

## NOTES AND DISCUSSION

### When is Enough, Enough? A Comment on Grodzinsky and Marek's "Algorithmic and Heuristic Processes Revisited"

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In this comment I argue that although Y. Grodzinsky & A. Marek's (1983, *Brain and Language*, 33, 216-225) criticism of the conclusions reached by A. Caramazza and E. B. Zurif (1976, *Brain and Language*, 3, 572-582) in their paper on comprehension disorders in so-called agrammatic patients is not entirely without technical merit, its impact on the claims made by Caramazza and Zurif is inconsequential. I show that there are deeper theoretical and methodological reasons which undermine the claims made by Caramazza and Zurif and the only superficially different proposal of Grodzinsky and Marek. © 1988 Academic Press, Inc.

In a critical note Grodzinsky and Marek (1987; henceforth G&M) argue that there is no evidence in support of the view proposed in Caramazza and Zurif (1976; henceforth C&Z) that "... at least for the Broca's aphasics, brain damage affects a general language processing mechanism that subsumes the syntactic component of both comprehension and production" (p. 581; quoted in G&M). I agree with *this* conclusion reached by G&M—a conclusion I have defended in several publications (Caramazza, 1984; 1986; Badecker & Caramazza, 1985) on the basis of arguments different from those presented by G&M. Given that G&M have reached a conclusion that is apparently similar to one I had already reached it would appear that I should welcome their critical note of C&Z and related studies. However, the apparent similarity between our respective conclusions is highly misleading: G&M use their critique of C&Z as a tram-

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poline for proposing what they mistakenly consider to be a significant variant of the thesis originally defended by C&Z; I, for my part, have argued that the original claim by C&Z is not even wrong, it is simply vacuous as are similar claims including the one proposed by G&M in their note. In this comment I briefly elaborate on this latter claim after having first analyzed the arguments presented by G&M.

There are two parts to G&M's argument: a "theoretical" and a technical part. The technical argument has some merit; the theoretical one is clearly inadequate. Briefly, the technical argument shows that C&Z's experimental design was incomplete for the intended purpose. These latter authors wanted to show that a clinically classified group of Broca's aphasics were impaired in syntactic processing in a comprehension task. They tested their patients in a sentence/picture verification task in which they manipulated sentence types and picture contrasts (see summary in G&M). C&Z wanted to show that their patients performed well when the relevant contrasts for "good" performance involved lexical/semantic information and poorly when the contrast required "normal" processing of syntactic structure (reversible sentences). The result they obtained "confirmed" their expectation. However, C&Z also advanced a stronger hypothesis. They argued that patients who, because of a syntactic processing impairment, perform poorly in a sentence/picture matching task with reversible sentences, should perform well with nonreversible sentences. The basis for this prediction was that the hypothesized impairment only involved syntactic processes and, therefore, patients could use their intact lexical/semantic ability to "construct" a semantic interpretation for the sentence as a whole. I will not enter into the merits of this reasoning, which I now find to be of dubious clarity and explicitness, since it is not relevant to the issue under consideration in this discussion. What is at issue is whether or not, within the framework that animated the hypothesis considered by C&Z, the experimental design used by these authors was adequate for the intended purposes.

G&M claim that the experimental design used by C&Z, as well as those used by Schwartz, Saffran, and Marin (1980), Heeschen (1980), Deloche and Seron (1981), Ansell and Flowers (1982), Linebarger, Schwartz, and Saffran (1983), Kudo (1984), Wulfeck (1984), Grodzinsky (1984), and Caplan and Futter (1986) were not adequate. In the specific case of the experimental design used by C&Z, the problem is that patients could have picked the correct picture not because that picture correctly represented the semantic representation the patient had constructed for the stimulus sentence but because the alternative picture choice was bizarre. Recall that the experiment involved having subjects pick one of two pictures in response to an aurally presented sentence. The sentences in question were center-embedded constructions that varied in terms of whether the two nouns could equally assume the thematic role of agent.

