

Grodzinsky's Latest Stand - or, just how specific are "lesion-specific" deficits?

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Abstract

Deficits observed in Broca's aphasia are much more general than Grodzinsky acknowledges. Broca's aphasics have a broad range of problems in lexical and morphological comprehension; furthermore, the classic "agrammatic" syntactic profile is observed over many populations. Finally, Broca's area is implicated in the performance of many linguistic and non-linguistic tasks.

Yosef Grodzinsky (YG) has penned a highly imaginative account of aphasic deficits and their neural correlates, the latest in a series of proposals that he has put forward in the last 15 years for a grammar-specific faculty in the human brain (e.g. Grodzinsky, 1984). His proposals are famous for their strength, clarity and falsifiability. Below we provide evidence that falsifies his latest stand.

First, YG claims that the receptive deficit in Broca's aphasia is restricted primarily (perhaps exclusively) to grammar (e.g. "the patients seem to have no impairment to their lexicon in comprehension, namely, the part of the lexicon that interacts with sentence grammar is intact." Section 2.1). This is misleading. It is well established that Broca's aphasics have marked deficits in both phonological and lexical processing, receptively and expressively (Goodglass, 1993). In fact, some of the first demonstrations of impaired lexical priming in Broca's aphasia were conducted at the same institution where YG conducts his English-language work (e.g. reduced, delayed or deviant word-word priming in Prather, Shapiro, Zurif, & Swinney, 1991; see also Milberg, Blumstein & Dworetzky, 1988).

Second, YG asserts (Section 2.1) that the grammatical comprehension deficit in Broca's aphasia is quite restricted, affecting syntactic movement operations while leaving other aspects of grammar intact (such as computation of agreement and case). This is simply untrue. There is now a large cross-linguistic literature showing that Broca's aphasics (and other groups as well) are markedly impaired in the use of agreement and case information to assign agent-patient roles (Bates, Friederici, & Wulfeck, 1987; Heeschen, 1980; MacWhinney, Osmán-Sági, & Slobin, 1991). Furthermore, although these patients often perform above chance on grammaticality judgment tasks, they are significantly less accurate in detecting subject-verb agreement errors than violations of movement (Devescovi et al., 1997;

Wulfeck, Bates, & Capasso, 1991).

Third, the core of YG's argument revolves around a specific type of syntactic deficit that is supposed to be unique to Broca's aphasia: a deficit in the movement operations associated with (inter alia) the processing of non-standard word order. This is supposed to result in chance performance on passives and object clefts despite above-chance performance on actives and subject clefts. In fact, this very pattern has been observed in all forms of aphasia. For example, Dick, Bates, Wulfeck and Dronkers (1998) compared a large number of anomics, Wernicke's, conduction, and Broca's aphasics and found cases with YG's signature "agrammatic profile" in all aphasic groups, including anomics (i.e. patients with word-finding deficits who do not display clinically significant signs of expressive agrammatism). The presence or absence of this agrammatic profile also failed to correlate with any particular lesion site, and appeared often in patients with lesions sparing Broca's area. We note that the same profile is observed in children who are still acquiring their language, and it can be reproduced in college students who have to perform exactly the same task under "stressful" conditions (e.g. a combination of low-pass filtering and compression of speech). In short, this profile has absolutely no localizing value.

Finally, YG insists that the neural tissue in and around Broca's area is specialized for and dedicated to these syntactic operations, declaring that "the neuro-linguistic localizing schema of language perception may not have permeated the clinical literature, yet it is currently accepted in cognitive neuroscience." In fact, very much the opposite is true. Not only do functional imaging studies show language-related activation in widely distributed and overlapping networks (see Müller, this volume, for further comments), but a steadily increasing number of studies show that regions in and around Broca's area are activated during non-

linguistic tasks, such as object manipulation, mental imagery of tools, and sequential finger tapping cued by a drawn hand (Krams, Rushworth, Deiber, Frackowiak, & Passingham, 1998; Rizzolatti & Arbib, 1998). Such "promiscuity" of activation does not lend much support to a language-specific role for Broca's area.

To summarize: The "core data" of agrammatism that YG uses to define the putative role of Broca's area is observed in a wide range of populations, with different etiologies, including normal adults processing under stress. Patients with damage in and around this region display a range of deficits inside and outside of the grammar. Finally, imaging studies of normals show that Broca's area itself is involved in many different linguistic and nonlinguistic tasks. In short, the pattern of selective deficits and activations that are essential to YG's proposal are not so selective after all.

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