

Grodzinsky believes that a problem with traces cannot derive from a processing disorder? In fact, both the difficulty with noncanonicity and the one with embedding can in principle be accounted for by a processing model that incorporates trace formation as a processing component (c.f. Haarmann et al. 1997). It is even possible that trace formation does not constitute an independent source of processing load and that the canonicity effect arises as the consequence of word-order strategies (c.f. Kolk & Hartsuiker 1999; Kolk & Weijts 1996). If this sounds too farfetched, it is important to realize that for Grodzinsky as well the difference between canonical and noncanonical sentences results from strategy use and not from the presence or absence of traces.

3. Just tree truncation? No! For agrammatic production, Grodzinsky puts forward the hypothesis that the agrammatic syntactic trees are truncated at the level of a particular node, typically the tense node. As support for this hypothesis, Grodzinsky points out that in Dutch agrammatic speech, verbs in main clauses not only appear uninflected, but are also in sentence-final position. He says this has to do with the fact that Dutch is an SOV-language, meaning that the base-generated position of the verb is after the object. To produce an inflected verb in a main clause (which has the SVO-order), the verb has to move and this movement is prohibited by the truncation. The result is an absence of inflection and no change in verb position. As supporting evidence, Grodzinsky refers to the study of Kolk and Heeschen (1992), which would show the predicted pattern. And indeed, the right side of Table 5 in Kolk and Heeschen (1992) does show the required pattern: uninflected verbs in final position. The table also has a left side, however. This left side demonstrates that these same patients produced an even larger set of utterances with inflected verbs in SVO position. Similar findings are reported in de Roo (1999). This means that although the patients make excessive use of root infinitives, they are – on the whole – by no means unable to produce inflected verbs in medial position (contrary to the claim made by Bastiaanse & van Zonneveld 1998). This is also demonstrated in the study by Hofstede and Kolk (1994), who showed that finiteness omission strongly decreased in a picture description task as compared to spontaneous speech. Parenthetically, Grodzinsky dismisses utterances with clause-final uninflected verbs as “ungrammatical.” Yet such utterances are certainly part of the elliptical register in normal speech (Kolk & Heeschen 1992).

There is another recent finding that seems hard to reconcile with the truncation hypothesis. This finding concerns the production of the passive, which is notoriously hard for aphasics. The truncation hypothesis would explain this difficulty as a consequence of an inability to perform movements to a position in the tree higher than the affected node. This account predicts that agrammatics cannot be induced to produce passives. However, Hartsuiker and Kolk (1998) elicited passives from agrammatic patients in a sentence priming task. They showed that repetition of an unrelated passive consistently led the patients to produce considerable numbers of passive picture descriptions, which they did not in spontaneous speech.

The brain does not serve linguistic theory so easily

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Abstract: It is a major move from the claim that the core linguistic problem in Broca’s aphasia is the inability to deal with traces, to the claim that this is the syntactic operation only and that it is exclusively supported by Broca’s region. Three arguments plead against this move. First, many Broca patients have no damage to Broca’s area. Second, it is not only passive, but also active jabberwocky sentences that activate the frontal operculum in a judgment task. Third, the same area is involved in a phrase-building production task that does not require tense processing.

Most of the evidence Grodzinsky marshals in support of his Trace Deletion Hypothesis (TDH) is aphasiological, the more or less carefully tested linguistic performance of Broca’s patients. For the sake of argument I will accept both the evidence and Grodzinsky’s linguistic analysis thereof. It is, however, a major additional step to attribute the core linguistic problem in Broca’s aphasia, the inability to deal with traces, to damage in Broca’s area and/or its immediate vicinity. Damage to that region is neither a necessary nor a sufficient condition for the syndrome of Broca’s aphasia to arise (Willmes & Poeck 1993). TDH predicts that Broca’s patients without damage to that region should be perfectly all right as far as dealing with traces is concerned. And damage to just that region, even if it does not result in Broca’s aphasia, should be sufficient to create the processing problems predicted by TDH. Is that really intended? If so, does the author have any evidence to support these strong claims?