Three types of center—embedded sentences were used: reversible (e.g., The cat that the dog is biting is black), improbable (e.g., The dog that the man is biting is black), and nonreversible sentences (e.g., The apple that the boy is eating is red). One of the experimental conditions required the patient to pick the correct choice from two pictures which differed in terms of the thematic roles of the participants in the action depicted in the pictures—one correctly depicted the stimulus sentence and the other depicted a situation in which the thematic roles of the two nouns in the stimulus sentence were inverted (e.g., for the sentence “The man that the horse is riding is fat,” one picture depicted a horse riding a fat man and the other depicted a man riding a fat horse). G&M argue that the Broca’s aphasics’ good performance with the nonreversible sentences could reflect no more than the patient’s bias for choosing nonbizarre pictures. They propose that in order to have tested for this possibility C&Z should have included an experimental condition in which the bizarre picture was the correct choice (i.e., presented sentences such as “The boy that the apple is eating is red”). However, the proposed remedy does not work. If patients *only* responded correctly because they systematically avoided picking the bizarre picture, then the inclusion of another sentence type in the experimental design could not have affected performance—patients would have picked the nonbizarre picture independently of the sentences presented. If we want to test issues concerning the role of reversibility in the comprehension processes and if G&M’s observation about the basis for patient’s performance in choosing bizarre pictures is correct, then we must adopt an experimental paradigm different from the one used by C&Z.

It must be emphasized that, even though the criticism raised by G&M concerning the limitations of the experimental design used by C&Z is technically correct, *this* limitation does not undermine the principal claim made by C&Z. Recall that these latter authors wanted to show that, contrary to the accepted view at that time, the patients they had classified as Broca’s aphasics were impaired in the comprehension of reversible sentences; that is, that Broca’s aphasics are impaired in syntactic processing in comprehension tasks. And, in this latter respect, the data they obtained confirmed their hypothesis. The crucial flaw in the conclusions reached by C&Z in their article does not concern problems with the experimental design they used, but involves instead deeper methodological and theoretical limitations. I will return to this point below.

In their critical note G&M not only present a technical critique of the research reported by C&Z but also raise more general methodological/substantive issues. One such issue concerns the proper interpretation of cognitive deficits consequent to brain damage. They raise the issue of whether we should consider cognitive deficits as reflecting either the total or partial disruption of a processing component. Obviously, this

issue cannot be meaningfully addressed in the abstract: the extent of damage will differ from patient to patient and, therefore, we cannot have a general position on this issue. But, without providing any motivation G&M attribute to C&Z the position of assuming the total disruption of a cognitive mechanism. This attribution is unjustified. C&Z asserted only that they considered their results as showing that their patients had suffered damage to the syntactic component of the language processing system; no claim was made concerning the extent of the hypothesized functional damage. It is unclear to me how such a misattribution was possible from a careful reading of C&Z. I can only assume that the misattribution served a purpose—that of motivating G&M's discussion of their hypothesis of the basis for "agrammatism." Specifically, these authors wished to promote the view that "the correct characterization of the syntactic abilities in agrammatic Broca's aphasia maintains only a partial impairment (sic). As a consequence, these patients have access only to incomplete syntactic representations..." (Grodzinsky & Marek, this issue). But, G&M could have presented their view without grossly misrepresenting C&Z. Their need to invent a strawman may reflect the fact that, to my knowledge, the distinction between partial and total disruption of a cognitive component has not played a significant role in any account of "agrammatism," no matter how misguided. G&M do not help their cause by raising the nonissue of partial vs. total disruption of syntactic processes by misattribution.

