et al., 2006). However, recent evidence from analysis of speech samples collected from 16 individuals with PNFA demonstrated a large preponderance of phonemic (i.e., errors that are well articulated and language appropriate) relative to phonetic (e.g., errors that result in sound/s that do not occur in the speaker's language) speech errors (Ash et al., 2010), which the authors contend reflects impairment of the linguistic phonological system. This evidence supports studies demonstrating that phonemic paraphasic errors are characteristic of production attempts in PNFA (Caselli & Jack, 1992; Mendez, Clark, Shapira, & Cummings, 2003). A phonological explanation for production deficits is supported further by observations of concomitant comprehension impairment in the form of deficient phonemic discrimination in patients with PNFA (Grossman et al., 1996), in which impairment to auditory-verbal short-term memory was also noted. Semantic memory has been shown to be relatively intact in early stages of PNFA (but see Reilly, Rodriguez, Peelle, & Grossman, 2011). As with the progression of SD, the constellation of deficits in PNFA gradually evolves from relatively focal production deficits linked to circumscribed brain atrophy to more diffuse impairments in memory, cognition, and motor function as more of the brain is compromised during the neurodegenerative process.

PREDICTIONS AND AIMS

We hypothesise that, in SD, patients will come to heavily rely on phonology for AVSTM and that semantic attributes of the target items assume waning importance as disease severity worsens. Conversely, we hypothesise that the progressive degradation of phonological representations in PNFA produces reliance on lexical-semantic properties with waning reliance on phonology. We contrasted item-level psycholinguistic attributes associated with lexicality errors relative to correctly recalled words and nonwords.

We defined a lexicality error as either (1) producing a nonword when the target is a word (e.g., stork → vrok) or (2) producing a real word when the target item is a nonword (e.g., vrok → stork). Our choice of psycholinguistic variables was constrained to factors that have both quantitative published norms and precedence as influencing recall in past investigations of neuropsychologically impaired populations. We examined the following lexical-semantic variables: imageability, familiarity, frequency, and the following phonological variables: word length, wordlikeness (i.e., the extent to which a nonword is subjectively rated as sounding like a real word), and phonological neighbourhood density. We predicted that, in the context of a semantic impairment (i.e., semantic dementia), patients would show greater sensitivity to phonological attributes of the target items relative to other psycholinguistics variables that characterise meaning. In contrast, a dominant phonological impairment (i.e., PNFA) would result in hypersensitivity to semantic properties of the target items.

METHOD

Participants

Participants with a diagnosis of either semantic dementia ($n = 2$) or progressive non-fluent aphasia ($n = 3$) were recruited from memory disorders clinics at the University of Florida. Diagnoses were subsequently confirmed by an interdisciplinary consensus review mechanism consisting of experienced clinicians in accord with published criteria for these conditions (Neary et al., 1998). Relevant neuropsychological and demographic data appear in Table 1.

Participants were heterogeneous in terms of disease severity, ranging from moderate to severe. Exclusionary criteria were co-morbid neurological conditions (e.g., stroke, tumour) and sedating medications. At the time of testing, all patients were undergoing pharmacotherapies including combinations of NMDA receptor antagonists (e.g., *Memantine*) and acetylcholinesterase inhibitors (e.g., *Donepezil*). Participants and/or

TABLE 1
Demographic and neuropsychological data

<i>P ID</i>	<i>Sex</i>	<i>Age</i>	<i>Dx</i>	<i>Ed.</i>	<i>BNT</i>	<i>MOCA</i>	<i>Trails A:B</i>	<i>Dig F:B</i>	<i>Letter fluency</i>	<i>Animal fluency</i>	<i>Pyr & palm word</i>	<i>Pyr & palm pic</i>
ZB	F	60	SD	18	13	21	31:68	9 : 8	8	9	46	48
BB	M	79	SD	16	17	8	199:t.o.	3 : 4	6	7	43	44
QR	M	74	PNFA	16	24	4	t.o.:t.o.	4 : 1	0	3	44	45
JS	M	70	PNFA	16	30	12	97:t.o.	2 : 2	3	4	44	38
LW	M	75	PNFA	20	58	26	52/116	6 : 3	6	12	52	51

Dx = diagnosis; Ed = years of education; BNT = Boston Naming Test Long, Form, test scores are out of 60 (Kaplan, Goodglass, & Weintraub, 1983); MOCA = Montreal Cognitive Assessment, test scores are out of 30 (Nasreddine et al., 2005); Trails A/B represents time in seconds to complete the Trail Making Test Versions A and B, a "t.o." indicates that we timed the patient out after 300 seconds; Dig F/B = Forward Digit and Backward Span (Wechsler Adult Intelligence Scale III; Wechsler, 1997); Letter Fluency = number of non-repeated words produced in 60 seconds that start with the letter "F"; Animal Fluency = number of non-repeated animal names produced in 60 s; Pyr & Palm Word/Pic = Pyramids and Palm Trees Test Word and Picture Versions (Howard & Patterson, 1992).

caregivers provided written informed consent in accord with protocol approved by the University of Florida's Institutional Review Board. We briefly describe each of the individual cases to follow.

