Annual Review of Applied Linguistics (2008) **28**, 73–90. Printed in the USA.
Copyright © 2008 Cambridge University Press 0267-1905/08 $16.00
doi:10.1017/S0267190508080045

4. LANGUAGE AND DEMENTIA: NEUROPSYCHOLOGICAL ASPECTS

Daniel Kempler and Mira Goral

This article reviews recent evidence for the relationship between extralinguistic cognitive and language abilities in dementia. A survey of data from investigations of three dementia syndromes (Alzheimer's disease, semantic dementia and progressive nonfluent aphasia) reveals that, more often than not, deterioration of conceptual organization appears associated with lexical impairments, whereas impairments in executive function are associated with sentence- and discourse-level deficits. These connections between extralinguistic functions and language ability also emerge from the literature on cognitive reserve and bilingualism that investigates factors that delay the onset and possibly the progression of neuropsychological manifestation of dementia.

Introduction

Language deficits are frequent in dementia: Patients with dementia demonstrate, among other signs, word-finding problems (anomia), sentence comprehension deficits, and lack of cohesion in discourse. Unlike aphasias that are due to focal brain damage, language deficits in dementia occur in the context of multiple cognitive impairments.

This article reviews recent findings regarding language impairment in three dementia syndromes with reference to the associated cognitive deficits in each. In particular, we attempt to highlight the instances in which language problems are due to a primary linguistic impairment versus decline in other neuropsychological functions.

We first review relevant data from three dementia syndromes: dementia of the Alzheimer's type (DAT) and two variants of frontotemporal dementia (FTD): semantic dementia (SD) and primary progressive nonfluent aphasia (PNFA). These three syndromes can be distinguished by their impairment patterns and distribution of neuropathology. DAT is characterized by a progressive deterioration of memory and at least two other cognitive domains (such as language, visuospatial perception, executive function). The neuropathology of Alzheimer's disease involves regions

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

throughout the brain, particularly the hippocampus and areas in the frontal cortex (Kempler, 2005; Mendez & Cummings, 2003). Two varieties of FTD are distinguished from other dementia syndromes by their marked language impairments.[1] One FTD variant, SD, is characterized by fluent speech output accompanied by anomia and comprehension impairments. The neuropathology of SD appears to be primarily temporal in distribution. SD can be clinically confused with DAT in those patients with DAT who show relatively early and circumscribed language impairment. Another FTD variant, PNFA, is characterized by nonfluent speech output and anomia alongside relatively preserved comprehension. The neuropathology of PNFA appears to be frontal in distribution. PNFA, due to the nonfluent speech output is rarely confused with the symptomatology of DAT. Although these two FTD syndromes are described as distinct, there are patients who demonstrate a mixed pattern with symptoms of both SD and PNFA (Grossman & Ash, 2004; Kempler, et al., 1990; Kertesz, Davidson, McCabe, Takagi, & Munoz, 2003; Mesulam, 1982; Rogalski & Mesulam 2007).

In the first part of this review we discuss the lexical, sentence, and discourse impairments associated with each syndrome. The data suggest that these three syndromes differ with regard to the relative contribution of (1) grammatical (including phonological and morphosyntactic), (2) semantic/conceptual and (3) extralinguistic deficits to the observed language impairments. In the second part, we review the literature on cognitive reserve and bilingualism, also highlighting the close relationship between language and cognitive changes in dementia.

Language Changes in Three Dementia Syndromes

Extralinguistic Deficits Account for the Early Lexical Impairments of Dementia of the Alzheimer's Type (DAT)

Lexical impairments in DAT have been studied for decades (e.g., Aronoff et al., 2006; Irigaray, 1967; Kempler, 1988). Patients with DAT have trouble recalling names and other words, often substituting pro-forms (e.g., "he," "it"), using conceptually related words (e.g., "dog" for "horse"), or pausing when they cannot generate a target word in conversation or in structured tasks. Anomia, at least in spontaneous speech and simple picture-naming tasks, could be due to extralinguistic deficits or a deterioration of the underlying semantic/conceptual system. Extralinguistic deficits can include inattention to the task, forgetting the target word, or being distracted by related competitor responses. A semantic/conceptual impairment, defined as a loss of underlying semantic memories, would be manifested in lexical production, as well as any other task that relies on that meaning, including comprehension, knowledge of category relationships, attributes, and the like.

Data from several sources can help shed light on this issue. Studies have shown that word-picture matching is relatively intact in DAT, even when naming is not, suggesting that the semantic system is sufficiently preserved to support distinguishing semantically related words from one another (e.g., Astell & Harley,

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

1998; Rogers, Ivanoiu, Patterson, & Hodges, 2006). Several other tasks have been used to probe the semantic system in DAT more deeply, including word definition (Astell & Harley, 2002) and similarity judgments in which participants sort or arrange words so that similar items are grouped together (e.g., Aronoff et al., 2006; Bonilla & Johnson, 1995; Ober & Shenaut, 1999). In both definition and similarity judgment tasks, patients with DAT have performed similarly to healthy controls, giving correct (although impoverished) definitions and grouping categorically similar words together.

Semantic priming is another method to examine underlying semantic knowledge. Priming results that resemble those obtained from healthy individuals suggest that automatic semantic activation is intact in individuals with DAT (e.g., Ober, 2002; Ober, Jagust, & Koss, 1991; Shenaut & Ober, 1996). However, it should be noted that priming studies of individuals with DAT have yielded contradictory results (i.e., lack of semantic priming, hyperpriming, or priming effects comparable to control participants). It has been noted that even partially degraded semantic information might result in intact semantic priming, making it difficult to ascertain the degree of semantic/conceptual loss (Giffard, Desgranges, & Eustache, 2005). These priming data and those documenting qualitatively intact performance on tasks of word comprehension, word definition, and similarity judgments imply that the well-documented and pervasive symptom of anomia in DAT can be attributed to extralinguistic cognitive deficits. That said, it is undeniable that as the disease progresses, data from a range of lexical semantic tasks, including those mentioned earlier, show deterioration of the semantic/conceptual system (e.g., Aronoff et al., 2006).

