

## Caramazza's Response to Discussants

Dr. Caplan made three points in his talk: 1) data from brain-damaged patients have little utility beyond serving as a source of ideas for theoretical refinement in Linguistics; 2) we should not do single-patient studies but patient-series studies; and 3) patient-group studies allow valid inferences about normal language processing.

I am not sure why Dr. Caplan would assign such a low status to research with aphasic patients. He offered no arguments in support of his assertion. He is entitled to his opinions but they should not be accorded the status of sound argument. We all hold strange opinions we are not able to defend. For example, I believe the moon is made of green cheese.

The second point raised by Dr. Caplan--that series of roughly homogeneous patients should be studied together, a proposal I made some years ago (Caramazza and Martin, 1983)--is interesting but irrelevant to the present discussion. The crucial issue is whether or not single-patient methodology is the only legitimate form of analysis of the performance of brain-damaged patients. If we agree that only single-patient methodology allows valid inferences from impaired performance to models of normal language processing, then whether one studies just one or several patients depends on the availability of relevant cases for study. But, if one studies more than one patient the methodology must remain that of single-patient studies if it is to allow valid inferences.

Dr. Caplan's third point is puzzling. He concludes that patient-group studies allow valid inferences to normal cognitive processes. This point is not made on the basis of argument but by discussing one of his research projects. I have no more to say about this matter than what I said in my paper--there are clear logical reasons for concluding that there are no nontrivial cases where averaging of patients' performance allows valid inferences to models of normal cognitive processing.

I would like to respond to two points made by Dr. Lemme. The first concerns an apparent circularity in my argument against classical aphasia classifications. If I understood Dr. Lemme's argument it goes as follows: I have argued that the pragmatic motivation, for believing that patterns of impaired performance consequent to brain damage are relevant to theories of normal language processing, is the observation that language impairment does not result in undifferentiated loss of language processing ability but, instead, it appears to reflect the selective loss of various processing functions. I have also argued that we should abandon theoretically vacuous aphasia classifications. She takes these two statements as leading to a circular argument. I fail to see the circularity--because I reject the accepted aphasia classifications it does not mean I do not value the observations of language dysfunction that have been reported in the literature. The observations in the literature and those we make in our laboratory are independent (well, not quite but it will do for our purposes) of the classification schemes we adopt. It is these observations that I claim provide the pragmatic support for my contention that acquired disorders of language may play an important role in the development of theories of normal language processing.

The second point I would like to address concerns Dr. Lemme's appeal to "severity" in evaluations of language disorders. Severity may be a dangerous concept unless used properly. It is a dangerous concept when severity is applied as a general measure of language impairment. If we assume that

language processing involves a complex set of independent processing mechanisms and that brain damage may selectively damage one or more of these processing components, then the indiscriminate grouping of different deficits under the general rubric severity misses all the relevant information about the precise nature of the language impairment in individual patients. This does not mean that one processing component may not be more severely damaged in one patient than in another. We can have severity measures but these must be for specific processing mechanisms.

Dr. Davis' comments have raised two issues that I would like to address briefly. One concerns the need for computational explicitness. By this I mean that our models of language processing must be articulated in detail and must make explicit the processing structure of the assumed components of processing.

The second issue raised by Dr. Davis concerns the relatively limited scope of the theoretical framework I proposed in my talk. He urged us to consider language processing in the wider framework of communicative competence. I agree that there are important theoretical and empirical issues regarding the pragmatics of language use. And, my presentation did not want to exclude this important aspect of language use. The inferential schema I discussed is equally applicable to this broader theoretical context.

In conclusion, I wish to reiterate a point I have made in several publications (e.g., Caramazza, 1986). When I claim that only single-patient studies allow valid inferences from impaired performance to models of normal language processing I am not claiming that we should study only a single patient. Obviously, the theories we develop must be theories of language processing for all normal individuals. These theories must be compatible with observations from all brain-damaged patients. The claim is that evidence from each patient studied must converge on a single best explanation of language processing.