Patient ZB. ZB is a 60-year-old female diagnosed with semantic dementia in 2010 approximately 1 year after the onset of symptoms. ZB was a surgical nurse with Master's-level schooling who reported first having difficulties distinguishing and naming surgical tools. This soon progressed to difficulties in discriminating medical conditions and communicating post-operative instructions to her patients. ZB now presents with severe anomia and moderate non-verbal semantic memory impairment (see Table 1). Although staging guidelines for the severity of SD are highly variable, patient ZB can reasonably be classified as mild-moderate based on disease duration and symptomatology. Serial structural neuroimaging scans over one year demonstrating the progression of ZB's atrophy appears in Figure 1.

Patient BB. BB is a 79-year-old male with a Bachelor's-level education who was diagnosed with semantic dementia in 2009 after 2 years of subtle language problems. BB is a retired police officer whose chief complaint is poor memory for words. His impairments in naming and verbal fluency bear these complaints out (see Table 1). On structural MRI, BB shows marked unilateral lobar atrophy (left > right). He also has begun to show evidence of nonverbal semantic impairment in activities of daily living (e.g., adding non-edible ingredients to recipes).

Of the patients we report here, BB is perhaps the most atypical in terms of representing a canonical diagnosis of semantic dementia. First, BB is somewhat aged for a diagnosis of frontotemporal dementia, whose average onset is typically early during the sixth decade of life with a tapering incidence during later years (Forman et al., 2006; Hodges et al., 2010). This led us to initially vacillate between diagnoses of atypical Alzheimer's disease versus an older onset of frontotemporal dementia. Ultimately, in our consensus review, we ruled in favour of semantic dementia in light of three primary sources of evidence: (1) At test, BB did not manifest severe anterograde episodic memory impairments that are a hallmark of Alzheimer's disease; (2) BB's MRI scan revealed an asymmetric progression of left hemisphere cortical lobar atrophy that is commonly reported in semantic dementia but has not to our knowledge been associated with Alzheimer's disease (Galton et al., 2001; Mummery et al., 2000);

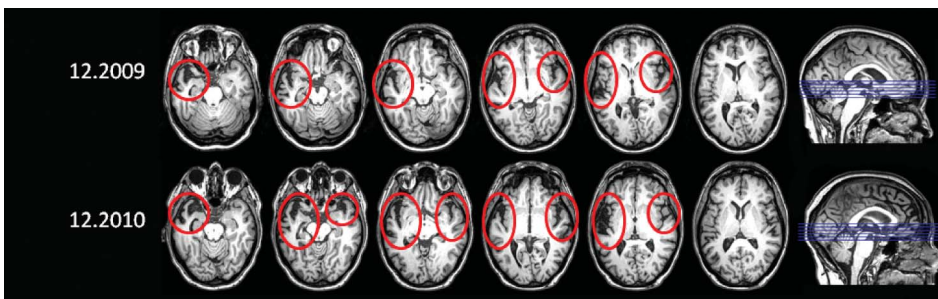


Figure 1. Serial structural magnetic resonance imaging of semantic dementia (patient ZB). Figure demonstrates serial temporal lobe atrophy in patient ZB over 1 year. To view this figure in colour, please see the online issue of the Journal.

and (3) BB's hippocampi did not show the disproportionate atrophy that often marks moderate to late stage Alzheimer's disease.

Patient QR. QR is a 74-year-old male retired engineer with a Bachelor's-level education. QR was diagnosed in 2010 with PNFA after approximately 1 year of naming difficulties. At the time of testing, QR's speech production was severely impaired, bordering on near mutism in spontaneous conversation. In addition to these speech output difficulties, QR is now alexic and agraphic.

Patient JS. JS is a 70-year-old male who was diagnosed with PNFA in 2010 after 2 years of progressive language and memory disturbance. JS is a retired mechanical engineer with a Bachelor's-level education. His speech is characterised by clipped one-word utterances, incessant restarts, hesitations, and audible struggle. JS recently began to experience reading impairment but is otherwise functionally independent.

Patient LW. LW is a 75-year-old male with a Doctoral degree in ecology who was diagnosed with PNFA after 6 months of progressive speech problems. LW has clear insight into these difficulties and has consistently described his impairment as "I can't speak." LW is a retired college professor and renowned wildlife author. He reports recent difficulties in high-level writing that have forced him to stop writing his regular column for a wildlife magazine.

