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Lexical competition effects in aphasia: Deactivation of lexical candidates in spoken word processing

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Abstract

Research has shown that Broca's and Wernicke's aphasic patients show different impairments in auditory lexical processing. The results of an experiment with form-overlapping primes showed an inhibitory effect of form-overlap for control adults and a weak inhibition trend for Broca's aphasic patients, but a facilitatory effect of form-overlap was found for Wernicke's aphasic participants. This suggests that Wernicke's aphasic patients are mainly impaired in suppression of once-activated word candidates and selection of one winning candidate, which may be related to their problems in auditory language comprehension.

Keywords: Inhibition; Aphasia; Auditory word recognition; Lexical processing; Negative priming; Lexical competition; Deactivation; Spoken word processing; Interference

1. Introduction

Aphasic patients have been shown to be impaired in speech perception and auditory comprehension tasks. Although identification of *vowel* contrasts is relatively unimpaired across aphasia types (Gow & Caplan, 1996), problems with identification or discrimination of *consonantal* stimuli are relatively common. Reasonably, researchers have tried to relate perceptual deficits in acoustic–phonetic analysis to auditory speech comprehension deficits. Wernicke's aphasic patients in particular may experience severe speech comprehension problems. However, the link between (low-level) perceptual deficits and (higher-level) speech comprehension is not at all clear-cut: several studies failed to find a strong relationship between perception ability and auditory speech comprehension (Basso, Casati, & Vignolo, 1977; Blumstein, Cooper, Zurif, & Caramazza, 1977; Csépe, Osman-Sági, Molnár, & Gósy, 2001; Miceli, Gainotti, Caltagirone, & Masullo, 1980; Yeni-Komshian & Lafontaine, 1983). Therefore, acoustic–phonetic analysis impairments alone cannot explain the auditory speech comprehension problems encountered by aphasic patients, Wernicke's patients in particular.

Milberg, Blumstein, and Dworetzky (1988) investigated lexical access by presenting aphasic patients with pairs of words (primes and targets). Subjects were asked to perform lexical decision on the second member of the pair. The first words in the pair were systematically changed by one or more phonetic features (the items *cat*, *gat*, and *wat* as primes for the target *dog*). The non-brain-damaged control group showed a monotonically decreasing degree of facilitation as a function of phonological distortion. Participants with fluent aphasia, however, showed mediated semantic priming in all phonological distortion conditions (*gat*/*wat* activating the lexical item *cat*, of which the activation then spreads to *dog*), relative to the unrelated

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baseline. The nonfluent group, on the other hand, showed priming only in the undistorted related condition. On the basis of these results, Milberg et al. (1988) suggested that whereas the nonfluent aphasic participants seem to have reduced lexical activation, compared to an age-matched control group, the fluent aphasic participants have increased lexical activation (or 'overactivation').

Connine, Blasko, and Titone (1993) also performed a mediated semantic priming study (with non-brain-damaged participants only) in which they varied the position of the changed phoneme and the phonological distance between the changed phoneme of a spoken non-word prime and the lexical item it is supposed to activate. As was found by Milberg et al. (1988), the size of the mediated priming effect depended on phonological distance in Connine et al. (1993). Mediated priming effects were found when the initial phoneme mismatch is minimal (e.g., only one phonetic feature). However, if the initial phoneme mismatch involves more than two phonological features (such as place, voicing, and manner features, e.g., the *maximal mismatch* prime ZANNER derived from MANNER), no cross-modal semantic priming was observed in Connine et al. (1993) (for, e.g., STYLE). This latter finding agrees with the results of Marslen-Wilson and Zwitserlood (1989) for initial maximal mismatches: no mediated priming effects was found for Dutch target *bij* ('bee') when rhyme word prime *woning* ('dwelling') or rhyme non-word prime *foning* were used to activate *honing* ('honey').

Gordon and Baum (1994) investigated whether the lack of mediated priming in nonfluent aphasic patients (Milberg et al., 1988) should be attributed to a lack of phonological priming or to the absence of (discernable) spreading of activation to semantic associates. Rhyme facilitation was found for the control group and for the nonfluent patient group. Leonard and Baum (1997) also found rhyme priming effects for a nonfluent patient group (*blood* facilitating lexical decision to *flood*, relative to an unrelated condition). The results of these latter two studies suggest that absence of mediated priming in the nonfluent patient group in the Milberg et al. (1988) study should be attributed to spreading of semantic activation, rather than to a lack of phonological priming. To replicate and extend the results of Gordon and Baum (1994), Baum (1997) investigated the effects of phonological, semantic, and mediated priming in control participants, fluent and nonfluent aphasic patients. Baum (1997) hypothesised that even if the control non-brain-damaged subjects do not exhibit mediated semantic priming, the fluent aphasics might, if lexical access of these patients should indeed be characterised as 'overactivation' (cf. Milberg et al., 1988). Importantly, however, no mediated priming was found in any of the subject groups, which contrasts with the Milberg et al. (1988) results.[1]

Yet, even though a lack of mediated priming for the nonfluent aphasic patients in Milberg et al. (1988) may not be decisive evidence, additional support for their proposal that nonfluent or Broca's aphasic patients suffer from a disturbance in the activation levels of lexical entries was found in later studies. Subphonetic variation has been found to affect lexical activation in that less prototypical stimuli produce a short-lived reduction in semantic priming for normal subjects, compared to more prototypical stimuli. Andruski, Blumstein, and Burton (1994) tapped lexical activation after the presentation of prototypical word forms such as *cat* (via lexical decision time to *dog* as a measure of activation of *cat*) and compared this to the amount of lexical activation elicited by acoustically degraded stimuli such as *c*at* in which voice-onset time is manipulated to be closer to the voiced–voiceless boundary. Even though subjects would still classify the poorer phonetic exemplar sound as [k], the less prototypical word form *c*at* produced less priming than the unmanipulated *cat*, yielding evidence for initial gradedness of activation. Utman, Blumstein, and Sullivan (2001) reasoned that Broca's aphasic patients might be more vulnerable to such subphonetic variations. This was confirmed by their results with Broca's patients, but *only* in conditions of competition: subphonetic degradations resulted in an even greater reduction in lexical activation than found for control subjects when words such as *c*oat* were presented (having the competitor *goat*). When there was no voiced competitor (*c*at*), a reduced priming effect was found for the Broca's aphasic patients, as was found for non-brain-damaged subjects in the Andruski et al. (1994) study. Similar effects of acoustic degradation and lexical competition were found in Misiurski, Blumstein, Rissman, and Berman (2005) who included unimpaired listeners and Broca's aphasic patients. Mediated priming effects had been found with acoustically modified items such as *t*ime* (altered [t] being closer to the voiced phonetic category boundary) for normal listeners: presentation of somewhat intermediate forms not only activate the intended lexical form, but also the competitor. For the non-brain-damaged listeners, a reduced but significant semantic priming effect was found for *penny* (through partial activation of *dime*). However, Broca's aphasic participants did not show any mediated priming for targets preceded by altered primes. These results support the claim that Broca's aphasic patients show a general

[1] Additional evidence that spreading of semantic activation can only be measured reliably in conditions in which the prime word is activated to a relatively high degree can also be found in three doctoral dissertations. They report unsuccessful attempts to investigate activation of multiple word candidates by the presentation of partial primes (cut off before offset: Chwilla, 1996; Janse, 2003; Jongenburger, 1996).