#### GENERAL DISCUSSION

Q: Dr. Caramazza, in your discussion, it seems that your basic assumption is dependent on this equation you gave at the beginning.  $M + L$  yields  $O$ . And, in that equation, as I understood it, your argument is that when we see a person with a lesion or a functional deficit, the change that we're seeing is in the functional deficit. Now, isn't it possible that in many individuals who have structural brain lesions that what's changed is actually "M" rather than "L" and that what you're dealing with in brain-damaged individuals is a change in the model, rather than a direct effect of a lesion, and in many situations this would explain a lot of the variations we see in individual performance; i.e., that the effect is on the model itself so that you actually have multiple models. And in this case then a single-case study would become a discussion in error if one is trying to talk about the normal, unless one could really define this separate model. That's issue number one.

The second issue is the "M." You assume that the model is the same in each of us. I think that that's probably an error and let me give you an example. I know nothing about linguistic theory. In listening to your talk and to Dr. Caplan's talk, I felt very aphasic during much of it. Part of it is that my background is quite different than yours. My education is different than yours. My capability in multiple language is also quite different. Won't these differences in my environment, environmental bringing-up (or whatever my social background is),

California rather than the east coast, all play into modifications and variations on a model, so that "M" actually has to be listed "M" with "I" under it, where "I" represents a number of variations on the theme?

A: You have raised two important issues with respect to the issue of why it is I assume that brain damage results in  $M + L \rightarrow 0$  as opposed to some modification of M, say M1. I think that there are good reasons for doing this. My assumption is that brain damage modifies an existing cognitive system along specifiable lines. Brain damage does not create de novo cognitive systems. If it did so, the data from brain-damaged patients could not be used to inform theories of normal cognitive functioning. I think that this assumption is well worth making. I cannot imagine how it would be possible for brain damage to create new cognitive processing systems.

Your second question concerns whether or not it is reasonable to assume that M's are equivalent in relevant respects as I have argued. Again, I think the assumption is well worth making. This does not mean that there are no individual differences in cognitive processing. However, these differences are not at the level of the organization of cognitive systems.

Q: Do you think that a new classification system will emerge from some presumably large number of your single-subject designs? If you do, how convinced are you that it will or won't resemble existing ones? And, if your answer is "no," we won't find a classification system out of some large number of single cases, doesn't that weaken the contribution that aphasia, or the study of aphasia, can make to the study of normal language?

A: I don't think that the development of a classification of language disorders is a necessary step in understanding normal language processing. Of course, given a theory of language processing we can make predictions about the types of language disorders that are likely to result from language processing. Furthermore, given such a theory we can determine on the basis of a patient's performance where the likely locus/loci of the functional deficit is in the cognitive system. This is equivalent to saying that we can specify, for a given theory, the conditions to be satisfied for a pattern of performance to constitute evidence that a patient has damage to some or other component of processing. So, in principle, a classification scheme is possible for a given theory of language processing. But the classification is theory-dependent and it must change as our theories change.

C: Your purpose in your work, is to develop models of cognitive processes. We develop models of cognitive processes and base treatment on that and then we also treat patients; we want to make patients better. I think that group studies are useful to us as clinicians, although we do not need to be theoretical in our approach to patients. So I think we need both kinds of designs in our work.

A: As I stated earlier, I do not think that there are any nontrivial cases where averaging patients' performance is useful or methodologically valid. But, I must reiterate that there may be theoretical questions which can only be addressed by determining whether, for a series of individual cases, two or more symptoms are associated. For such cases we also need single-patient studies for series of patients.

Q: I didn't understand Dr. Lemme to be talking about severity of a person who is aphasic and also has certain visual problems, but the range of severity we see within aphasia. We know that people improve and that the existing model or classification systems do not cope with that improvement very well, and that people do change their type of aphasia over time. Nonfluent patients become fluent. And in terms of brain-behavior relationships, I'm not convinced the lesions pack their bags and move to another part of the brain. So, those existing systems do not explain change in severity. How would your model cope with severity? If you look at a single subject, you're going to catch them at different points in severity over time.

A: This is an important question. I am afraid I have not given much thought to the problem you raise. Our research is carried out with patients whose impaired performance remains stable over the course of our research. By stable I mean that the qualitative pattern of performance remains constant over time. Quantitative variation (severity?) does not play a crucial role in theory development at the level at which we have been concerned.