Materials and procedure

Participants first underwent a battery of neuropsychological and language assessments (see Table 1). Then over multiple sessions we administered specific subtests of the *Temple Assessment of Language and Short Term Memory in Aphasia (TALSA)* (Kalinyak-Fliszar, Kohen, & Martin, 2011; Martin, Kohen & Kalinyak-Fliszar, 2010). In an effort to reflect the diversity of both semantic and phonological relatedness effects on recall we presented subtests of the TALSA varied by list relatedness. Patients were requested to repeat lists of either words or nonwords, and these lists were presented in discrete blocks (i.e., exclusively words or exclusively nonwords).

For the word lists, items were (1) semantically and phonologically unrelated (e.g., *skunk, car*), (2) semantically related and phonologically unrelated (e.g., *skunk, beaver*), or (3) phonologically related but semantically unrelated (e.g., *skunk, skull*). Items in the phonologically related word strings shared onsets. Words varied in length from one to three syllables; however, word length was matched overall across trials. For example, if a particular three word list of semantically related words had nine constituent syllables, all other trials within that list also had nine syllables. Importantly, items in the TALSA parametrically vary on the following psycholinguistic dimensions known to influence lexical processing: word frequency, age of acquisition, familiarity, imageability, phoneme length, phonological neighbourhood density, and wordlikeness. In addition to real words the TALSA also contains a nonword list repetition condition. Nonwords were derived by changing one to three phonemes of the real word items, sampling equally from initial, medial, and final positions of the original word. This procedure generated a wide range of nonword stimuli varying in wordlikeness and phonological neighbourhood density. Psycholinguistic attributes of the target items appear in Table 2.

TABLE 2
Psycholinguistic attributes of the stimuli

	<i>List length</i>										
	<i>1</i>		<i>2</i>		<i>3</i>		<i>4</i>		<i>5</i>		
	M (SD)		M (SD)		M (SD)		M (SD)		M (SD)		
	<i>Word</i>	<i>Nonword</i>	<i>Word</i>	<i>Nonword</i>	<i>Word</i>	<i>Nonword</i>	<i>Word</i>	<i>Nonword</i>	<i>Word</i>	<i>Nonword</i>	<i>Total</i>
Freq	1.12 (.53)	n/a	.99 (.75)	n/a	1.00 (.61)	n/a	1.51 (.62)	n/a	1.74 (.58)	n/a	1.14 (.70)
AOA	227.00 (50.08)	n/a	259.48 (56.86)	n/a	268.97 (41.02)	n/a	225.70 (45.44)	n/a	270.93 (48.91)	n/a	260.85 (49.77)
Fam	544.49 (54.69)	n/a	538.23 (59.77)	n/a	529.11 (49.91)	n/a	559.42 (49.48)	n/a	568.19 (46.24)	n/a	541.76 (54.63)
Imag	592.91 (47.39)	n/a	594.55 (31.46)	n/a	595.26 (32.24)	n/a	592.73 (35.40)	n/a	599.02 (22.93)	n/a	595.08 (32.43)
P-Length	4.74 (1.59)	3.72 (.89)	4.98 (1.44)	3.71 (.75)	4.57 (1.38)	3.87 (.97)	3.51 (1.22)	3.60 (1.06)	3.91 (1.04)	4.02 (.80)	4.36 (1.26)
Density	10.08 (9.93)	n/a	5.87 (7.57)	n/a	8.69 (8.85)	n/a	15.09 (10.37)	n/a	8.84 (6.89)	n/a	8.54 (8.90)
Wordlike	n/a	4.64 (.77)	n/a	4.31 (1.21)	n/a	4.33 (1.05)	n/a	3.90 (.99)	n/a	4.13 (.75)	4.23 (1.01)

Freq = frequency, AOA = age of acquisition, Fam = familiarity, Imag = imageability, P-Length = phoneme length, Density = neighbourhood density, Wordlike = wordlikeness.

Testing procedure. Patients were seated at a desktop computer in a quiet setting. We standardised stimulus presentation using E-Prime 2.0 Professional software (Psychology Tools Inc, 2010). Upon a brief familiarisation sequence, E-Prime presented auditory stimuli at a rate of one word per second (1000 ms interstimulus interval) as wavefiles over external speakers. Stimulus lists began at the one-item level and ascended in length until attaining the individual patient's maximum span. We operationally defined span as the list length at which a patient was unable to correctly recall more than 50% of items in either free or serial order. Immediately upon hearing each stimulus list, patients received a brief audiovisual cue prompting them to repeat the list in order. We digitally recorded video and audio for each session and scored all responses offline. We administered a range of nonword and word lists varied by specific psycholinguistic attributes described to follow. Experiment order was counter-balanced across both lexicality (i.e., word or nonword) and list relatedness condition (i.e., phonological, semantic, or unrelated lists). All testing was conducted over an approximately 2-month period, and all neuropsychological measures were collected within a window of six months contemporary with the WM testing.

