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Semantic interference during object naming in agrammatic and logopenic primary progressive aphasia (PPA)

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ABSTRACT

This study examined the time course of object naming in 21 individuals with primary progressive aphasia (PPA) (8 agrammatic (PPA-G); 13 logopenic (PPA-L)) and healthy age-matched speakers ($n = 17$) using a semantic interference paradigm with related and unrelated interfering stimuli presented at stimulus onset asynchronies (SOAs) of −1000, −500, −100 and 0 ms. Results showed semantic interference (SI) (i.e. significantly slower RTs in related compared to unrelated conditions) for all groups at −500, −100 and 0 ms, indicating timely spreading activation to semantic competitors. However, both PPA groups showed a greater magnitude of SI than normal across SOAs. The PPA-L group and six PPA-G participants also evinced SI at −1000 ms, suggesting an abnormal time course of semantic interference resolution, and concomitant left hemisphere cortical atrophy in brain regions associated with semantic processing. These subtle semantic mapping impairments in non-semantic variants of PPA may contribute to the anomia of these patients.

1. Introduction

Primary progressive aphasia (PPA) is a clinical dementia syndrome cause by neurodegenerative disease, which affects the language network and language functions while preserving attention, memory and other aspects of cognitive processing during the initial stages (Mesulam, 2003). One of the most prominent deficits seen in PPA is anomia manifested by word finding and object naming difficulty (Mesulam, 1982). The most severe anomia is seen in the semantic subtype of PPA (PPA-S), where word comprehension is also severely impaired (Adlam et al., 2006; Gorno-Tempini et al., 2004; Mesulam et al., 2009a,b; Neary et al., 1998). However, patients with agrammatic (PPA-G) and logopenic (PPA-L) variants of PPA, who have relatively spared word comprehension, also have naming impairments thought to stem from faulty word retrieval (e.g., phonological and/or articulatory deficits) rather than faulty semantic processes (Mesulam et al., 2009a,b).

According to models of spoken word production, to name an object it is necessary to recognize the object, access the meaning or semantic representation of the object, selectively link the two representations, and activate the phonological form of the associated lexical item. These interrelated processes must unfold rapidly and in a manner that protects them from disruptive interference (Levelt, 1992; Levelt, Roelofs, & Meyer, 1999; Rapp & Goldrick, 2000; Roelofs, 1992; also see Mesulam et al., 2009b). One method for investigating these aspects of naming is the picture–word interference paradigm, an adaptation of the Stroop task (Stroop 1935), which involves presentation of pictures to name coupled with lexical probes or interfering stimuli (IS, hereafter) presented with variable time relationships to the object. Normally, when the IS is semantically (categorically, but not associatively) related to the target picture, a semantic interference effect occurs. That is, picture naming is slowed compared to when the IS is unrelated to the target item. For example, naming a picture of a *fox* is slower in the presence of the IS *goat* compared to *globe*. This slowing occurs because the lexical-semantic network associated with the IS is automatically activated when it is encountered; in turn, the target picture activates its network, which includes the IS. Thus, competition occurs and the need to deactivate the IS is required (La Heij, 1988; Schriefers, Meyer, & Levelt, 1990; Starreveld & La Heij, 1995, 1996). Whether the source of the semantic interference effect is associated with semantic and/or lexical competition is unclear. Some suggest that both the IS and target are automatically lexicalized and, hence, available for production. The phonological form of

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the latter must, therefore, be inhibited in order for the target word to be produced (Bloem and La Heij; 2003; Bloem, van den Boogaard, & La Heij, 2004; Finkbeiner & Caramazza, 2006; Janssen, Schrim, Mahon, & Caramazza, 2008; Mahon, Costa, Peterson, Vargas, & Caramazza, 2007). In either case, competition must be resolved before naming of the target item can be accomplished.

A parameter of importance for this task is the temporal relation between presentation of the probe and the picture to be named, that is, stimulus-onset asynchrony (SOA). At some SOAs semantically related IS hinder naming through the process of competitive interference as described above, whereas, at others the probe could conceivably facilitate naming by bringing the correct word closer to a retrieval threshold. Semantic interference effects are the most common and have been shown in healthy speakers when IS are presented at SOAs ranging from −300 ms to +100 ms (i.e., up to 300 ms prior to picture presentation or 100 ms following it) (Glaser & Düngelhoff, 1984; La Heij, Dirkx, & Kramer, 1990; Lupker, 1979; Rayner & Springer, 1986; Starreveld & La Heij, 1995, 1996; Underwood, 1976).

Semantic interference is a normally occurring phenomenon. However, its magnitude and duration can be influenced by brain damage. In fact, the SI paradigm can be used to test the robustness of information processing routines that underlie object naming. Only a few studies have used the picture–word interference paradigm for this purpose in neurologically impaired patients with naming deficits. In a study by Hashimoto and Thompson (2010), involving 11 individuals with mild aphasia resulting from stroke, aphasic participants showed abnormally heightened semantic interference effects at SOAs of −300 and 0 ms. But at SOA = +300, semantic interference effects disappeared as in normal age-matched speakers. In another study examining semantic interference with one aphasic individual, Wilshire, Keall, Stuart, and O'Donnell (2007) also found abnormal effects, albeit patterns that differed from those seen by Hashimoto and Thompson (2010). That is, the semantic interference effect was absent at SOA = 0 ms, but trends toward semantic interference were found at SOAs of +200 and +400 ms. Interestingly, none of the patients in Hashimoto and Thompson (2010) or in Wilshire et al. (2007) showed strong evidence pointing to semantic deficits in off-line testing.