Another misattribution, masquerading as an issue, offered by G&M concerns the issue of "task analysis." These authors state that "Only an analysis of the response type and the solution space, coupled with the syntactic construction in question, constitutes the actual 'task analysis.' This, we believe, is the only way to assign a coherent interpretation to data from the comprehension of brain damaged people. This, of course, is against common wisdom in neuropsychology, where tasks are always equated with *activities* such as reading, writing, repetition, etc., and syntactic form and response options are lumped together. We are thus putting ourselves in opposition to many interpretations of aphasic performance (see, for example, Caramazza & Berndt, 1985; Caramazza, Berndt, Basili, & Koller, 1981, and many, many others)." (Grodzinsky & Marek, 1987).

Although from the text it is not entirely clear what windmill the authors are tilting at, we may make an educated guess. They seem to be suggesting that the position that my colleagues and I have taken (as well as the unspecified many, many others) with respect to the analysis of cognitive disorders is that we mechanically equate poor performance on a specific task with total disruption of a cognitive faculty. Surely this is a preposterous misattribution! What in the papers cited could have led G&M to such a strange conclusion? Clearly not a careful reading of those papers nor of

the recent literature in cognitive neuropsychology. And, yet, I am not willing to believe that these authors are simply unfamiliar with the recent literature in cognitive neuropsychology. There must be something in this literature that has led them to their unjustified misattribution. All I can think of is that the recent emphasis on the need to develop detailed models of such cognitive functions as sentence comprehension, oral reading, naming, etc., in order to interpret a pattern of impaired performance, has led G&M to confuse "cognitive model for the performance of a task" with "task."

If there is a major gain in recent neuropsychological research it is the effort to provide increasingly explicit cognitive models to guide our research. Since cognitive systems are intrinsically extremely complex, it is not easy to infer the correct relationship between impaired performance and functional lesion(s) to the cognitive system that supports a task. The appreciation of this complexity has led neuropsychologists to strive for increasingly better articulated models of the cognitive systems that support performance in various tasks, and for correspondingly more detailed analyses of cognitive performance. A corollary of this development has been the greater emphasis that has been placed on what we may loosely call a "systems" approach in neuropsychological research. That is, the focused investigation of a patient's performance that takes in consideration all the relevant cognitive components that may plausibly be assumed to underlie a patient's poor performance in any task of interest. Thus, for example, if we were to be interested in the development of a model of sentence comprehension through the analysis of comprehension disorders, the investigation of the comprehension disorder in any given patient must consider all theoretically plausible contributions to the observed disorder. The obvious implication of this stance is that the comprehension task of interest (the one which gave rise to the impaired performance one is trying to use for the purpose of constraining a model of normal cognitive processing) must be analyzed in sufficient detail to allow the correct localization of a functional lesion to the comprehension process. The position I have outlined here seems to me to be what G&M call the "common wisdom in neuropsychology." To my mind, this is not a bad position at all. G&M may want to "put themselves in opposition" to it but they should do so with sound argument and not by misattribution.

I would now like to return to the principal issue under consideration in this discussion; viz., the issue of what claims may justifiably be made on the basis of results such as those reported by C&Z. I have already noted that although G&M are technically correct in the criticism they directed to the C&Z study, such a criticism does not, on its own, undermine the principal claim made by C&Z in their paper. The criticism one should direct to C&Z is much more serious and it applies equally to the "alternative" proposal of the basis for "agrammatism" offered by G&M.

The criticism has both theoretical and methodological dimensions; it concerns the very nature of the claim being made and the methodological foundations on which such claims rest.