Patients were cued to repeat separate lists of words and nonwords beginning with one item and ascending to maximum span. Thus, differences in span dictated the total number of stimulus items attempted by each patient. At each length beyond one target item, patients completed 10 trials. For example, at the two-word level, patients were cued to repeat 10 separate lists such as "shoe . . . girl". If that patient exceeded 50% accuracy, she would receive a set of 10 three-word lists, and this process would continue until attaining span.

Data analyses. In the analyses to follow we exclusively examined lexicality errors. A lexicality error can hypothetically occur in either of two directions. *De-lexicalisation* occurs when a patient produces a neologism when attempting to recall real word (e.g., dog, cat, bat → bod, dat, cov), whereas *lexicalisation* occurs when a patient erroneously produces a real word when attempting to recall a nonword (e.g., blat, vram, flob → bat, bomb, flop). We isolated both types of lexicality errors by first collapsing *all* observed errors into a single matrix. We then coded each error as either lexical or non-lexical in nature. Non-lexical errors included phonemic distortions that shared at least one syllable overlap with the target (e.g., umbrella → umbellug), semantically and visually related substitutions (e.g., umbrella → mushroom), omissions (e.g., umbrella → "I don't know"), and other non-lexical errors (for further discussion of error coding schema as pertains to phonological errors, see also Reilly, Peelle, et al., 2011; Reilly, Rodriguez, et al., 2011). We defined a lexicality error as one in which the patient produced a nonword that shared no syllable overlap with the target OR when the patient produced a real word in place of a target nonword.

We then conducted a series of planned contrasts examining psycholinguistic attributes of correctly recalled responses to lexicality errors. We obtained word frequency values (normalised per million words) from SubtLexUS psycholinguistic database (Brysbaert & New, 2009). We obtained values for age of acquisition, familiarity, imageability, and phoneme length from the MRC Psycholinguistic database (Coltheart, 1981). We obtained phonological neighbourhood density values (i.e., the number of real word neighbours that can be generated by deletion, substitution, or addition of any single phoneme) (Luce & Pisoni, 1998) from the Washington

University Speech & Hearing Lab Neighbourhood Database (Sommers, 2011). We derived our own in-house measure of the wordlikeness (phonological plausibility of a nonword) by querying 19 independent raters (age $M = 27.74$), who rated each nonword's similarity to a real word on a Likert scale from 1 (not at all plausible as an English word) to 7 (highly plausible as an English word). Additional planned contrasts involved assessing psycholinguistic properties of recalled relative to forgotten items as functions of disease identity (i.e., PNFA vs SD) and disease severity (mild-moderate or severe).

RESULTS

It is critical to note that all patients showed limited recall for both words and nonwords and accordingly committed many errors. The average word list span was 2.6 (range 1–4), and the average nonword list span was 1.2 (range 0–3). All patients showed an advantage in recall accuracy for words relative to nonwords as confirmed by a significant Wilcoxon signed rank test contrasting word-nonword recall span differences (Wilcoxon $p = .03$). The magnitude of the word-nonword recall accuracy difference did not differ as a function of disease aetiology when contrasting PNFA versus SD (Mann Whitney U Test $p = .74$).

Individual patient performance is enumerated in Table 3, and Figure 2 illustrates each patient's distribution of recall errors collapsed across all list lengths. As is evident in Figure 2, lexicality errors were common among all patients, accounting for 18.5% of all errors. However, there was no reliable correlation between the relative proportion of observed lexicality errors and either disease severity or nosology. That is, patients ZB, BB, JS, and LW all committed grossly similar relative proportions of lexicality errors (11.5, 17.93, 14.41, and 14.46). However, patient QR (severe PNFA) was unique among this cohort, producing almost double the relative proportion of lexicality errors as the others (i.e., 30%).

Severity of semantic impairment was a stronger predictor than disease aetiology (PNFA or SD) with respect to the directionality of nonword errors (see Table 3). That is, the more severely semantically impaired patients were more likely to commit an error in the direction of producing a neologism when the target was a real word ($z = -1.77$, $p = .08$), irrespective of their diagnosis. In contrast, the more mildly impaired patients trended towards a higher likelihood of "lexicalising" nonwords (i.e., turning a nonword target into a real word) ($z = -1.73$, $p = .08$).

TABLE 3
Serial recall error distributions

P ID	W Span	NW Span	Lex error	% of Total error	% NW-W	% W-NW	Non Lex error	% of Total error	Total error	Correct	Total
ZB	4	3	13	11.50	8.67	0.33	100	88.50	113	342	455
BB	2	0	52	17.93	20.67	7.93	238	82.07	290	152	442
QR	2	0	71	29.96	26.67	20.98	166	70.04	237	98	335
JS	1	0	49	14.41	20.67	5.90	291	85.59	340	115	455
LW	4	3	12	14.46	6.67	1.64	71	85.54	83	372	455

W Span = average word span, NW Span = average non-word span, P ID = patient ID, Lex Error = lexical error, Non-Lex Error = non-lexical error. We operationally defined maximum span (span length) as the list length at which a participant was unable to recall > 50% of all items.

