

Clinical Syndromes Are Not God's Gift to Cognitive Neuropsychology: A Reply to a Rebuttal to an Answer to a Response to the Case against Syndrome-Based Research

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In this paper we consider several issues about single-patient versus syndrome-based research in response to E. Zurif, D. Swinney, and J. A. Fodor's (1991, *Brain and Cognition*, 16, 198–210) criticism of A. Caramazza and W. Badecker (1989, *Brain and Cognition*, 10, 256–295). We argue that these authors have failed to provide convincing arguments in favor of syndrome-based research. In particular, we show that the specific example—a study by D. Swinney, E. Zurif, and J. Nicol (1989, *Journal of Cognitive Neurosciences*, 1, 25–37)—given by these authors as a demonstration of the usefulness of syndrome-based research to inform theories of normal language processing does not in fact serve this purpose. © 1991 Academic Press, Inc.

In various writings we and other researchers have maintained that one is compelled to use single-patient methodology whenever one wishes to employ data from acquired cognitive impairments to motivate specific claims about normal processing mechanisms or to explain patients' impairments in terms of some hypothesized damage to the normal cognitive system. The details of the arguments supporting these contentions may be found in Badecker and Caramazza (1985), Caramazza (1984; 1986), Caramazza and McCloskey (1988), and McCloskey and Caramazza (1988). In Caramazza and Badecker (1989) we answered Zurif, Gardner, and Brownell's (1989) objections to the conclusions of our arguments, and Zurif, Swinney, and Fodor (preceding article; hereafter ZSF) have provided a rebuttal. In the present paper we undertake a reply to their comments.

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THE DUHEM/QUINE THESIS

One of ZSF's primary objections to our position is that, in their view, it sets cognitive neuropsychology apart from other sciences by erecting a false division between what can and cannot be controlled in an experiment. In the course of pointing out the common basis for reasoning from psychological and neuropsychological experiments, Caramazza (1986) observed that a patient's impairment may be thought of as an experimental condition that is imposed on the normal cognitive system. However, unlike those conditions of the experiment that are manipulated by the experimenter (e.g., the materials, the task, the details of stimulus presentation), the cognitive impairment is imposed by nature and is therefore unknown in a sense that has important consequences for methodology. For example, the significance of these facts for the single-case versus group-study issue is that the scientist cannot interpret the average performance of a group of patients as an indication of how any member of that group actually performed, since the patients included in the group may have damage to different cognitive mechanisms. In response to this argument, ZSF point out that the simple fact that one does not start with the correct theory about the "true" experimental conditions in an experiment does not entail any special status for performance under those conditions. Thus, ZSF observe that, in any science, the experimental conditions may be unknown to the experimenter in that he/she may not have warrant for the theory that motivated the experimental design. But even if the experimenter is essentially ignorant of the true experimental conditions, they argue, this does not prevent him/her from drawing valid conclusions from the average performance of subjects who performed a task under those conditions. From this ZSF conclude that the experimenter's ignorance about *what* conditions are imposed by brain damage cannot invalidate the group-study methodology.

We do not disagree with ZSF about the epistemological features of the experimental situation, but these considerations have little bearing on our original arguments about the limitations of patient-group methodology. When an experimenter performs a particular manipulation in an experiment, there may in fact be theoretical consequences that he/she did not foresee. This by itself would not render group averaging invalid. That is, it is not a requirement for the valid interpretation of group averages that one know in advance *what* the true cognitive manipulation is that has been introduced in an experiment. On this point we are in full agreement with ZSF. However, the experimenter does rely on imposing the *same* experimental conditions (whatever their true effects) when group averaging is employed. If the conditions under which subjects perform a task are not equivalent (in relevant respects), then we are not licensed to take the average of their score as a measure of any subject's performance on

that task, or as a faithful estimate of the performance of an ideal population. Thus, what matters for the averaging method is that the subjects whose performance is to be averaged will have been imposed *equivalent* experimental manipulations (as far as the experimenter can determine), even though one may have the wrong theory about the true effects of the manipulation. And, if we consider brain-damaged subjects to be “experiments of Nature,” the issue becomes one of determining under what conditions we can assume that the same experimental manipulation has been introduced in different experiments; that is, under what conditions can we assume that two patients have equivalent cognitive deficits? If we are ignorant of the particular cognitive impairments that two patients present with, and if we have no real basis for believing that they present with the same cognitive deficit, then their average performance *cannot* be taken as a meaningful reflection of their cognitive impairments. More generally, if we have reason to suppose that the behavioral criteria for clinical categorization are consistent with more than one underlying cognitive impairment, then groupings of patients based on clinical standards cannot serve as the starting point for reasoning about the normal system, nor can they be used to motivate explanations of acquired deficits in terms of damage to specific cognitive mechanisms. We do not see that these consequences are in any way called into question by the observations that ZSF make. Note, furthermore, that the considerations presented here are *not* unique to cognitive neuropsychology and do *not* distinguish it from any other science. Nonetheless, because there seems to be some confusion about the nature of our argument, an example may help remove these misunderstandings.

