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Susan Edwards and David Lightfoot (2000). Intact Grammars but Intermittent Access. *Behavioral and Brain Sciences*, 23(1), pp 31-32.
doi: <http://dx.doi.org/10.1017/S0140525X00332393>

Collection Permanent Link: <http://hdl.handle.net/10822/707730>

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Broca's area. This assumption reflects a long-standing and common misinterpretation of the role of Broca's area in Broca's aphasia and deserves to be clarified.

It is true that many neurology textbooks state that lesions to Broca's area result in Broca's aphasia, but the fact is that lesions restricted to Broca's area alone never lead to a persistent Broca's aphasia. Even surgical removal of Broca's area leads only to a transient mutism, after which the patient returns to normal. Grodzinsky correctly cites Mohr (1976), who established that persistent Broca's aphasia results only from lesions that extend beyond Broca's area and generally include surrounding frontal cortex, the insula, and underlying white matter. In our work, we have found that chronic Broca's aphasics do indeed tend to have larger lesions. However, we find that only 50 to 60% of our patients with lesions including Broca's area have persistent Broca's aphasia (Dronkers & Jovanovich, forthcoming; Dronkers et al. 1992).

In addition, we have seen several patients with chronic Broca's aphasia (as determined by the Western Aphasia Battery and the Boston Diagnostic Aphasia Exam) whose lesions completely spare Broca's area. In fact, 15% of our right-handed chronic stroke patients with single, left hemisphere lesions and Broca's aphasia do not have lesions in Broca's area at all. If the relationship between Broca's area and Broca's aphasia is so poor, why has the idea of a perfect relationship been promoted for so long?

The persistence of this idea stems from a long history of misinterpretations. First, most people assume that Broca regarded the posterior inferior frontal gyrus as a language area. This is not the case. Broca never stated that this area supported language functions. He concluded that it had something to do with articulation, as his two patients had difficulty in speech production. He was quite convinced that they understood everything that was said to them and that their language was therefore intact. He refused to call the disorder an "aphasia," rather calling it an "aphemia," from the term "phemi," "I speak," "I pronounce" (Broca 1861b). It was Trousseau who later coined the term "aphasia," triggering a letter from Broca who felt the term was not appropriate for this articulation deficit (Broca 1864).

Second, we assume that Broca had numerous patients to support his claim. In fact, his case was based largely on two initial patients whose brains he preserved (Broca 1861a; 1861b). Other cases were presented to him as instances of a speech disorder with involvement of the posterior inferior frontal gyrus. These cases are not all documented and the extent of the lesions is often not known. The damage most likely involved neighboring frontal regions, including the underlying insula and white matter. In fact, Broca's first case is now known to have had a much larger lesion than was apparent on the surface of the brain and also involved these underlying areas, not just Broca's area (Signoret et al. 1984).

A third assumption we make is that no one ever refuted Broca's claim by presenting contradictory cases. Actually, during the century numerous cases were presented with lesions to Broca's area and no Broca's aphasia, or, Broca's aphasia with no lesion to Broca's area (Bramwell 1898; Marie 1906; Mohr 1976; Moutier 1908). These cases, like the ones discussed above, were not uncommon. Because they do not fit the traditional model and no new model has been introduced, they tend to be dismissed as exceptions. Nevertheless, they represent a significant number of cases.

A fourth assumption is that new functional neuroimaging data support the traditional model of language in Broca's area. Actually, Broca's area has been implicated in everything from speech production and episodic memory encoding to gesture recognition and mirror drawing. In truth, Broca's area is probably activated any time a task requires subvocalizing, and this does not support its role exclusively in any particular cognitive function aside from articulation. In addition, neuroimaging studies of language activate many other brain regions besides Broca's area but these are frequently ignored. Studies that focus only on regions of interest such as Broca's area minimize the importance of other integrated brain regions that also contribute to the intricacies of language.

Finally, we often forget that Broca's aphasia is not a single entity with a solitary deficit. It is a syndrome with many individual deficits. Broca's aphasics have difficulty not only with complex grammar, but with naming, articulatory planning and the execution of articulatory movements, repetition, reading, and writing. It would be foolish to assume that all these functions could be located in one brain region. Indeed, at least one of them (articulatory planning) has been shown to involve a discrete area of the precentral gyrus of the insula (Dronkers 1996), not Broca's area at all. Thus we see that localizing Broca's aphasia to one area is too simplistic; rather, several individual brain areas may subserve the different functions affected in patients with Broca's aphasia.