The overall picture that emerges from the literature at this point is one of a slow stepwise deterioration of a semantic system. Early on in the process, the loss of semantic features is minimal and does not interfere with performance on a wide range of semantic tasks (e.g., word comprehension and definition, similarity judgment, priming). At this point, memory and attention are often moderately impaired and do interfere with task performance. However, as the semantic deterioration progresses, naming errors change in quality (early on, contrast coordinates: “apple” for “pear”; later, superordinates: “fruit” for “apple”; still later: “I don’t know”), comprehension deteriorates and deficits in other lexical tasks will become more apparent as well (see Aronoff et al., 2006, and Gonnerman, Andersen, Devlin, Kempler, & Seidenberg, 1997, for discussion of how this progression can be conceptualized and map onto specific task performance). At these later points in the dementing process, the semantic/conceptual system is no longer intact, and errors in naming and other lexical tasks can be attributed to a combination of extralinguistic and semantic/conceptual deficits.

Extralinguistic Deficits Explain Sentence and Discourse Comprehension and Production Impairments of DAT

Sentence production in DAT is characterized by intact morphosyntactic structure (i.e., subject-verb agreement, well-formed plural and tense markings) (Kavé,

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

2003; Kavé, Leonard, Cupit, & Rochon, 2007; Kemper, LaBarge, Ferraro, Cheung, & Storandt, 1993; Kempler, Curtiss, & Jackson, 1987). Although sentence production in DAT is not error-free, Altmann, Kempler, and Andersen (2001) demonstrated that grammatical errors made by patients with DAT are similar in type and proportion to those made by healthy elderly, suggesting that similar processes are operating in the grammar of both groups.

Sentence comprehension can appear impaired in offline tasks that involve listening to and remembering instructions while selecting one of several choices in a response array or responding to information questions about the material presented (Kempler, Almor, Tyler, Andersen, & MacDonald, 1998; Rochon, Waters, & Caplan, 2000). The fact that patients with DAT do not show effects of syntactic complexity and that their performance correlates with measures of working memory have led authors to conclude that sentence comprehension impairments can be attributed to extralinguistic deficits in executive function (e.g., working memory). If offline sentence comprehension deficits are due to memory impairment, performance on online comprehension tasks, which minimize extralinguistic task demands, should be intact. Indeed, Almor, MacDonald, Kempler, Andersen, and Tyler (2001) demonstrated in an online cross-modal naming paradigm that patients with DAT performed similarly to healthy elderly in processing subject-verb agreement. Small, Andersen, and Kempler (1997) showed that speech rate alteration can modulate sentence comprehension for patients with DAT, suggesting, again that extralinguistic factors can play a significant role in sentence processing for this population. Consistent with these data, Kavé and Levy (2003) demonstrated in both online and offline tasks that participants with DAT, like healthy controls, were sensitive to violations of tense and person. Taken together, these data suggest that grammatical processing may be grossly intact in DAT, at least with respect to relatively simple and robust grammatical phenomena (e.g., subject-verb agreement).

However, the data with regard to grammatical comprehension are not altogether uniform. For instance, Price and Grossman (2005) collected data from patients with DAT using an online word detection paradigm. They found that although patients with DAT were sensitive to violations of verb transitivity, they were not sensitive to violations of thematic role assignment. Therefore, sensitive and specific tasks may reveal islands of impairment within the sphere of grammatical processing in DAT. Overall, the bulk of studies suggest that impairment in sentence comprehension is not the result of grammatical deficits per se, but rather can be attributed to extralinguistic factors, in particular, impairments in attention and working memory.

Extralinguistic processing deficits have also been used to explain discourse impairments in DAT. Patients with DAT are known to have difficulty constructing an informative and coherent narrative. Their narratives are often repetitive with topic changes, unclear references (e.g., “he,” “there”), and lack of coherence and informativeness (Chapman, Highley, & Thompson, 1998; Laine, Laakso, Vuorinen, & Rinne, 1998; Ripich & Terrell, 1988). The association between discourse and working memory in DAT was made explicit by Almor and colleagues. Almor, Kempler, MacDonald, Andersen, and Tyler (1999) demonstrated that the speech of patients

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

with DAT contained more pronouns (compared to full nouns) than the speech of healthy participants. Furthermore, the researchers demonstrated in an online cross-modal naming paradigm that patients with DAT were less sensitive than healthy participants to the appropriateness of pronominal references in short discourses. Sensitivity to pronoun appropriateness positively correlated to working memory scores, providing further evidence for the contribution of extralinguistic deficits to the discourse impairment observed in DAT.

Semantic/Conceptual Deficits Explain the Early Lexical Impairments of Semantic Dementia (SD)

Anomia and word comprehension deficits are the hallmark of SD. Unlike DAT, in which initial anomia occurs in the context of relatively intact semantic/conceptual knowledge, in SD, anomia is generally accompanied by impairments in other semantic/conceptual tasks from the earliest point. This has prompted several scholars to propose an underlying modality-independent semantic/conceptual deficit in SD.

In SD, word retrieval deficits are usually accompanied by word comprehension impairments, although the naming deficit is generally more severe than the comprehension deficit (e.g., Rogers et al., 2006). Additionally, patients with SD present deficits in various tasks that rely on underlying semantic/conceptual representations, including defining words (Warrington, 1975), sorting objects (Hodges & Patterson, 1996), and demonstrating the use of objects (Hodges, Bozeat, Lambon, Patterson, & Spatt, 2000). Rogers et al. recently reported results of verbal and nonverbal semantic/conceptual tasks from 42 patients with SD. The patients with SD, in addition to being predictably impaired on a naming task, were also impaired on comprehension tasks using words (e.g., word-picture matching and the word version of the Palm Trees and Pyramids Test) and a semantic conceptual task with pictures (the picture version of the Palm Trees and Pyramids Test). In this and other studies, the naming deficit in SD appears to emerge early in the course of the disease and correlate significantly with performance on other semantic/conceptual tasks (Grossman & Ash, 2004).