Probably, only functional neuroimaging can provide the critical test, but the available studies are too few and not specific enough to test TDH. The only conclusion they allow Grodzinsky to draw is that the evidence does not speak against the hypothesis. Two comments are in place here, both concerning recent neuroimaging studies. First, there is the follow-up study to Stromswold et al. (1996) by Caplan et al. (1998). In this PET subtraction study, subjects read sentences that contained a center-embedded or right-branching relative clause and judged them for their plausibility. As in the Stromswold et al. study, the center-embedded condition triggered more activation in the pars opercularis of Broca’s area than the easier right-branching condition did. Clearly, because these sentence types differ in the distance between phrase and extraction site, the additional activation of Broca’s area in handling center-embedding structures is supportive for TDH. It should be added, though, that there are also straightforward phrase structural differences between the two types of sentence. If the specific expertise of Broca’s area is phrase structural (contrary to TDH), then these differential activations were still to be expected.

Second, two even more recent studies do test a critical aspect of TDH, namely, that Broca’s region does *not* support any other syntactic operations than the specific ones formulated in TDH (“only these,” target article, sect. 4). These two tests violate TDH. One study, an event-related fMRI experiment by Meyer et al. (1999) compared (among other things) a syntactic jabberwocky condition (in German) to a rest condition. Subjects judged the test sentences on “being syntactic” and on “containing pseudowords.” Broca’s region, in particular, the left frontal operculum (i.e., the small region directly caudal to Broca’s area), was activated by both active SVO sentences and by their passive equivalents. There was no statistical difference between the two conditions. The activation by passive jabberwocky is predicted by TDH, the activation by active SVO jabberwocky violates the “only” clause of TDH.

The other study, by Indefrey et al. (1999b), is a PET study of syntactic production. Here, subjects saw Michotte-type launching events. In condition 1 they provided full-sentence responses (such as the German equivalent of *the red square launches the blue ellipse*). In condition 3 they provided word list responses (such as *square red ellipse blue launch*). A single, highly specific difference

activation was obtained for the full syntactic response – in the frontal operculum. The major syntactic difference between the two response types is phrase-structural, not transformational. Still, Grodzinsky could argue that in a production task TDH predicts involvement of Broca's region in tense processing, moving the German clause-final verb to its tensed second position. But here condition 2 of the experiment gets in the way. The subjects' task was restricted to NP building, producing responses of the type *red square*, *blue ellipse*, *launch* (notice that in German, this involves establishing gender agreement between noun and adjective). This condition also evoked significantly stronger activation of the frontal operculum (and of no other area) than the word list condition did (though less so than the full sentence condition). I see no way of reconciling these results with TDH.

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Agrammatism, syntactic theory, and the lexicon: Broca's area and the development of linguistic ability in the human brain

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Abstract: Grodzinsky's Tree-Pruning Hypothesis can be extended to explain agrammatic comprehension disorders. Although agrammatism is evidence for syntactic modularity, there is no evidence for its anatomical modularity or for its localization in the frontal lobe. Agrammatism results from diffuse left hemisphere damage – allowing the emergence of the limited right hemisphere linguistic competence – rather than from damage to an anatomic module in the left hemisphere.

Using the Principles and Parameters framework, Grodzinsky tries to show that agrammatism is the result of damage either to a primary linguistic processor devoted to syntactic transformational operations or to a nonlinguistic slave system critical for running specific syntactic routines (e.g., phonological short-term memory). He further argues that Broca's area (or a topographically extended "grand Broca" anterior perisylvian area) would be the crucial site for this functional processor. On the one hand we would like to push further the implication of his Tree-Pruning Hypothesis; on the other, we wish to discuss his historical premises, and to reconsider his sharp assumptions in favor of a left frontal localization of the syntactic processor.

1. Linguistic perspectives on agrammatism. Grodzinsky's Trace Deletion Hypothesis (TDH) explains the failure of agrammatic patients to comprehend constructions involving transformations such as reversible passives and SO reversible relative clauses. However, it falls short of explaining the nonmandatory character of this failure (Berndt et al. 1996; Luzzatti et al. 1999; Miceli et al. 1983). Agrammatism is a condition in which production is also severely affected. In spontaneous speech and in reading (phonological dyslexia), agrammatic patients often omit unbound functional morphemes (e.g., articles, prepositions) or substitute inflectional affixes so as to produce less marked lexical forms. Although Grodzinsky does not focus on this, he tackles the production weaknesses from a particular perspective that has proven very fruitful in accounting for language acquisition (Wexler 1994) and specific language impairment (SLI) in childhood (see Clahsen et al. 1997; Rice & Wexler 1996).