In short, the relationship between Broca's area and Broca's aphasia is not as straightforward as we once thought. Instead, with the contributions of linguistics and psycholinguistics our concept of Broca's aphasia has grown and become more intricate. Our knowledge about the role of Broca's area has been enhanced by technologies that allow us to view the brain areas affected by the injury *in vivo* and then make educated statements about the relationships between brain regions and functional deficits. Behavioral studies that provide structural neuroimages or reconstructions of patients' individual lesions (rather than broad general descriptions) are particularly helpful in drawing conclusions about the neurology of certain behavioral functions. Whether intrasentential computations reside in an area of frontal cortex remains to be seen, but we would be hindering our progress in understanding brain-behavior relationships if we assumed that Broca's area subserved this function merely because it might be affected in patients with Broca's aphasia.

ACKNOWLEDGMENT

This work was supported by the Department of Veterans Affairs Medical Research and the National Institute of Neurological Disorders and Stroke.

Intact grammars but intermittent access

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Abstract: Grodzinsky examines Broca's aphasia in terms of some specific grammatical deficits. However, his grammatical models offer no way to characterize the distinctions he observes. Rather than grammatical deficits, his patients seem to have intact grammars but defective modules of parsing and production.

It is a fact about natural languages that words and phrases may be pronounced in displaced positions. In *What did you buy?*, *what* functions as the direct object of *buy*, but does not occur in the usual direct object position, to the right of the verb. Some generative models have used a movement operation to characterize this phenomenon. Here, Grodzinsky claims to have shown that language is not located in Broca's area. Rather, this area is more specialized than previously thought and deals with two syntactic functions: the movement operation alluded to above insofar as it affects the understanding of language (sect. 2.1) and "the construction of higher parts of the syntactic tree in speech production" (Abstract). There are problems at two levels.

First, Grodzinsky claims (sect. 2.4.1) that the key comprehension deficit of Broca's aphasics relates to the movement of phrasal constituents, NPs and *wh*-phrases, but not of heads. There are difficulties in recovering the movement of phrases, but not the movement of heads: He writes that "agrammatic aphasics are capable of representing traces of [head] movement" (sect. 2.4.1). His distinction is based on a grammaticality judgment test but, because

strategies independent of traces may be involved (for example of the type he sketches in sect. 2.6), there could be other explanations. However, his claim taken at face value raises a problem: There is no straightforward way of isolating movement of phrases from the movement of heads in the 1980s-style Government-Binding (GB) model he uses. Such models postulate an operation Move Alpha, by which both phrases and heads move, leave traces, and are subject to the same locality restrictions.

Grodzinsky's data do not fit neatly with his GB model, which does not cut the empirical pie in the way he needs. The problem is compounded under more recent Minimalist approaches. Not only is there no ready distinction to be found here between the movement of heads and phrases – there is no movement operation at all. Minimalist analyses dispense with a distinct level of D-structure and therefore with D-structure-to-S-structure mappings. They also dispense with familiar top-down phrase-structure rules. Instead, phrase structure is built bottom-up through the successive merger of elements from an “array” of lexical items. For a sentence like *I will visit London*, *visit* is merged with the NP *London* to yield a VP; then the inflectional element *will* is merged with that VP to yield an IP, and so on. One type of merger involves copying an element. For the expression *What did you buy?*, *what* is copied and merged with [*did you buy what*], yielding [*what [did you buy what]*], with subsequent deletion of the lower *what* (we omit structural labels). This is how “movement” phenomena are handled but Minimalist approaches involve no movement as such. There is no unitary operation corresponding to GB movement. Elements are merged successively, sometimes copied and sometimes deleted; it is hard to imagine what particular function would be compromised in the people Grodzinsky describes. He notes that functions that build phrase structure are not compromised in his subjects, but in Minimalist syntax there is little beyond structure-building functions.