Data suggest that the underlying semantic/conceptual deficit in SD, like DAT, progresses. Early in the course of SD, fine distinctions are first lost between items that share many perceptual and conceptual features (e.g., “apple” and “pear”), leading to naming errors at coordinate level. Distinctions between dissimilar objects remain clearer, preserving the ability to select, for instance, the odd-man out from an array, as in the Palm Trees and Pyramids Test. At this early stage in the dementia, sufficient semantic information remains in the system to allow accurate performance on tasks of attribute and category structure (e.g., “Is pear a fruit?”) and many comprehension tasks. As damage to the system progresses, more general distinctions are lost (animate vs. inanimate, abstract vs. concrete, category knowledge), interfering with an increasing number of semantically based tasks. Verfaellie and Giovanello (2006) recently documented in a 4-year longitudinal study of a patient with SD this pattern of (1) an early and persistent semantic impairment in word generation and (2) a

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

progression of impairment on a task of implicit attribute (concrete/abstract) identification.

Taken as a whole, the data suggest that the lexical deficit in SD seems to progress from one predominated by anomia to one in which an underlying semantic deficit is undeniable (see Grossman & Ash 2004, for review). Unlike DAT, the deterioration of an underlying semantic/conceptual system appears earlier and across a variety semantic/conceptual tasks.

It should be mentioned that there are data that suggest the semantic/conceptual deficit in SD may not be as pervasive or as general as just stated. Several authors have proposed that visual feature information is disproportionately affected in SD. This would explain patients' inability to make judgments with regard to categories that crucially rely on perceptual information (e.g., fruits and vegetables) and their relatively preserved abilities to do so with categories that rely less on perceptual distinctions (e.g., numbers and other abstract concepts) (e.g., Crutch & Warrington, 2006; Halpern et al., 2004; Vesely, Bonner, Reilly, & Grossman, 2007).

Sentence and Discourse Abilities Appear Preserved in SD

In contrast to the progressive deterioration of the semantic/conceptual system underlying lexical processing, syntactic and morphological aspects of connected speech remain essentially unaffected by SD. Most reports of patients with SD describe speech output as fluent and paraphasic, but with little or no mention of grammatical construction problems. It can be argued that SD leads to a dissociation between impaired conceptual knowledge and intact language structure in production and comprehension. For example, a longitudinal case study by Kavé and colleagues (2007; Rochon, Kavé, Cupit, Jokel, & Winocur, 2004) found that, despite clear semantic deficits that affected message content, the patient demonstrated intact morphological and syntactic structure in language performance. Grossman, Rhee, and Moore (2005) studied three patients with SD using an online measure of sentence comprehension (i.e., sensitivity to grammatical violations) and found that patients with SD performed similarly to healthy control participants. Preservation of language structure appears to extend to discourse production (Ash et al., 2006), but there is little relevant discourse data available to date.

Lexical Deficits of Progressive Nonfluent Aphasia (PNFA) Are Attributed to a Phonological Access Impairment in Word Production

Anomia is also a hallmark of PNFA. Anomia in PNFA is apparent in conversational speech, confrontation naming, word reading, and word repetition (Clark, Charuvastra, Miller, Shapira, & Mendez, 2005; Zakzanis, 1999). In contrast to errors found in SD, the speech errors of individuals with PNFA are more likely to be phonological than semantic paraphasias (Clark et al., 1999; George & Mathuranath, 2005). Furthermore, the impairment is typically seen in tasks that involve word production, whereas relatively good performance characterizes tasks that do not

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

require language production (e.g., single-word comprehension, verbal and visual versions of the Palm Trees and Pyramids Test) (Rogers et al., 2006).

In addition to preserved word comprehension abilities, certain nonlanguage cognitive abilities also remain relatively preserved, at least in mild and moderate stages of PNFA. In one recent study, individuals with PNFA were impaired on naming, verbal fluency tasks, and memory for words (word list recall), but not on visual recall and figure drawing (Zahn et al., 2005). Relatively preserved or stable performance has also been documented on a range of nonverbal tasks, including a modified Visual Verbal Test, a visual reproduction test, face recognition, and Raven's Progressive Matrices (Weintraub, Rubin, & Mesulam, 1990; Wicklund, Johnson, & Weintraub, 2004).

These data suggest the lexical deficit in PNFA is confined to lexical production processes and is likely due to impaired access to phonological representations and/or motor programming more than a semantic impairment (Clark et al., 2005; George & Mathuranath, 2005, case 2; Hodges & Patterson, 1996; Rogers et al., 2006; Weintraub et al., 1990).

Morphosyntactic and Extralinguistic Impairments Both Contribute to Sentence Processing Deficits of PNFA

Patients with PNFA exhibit grammatical deficits in both production and comprehension. The nonfluent language production of patients with PNFA is characterized by short sentences with phonological and grammatical errors in spontaneous speech. Difficulties are also present in structured language production tasks, such as sentence completion and picture description (Grossman & Ash, 2004; Grossman et al., 1996; Hodges & Patterson, 1996). Discourse production is impaired, often characterized by limited output and omission of information (Ash et al., 2006).

Sentence comprehension impairments appear in explicit, offline tasks, such as sentence-picture matching with grammatically complex sentences and part V of the Token Test (e.g., Grossman et al., 1996; Thompson, Ballard, Trait, Weintraub, & Mesulam, 1997). Such deficits can be attributed to morphosyntatic impairment or to extralinguistic task-performance factors, including attention and memory. To address the role of attention and memory in sentence comprehension, Grossman and colleagues (2003, 2005) directly compared the relationship between sentence comprehension and extralinguistic cognitive abilities in PNFA. They reported that poor performance on offline sentence comprehension tasks correlated with impairments in auditory short-term memory. These results along with slowed processing in online tasks, have led them to reasonably conclude that slowed processing time allows sentence-level material to degrade in short-term memory during the comprehension process. Thus, the combination of slowed processing and impaired short-term memory results in impaired sentence comprehension.