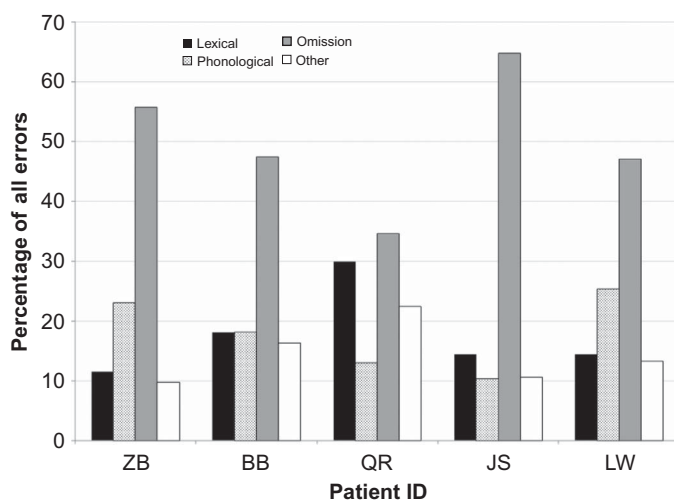


Figure 2. Proportion of errors across patients. Graph reflects the proportion of each error type relative to all errors. *Other* errors denote perseverations, semantic errors, mixed errors, and other unclassifiable responses.

Item-level results

We also examined item-level psycholinguistic properties of the words and nonwords in an effort to discern which variables were predictive of making a nonword response error. Using parametric statistical procedures we treated words and nonwords as independent observations within each patient. For example, patient BB made 52 nonword errors in the context of 152 correct responses. We contrasted the attributes of BB's sample of correct responses ($n = 152$) to those of his incorrect responses ($n = 52$) assuming independence of the item-level response data within each patient.

When inspecting the item-level data distribution we found that word frequency violated assumptions of normality and homogeneity of variance. We therefore recomputed word frequency using a log transformation. All additional contrasts satisfied the assumptions of variance and normality and were conducted using the original raw values. Table 4 lists the summary statistics for each of the psycholinguistic variables described individually to follow. Patient ZB (mild semantic dementia) made very few errors (i.e., 1 total) in the direction of a word-to-nonword error. Therefore the individual patient contrasts below do not reflect AOA, familiarity, imageability, and neighbourhood density for patient ZB. Likewise we were unable to evaluate effects of age of acquisition for AOA because his errors included words for which no published norms are available.

Word frequency

Figure 3 reflects mean differences in word frequencies for correctly recalled items relative to targets in which a lexicality error occurred. Patient JS (severe PNFA) made more lexical errors on low relative to high frequency words, $t(119) = -2.50$, $p = .01$. No other patient differed significantly with respect to frequency. Contrasts of ZB's performance were precluded due to only one observed error in the direction of a word-to-nonword error.

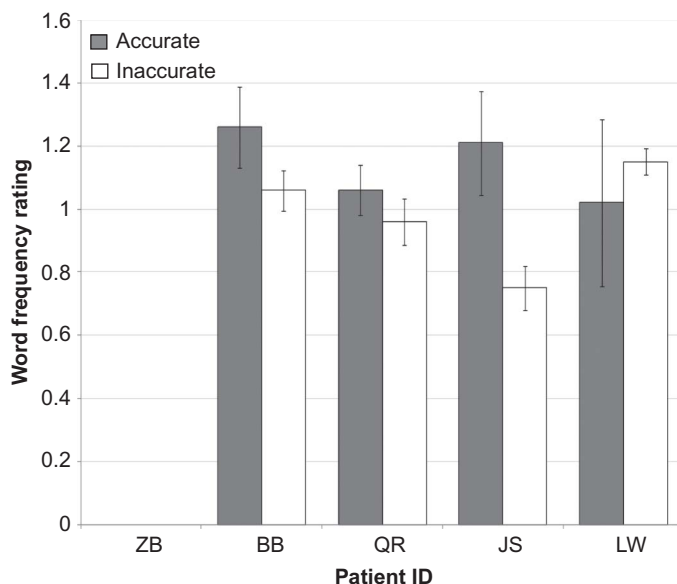


Figure 3. Mean frequency rating for accurately versus inaccurately guessed target words across patients. Graph displays log transformed word frequency ratings for accurate relative to inaccurately guessed target words. Frequency ratings are based on frequency per million words. Frequency ratings have possible range of 0 to 41857 with a mean of 25.23 (Brysbaert & New, 2009).