With respect to the nature of the claims themselves, it is my contention that statements such as the two that follow—statements that succinctly summarize the theoretical positions of C&Z and G&M—are theoretically vacuous: “at least for the Broca’s aphasics, brain damage affects a general language processing mechanism that subserves the syntactic component of both (language) comprehension and production.” (Caramazza & Zurif, 1976; p. 581), or “the correct characterization of the syntactic abilities in agrammatic Broca’s aphasia maintains only a partial impairment (sic). As a consequence, these patients have access only to incomplete syntactic representations, which explains why their differential performance correlates with specific types of syntactic constructions.” (Grodzinsky & Marek, this issue). Such claims are vacuous because clinically defined patient types (e.g., Broca’s aphasia, agrammatism, deep dyslexia, and so forth) cannot serve as the basis for theoretical statements concerning the nature of cognitive deficits. Briefly, the basis for this contention is as follows. Since clinically defined patient categories are ill-defined—that is, we can specify neither the extension nor the intention of these sets—it is totally unclear what it means to say that patients of such and such a category have a deficit to some component or other of a cognitive system (Caramazza, 1984; Marshall, 1986). Thus consider what it might mean to say that agrammatic Broca’s aphasics are impaired in syntactic processing. Which features of the complex clinical picture that characterizes the syndrome of Broca’s aphasia are the result of the hypothesized functional lesion? And, even if one were able to specify the range of such features (i.e., have a relatively well-defined description of the range of relevant facts the theory is supposed to account for), in what way do the claims of a syntactic deficit account for the identified pattern of impairment? Thus, in what way does the hypothesis of a syntactic deficit as the basis for relevant features of so-called agrammatism, say the morphological errors in the speech of some such patients, provide a meaningful account of such performance? It should be apparent that no nontrivial answers have been given to such queries—indeed, none can be forthcoming either (see Badecker & Caramazza, 1985, for detailed discussion of these issues).

As I have argued in some detail elsewhere (Caramazza, 1986), if we can account for a pattern of impaired performance by postulating a functional lesion to a model or theory of language processing, then we can take the obtained result as empirical support for the proposed model over some alternative account. It must be emphasized here, if there is any need, that the postulated model must be consistent not only with the specific pattern of impaired performance under consideration, but

also with other clearly established patterns of impairment and normal performance that are theoretically relevant. Thus, C&Z and G&M would be justified in concluding that a patient has a functional lesion to a syntactic processing component or to a mechanism that establishes traces in a syntactic structure, respectively, if the postulation of these functional lesions allows them to account for relevant patterns of comprehension performance. However, both C&Z and G&M draw much stronger conclusions from their respective data than is legitimately possible (here I am concerned neither with the details of the particular theories proposed by either set of authors nor with the reliability or validity of the results on which they base their claims).

C&Z and G&M conclude that their claims are claims about a patient category called agrammatic Broca's aphasia. It can easily be shown, however, that these conclusions are unwarranted. Even ignoring the fact that "agrammatism" is not a theoretically coherent notion, there is the issue of what these authors might mean by their respective claims about agrammatism—that is, by their claims that the functional lesions to the language processing system they hypothesize can account for the features of the impaired performance that is used to identify the patient population they are referring to. Obviously, they cannot mean that brand of agrammatism identified by reference to a particular pattern of language production difficulties (i.e., "productive" agrammatism). For if this were what they meant then the claims are empirically unmotivated and theoretically vacuous. The claims are empirically unmotivated because they have not shown that the comprehension failure they have analyzed is a standard feature of the patient type they wish to make claims about; that is, they have not shown that a specific pattern of production impairment is necessarily related to a specific pattern of comprehension impairment. Furthermore, this claim is empirically false (e.g., Miceli, Mazzucchi, Menn, & Goodglass, 1983; although this empirical fact was not known at the time C&Z published their work, G&M cannot plead ignorance!). C&Z's and G&M's claims are vacuous because they have not established a theoretical link between the claims concerning the comprehension process and those concerning the production process. Thus, consider, for example, G&M's claim about trace deletion and agrammatism. What relationship is there between these authors' claim concerning trace deletion and so-called agrammatic production? Patently, the details of agrammatic production, whatever these might be, are totally unrelated to the details of the claim concerning the hypothesis of trace deletion in the comprehension of these patients. In the absence of clear statements on this issue the claims about "agrammatism" remain contentless.