Recent evidence provided by Grossman and his colleagues, however, suggests that at least some individuals with PNFA experience syntactic impairment

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

per se (Grossman & Ash, 2004; Grossman & Moore, 2005; Peelle, Cooke, Moore, Vesely, & Grossman, 2007). For example, Peelle et al. demonstrated that individuals with PNFA, as compared to healthy older participants, were insensitive to grammatical errors despite their reasonable sensitivity to thematic role violations in a word-monitoring task. In an online monitoring task, extralinguistic factors are not expected to affect performance and therefore cannot account for the impairment observed in individuals with PNFA.

Insights from Cognitive Reserve and Bilingualism

The data reviewed so far show that there is a close relationship between cognitive deficits and language impairments in dementia. If the relationship is causally linked, the association observed in deficits should also show up in patterns of preservation. Therefore, it can be hypothesized that superior cognitive abilities would be associated with relatively preserved (related) language abilities. This is precisely the assumption underlying the concept of "cognitive reserve" and the literature investigating those cognitive factors that might mitigate cognitive and language decline in dementia. In this section we summarize two areas of dementia research relevant to this notion: cognitive reserve and bilingualism.

Individuals with Premorbid High Levels of Education, Literacy, and Mental Activity Show a Delayed Onset of Dementia Symptoms

Cognitive reserve refers to particularly rich neuronal connections in the brains of individuals who have achieved high education levels and engaged in complex mental activity across the life span. It is theorized that cognitive reserve offers protection against the cognitive changes associated with the pathology of dementia (Stern, 2002). That is, despite neural dysfunction, individuals with higher levels of cognitive reserve may not show behavioral symptoms of dementia as early or as significantly as those with less cognitive reserve. The protection afforded by cognitive reserve manifests as lower incidence rates and delayed onset of the dementia symptoms (e.g., Manly, Schupf, Tang, & Stern, 2005; Perneczky, Diehl-Schmid, Pohl, Drzezga, & Kurz, 2007; Valenzuela & Sachdev, 2006). For example, Valenzuela and Sachdev conducted a meta-analysis of 22 longitudinal studies of cognitive reserve and incidence of dementia and found that premorbid higher education, higher IQ, and high levels of leisure and mental activities all were associated with lower (up to 47% lower) incidence of dementia.

Although the protection value of cognitive reserve has been consistently associated with lower incidence of dementia, controversy remains regarding the relation between cognitive reserve and rate of cognitive decline once dementia is recognized (e.g., Scarmeas, Albert, Manly, & Stern, 2006). Higher cognitive reserve has been associated with slower decline in some studies (e.g., Manly et al., 2005; Wilson et al., 2004) but with faster decline in others (e.g., Scarmeas et al.; Stern, Albert, Tang, & Tsai, 1999). Those who found faster decline with higher reserve suggest that the protective effect of cognitive reserve delays the appearance of the dementia symptoms; therefore, by the time the symptoms do appear, the progression

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

of the underlying neuropathology is more advanced and is manifested relatively abruptly.

The variables that compose cognitive reserve include level of education and occupation, social engagement, stimulation in the environment, and levels of intelligence and literacy. Manly and her colleagues (e.g., Manly, Touradji, Tang, & Stern, 2003, 2005) have argued that literacy level is a better measure of reserve than education level, especially for an ethnically varied population. In an epidemiological study of over 3,000 randomly sampled individuals older than 65 years, Manly et al. (2005) found that literacy level (measured as reading levels in English, using subtests of the Wide Range Achievement Test-Version 3) predicted decline in language, memory, and executive function better than did years of formal education. Specifically, low literacy was associated with faster decline in memory and language scores.

A relation between premorbid literacy skills and cognitive decline was reported by Kemper and her colleagues (e.g., Kemper, Greiner, Marquiz, Prenovost, & Mitzner, 2001). In their longitudinal study of a large sample of nuns, Kemper and her colleagues found that higher linguistic skills in early adulthood, as measured by the proportions of complex sentences (in terms of linguistic structure as well as density of ideas) in writing samples, were associated with lower incidence rates of dementia. They did not find that the number years of education of the studied individuals was related to the rate of decline in their linguistic skills over time. Taken together, these data suggest premorbid language or literacy abilities predict the onset of cognitive decline in dementia, highlighting the interrelation of language and other cognitive resources in dementia.

An additional protective variable, proposed by Bialystok, Craik, and Freedman (2007), pertains to premorbid proficiency and use of more than one language. Their study suggests that the special cognitive and language behaviors associated with bilingualism can mitigate the onset of the dementia symptoms. We briefly review data from studies of dementia in bilingual speakers. The first group of data reviewed pertains to decline associated with dementia, whereas the second pertains to preserved abilities.

Deficient Executive and Switching Skills Explain Patterns of Language Impairments in Bilinguals with Dementia

The languages of bilingual and multilingual speakers who are proficient in their languages are assumed to be processed by largely overlapping neural networks (e.g., Abutalebi & Green, 2007). Consequently, dementia-related language impairment would be expected to affect all languages, preserving premorbid levels of relative proficiency. Support for this can be found in several studies of bilingual dementia (e.g., Filley et al., 2006; Hyltenstam & Stroud, 1989; Salvatierra, Rosselli, Acevedo, & Duara, 2007). For example, Filley et al. reported the language difficulties of an English-Chinese bilingual woman with progressive aphasia. Her language deficits included paraphasic errors and impaired repetition abilities equally in both

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

languages. Similarly, Friedland and Miller (1999) reported impairments reflecting levels of pre-onset proficiency in four English-Afrikaans speakers with mild to moderate dementia, diagnosed with DAT.

However, differential impairment of the two languages of bilinguals with dementia was reported in the two cases described in Mendez, Saghafi, and Clark (2004). One case was an English-Spanish-German speaker who experienced difficulty with naming and word comprehension. His deficits were greater in Spanish and German than in English (presumably his L1). The second case was a Spanish-English-Polish trilingual who had experienced progressive difficulty with word retrieval and word comprehension. His performance on language testing was worse in Polish, better in English (L2), and best, although also impaired, in Spanish (L1). The authors concluded that these data suggest differential impairment of the languages of multilingual speakers and differential processing of these languages in the multilingual brain. But, as has been proposed for bilingual aphasia (e.g., Green, 1998), what appears to be differential language impairment could reflect executive dysfunction, which might prevent appropriate language or code switching by impairing the ability to activate and inhibit the languages according to the communication situation.