Consider an experiment designed to evaluate a hypothesis about a specific property of the lexical access mechanism. Suppose, for example, that one believed that all the senses of a polysemous word are obligatorily, fleetingly activated upon presentation of a stimulus, but that only one sense is subsequently selected on the basis of context (e.g., Swinney, 1979; Tannenhaus, Leiman, & Seidenberg, 1979). Given this hypothesis, one might carry out an experiment designed to determine the meanings available to a subject by probing either shortly after (say 500 msec) or at some later point (say 1500 msec) after the presentation of an ambiguous word in a disambiguating sentence context. The expectation derived from the hypothesis is that both meanings are available at the shorter stimulus onset asynchrony (SOA), but that only the context-appropriate meaning is available at the longer SOA. The results of such an experiment could be used to assess (part) of the stated hypothesis of lexical access.

Now, suppose that in such an experiment one failed to find support for the hypothesis; that is, suppose that there was no evidence that both meanings of an ambiguous word are available at the short SOA. Can we reject the hypothesis that all senses of a word are obligatorily accessed

upon initial presentation of a stimulus? Not necessarily. Although the results do not support the hypothesis, it is unclear whether they require that we reject it.¹ Of course, it could be that presentation of a polysemous word does not, in fact, obligatorily lead to access of all senses, but it is also possible that we were mistaken about the time parameters of activation or that we were mistaken about some other assumption. Thus, it could be that at 500 msec selection of a specific meaning has already taken place. In such a case the negative result would merely indicate that we were mistaken about the relevant experimental parameter we should have used in our experiment (i.e., we should have probed earlier, say at zero msec). The point here—the Duhem/Quine thesis as used by ZSF—is that a negative result does not unambiguously determine which part of a theory may be faulty (including the possibility of faulty assumptions about the effects of an experimental manipulation).

The above case should be contrasted with the situation where the experimenter may be mistaken not about the possible effects of an experimental manipulation, but about the very manipulations that are introduced in an experiment. Thus, suppose that the program used to present the stimuli in an experiment with a single intended SOA had a bug such that some subjects received the probe at SOAs of 500 msec and some at SOAs of 1500 msec after the stimulus word (assuming for the sake of argument that the former time lag represents a point at which both meanings would be available and the latter time lag represents an experimental condition where meaning selection on the basis of context has taken place). In this case, the experimental conditions imposed by the experimenter are not equivalent across subjects, and averaging their performance would be a meaningless exercise. That is, the averaged performance would represent a theoretically meaningless mixture of a condition where all meanings are available (short SOA) and a condition where only the context-appropriate meaning (long SOA) is available to a subject. It is this latter situation—one characterized by a theoretically meaningless mixture of experimental conditions—that is relevant to the patient-group methodology (and not the one described above in which there might be epistemological uncertainty about the effects of an experimental condition). Note that, contrary to ZSF, the Duhem/Quine thesis has no bearing on the latter issue.

In short, we think that it is misguided to appeal to the Duhem/Quine thesis in order to motivate the claim that we have provided an unrealistic portrayal of the “ideal” of experimental method. Although the finer points of the philosophy of science surely escape us, we understand the Du-

¹ Of course, if there were no other grounds for believing in the hypothesis in the first place, the obtained result would be given considerably more weight in casting doubt on the hypothesis than if we had other reasons for believing in it.

hem/Quine thesis to speak to the indeterminacy of a given negative result for falsifying a theory. Specifically, the thesis holds that given a result that is inconsistent with a theory, such a result does not allow one to unambiguously locate the faulty part of the theory. ZSF appeal to this thesis in support of their claim that “. . . it is always an open question whether an experimenter has the right theory about that which he is manipulating” (ZSF, 1919). While it may be true that such doubt is probably incorrigible, we do not see its relevance to the present context. Even if one were to accept the Duhem/Quine thesis (and we are not competent to judge its correctness), it is easy to see that it is irrelevant to the present discussion. The issue here is not whether the experimenter has the right theory about some manipulation he/she has introduced in an experiment (for which doubt may remain), but whether he/she has introduced the *same* manipulation in different trials or replications of an experiment. That is, the experimenter’s confidence that he/she has introduced the *same* manipulation in different trials of an experiment is independent of the confidence he/she might have about the theory of *what* he/she is manipulating. At the risk of being repetitious, the issue here concerns the inappropriate though remediable application of a methodological practice (syndrome-based research) and not an incorrigible epistemological doubt about the effects of experimenter-imposed conditions.

ON GOD’S GIFTS TO COGNITIVE NEUROPSYCHOLOGY

Setting this issue aside for the moment, there is another argument that ZSF make for syndrome-based research: Even if we lack theoretically sound criteria for grouping patients, syndromes provide the basis for an observational science. In their own words “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” (ZSF, 1919).² The difficulty this statement poses lies in the intended sense of the phrase “to constrain theory.” What is the object of the theories that can be constrained by syndrome-based data? What is one trying to explain? If the cited passage is intended to defend clinical syndromes as objects of study in and of themselves, then one certainly could not fault it for making false claims. On the other hand, if the theories that ZSF refer to include theories of how the normal system operates, or how patterns of acquired impairment

² We are puzzled by ZSF’s claim that “syndromes are what the world gives us” (ZSF, 1991). Do they mean by this that syndromes are *natural kinds*? What motivates such a belief? We have argued extensively (e.g., see Badecker & Caramazza, 1985) that there are no grounds for such a belief. Syndromes are historically determined, theoretically arbitrary categories reflecting only pretheoretic intuitions about the nature of language and cognition.

can be explained in terms of underlying deficits to the normal system, then we have serious problems with their reasoning.