Age of acquisition

Figure 4 reflects mean differences in word AOA for correctly recalled items relative to targets in which a lexicality error occurred. QR (PNFA severe) trended toward making more lexical errors on words with a later AOA relative to words with an earlier AOA, $t(48) = 1.89$, $p = .07$. No other patient showed an age of acquisition advantage.

Familiarity

Figure 5 reflects mean differences in word familiarity for correctly recalled items relative to targets in which a lexicality error occurred. Both BB (moderate semantic dementia), $t(132) = -1.77$, $p = .08$, and QR (PNFA severe), $t(122) = -1.87$, $p = .06$, trended towards making more lexical errors on low familiarity words.

Imageability

Figure 6 reflects mean differences in word imageability for correctly recalled items relative to targets in which a lexicality error occurred. JS (PNFA severe), $t(93) = -1.98$, $p = .05$, and LW (mild PNFA), $t(220) = -2.00$, $p = .05$, produced more nonword errors for less-imageable (i.e., abstract) words relative to high-imageability (concrete) words.

Phoneme length

Figure 7 reflects mean differences in phonemic length for correctly recalled items relative to targets in which a lexicality error occurred. ZB (mild semantic dementia) produced more nonword errors for shorter words, $t(21.6) = -5.34$, $p = .01$.

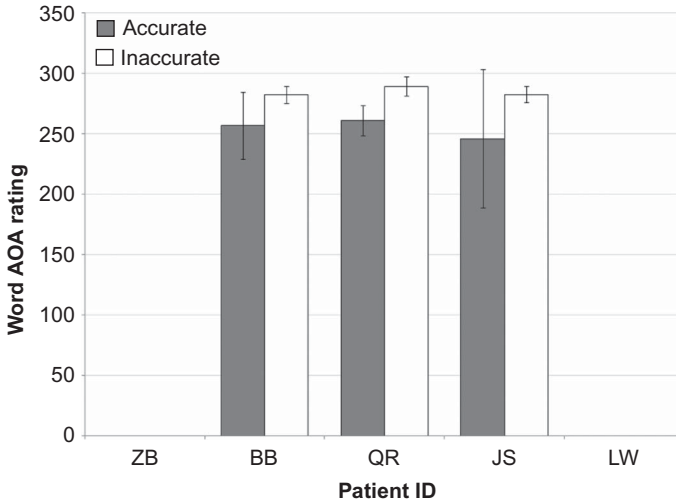


Figure 4. Mean AOA for accurately versus inaccurately guessed target words across patients. Graph displays AOA ratings for accurate relative to inaccurately guessed target words. AOA ratings lie within the range of 100 to 700 with a mean of 405 (Coltheart, 1981).

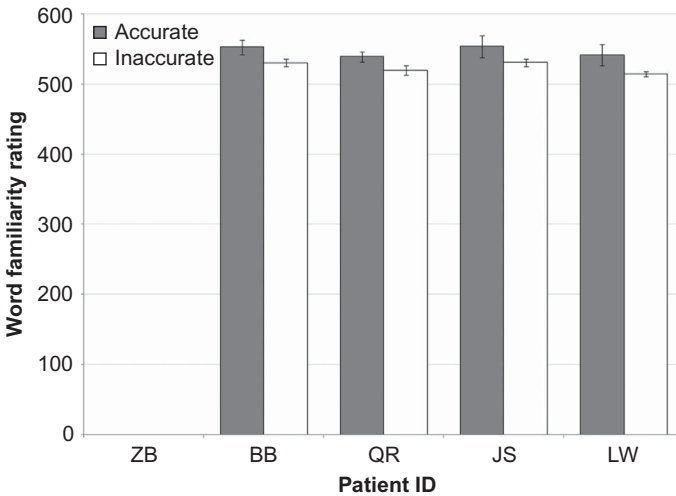


Figure 5. Mean familiarity rating for accurately versus inaccurately guessed target words across patients. Word familiarity ratings for accurate relative to inaccurately guessed target words. Word familiarity ratings lie within the range of 100 to 700 with a mean of 488 (Coltheart, 1981).

Phonological neighbourhood density

Figure 8 reflects mean differences in neighbourhood density for correctly recalled items relative to targets in which a lexicality error occurred. No patient showed an effect of phonological neighbourhood density for erred relative to correct responses.