Furthermore, evidence from bilingual dementia suggests that some (although not all) individuals with dementia tend to switch between their languages inappropriately, that is, even when their interlocutor does not speak or understand one of the languages (De Santi, Obler, Sabo-Abrahamson, & Goldberger, 1990; Friedland & Miller, 1999). This is in contrast to code switching typical of bilingual conversations. In most cases, bilingual speakers with dementia tend to switch to their first language (Friedland & Miller, 1999; Mendez, Perryman, Pontón, & Cummings, 1999). Mendez et al. suggested that switching to the first language in dementia is explained by the fact that earlier learned information is more resistant to decline than later learned information. However, the inappropriate or involuntary switching to one language can also be explained by impaired cognitive function in executive control. According to this account, appropriate and inappropriate language choice in bilinguals is related to preserved or impaired executive skills.

Use of More Than One Language Can Enhance Cognitive Reserve and Delay Onset of Dementia

Bilingual and multilingual individuals, who have years of experience switching between languages and inhibiting or activating their languages in accordance to the communication situation, might have particularly enhanced executive function abilities (Bialystok, Craik, Klein, & Viswanathan, 2004). Support for this is found in a recent article by Bialystok and colleagues who demonstrated that enhanced executive function associated with bilingualism might be part of cognitive reserve discussed earlier. Bialystok et al. (2007) reviewed the records of 184 patients who received a diagnosis of dementia and could be classified as either monolinguals ($n = 91$) or bilinguals ($n = 93$). The bilinguals varied in the age of second language learning and in the languages spoken (25 different first languages; most common

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

languages other than English included Polish, Yiddish, German, Rumanian, and Hungarian). They found that the age of onset of dementia symptoms (on the basis of family report during the initial neurological assessment) of the bilingual speakers was significantly later (4.1 years) than that of the monolinguals. This was true despite the significantly fewer average number of years of education for the bilingual group as compared to the monolingual group. There was no difference between the two groups in rate of decline 4 years after the initial testing. The authors proposed that constant use of two (or more) languages could be taken as a type of complex mental activity and associated with delayed onset of dementia symptoms. The authors interpreted their findings of later onset of dementia symptoms in bilinguals as compared to monolinguals as the result of well-practiced attentional control that accompanies years of using two or more languages, attending to one and inhibiting another appropriately. The research leaves the question of whether and how the lifelong use of two languages provides a unique contribution to cognitive reserve unanswered. Clearly, these ideas are preliminary and need to be replicated.

Conclusion

The data reviewed here suggest that many language impairments seen in dementia are due to extralinguistic rather than linguistic deficits. Problems with memory and attention disrupt word finding in early and moderate in DAT; Decreases in executive function and memory cause sentence-level processing problems seen in all three dementia syndromes. In contrast with these examples of the close connection between linguistic and non-linguistic neuropsychological functions, deficits in the semantic/conceptual system itself are predominantly responsible for the naming and word comprehension impairments in SD and in later-stage DAT.

The close relationship among executive function, language abilities, and bilingualism supports the concept that cognitive reserve can delay the onset of dementia. Although the concept of cognitive reserve and preliminary data are intriguing, they raise many questions. For instance, does cognitive reserve simply refer to higher levels of skill development or to the development of additional and unique cognitive abilities, possibly metacognitive skills? Furthermore, it is unclear how cognitive reserve is acquired and the role of such factors as genetic predispositions, formal education, sociolinguistic environment, and so forth.

Note

1. The term *primary progressive aphasia* is often used to describe two distinct frontotemporal dementia (FTD) syndromes that are characterized by language deficits early in the course of the dementia. This should not be confused with the more typical use of the word *aphasia* to indicate abrupt-onset nonprogressive communication impairments often caused by cerebrovascular accidents. In this review, we refer to these FTD syndromes using the terms *semantic dementia* and *progressive nonfluent aphasia*.

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

ANNOTATED REFERENCES

Almor, A., Kempler, D., MacDonald, M. C., Andersen, E. S., & Tyler, L. K. (1999). Why do Alzheimer's patients have difficulty with pronouns? Working memory, semantics, and reference in comprehension and production in Alzheimer's disease. *Brain and Language, 67*, 202–227.

Although many authors have noted the lack of cohesion of narratives and conversations produced by patients with dementia, this article is unique insofar as the authors linked discourse impairments in spontaneous speech, memory performance, and experimental manipulations of discourse comprehension. Furthermore, this article combines basic research into the source of discourse deficits with practical application by investigating how we might improve discourse comprehension in this population. The authors document that patients with DAT overuse pronouns in spontaneous speech and connect that observation with the fact that patients with DAT are relatively insensitive to incorrect pronouns being used in discourse directed to them. Furthermore, these impairments in discourse production and comprehension correlate with scores on working memory tasks in the expected direction (patients with poor working memory perform less like healthy elderly than those with better working memory). The final experiment tackles a potential intervention strategy by demonstrating that discourse processing of patients with DAT can be improved by use of full noun phrases—rather than pronouns—throughout a discourse. Interestingly, this type of discourse is relatively unnatural and was shown to be disadvantageous for healthy elderly listeners in the same experimental paradigm.

Bialystok, E., Craik, F. I. M., & Freedman, M. (2007). Bilingualism as a protection against the onset of symptoms of dementia. *Neuropsychologia, 45*, 459–464.

Although this study is a retrospective examination of medical records, its findings are compelling and will no doubt inspire further investigation. Bialystok and her colleagues reviewed consecutive records of 228 patients referred to a memory clinic in Toronto, Canada. On the basis of information about place and date of birth, year of immigration to Canada, languages used, and English proficiency, the authors identified 91 monolingual and 93 bilingual patients diagnosed with dementia. When they compared the monolinguals and bilinguals in the sample, they found a 4.1-year difference in the age of onset of the dementia symptoms. The bilinguals demonstrated a later age of onset, and this difference was significant even though the bilinguals had, on average, fewer years of formal education. The notion that active use of two (or more) languages throughout the life span offers protection against cognitive changes associated with dementia is worth further investigation.