Abnormal patterns of semantic interference also were seen in a group of individuals with primary progressive aphasia (PPA) studied by Vandenberghe et al. (2005). Like the stroke-induced aphasic speakers studied by Hashimoto and Thompson (2010) and Wilshire et al. (2007), abnormal semantic interference effects were found for the PPA speakers even though no word comprehension deficits were noted on behavioral testing. In contrast to unimpaired participants, who showed semantic facilitation (i.e., faster naming for related compared to unrelated words) at −750 ms SOA, the PPA group showed semantic interference in this condition. This pattern suggested that individuals with PPA successfully activate semantic representations in response to IS, however, deactivation is slowed compared to normal, leaving the competitor network active when the target is to be named. Alternatively, this pattern could suggest a delay in the time course of lexical activation, that is, later than normal rise time. In either case, the subsequent iterative process of linking the target object to its name was abnormally vulnerable to the interference of competing representations.

Priming studies provide some insights into how lexical-semantic activation proceeds in aphasic speakers. Individuals with Broca's aphasia resulting from stroke, typically involving anterior brain tissue, have shown reduced levels of activation and/or slow rise time; whereas, those with Wernicke's aphasia, associated with posterior brain lesions, show greater activation than normal as well as delayed deactivation (Blumstein & Milberg, 2000; Janse, 2006; Prather, Zurif, Love, & Brownell, 1997; Swinney, Zurif, & Nicol, 1989, Yee, Blumstein, & Sedivy, 2008). Both priming and word-interference paradigms operate on the premise that the functional architecture of the lexical processing system involves spreading activation across linguistic units or nodes (Dell, 1986; Masson, 1995), which results in competition among potential candidates. Hence, activation of primes in priming paradigms and that for the IS presented in word-interference paradigms involve similar processes; however, the word-interference paradigm requires an addition step – naming of a related or unrelated competitor – following this activation.

The present experiment was designed to examine the effects of semantic interference on naming in individuals with PPA-G and PPA-L and to examine patterns of cortical atrophy in participants with abnormal semantic interference effects. We enrolled PPA patients and age-matched healthy controls in a word interference paradigm with SOAs of −1000, −500, −100, and 0 ms. Based on similarities between cerebrovascular lesion sites and the location of peak atrophy involving the inferior frontal gyrus, we hypothesized that reduced or delayed activation may be seen for the PPA-G group, akin to the performance of individuals with stroke-induced Broca's aphasia. In the case of reduced activation, semantic interference effects would be either absent or weak across all SOAs, whereas a pattern of delayed activation would result in semantic interference effects only in SOA = −500 and/or −1000 ms conditions. Conversely, we conjectured that PPA-L participants, who often show involvement of posterior brain structures (Gorno-Tempini et al., 2004, 2008; Mesulam et al., 2009b), may show patterns of excessive IS-induced activation, or delayed deactivation, similar to those observed in patients with stroke-induced Wernicke's aphasia. In the former case, the magnitude of semantic interference would exceed that of normal control participants across conditions, whereas in the latter, semantic interference effects would be present in longer SOA conditions, i.e., at −1000 ms. We also entertained the possibility that both patient groups would show normal patterns of semantic interference if the source of their naming deficit stemmed strictly from post-semantic phonological and/or articulatory impairments.

2. Method

2.1. Participants

Participants included 21 individuals with PPA (8 PPA-G and 13 PPA-L) and 17 cognitively intact volunteers. All were recruited through the Cognitive Neurology and Alzheimer's Disease Center (CNADC) at Northwestern University (Chicago, IL) and tested in the Aphasia and Neurolinguistics Research Laboratory at Northwestern (Evanston, IL). The three participant groups were matched for age (PPA-G: $M = 62$ years, sd = 6.13; PPA-L: $M = 64$ years, sd = 7.29; control group: $M = 63$ years; sd = 6.4) ($\chi^2(2, N = 38) = .626$; $p = .731$, Kruskal–Wallis Test) and education (PPA-G: $M = 17$ years, range = 14-20; PPA-L: $M = 16$ years, range = 12–20; controls: $M = 16$ years, range = 11-20) ($\chi^2(2, N = 38) = .353$; $p = .838$, Kruskal–Wallis Test). All were monolingual English-speaking, passed a pure-tone audiometric hearing screening, and were right handed, with the exception of two participants with PPA who were left handed (PPA-G1 and PPA-L8). All also presented a negative history of prior neurological or psychiatric deficits. Compensation for participation in the study was provided and informed consent was obtained prior to participation. The Institutional Review Board at Northwestern University approved this study.

The diagnosis of PPA was based on neurological, neuropsychological and neurolinguistic testing, showing an absence of neurological signs other than progressive language deficits. Symptom onsets ranged from 2 to 10 years prior to testing, however, the two PPA groups were matched for symptom duration (PPA-G: