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NOTES AND DISCUSSION

An Evaluation of Assumptions Underlying the Single-Patient-Only Position in Neuropsychological Research: A Reply

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This note challenges the position adopted by A. Caramazza and W. Badecker (1989, *Brain and Cognition* 10, 256-295) that, since the a priori classification of patients can only be theoretically arbitrary, the basic unit of analysis in cognitive neuropsychology must be the individual patient. We argue that even if there is no prior theory to justify patient classification, this does not preclude group studies; syndromes are what the world gives us-they constrain theory, permitting groups to be formed for research purposes. We also reexamine a particular example of group-based research that was extensively criticized by Caramazza and Badecker. We confront each of their criticisms, and, again, demonstrate the validity of group-based research.

Caramazza and his colleagues have now written quite a few articles that attempt to defend the position that *only* single-patient studies allow valid inferences about the structure of cognitive mechanisms from the analysis of impaired performance (e.g., Caramazza, 1986; Caramazza & McCloskey, 1988). We believe that they are wrong about this issue, and, by extension, about the type of scientific endeavor represented by cognitive

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neuropsychology. Also, we think that a proper resolution of the matter requires that a consideration of the general methodological/scientific issues be kept separate from any discussion of specific empirical issues. Accordingly, we deal first with the general scientific/methodological considerations that' bear on the controversy. Our arguments in this respect are fashioned largely in response to the notions expressed in Caramazza and Badecker (1989), this paper being the latest in their single-case-only series, and one that was written in direct response to a paper by Zurif, Gardner, and Brownell (1989) defending group studies. We then examine a particular experimental result that figured in Zurif et al.'s defense of group studies and that Caramazza and Badecker attacked in their response.

GENERAL SCIENTIFIC/METHODOLOGICAL ISSUES

The linchpin in the Caramazza and Badecker position is that the procedures in cognitive neuropsychology differ in a fundamental way from typical experimental procedures in other sciences. As they state it, in contrast to scientists in other domains, cognitive neuropsychologists cannot *control* all relevant experimental manipulations; brain-damaged subjects are experiments of nature in which one experimental condition (the "functional" lesion) cannot be determined in advance, but rather must be inferred from the individual patient's performance. Caramazza and Badecker take this to indicate that the a priori classification of patients into clinical categories can only be theoretically arbitrary and that the only nonarbitrary classification of patients possible is, thus, a posteriori. That is, in their account, the only classification that is possible is one that is based after-the-fact on those theoretically relevant performance characteristics that allow the identification of a functional lesion in a cognitive system. And this, they continue, is equivalent to the claim that patient classification cannot play any significant role independently of the single-patient research projects that are required to determine that each of the patients in question has the appropriate functional lesion for a posteriori classification. In short, ". . . the basic unit of analysis in cognitive neuropsychology must be the individual patient" (Caramazza & Badecker, 1989).

There are a number of points about this general position that require discussion. The first, but by no means the most central, is that the contrast of the "ideal" of experimental method portrayed for other sciences compared with cognitive neuropsychology is far from realistic. Even when the "manipulated" variable (the strength of the scientific experimental method) *is* manipulable, as in standard experimentation, it is always an open question whether the experimenter has the right theory about that which he is manipulating. It is a truism of the philosophy of method that the theory of the experimental manipulation, as well as the theory that

the experiment is designed to test, can, in principle, be revised in the face of recalcitrant data. (This is an immediate consequence of the Duhem (1962)/Quine (1961) thesis which argues that, in principle, *any* statement of a theory is revisable in the face of recalcitrant data.) The fact that the scientist claims, in all sincerity, to have performed a certain manipulation does not entail that he has actually done so. In this sense, then, the experimental conditions that are manipulated can *never* be "determined in advance" (i.e., in advance of the best explanation of the experimental outcome). So it is not at all clear, as Caramazza and Badecker would have it, that there is a principled difference between neuropsychology and other cognitive sciences with respect to control over experimental manipulation.

The central points of the Caramazza and Badecker position are that a priori classification must be theoretically arbitrary and that this being so a priori patient groupings are inadmissable.

We do not concede the first point as the expression of some sort of principle. One can imagine circumstances in which an experimenter might have an ironclad. theoretical reason to suppose that if a brain-damaged subject exhibits an impairment in one capacity, he will exhibit a syndrome involving other capacities. For example (in an extreme case), it seems very likely that a subject who is completely insensitive to light will have subnormal color discrimination.

An even more important consideration, however, has to do with the consequences of theoretically arbitrary a priori classification. In opposition to Caramazza and Badecker, we claim that even where there is no prior theory to justify a particular taxonomy, there need be no impediment to rational inquiry. Our point here is that taxonomies in cognitive neuropsychology do not have to be theoretically motivated; they have to be expirically motivated. And this brings us to the heart of the matter: cognitive neuropsychology, like, for example, astronomy, is an *observational* science. Its practitioners get by without actively manipulating functional lesions or, for that matter, brain lesions. In this framework, syndromes (even loosely defined ones like nonfluent, agrammatic Broca's aphasia) are what the world gives us; they are there to constrain theory and, to this end, to allow groups to be formed for research purposes.

The way such observational science works is generally held to be as follows: through observation an investigator notices a number of symptoms or signs that appear together (perhaps this is guided by some rudimentary belief about the object being observed, perhaps not); this emerging syndrome suggests an etiology to the investigator; the etiological model predicts previously unnoticed components of the syndrome; examination of the object for these additional components proceeds, either sustaining the hypothesized etiology or not (usually not); the new observations add to the syndrome leading to new hypothesized etiologies; etc. Thus, there is

essentially a two-stage process at work here: first, the reliability of a syndrome (or that part of it that is intuitively relevant to one or another theoretical concern) is established, and then there is the step commonly called "saving appearances," where a model to account for the syndrome is constructed.[1] (For a nonbiological example, consider that observation of the movements of the planets might suggest a heliocentric model to an observer. This in turn might predict stellar parallax, a previously unnoticed "appearance.") The basic questions in this observational enterprise, then, concern the empirical matters of whether the data sustain a particular classification and whether the model chosen to explain the classification is reasonable (and continues to be so).

Zurif et al. provided an illustration from medicine of these inductive stages. Specifically, they described the process by which "Legionnaire's disease" was studied-from the initial and tentative grouping of patients who shared symptoms, through inferences supported by group-based research, to the present understanding of the disease. This illustration was criticized by Caramazza and Badecker as faulty reasoning-by-analogy (as possibly a rhetorical ploy to suggest that good medical practice cannot be bad neuropsychology). But it was not reasoning-by-analogy. It was a description of methods common to observational sciences. If Caramazza and Badecker think that there are important differences between the illustration provided in Zurif et al. and cognitive neuropsychology, they must show what these relevant differences are. We do not think that there are any, for this is the way observational sciences function.

The point is that in cognitive neuropsychology, one is not concerned with whether a classification is valid. It simply is not in the nature of the science to be able to do so. Rather the goal is to determine whether inferences from observations are reasonable (whether they continue to "save appearances") and whether observations are reliable. To argue, therefore, that a failure to provide an a priori theoretical justification for patient classification ought to preclude group studies is simply to miss the point of observational science. One will naturally tend to group observations (patients, in this case) as a way of determining whether the observations are reliable and whether the model continues to support the observations (across and within patients).[2,3]

[1] This is, of course, a rational reconstruction of "stages" of the process; in practice, both go on together or, at least, in arbitrary order.

[2] This is not to suggest that such observation, grouping, or model-building will be easy. It is clearly not so. However, the process is one that is approached through both case studies and group studies in all such sciences, and there is simply no principled way to eliminate group studies from the enterprise.

[3] Along these lines, it should be noted that single-case studies themselves provide no guarantee of getting theoretically correct descriptions. No matter how carefully you examine one patient you will still run the risk of that patient being a statistical outrider. That is,

AN EXAMPLE OF GROUP-BASED RESEARCH

In addition to criticizing the logic of the single-case-only position, Zurif et al. also attempted to mount positive support for group-based research. This was in the form of two research examples. One example had to do with real-time lexical access in aphasia, the other, with syntactic limitations of relevance to sentence comprehension. Caramazza and Badecker argue that neither example works. In what follows, we discuss the first of the two examples in detail with the aim of demonstrating that Caramazza and Badecker are wrong. (A discussion of the other example in the context of supporting group-based research is the focus of another paper (Grodzinsky, 1990b).)

The example we reexamine here is of a study (Swinney, Zurif, & Nicol, 1989) that examined in brain-damaged patients a processing component involved in sentence comprehension: namely, a lexical access component that provides data on word meanings for the rest of the comprehension system. One focus of this study concerned a general theory about perceptual processing in intact adults known as the modularity thesis (Fodor, 1983). A central component of this thesis is that input processing systems are constituted as fast-acting devices that are not penetrated by rational considerations. That is, these devices are considered to be contextually impenetrable.

Support for this thesis comes in part from findings referred to in Zurif et al. To repeat one such finding here, Swinney (1979) has observed, via the application of a cross-modal lexical priming technique, that immediately upon hearing a polysemous word in a sentence the normal listener accesses all of that word's meanings, not just the one relevant to the sentence's context. Only after a time delay of approximately a second does context exert its effect; only after that time does context damp all of the word's meanings save the relevant one. One general lesson from this, then, is that such processing does not consist of a set of maximally interactive processes. Rather, context seems to have an effect on lexical processing only following normal exhaustive access.

In line with this form of inquiry, the study of asphasia carried out by Swinney et al. (1989) and described in Zurif et al. showed that nonfluent agrammatic Broca's aphasic patients had a module-specific disruption to lexical access that could reasonably be implicated in their real-time comprehension problems.[4] However, these patients did not appear to lose the

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every patient that reliably demonstrates the lack of some cognitive ability is actually a check that cannot yet be cashed; one cannot tell, in individual cases alone, if a single case (that might, let us say, demonstrate a behavior NOT a part of an hypothesized syndrome under investigation with that patient) represents an anomalous (outlier) subject or a case that can be used to disprove that hypothesis.

[4] Here and throughout the rest of this paper we use the term nonfluent, agrammatic

modular properties of lexical access. They did not, that is, access only contextually relevant word meanings. Rather their disruption seemed to preserve the fundamental modular property of cognitive architecture, but to render the internal operations of the access module, itself, less efficient. Namely, only the most frequent meaning of the ambiguity was initially accessed, independently of the contextual bias.

We note here also that this result arose in the context of what appeared to be good comprehension of the test sentences. Specifically, twice during the course of each experimental session-four times in all-we asked the patients to paraphrase what they had just heard, and in each instance they were able to do so. Moreover, our test of comprehension was more rigorous than is usual in aphasia research, requiring not only understanding but also the capacity to organize the gist of what was understood for an appropriate response. Also of importance here, we used only structurally simple sentences-sentences of the type that nonfluent, agrammatic Bro-

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Broca's aphasia to refer to patients who show particular comprehension and production problems. With respect to comprehension, they show broadly normal understanding at the level of conversation, but are impaired at the syntactic level, at the least, in the understanding of sentences in which nouns are ordered noncanonically (e.g., the passive). With respect to production, they present with noticeable syntactic simplification and show imperfect control of grammatical morphemes-all in the context of nonfluency.

In the framework of this characterization, we note that a recent analysis by Miceli, Silveri, Romani, & Caramazza (1989) has shown great variation in the proportion of grammatical morphemes omitted and substituted in a group of nonfluent aphasics. To our minds, however, the numerical values Miceli et al. (1989) cite are simply irrelevant to the issue of categorizing this patient group for the exploration of psycholinguistic issues. By seeking to characterize nonfluent, agrammatic Broca's aphasia as a quantifiable entity, they do nothing so much as enter the unnecessarily complicating factor of severity. And by focusing on differences among such patients, they miss the point that these patients also share grammatical features that allow categorization in the first place (even for their own study). They miss the point that every patient they tested showed abnormal control of grammatical morphemes. In effect, Miceli et al. (1989) seem unwilling to accept loosely defined patient groupings in advance of some theory, even if these groupings can help researchers form and sharpen theoretical models (see also Grodzinsky, 1990a, 1990b). And this we have argued above is the situation observational scientists most often must confront.

At any rate, the research example of ours that we discuss in this paper employs also a second group of aphasic patients-Wernicke's aphasic patients. Like nonfluent, agrammatic Broca's patients, they also show comprehension problems. But the two groups contrast in terms of production: While Broca's are nonfluent, Wernicke's are fluent. Moreover, an important point of our research example is that the two groups contrast in terms of lesion site: Wernicke's aphasia implicates left-posterior cortex; nonfluent, agrammatic Broca's aphasia implicates left anterior brain regions (often, apparently, including the anterior insula) (Blunk, DeBleser, Willmes, & Zeumer, 1981; Mazzocchi & Vignolo, 1979; Naeser & Hayward, 1978; Tramo, Baynes, & Volpe, 1988). These boundaries are inexactly drawn at present and, even so, there are clear exceptions. But, then, the behavioral indices entered in the brain-language correlation are, themselves, far from precise; and it remains an empirical issue whether disruptions stated in real-time terms might not have more exact lesion localizing value.

ca's are known to comprehend such that, clinically, they show "relatively intact" comprehension.

To reconcile the nonfluent, agrammatic Broca's patients' aberrant lexical access pattern with the strong likelihood that they understood each sentence, Swinney et al. (1989) speculated that the patients were slower at-but not disbarred from-activating relevant word meanings. Specifically, they speculated that the lexical access component operates with a slower-than-normal rise time: although the patients eventually retrieve the word meanings relevant for comprehension, they are, nonetheless, unable to provide the normal lexically based information to other components of the language processing device (e.g., they syntactic parser) at the correct (necessary) temporal point in the comprehension sequence.[5]

At any rate, Swinney et al. (1989) observed that this module-specific disruption did not hold across all aphasic patients with comprehension limitations. Nonfluent agrammatic Broca's aphasic patients with predominantly anterior lesions and fluent Wernicke's aphasic patients with lesions in left posterior cortex were different from each other. The former showed the disruption already described; the latter showed normal contextually independent, exhaustive access. So, Zurif et al. claimed that apparently similar comprehension failures can have different antecedents from a real-time processing perspective and, further, that these different antecedent disruptions can be aligned roughly with different lesion sites: anterior lesions being implicated in the real-time lexical access problem, posterior lesions causing some other problem for the comprehension system.

Caramazza and Badecker's first complaint with this study is that the possibilities are misconstrued to begin with-that if lexical access is encapsulated in the normal case and that if brain damage "unencapsulates" this access, then we have supposed the possibility that brain damage can result in the reorganization of the functional system. And this possibility-a "straw man" in their words--disallows us from contributing to a theory of the normal processing system. But that complaint is without merit: a

[5] This speculation has since been scrutinized experimentally via a detailed study of lexical access in a nonfluent, agrammatic Broca's aphasic patient with primarily left-anterior damage (Prather, Zurif, Stern, & Rosen, 1990). Prather et al. (1990) did not test for word access in the service of sentence processing, but rather, used a list priming paradigm in which words were presented continuously at different rates in different conditions. This list paradigm mimics one of the demands of sentence processing, specifically, the need to process words that follow each other in rapid succession.

The findings were these: elderly normal subjects were observed to prime at a short interstimulus interval (500 msec), but the patient did not show priming until an interstimulus interval of 1500 msec. Moreover, the patient's priming effect diminished at 1800 msec, strongly suggesting that the abnormality had to do with automatic processing, uncontaminated by strategy-driven effects. Clearly, these data are compatible with the suggestion raised here: namely, that nonfluent, agrammatic Broca's aphasia implicates slower-than-normal lexical access.

disruption to one processor, such as we were examining for, does not require that the remaining comprehension processing devices be reorganized. In fact, the central assumption in inferring functional localization from pathological material is that a functional lesion to one or another processor leaves the rest of the system intact (e.g., Caramazza, 1986).

We are also not persuaded by Caramazza and Badecker's second comment concerning "unencapsulation" or "demodularization." This is their claim that even if Swinney et al. (1989) had shown contextual infiltration of lexical access, they still could not have claimed that brain damage destroys a module. And this is so, Caramazza and Badecker state, because contextual influence is also shown in normal processing, namely, in the particular case when meaning for the lexical item is probed "downstream" from its appearance in the sentence. But, this comment simply represents a misunderstanding of both the modularity claim and the entire reason for the use of on-line experimental method in the examination of human cognition.[6] The issue with regard to modularity is whether context affects *initial* perceptual processing-that which we call lexical *access*. (No one doubts that context *eventually* has an effect on interpretation, but the evidence is that it has such an effect *after* modular lexical access.) So, had Swinney et al. (1989) shown immediate penetration of context in nonfluent agrammatic Broca's aphasia, they could reasonably have argued for demodularization of one aspect of sentence processing.

In any case, however, that is not what Swinney et al. (1989) found. Again, what they found for nonfluent agrammatic Broca's patients was that the modularity of lexical access remains intact; the functionally modular property of access is not absent, but, rather, the internal operation of that module works abnormally, most likely in terms of its temporal operating characteristics.

Caramazza and Badecker, however, are none too accepting of the actual result either. First, they ask what would have happened if one of Swinney et al.'s (1989) four patients in this group had shown a different pattern of performance from the other three. And the response they favor is that

[6] Caramazza and Badecker are also wrong in arguing that because "on-line" studies carried out by Tyler (1985, 1988) did not require group analyses, it cannot be true that such methodology mandates the averaging of data from different subjects. But Zurif et al. never claimed that it did. Quite simply, the notion "on-line task" is neither a uniform nor a monolithic concept: the tasks of Tyler and Swinney et al. were different and required different analysis. More to the point, and consistent with what Zurif et al. actually claimed, the method employed by Swinney et al. (1989) *does* require multiple subjects. Given that one exposure to contextually biased ambiguities alters subsequent interpretation of those ambiguities, subjects cannot, in principle, be allowed to be in more than one of the several biasing conditions of these experiments. If they are, their behavior in subsequent experimental conditions will not be independent of their earlier exposure. (In practice, with brain-damaged subjects it may be that they are not responsive to such exposure in the "normal" way; however, in principle, one cannot depend on that possibility.)

the category of nonfluent, agrammatic Broca's aphasia would have to be considered heterogeneous at the level of the generalization sought. Our response is different. It is the standard one: that experimentation is an uncertain effort and that in consequence, statistical tests provide us with some idea of the strength of a hypothesis in the context of the particular paradigm used and the current state of knowledge. This connects with points made earlier: One of the reasons (not the only one) for using statistical procedures in experimental analysis is that one often does not have a detailed understanding of the experimental manipulation. For example, does it affect all of the subjects equally? Does it affect all of the subjects in the same way? This introduces an uncertainty over and above the one that the experiment is intended to resolve: namely, the nature of the contingency between the putative manipulated variable and the putative dependent variable. To our minds this use of inferential statistics in acknowledgment of variability and uncertainty is not too different (maybe a little more precise) from the way in which descriptive generalizations are handled in the single-case-only approach. There, too, isolated results are not likely to render null the effect of the rest of the available evidence.

As it happens, however, all of the patients in Swinney et al. (1989) did show the same performance pattern. So Caramazza and Badecker's next move is to compare the Swinney et al. data to various other findings. The first of these comparisons is with Milberg and Blumstein's finding (1981; also Milberg, Blumstein, & Dworetsky, 1987) which is that nonfluent agrammatic Broca's patients show no automatic access characteristics whatsoever. By focusing on this difference, however, Caramazza and Badecker miss a significant generalization: namely, that in each of these studies nonfluent, agrammatic Broca's patients showed aberrant lexical access, while Wernicke's patients did not. Moreover, Milberg and Blumstein (1981) and Milberg et al. (1987) looked at access for isolated words presented in pairs or as triplets, a situation that has been demonstrated to elicit different processing characteristics from that for words in sentences (see, e.g., Swinney, 1979). Caramazza and Badecker acknowledging this, nonetheless ask why the sentence/nonsentence difference should matter, given that Swinney et al. found sentence contexts not to exert any influence. But, this is sophistic. Since the nonfluent agrammatic Broca's patients appeared to understand the sentences in which lexical access was probed for, it seems reasonable to assume that context did exert an eventual effect and that, as mentioned earlier, the patients' abnormality is to be stated in terms of a delay in providing lexical information to other sentence processing constituents. Quite simply, none of Milberg and Blumstein's experimental manipulations were geared to addressing this possibility (cf., Prather, Zurif, Stern, & Rosen, 1990 and footnote 5). We find

no reason, therefore, for thinking that their work undermines or challenges that of Swinney et al. (1989).

Caramazza and Badecker's second comparison-that between Swinney et al.'s (1989) fluent and nonfluent patients-misses the point entirely. As we understand it, their complaint is that (1) since asyntactic comprehension (their term, not ours) is a feature of both the nonfluent agrammatic Broca's and the fluent Wernicke's groups and (2) since only the former showed an abnormal lexical access pattern, there is nothing particularly compelling about the connection between the disruption to the lexical access system and the comprehension limitation. Their complaint is that Swinney et al. (1989) were being arbitrary in singling out such a connection. But there is nothing arbitrary about this connection: it holds only for one group and not the other, and each group was initially formed on independent clinical grounds far removed from the experimental facts that later emerged. Again, the point of the Swinney et al. (1989) finding in this argument is that apparently similar comprehension failures can mask different real-time processing disruptions and that these different processing disruptions can be aligned with different clinical pictures-agrammatic, nonfluent Broca's patients vs. fluent Wernicke's aphasic patients-that roughly implicate different lesion sites.