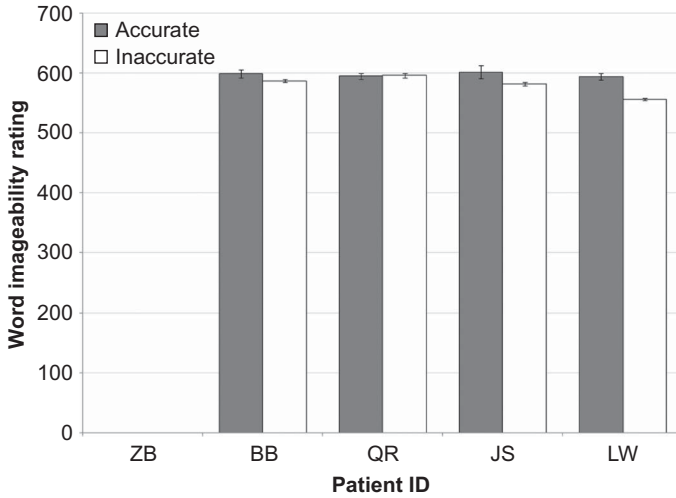


Figure 6. Mean imageability rating for accurately versus inaccurately guessed target words across patients. Graph displays word imageability ratings for accurate relative to inaccurately guessed target words. Word imageability ratings lie within the range of 100 to 700 with a mean of 450 (Coltheart, 1981).

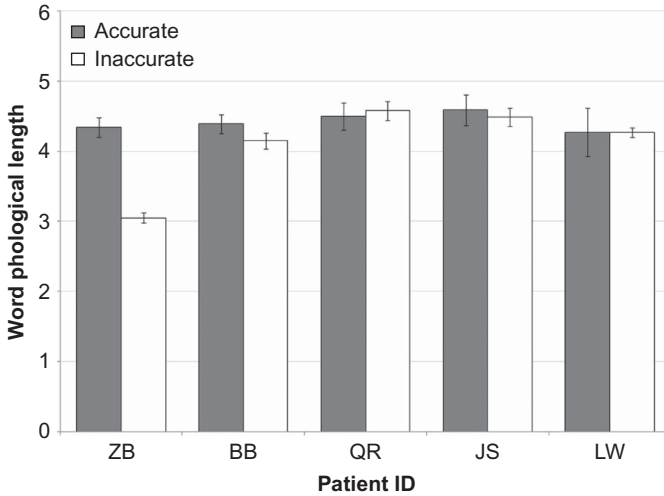


Figure 7. Mean word length for accurately versus inaccurately guessed target words across patients. Graph displays word length for accurate relative to inaccurately guessed target words. Word length here reflects total number of phonemes in the word.

Wordlikeness

Figure 9 reflects mean differences in wordlikeness for correctly recalled items relative to targets in which a lexicality error occurred. BB (moderate semantic dementia) produced more errors for less wordlike targets, $t(41) = -2.12, p = .04$.

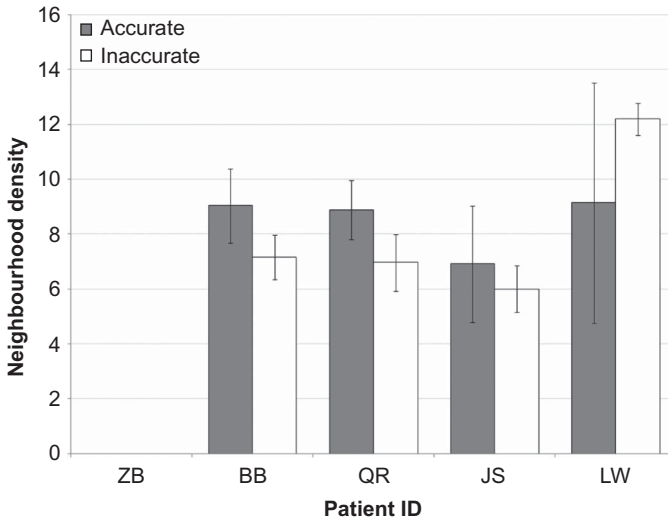


Figure 8. Mean neighbourhood density for accurately versus inaccurately guessed target words across patients. Graph displays word neighbourhood density for accurate relative to inaccurately guessed target words. Neighbourhood density is derived from the number of real word neighbours that can be generated by deletion, substitution, or addition of any single phoneme (see Sommers, 2011).

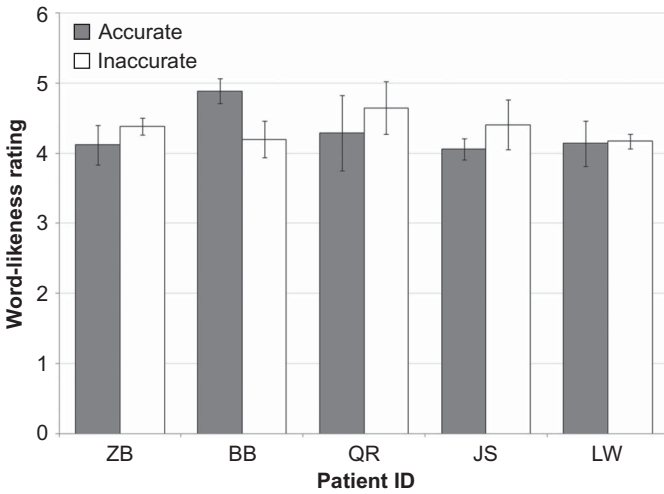


Figure 9. Mean subjective wordlikeness rating for accurately versus inaccurately guessed target words across patients. Graph displays the wordlikeness ratings for accurate relative to inaccurately guessed target words. Wordlikeness ratings reflect a 7-point Likert scale (mean = 4.24).