Grossman, M., Rhee, J., & Moore, P. (2005). Sentence processing in frontotemporal dementia. *Cortex, 41*, 764–777.

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

Sentence comprehension deficits have been documented across dementia syndromes and can be attributed to either language impairments or executive/attentional dysfunction. Grossman and colleagues addressed this issue by comparing offline and online performance of sentence comprehension and nonlinguistic measures of short-term memory in patients with semantic dementia (SD) and progressive nonfluent aphasia (PNFA) Although similar methods have been used with patients with DAT and aphasia, the authors are among the first to use the direct comparison of offline versus online paradigms to investigate sentence processing in SD and PNFA. Their data on reaction time and test performances are reported for individual patients, illustrating not only the heterogeneity of the syndromes, but also the fact that different underlying neuropsychological deficits can result in superficially similar sentence comprehension impairments: Patients with SD appear to have impaired sentence processing due to task demands of the offline measure but are able to process grammatical structures online when task demands are minimized; patients with PNFA, conversely, have difficulty with both off- and online sentence processing, attributed to slowed grammatical processing.

Rogers, T. T., Ivanoiu, A., Patterson, K., & Hodges, J. R. (2006). Semantic memory in Alzheimer's disease and frontotemporal dementias: A longitudinal study of 236 patients. *Neuropsychology, 20*, 319–335.

Rogers and colleagues' article is notable for the particularly large ($n = 236$) and clearly delineated sample of patients with dementia including the three subtypes reviewed in the body of this article (DAT, SD, PNFA). The large number of participants provides sufficient experimental power to allow comparison of dementia syndromes with a greater degree of authority than many authors. The research is also notable for the systematic approach comparing the participant groups' performances on semantic and nonsemantic tasks matched in task construction (e.g., naming; word vs. picture versions of palm trees and pyramids) and other neuropsychological assessments (e.g., digit span, object/space perception). The results facilitate cross-group comparisons and convincingly document the relative severity of semantic and other abilities in each of the dementia syndromes. Although the results are not necessarily surprising (e.g., patients with SD have a severe semantic impairment; patients with PNFA suffer from a speech production deficit), the impairments are documented and discussed in a clear, convincing, and illustrative manner. To date, this is one of the most clearly written and interpretable studies of factors that contribute to the lexical deficits in dementia syndromes.

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society, 8*, 448–460.

In this critical review, Stern attempted to define and clarify the concept of reserve. The author reviews models of passive (such as the

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

threshold model) and active (such as cognitive reserve and compensation) reserve. He defined cognitive reserve as "the ability to optimize or maximize performance through differential recruitment of brain networks, which perhaps reflect the use of alternate cognitive strategies." (p. 451). Stern discussed evidence for two possible accounts for cognitive reserve, namely, the recruitment of alternate or same networks for the completion of a task. The recruitment of the same brain networks entails increased activation of areas that are involved in the completion of (an easier version of) the task. By contrast, it is possible that cognitive reserve is associated with the ability to recruit alternate brain networks to perform the task. Stern's delineation of measures of impairment (e.g., prevalence of Alzheimer's disease) and of reserve (e.g., education), and the implications of the reserve concept (e.g., whether higher education level modifies brain anatomy) are extremely useful in our understanding of brain damage associated with dementia and its neuropsychological consequences.

OTHER REFERENCES

Abutalebi, J., & Green, D. (2007). Bilingual language production: The neurocognition of language representation and control. *Journal of Neurolinguistics, 20*, 242–275.

Almor, A., MacDonald, M. C., Kempler, D., Andersen, E. S., & Tyler, L. K. (2001). Comprehension of long distance number agreement in probably Alzheimer's disease. *Language and Cognitive Processes, 16*, 35–63.

Altmann, L. J. P., Kempler, D., & Andersen, E. S. (2001). Speech errors in Alzheimer's disease: Reevaluating morphosyntactic preservation. *Journal of Speech, Language, and Hearing Research, 44*, 1069–1082.

Aronoff, J. M., Gonnerman, L. M., Almor, A., Arunachalam, S., Kempler, D., & Andersen, E. S. (2006). Information content versus relational knowledge: Semantic deficits in patients with Alzheimer's disease. *Neuropsychologia, 44*, 21–35.

Ash, S., Moore, P., Antani, S., McCawlet, G., Work, M., & Grossman, M. (2006). Trying to tell a tale: Discourse impairments in progressive aphasia and frontotemporal dementia. *Neurology, 66*, 1405–1413.

Astell, A. J., & Harley, T. A. (1998). Naming problems in dementia: Semantic or lexical? *Aphasiology, 12*, 357–374.

Astell, A. J., & Harley, T. A. (2002). Accessing semantic knowledge in dementia: Evidence from a word definition task. *Brain and Language, 82*, 312–327.

Bialystok, E., Craik, F. I. M., Klein, R., & Viswanathan, M. (2004). Bilingualism, aging, and cognitive control: Evidence from the Simon task. *Psychology and Aging, 19*(2), 290–303.

Bonilla, J. L., & Johnson, M. K. (1995). Semantic space in Alzheimer's disease patients. *Neuropsychology, 9*, 345–353.

Chapman, S. B., Highley, A. P., & Thompson, J. L. (1998). Discourse in fluent aphasia and Alzheimer's disease: Linguistic and pragmatic considerations. *Journal of Neurolinguistics: Special Issue, 11*, 55–78.

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

Clark, D. G., Charuvastra, A., Miller, B. L., Shapira, J. S., & Mendez, M. F. (2005). Fluent versus nonfluent primary progressive aphasia: A comparison of clinical and functional neuroimaging features. *Brain and Language, 94*, 54–60.

Crutch, S. J., & Warrington, E. K. (2006). Partial knowledge of abstract words in patients with cortical degenerative conditions. *Neuropsychology, 20*, 482–489.

De Santi, S., Obler, L. K., Sabo-Abrahamson, H., & Goldberger, J. (1990). Discourse abilities and deficits in bilingual dementia. In Y. Joanette & H. H. Brownell (Eds.), *Discourse ability and brain damage: Theoretical and empirical perspectives*. New York: Springer.