Now to the second problem. If GB and Minimalist syntacticians do not have the means to capture naturally the descriptive generalizations that Grodzinsky reports, how should they revise their claims about grammars? They will surely want to know on what basis the descriptive generalizations are made. In his discussion of grammaticality judgments involving movement of phrasal constituents (sect. 2.4.1), he tells us that he “tested four nonfluent, agrammatic Broca's aphasics . . . with lesions in and around Broca's area, including white matter deep to it, ranging from the operculum, to the anterior limb of the internal capsule, to the periventricular white matter.” So, even for just these four patients, the anatomical damage covers more than Broca's area and includes subcortical tissue. His patients show comprehension deficits relating to the movement of phrases and production deficits relating to the higher elements of tree structures. He does not argue that there are production deficits relating to movement, or comprehension deficits relating to the topmost parts of phrase structure. So movement needs to be there to account for production capabilities and the full tree needs to be available to characterize comprehension functions. If a grammar is a representation of an individual's linguistic knowledge, which can be used for various purposes, then Grodzinsky's results actually suggest that there is no damage to the grammar, but rather two defects in the way it is used for the purposes of parsing and production, presumably two distinct modules.

Grodzinsky's claims are based on proportions and tendencies, often just “chance” versus “above chance.” For constructions involving movement of phrasal constituents, “error rates were about 40%.” (that means 60% correct), as opposed to 10% in comparable constructions not involving that kind of movement (90% correct; sect. 2.4.1). Similarly, the data on agreement and tense errors (sect. 2.7.3) show a difference in frequency (3.9% versus 42.4%); the difference is not absolute, and if tense is available 57.6% of the time, despite the “pruning,” it is available more often than not. There is a good deal of variation being glossed over, both linguistic and anatomical, which shows that he has not yet met his goal of “a new, highly abstract and precise approach” (sect. 0). In ad-

dition, there is much normal behavior: If subjects achieve 60% correct on relevant tasks, there is no basis for saying that they lack the relevant parts of the grammar (see Crain & Thornton 1998 for an enlightening discussion of such statistics). Grodzinsky presents two important and interesting features of agrammatism, but there are other salient features that his proposals do not address. If “most human linguistic abilities, including most syntax, are not localized in the anterior language areas” (sect. 4), why do patients who have sustained lesions in these areas have problems with verb retrieval and why is their speech typically “effortful, nonfluent, and telegraphic” (sect. 2.7.1)? Broca's aphasics generally have slow reaction times (Shapiro & Levine 1990) and it is clear from agrammatism samples in any introductory aphasia text that there is far more going on than what Grodzinsky describes. Therefore, there must be more ways to cut the empirical pie.

Grodzinsky has certainly made a healthy innovation in examining aphasias in terms of grammatical deficits, integrating work on pathologies with grammatical theory. However, what the variability suggests – both within and across individuals, both anatomically and in linguistic behavior – is not a precise grammatical deficit. His grammatical models offer no obvious way to characterize his empirical distinctions. Furthermore, if individuals often behave in accordance with a normal, intact grammar, then they must have an intact grammar, but with somewhat intermittent access to it. Perhaps the modules that use the grammar (parsing and production) are defective in some fashion. In saying this, we recognize that we assume a burden of argument that goes beyond the scope of a *BBS* commentary.

Syntax in the brain: Linguistic versus neuroanatomical specificity

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Abstract: We criticize the lack of neuroanatomical precision in the Grodzinsky target article. We propose a more precise neuroanatomical characterization of syntactic processing and suggest that syntactic procedures are supported by the left frontal operculum in addition to the anterior part of the superior temporal gyrus, which appears to be associated with syntactic knowledge representation.

The title of Grodzinsky's target article, “The neurology of syntax: Language use without Broca's area,” suggests that it provides detailed information about the functional neuroanatomy of syntactic abilities. The description he presents, however, is detailed only with respect to the psycholinguistic aspects of syntactic processing he proposes; it clearly lacks a similar precision with respect to neuroanatomy.

This is rooted in the fact that Grodzinsky's main empirical evidence for the claim that Broca's area is the “neural home to mechanisms involved in the computation of transformational relations between moved phrasal constituents and their extraction sites” (sect. 0) stems from lesion-based studies. Nature (in almost all cases) fails to offer lesions circumscribed enough to allow a precise description of the language-brain relationship. Thus the claim Grodzinsky formulates with respect to the Broca's area – as he acknowledges – can only hold for Broca's area as a large area (including surrounding left anterior neural tissue). He defines this area according to Mohr (1976) to “encompass most of the operculum, insula, and subjacent white matter” (sect. 0). These subregions in the left frontolateral region, however, subserve a number of different linguistic and nonlinguistic functions.

The functional neuroimaging evidence Grodzinsky cites is restricted to those studies investigating syntactic aspects (Bavelier et al. 1997; Just et al. 1996; Mazoyer et al. 1993; Stromswold et al.