Caramazza and Badecker also criticize Zurif et al. for their failure to specify the link between the processing account offered in Swinney et al. and the representational account offered by Grodzinsky (1986; 1990a). (The latter refers to the other study discussed in Zurif et al. as an example of the kinds of claims supported by group-based studies.) And, relatedly, they criticize Zurif et al. for the fact that these two studies do not specify the "same range of facts" about agrammatism. This criticism is completely off the mark: Grodzinsky's (1986, 1990a) account is of computational goals at the level of syntax, while Swinney et al. (1989) provide a processing account, focusing on how a different part of the system operates in real time. If nothing else, the descriptive apparatus is different for these two lines of inquiry. They certainly cannot be required to account for the same "facts."

We are, however, actively pursuing an account of a link between these two characterizations. Without entering the matter fully, we note that in Grodzinsky's (1986, 1990a) account, the kinds of syntactic features that agrammatic, nonfluent Broca's patients have trouble representing are those involved in constituent movement: Specifically, the patients are claimed to be unable to link the "trace" left by the moved noun or noun phrase with that noun or noun phrase. This seems to us to be an operation that is implemented under strict time constraints-stricter than those involved in integrating and representing the gist of an utterance. In real-time terms, the limitation Grodzinsky (1986, 1990a) argues for can be

viewed as an inability to reactivate the lexical semantic information of the moved noun or noun phrase at the normal time in the processing sequence-in time, that is, to fill the gap left by the moved constituent and indexed by the "trace." And this possibility is a straightforward ramification of the problem indicated in Swinney et al. (1989)-the problem of a slow rise time in lexical access in nonfluent, agrammatic Broca's aphasia. In this view, the cortical area implicated in this aphasia is not necessarily the locus of syntactic representations, but rather is crucially involved in sustaining fast-acting access systems that are, in turn, involved in building such representations in real-time.

This may not be the right story. But we are not just idly speculating. We are currently exploring the temporal characteristics of the gap-filling operation-the reactivation of antecedent lexical reference-in sentence parsing, both in normals and in nonfluent, agrammatic Broca's aphasic patients. And there is no reason to suppose that we will not eventually have information on the time-based link between early-stage lexical access disruptions and structural disruptions of the sort described by Grodzinsky (1986; 1990a). Moreover, even as we anticipate providing a real-time processing explanation for Grodzinsky's (1986; 1990a) analysis of sentences featuring constituent movement and gaps, we are positioned to determine if intrasentential indexing (and semantic reactivation) breaks down on a wider scale, affecting also the interpretation of reflexives-to determine, in effect, if Grodzinsky's analysis, indeed, covers all of the relevant syntactic features that the patients fail to comprehend normally.

But whether or not we are successful in this respect, the attempt to describe processing limitations need not await a full appreciation of the processing and representation of sentences. It is patent that not all connections and details need be appreciated before empirical explorations can begin. So far as showing the value of group-based research is concerned, we think that it suffices that the Swinney et al. (1989) study has provided an analysis of some relevance to current theorizing concerning modularity, that it has pointed out possibilities for programmatic research on the relation between access mechanisms and particular syntactic processing capacities, and that the patient groups were sufficiently well specified to permit experimental inquiry of our claims in other laboratories.

Finally, we return to the general point of this paper, which is that insofar as we are constrained to deal with the syndromes that nature provides, neuropsychology is an observational science; and, this does not require providing a priori theoretical justification for those syndromes. In effect, syndromes exist apart from what we make of them. So, nonfluent, agrammatic Broca's aphasia, first focused upon in terms of classical (disconnectionist) theory, is now examined from a very different perspective. Now we attempt to mine the syndrome in order to develop a model that ties the capacity to represent certain syntactic features to the integrity of

specific cortical sites-a model that connects the two via the real-time operation of information access systems that are, themselves, localizable.

In this respect, we claim only that the differences between nonfluent, agrammatic Broca's aphasic patients and other aphasic groups appear potentially revealing. We do not claim to understand the significance of all of the individual features constituting the syndrome, nor to understand the significance of feature variability, nor to understand the significance of "anomalous" cases within the nonfluent, agrammatic Broca's group. For that matter, it is our hope that the model we are currently developing will eventually do away with the need even to grapple with the clinical signs of this aphasia-that it will allow us to form groups directly on the basis of lesion site. But, for the present, however much our classification of nonfluent, agrammatic Broca's aphasia depends upon a curious mix of production and comprehension factors and lesion localization considerations, the fact remains that such classification is possible and, directly to the point of this paper, helpful for theoretical work.

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