Equally problematic would be the conclusions drawn by C&Z and G&M even were we to understand "agrammatic aphasic" in the restricted sense of "agrammatic comprehension aphasic" (i.e., if we were to ignore altogether aspects of sentence production). For here the claims would

not be interesting, since presumably the identification of agrammatic comprehension would be coextensive with the reported patterns of comprehension failure; in other words, there would be no more to the category "agrammatic comprehension" than the pattern of performance described in the experiments reported by the two sets of authors. So, either the locution "agrammatic aphasic" is coextensive with the reported patterns of comprehension failure, and hence tautologous, or it is not in which case no theoretical proposal has been offered for the set of comprehension features that will (independently) identify the category "agrammatic aphasic." The point here is that no independently well-defined category of patient performance has been identified over which theoretical claims could be made. Hence, once again the claims remain contentless.

We have seen in the preceding that the claims made by C&Z and by G&M are theoretically indefensible. One critique that has been raised against these authors concerns their failure to provide independently well-defined criteria for the identification of the patient category over which they wish to make their claims. This failure is not accidental but reflects instead a necessary "limitation" in research with brain-damaged patients. I have presented extensive arguments in support of this latter contention elsewhere (Caramazza, 1986). These arguments are too complex to be repeated in detail here. Briefly, the point is that meaningful neuropsychological research cannot be based on a priori classification of patient types. An analysis of the conditions needed for drawing legitimate inferences about normal cognitive processes (and ultimately about the neural mechanisms that implement these processes) from patterns of impaired performance consequent to brain damage has shown that such inferences must be mediated by the postulation of a functional lesion (or lesions) to the normal system. In a crucial sense we may consider a brain-damaged patient to constitute an "experiment of nature" where the functional lesion that results from brain damage represents a set of *unknown* experimental conditions that have been preset by nature. Our task consists in determining the nature of this functional lesion (the preset experimental conditions) in some hypothesized model of a cognitive system. Success in this task for a particular model may be taken as support for this model over alternative models which could not be functionally lesioned in such a way as to provide an account of the observed performance. However, a functional lesion can only be specified a posteriori—that is, on the basis of all the relevant evidence needed for determining the kind of transformation that the hypothesized model of the cognitive system has undergone as a result of brain damage. Thus, there can be no theoretical merit to a priori classificatory schemes that are based on any arbitrary subset of a patient's performance—all of a patient's relevant performance is needed to motivate the hypothesis of a functional lesion to a cognitive system. This analysis has an important methodological consequence: only single-patient studies allow valid inferences about the structure of normal

cognitive systems from the analysis of impaired performance in brain-damaged patients (see Caramazza, 1986, for detailed discussion). Since the results used by C&Z and by G&M to reach their respective conclusions about the nature of the deficit underlying agrammatism were based on patient-group data we have additional reasons for being skeptical about the "theoretical" claims made by these authors.

To conclude, I have tried to show that G&M's critique of C&Z is off the mark. There are deep theoretical and methodological reasons for rejecting claims such as those made by C&Z (and repeated in only a superficially more sophisticated form by G&M) that go well beyond the technical problems raised by G&M. Neuropsychology is fast shedding itself of an historically important but theoretically and methodologically inadequate framework for the analysis and interpretation of cognitive disorders. We are beginning to make efficient use of the rich patterns of impaired performance, which nature provides to us in the form of clinical disorders, for constraining theories of cognitive processing. The increasingly articulated models of cognitive processing that are emerging in the field of neuropsychology give us a privileged opportunity for laying the foundations for a nontrivial theory of the relationship between human cognitive processes and brain mechanisms. Surely the time has come to put an end to vague claims about clinical categories of aphasia such as that advanced by C&Z and the variant repropounded by G&M. When is enough, enough? Why is it still possible to publish papers which purport to make theoretically coherent claims about patient categories such as deep dyslexia, Wernicke's aphasia, or agrammatic Broca's aphasia?

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