Filley, C. M., Ramsberger, G., Menn, L., Wu, J., Reid, B. Y., & Reid, A. L. (2006). Primary progressive aphasia in a bilingual woman. *Neurocase, 12*, 296–299.

Friedland, D., & Miller, N. (1999). Language mixing in bilingual speakers with Alzheimer's dementia: A conversation analysis approach. *Aphasiology, 13*, 427–444.

George, A., & Mathuranath, P. S. (2005). Primary progressive aphasia: A comparative study of progressive nonfluent aphasia and semantic dementia. *Neurology India, 53*, 162–166.

Giffard, B., Desgranges, B., & Eustache, F. (2005). Semantic memory disorders in Alzheimer's disease: Clues from semantic priming effects. *Current Alzheimer Research, 2*, 425–434.

Gonnerman, L. M., Andersen, E. S., Devlin, J. T., Kempler, D., & Seidenberg, M. S. (1997). Double dissociation of semantic categories in Alzheimer's disease. *Brain and Language, 57*, 254–279.

Green, D. W. (1998). Mental control of the bilingual lexico-semantic system. *Bilingualism: Language and Cognition, 1*, 67–81.

Grossman, M., & Ash, S. (2004) Primary progressive aphasia: A review. *Neurocase, 10*, 3–18.

Grossman, M., D'Esposito, M., Hughes, E., Onishi, K., Biassou, N., White-Devine, T., et al. (1996). Language comprehension profiles in Alzheimer's disease, multi-infarct dementia, and frontotemporal degeneration. *Neurology, 47*, 183–189.

Grossman, M., Koenig, P., DeVita, C., Glosser, G., Moore, P., Gee, J., et al. (2003). Neural basis for verb processing in Alzheimer's disease: An fMRI study. *Neuropsychology, 17*, 658–674.

Grossman, M., & Moore, P. (2005). A longitudinal study of sentence comprehension difficulty in primary progressive aphasia. *Journal of Neurology, Neurosurgery and Psychiatry, 76*, 644–649.

Halpern, C. H., Glosser, G., Clark, R., Gee, J., Moore, P., Dennis, K., et al. (2004). Dissociation of naming nouns versus verbs in primary progressive aphasia. *Neurology, 62*, 1163–1169.

Hodges, J. R., Bozeat, S., Lambon, R. M. A., Patterson, K., & Spatt, J. (2000). The role of conceptual knowledge in object use evidence from semantic dementia. *Brain: A Journal of Neurology, 123*, 1913–1925.

Hodges, J. R., & Patterson, K. (1996). Nonfluent progressive aphasia and semantic dementia: A comparative neuropsychological study. *Journal of International Neurological Society, 2*, 511–524.

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

Hyltenstam, K., & Stroud, C. (1989). Bilingualism in Alzheimer's dementia: Two case studies. In K. Hyltenstam & L. K. Obler (Eds.), *Bilingualism across the lifespan: Aspects of acquisition, maturity, and loss* (pp. 202–206). Cambridge, England: Cambridge University Press.

Kavé, G. (2003). Morphology in picture descriptions provided by persons with Alzheimer's disease. *Journal of Speech, Language, and Hearing Research, 46*, 341–352.

Kavé, G., Leonard, C., Cupit, J., & Rochon, E. (2007). Structurally well-formed narrative production in the face of severe conceptual deterioration: A longitudinal case study of a woman with semantic dementia. *Journal of Neurolinguistics, 20*, 161–177.

Kavé, G., & Levy, Y. (2003). Sensitivity to gender, person, and tense inflection by persons with Alzheimer's disease. *Brain and Language, 87*, 267–277.

Kemper, S., Greiner, L. H., Marquiz, J. G., Prenovost, K., & Mitzner, T. L. (2001). Language decline across the life span: Findings from the nun study. *Psychology and Aging, 16*, 227–239.

Kemper, S., LaBarge, E., Ferraro, F. R., Cheung, H., & Storandt, M. (1993). On the preservation of syntax in Alzheimer's disease. Evidence from written sentences. *Archives of Neurology, 50*, 81–86.

Kempler, D. (1988) Lexical and pantomime abilities in Alzheimer's disease. *Aphasiology, 2*, 147–159.

Kempler, D. (2005). *Neurocognitive disorders in aging*. Thousand Oaks, CA: Sage.

Kempler, D., Almor, A., Tyler, L. K., Andersen, E. S., & MacDonald, M. C. (1998). Sentence comprehension deficits in Alzheimer's disease: A comparison of off-line vs. on-line sentence processing. *Brain and Language, 64*, 297–316.

Kempler, D., Curtiss, S., & Jackson, C. (1987). Syntactic preservation in Alzheimer's disease. *Journal of Speech and Hearing Research, 30*, 343–350.

Kempler, D., Metter, E. J., Riege, W. H., Jackson, C. A., Benson, D. F., & Hanson, W. R. (1990). Slowly progressive aphasia: Three cases with language, memory, CT and PET data. *Journal of Neurology, Neurosurgery and Neuropsychiatry, 53*, 987–993.

Kertesz, A., Davidson, W., McCabe, P., Takagi, K., & Munoz, D. (2003). Primary progressive aphasia: Diagnosis, varieties, evolution. *Journal of the International Neuropsychological Society, 9*, 710–719.

Irigaray, L. (1967). Psycholinguistic approach of the language of the demented. *Neuropsychologia, 5*, 25–52.

Laine, M., Laakso, M., Vuorinen, E., & Rinne, J. (1998). Coherence and informativeness of discourse in two dementia types. *Journal of Neurolinguistics, 11*, 79–87.

Manly, J. J., Schupf, N., Tang, M., & Stern, Y. (2005). Cognitive decline and literacy among ethnically diverse elders. *Journal of Geriatric Psychiatry and Neurology, 18*, 213–217.

Manly, J. J., Touradji, P., Tang, M., & Stern, Y. (2003). Literacy and memory decline among ethnically diverse elders. *Journal of Clinical and Experimental Neuropsychology*, *5*, 680–690.