Basing his position on linguistic theory Grodzinsky claims that agrammatic patients are selectively impaired in the use of tense but not of agreement morphemes. This selective impairment has a structural implementation that is expressed by the tree-pruning hypothesis, according to which the tense node that dominates the

Agreement node is pruned (see his Fig. in Ex. 28). Consequently, every projection above tense phrase, TP (for example, complementizer phrase, CP) is deemed to be absent from an agrammatic grammar. Whether or not the tree-pruning hypothesis is correct and generalizable to other aspects of the agrammatic disorder (see above), it paves the way for a whole range of interesting questions and falsifiable predictions.

One can attempt a unification of the comprehension and production disorders by exploiting the consequences of the tree-pruning hypothesis. Assuming the correctness of Grodzinsky's assumptions about the clausal architecture (but see Belletti 1990; Guasti & Rizzi 1999), the tree-pruning hypothesis offers an immediate explanation for the difficulties that agrammatic patients have in comprehending relative clauses. The syntactic representation of agrammatic patients cannot include the tense node or any higher one; specifically, it cannot include the CP, a node that is required to accommodate relative clauses. If agrammatics cannot assign the appropriate structure to relative clauses, they can hardly interpret them correctly, regardless of their ability to handle traces. An explanation along similar lines can be devised for passives. The subject of a sentence must move to the specifier of TP (see the tree in Ex. 28 in Grodzinsky's target article). Again, if TP cannot be projected, subjects cannot be moved there. Whatever representation agrammatic patients assign to a passive sentence, it is not the correct representation, and this suffices to explain their failure to interpret reversible passives. This perspective raises different theoretical and empirical questions, but it is a natural development of Grodzinsky's approach.

2. Historical background and coda. Paul Broca was neither a connectionist nor a diagram maker but a surgeon with rough psychological knowledge who had the opportunity to make the post-mortem observation of Monsieur Tan-Tan's cerebral lesion and tried to support Gall's and Bouillaud's functional localization of the speech faculty in the frontal lobes. He also was the first in demonstrating the left-right functional asymmetry of language processing in the brain.

A decade later, Wernicke drew his famous diagram in which he introduced the dichotomy between auditory and motor images of words. Since Wernicke, and during the next 30 years, Broca's area was for the German scholars the site of what we now call the phonological output lexicon, and for most French scholars (e.g., Lecours & Lhermitte 1976; Marie 1906) it was a center for articulatory motor control. This characterization of Broca's area as a center for the motor control of articulation is extremely reasonable, because Broca's area is right in the middle of the associative cortex for the bucco-pharyngeal and laryngeal praxic motor control. The association between morpho-syntactic disorders and frontal lesions was first made at the beginning of this century after Bonhoeffer's (1902) description of agrammatism.

3. Agrammatism and syntactic modularity. Grodzinsky claims, and we fully agree, that agrammatism is evidence for a modular organization of language in the left hemisphere. We doubt, however, that agrammatism provides clear support for functionally localizing a single linguistic processor in the left frontal lobe that is devoted to specific syntactic computations. A century of anatomy-function correlations in aphasia and more recent brain imaging (PET, fNMR) studies have provided scant evidence for the localization of single aphasic features or of clusters of symptoms (e.g. Caplan et al. 1996; Vanier & Caplan 1990).

Agrammatism seems to reflect extensive damage to the left hemisphere (LH) linguistic representations (functionally - but not necessarily anatomically - modular), which causes the emergence of less developed right hemisphere (RH) linguistic abilities. These are evident (1) at the lexical level in word class (nouns > verbs > function words), word frequency and imageability effects, (2) at the morphological level in a limited ability to process bound morphemes, (3) at the syntactic level (TDH, etc.), and (4) at the level of short-term memory in reduced short-term phonological capacity.

In this perspective the variability observed among aphasic (and agrammatic) patients is not the result of the isolated involvement