GENERAL DISCUSSION

We examined lexicality errors in SD and PNFA with attention to the specific psycholinguistic attributes of the target items that elicited an error. As a general trend, SD patients showed more sensitivity to phonological relative to semantic variables. In contrast, PNFA patients were swayed more by lexical-semantic attributes such as

familiarity, age of acquisition, and frequency. Across all patients the severity of an individual's semantic impairment was a stronger predictor of directionality of a non-word error than was disease aetiology. That is, patients with a more severe semantic impairment were more likely to produce a neologistic error in place of a real word target (e.g., *stork* → *vrom*). In contrast, patients with a more mild semantic impairment tended to err in the opposite direction, i.e., producing a real word when the target item was a nonword (e.g., *vrom* → *stork*). This constellation of findings is generally consistent with the hypothesis that in the context of degraded knowledge in one domain (e.g., phonology or semantics), an individual will revert to a dominant influence of the preserved domain. Thus, during a word list repetition task such as we employed here, patients showed parallels to impairments that are also evident in reading (e.g., surface dyslexia) and in single word repetition disorders (e.g., surface dysphasia).

Concluding remarks & treatment ramifications

The current results support models of language and memory premised upon a highly interactive contribution of semantics and phonology to vSTM (Acheson et al., 2010; Martin, Saffran, & Dell, 1996). These findings also have clinical relevance towards informing treatments for the profound language impairments associated with primary progressive aphasia. Importantly, we hypothesise that semantic dementia produces reliance on surface-level properties of words. In earlier related work we showed that patients with this disorder spontaneously exploit phonological regularities of words such as length and syllable stress placement when making explicit judgments of meaning and grammatical class (Reilly, Cross, Troiani, & Grossman, 2007). The exploitation of such cues demonstrates preserved bootstrapping abilities in SD (i.e., using cues at one level of linguistic processing to make inference about another level) that may spontaneously emerge as a way of prolonging language functioning. For this reason phonology becomes a critical factor in facilitating communication in SD and one that in fact appears to remain a residual strength that can be capitalised on for language therapy in this population.

As semantic knowledge degrades in SD we have hypothesised that patients spontaneously see a steady, graded shift towards reliance on preserved phonological processing, ultimately evolving to near complete formal (phonological or surface level) dominance for memory encoding (see also the compensatory processing account of Jefferies, Crisp, & Lambon Ralph, 2006). Such phonological reliance (as evident by sensitivity to a wordlikeness effect in BB) can theoretically be either adaptive or detrimental. For example, SD patients with surface dyslexia are typically able to exploit preserved grapheme-to-phoneme correspondence to successfully read orthographically irregular words. Yet the misapplication of this processing heuristic when encountering strange cases (e.g., low-frequency, orthographically irregular words) is likely to produce breakdowns in communication (see also Reilly et al., 2007, for a discussion of the misapplication of this heuristic in the context of making judgements of single word meaning).

Both PNFA and SD patients are likely continue to benefit from the advantage of real words relative to nonwords at least until very late stages during the course of their disease progression (see also Rogers, Lambon Ralph, Hodges, & Patterson, 2004). All of the patients we reported here retained a lexicality advantage by showing superior memory span for words relative to nonwords. Moreover, this recall advantage was not diminished as a function of either disease entity or severity. This finding suggests

that words do not simply devolve into nonwords in PNFA and SD as these patients experience progressive fading of lexical-semantic support. Instead it seems likely that this advantage enjoyed by real words results from sensitivity to a combination of cues, including lexical-phonological familiarity, word frequency, and idiosyncratically residual semantic knowledge,

Although the source of impairment in SD or PNFA may be different from that in stroke aphasia, the cognitive-behavioural shift of dependence towards residual language abilities is in many ways similar. Yet these conditions are also distinct, in that stroke produces a static or improving language impairment relative to the inexorable loss seen in progressive aphasia. During recent years the field of language rehabilitation has seen rapid advances in the treatment of progressive language disorders (Gonzalez-Rothi et al., 2009; Jokel, Rochon, & Leonard, 2006). Improved treatment specificity will demand consideration of the role of optimising vSTM in lexical reacquisition while also determining the most effective means of capitalising on residual language abilities (e.g., phonological representations).

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