Mendez, M. F., & Cummings, J. L. (2003). *Dementia: A clinical approach*. Philadelphia: Butterworth-Heinemann.

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

Mendez, M. F., Perryman, K. M., Pontón, M. O., & Cummings, J. L. (1999). Bilingualism and dementia. *Journal of Neuropsychiatry and Clinical Neuroscience, 11*, 411–412.

Mendez, M. F., Saghafi, S., & Clark, D. G. (2004). Semantic dementia in multilingual patients. *Journal of Neuropsychiatry and Clinical Neuroscience, 16*, 381.

Mesulam, M. M. (1982). Slowly progressive aphasia without generalized dementia. *Annals of Neurology, 11*, 592–598.

Ober, B. A. (2002). TR and non-RT methodology for semantic priming research with Alzheimer's disease patients: A critical review. *Journal of Clinical and Experimental Neuropsychology, 24*, 883–912.

Ober, B. A., Jagust, W. J., & Koss, E. (1991). Visuoconstructive performance and regional cerebral glucose metabolism in Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology, 13*, 752–772.

Ober, B. A., & Shenaut, G. K. (1999). Well-organized conceptual domains in Alzheimer's disease. *Journal of the International Neuropsychological Society, 5*, 676–684.

Peelle, J. E., Cooke, A., Moore, P., Vesely, L., & Grossman, M. (2007). Syntactic and thematic components of sentence processing in progressive nonfluent aphasia and nonaphasic frontotemporal dementia. *Journal of Neurolinguistics, 20*(6), 482–494.

Perneczky, R., Diehl-Schmid, J., Pohl, C., Drzezga, A., & Kurz, A. (2007). Non-fluent progressive aphasia: Cerebral metabolic patterns and brain reserve. *Brain Research, 1133*, 178–185.

Price, C., & Grossman, M. (2005). Verb agreements during on-line sentence processing in Alzheimer's disease and frontotemporal dementia. *Brain and Language, 94*, 217–232.

Ripich, D. N., & Terrell, B. Y. (1988). Patterns of discourse cohesion and coherence in Alzheimer's disease. *Journal of Speech and Hearing Disorders, 53*, 8–15.

Rochon, E., Kavé, G., Cupit, J., Jokel, R., & Winocur, G. (2004). Sentence comprehension in semantic dementia: A longitudinal case study. *Cognitive Neuropsychology, 21*, 317–330.

Rochon, E., Waters, G. S., & Caplan, D. (2000). The relationship between measures of working memory and sentence comprehension in patients with Alzheimer's disease. *Journal of Speech, Language, and Hearing Research, 43*, 395–414.

Rogalski, E., & Mesulam, M. (2007). An update on primary progressive aphasia. *Currently Neurology and Neuroscience Reports, 7*, 388–392.

Salvatierra, J., Rosselli, M., Acevedo, A., & Duara, R. (2007). Verbal fluency in bilingual Spanish/English Alzheimer's disease patients. *American Journal of Alzheimer's Disease and Other Dementias, 22*, 190–201.

Scarmeas, N., Albert, S. M., Manly, J. J., & Stern, Y. J. (2006). Education and rates of cognitive decline in incident Alzheimer's disease. *Journal of Neurology, Neurosurgery and Psychiatry, 77*, 308–316.

Shenaut, G. K., & Ober, B. A. (1996). Methodological control of semantic priming in Alzheimer's disease. *Psychology and Aging, 11*, 443–448.

Small, J. A., Andersen, E. S., & Kempler, D. (1997). Effects of working memory capacity on understanding rate-altered speech. *Aging, Neuropsychology, and Cognition, 4*, 126–139.

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press

Stern, Y., Albert, S., Tang, M. X., & Tsai, W. Y. (1999). Rate of memory decline in AD is related to education and occupation: Cognitive reserve? *Neurology, 53*, 1942–1947.

Thompson, C. K., Ballard, K. J., Trait, M. E., Weintraub, S., & Mesulam, M. (1997). Patterns of language decline in non-fluent primary progressive aphasia. *Aphasiology, 11*, 297–331.

Valenzuela, M. J., & Sachdev, P. (2006). Brain reserve and dementia: A systematic review. *Psychological Medicine, 36*, 441–454.

Verfaellie, M., & Giovanello, K. S. (2006). Conceptual priming in semantic dementia: A window into the cognitive and neural basis of conceptual implicit memory. *Cognitive Neuropsychology, 23*, 606–620.

Vesely, L., Bonner, M. F., Reilly, J., & Grossman, M. (2007). Free association in semantic dementia: The importance of being abstract. *Brain and Language, 103*, 154–155.

Warrington, E. K. (1975). The selective impairment of semantic memory. *Quarterly Journal of Experimental Psychology, 27*, 635–657.

Weintraub, S., Rubin, N. P., & Mesulam, M. M. (1990). Primary progressive aphasia. Longitudinal course, neuropsychological profile, and language features. *Archives of Neurology, 47*, 1329–1335.

Wicklund, A. H., Johnson, N., & Weintraub, S. (2004). Preservation of reasoning in primary progressive aphasia: Further differentiation from Alzheimer's disease and the behavioral presentation of frontotemporal dementia. *Journal of Clinical and Experimental Neuropsychology, 26*, 347–355.

Wilson, R. S., Fleischman, D. A., Myers, R. A., Bennet, D. A., Bienias, J. L., Gilley, D. W., et al. (2004). Premorbid proneness to distress and episodic memory impairment in Alzheimer's disease. *Journal of Neurology, Neurosurgery and Psychiatry, 75*, 191–195.

Zahn, R., Buechert, M., Overmans, J., Talazko, J., Specht, K., Ko, C. W., et al. (2005). Mapping of temporal and parietal cortex in progressive nonfluent aphasia and Alzheimer's disease using chemical shift imaging, voxel-based morphometry and positron emission tomography. *Psychiatry Research, 140*, 115–131.

Zakzanis, K. K. (1999). The neuropsychological signature of primary progressive aphasia. *Brain and Language, 70*, 70–85.

https://doi.org/10.1017/S0267190508080045 Published online by Cambridge University Press