Our own view is that clinical syndromes are not God's gift to cognitive neuropsychology. Clinical syndromes are constructs based on aspects of behavior that clinicians or other researchers have chosen to focus on in the face of a large set of cooccurring behavioral features (and/or site of brain damage). This is not to say that one cannot approach syndromes scientifically; but it does follow that, in syndrome-based studies, one can only derive information about the consequences of the clinical categorization. That is, one may have an interest in answering such questions as "What is the relative probability that non-word reading and word reading will be impaired in someone clinically categorized as an agrammatic Broca's aphasic?" One could certainly approach this question by examining the word and nonword reading performance of a number of patients categorized as such. The difficulty, as we have indicated (see Caramazza, 1986), is that although syndrome-based reasoning can provide answers to questions about the relative predictability of the behavioral features of this or another clinical group, these answers cannot constrain, or in some other way contribute to, an explanation of any group member's underlying cognitive deficit. They could not do so unless there were some basis for attributing the same cognitive impairment to all the members of the group to begin with. An example may help make this point clearer.

Suppose that you wanted to know whether patients with agrammatic production are also likely to present with comprehension impairment of some sort or other. In such a case you might select a group of agrammatic patients and test them in comprehension tasks to determine whether they are in fact impaired in understanding sentences. You might find that as a group the agrammatic patients show poor sentence comprehension. If you had a large enough sample you might even have considerable confidence about the strength of the association between the two symptoms. This result might even be of clinical (actuarial) value—it tells you that given the feature agrammatic production there is a reasonable probability that it is associated with comprehension difficulty. However, no further inferences about normal cognition or the underlying cause of the noted deficits are warranted. Thus, for example, you could not conclude from the group study that the production and comprehension disorders have a common underlying cause. For the latter type of inference you would at the very least need to show that agrammatic production is invariably associated with a comprehension deficit. If only 85% of the patients show the association between symptoms, even though statistically reliable given an appropriate sample size, this result could not be taken as support for the hypothesis of a common underlying deficit for agrammatic production and the associated comprehension disorder.

Of course, one could argue that for the 85% who show the association

there may in fact be a common underlying cause.³ Note, however, that in the latter case the relevant information is not the group result but the *individual* patients' results concerning the association of the two symptoms. Note further that one must provide explicit arguments about why it is that the remaining 15% do not show the correlation between symptoms. That is, one would have to argue, for example, that there are two distinct types of agrammatic production—one associated with comprehension difficulties and one associated with normal comprehension. This implies that the superficially similar disorders of agrammatic production are in fact the result of damage to different linguistic/cognitive processes. In the latter case, we would have to conclude that the critical features that determine membership in the category of agrammatism fail to distinguish between superficially similar but underlyingly different forms of damage to cognitive/linguistic mechanisms. Averaging the performance of patients selected on the basis of such criteria is not different from the equally unacceptable practice of averaging the performance of subjects (normal or otherwise) subjected to different experimental conditions (e.g., averaging the performance of subjects who have been presented probes at SOAs of 500 and 1500 msec in the experiment discussed in the previous section). In other words, when the goal of research is to characterize the nature of the normal mechanisms which when damaged result in particular patterns of deficits, then, syndrome-based research does not provide a defensible basis for empirical or theoretical generalizations.

THE MEDICAL ANALOGY

Zurif et al. (1989) argued that the methods of the syndrome-based approach to acquired cognitive impairments coincide with what a medical researcher does when he/she identifies a pathological syndrome and hypothesizes a common origin for the symptoms that outwardly define the syndrome. They suggested that the success of the medical method of research should suffice to support the analogous approach to acquired cognitive deficits. To the criticism that this is faulty reasoning by analogy (Caramazza & Badecker, 1989), ZSF reply that the argument merely describes methods that are common to observational sciences. They identify the goal of observational sciences as determining "whether inferences from observations are reasonable . . . and whether observations are re-

³ In order to sustain the thesis that the two associated symptoms have a common underlying cause, as opposed to the mere association of two independent deficits, one must at the very least be able to articulate an explicit hypothesis about the mechanisms which when damaged would result in the specific forms of associated deficits. Otherwise the claim of a common underlying disorder is vacuous—it would amount to no more than a restatement of the observed result.

liable,” stating that this brand of science is “not concerned with whether a classification is valid” (ZSF, 1991).⁴

As we have already indicated, we are in agreement with ZSF on one interpretation of this point: It is possible to apply scientific means in order to determine what one will “always,” often, or seldom observe in the performance of subjects who have been grouped on the basis of one or more behavioral measures. However, we suggest that one should take pains to distinguish this particular reading from some of the more general claims that one might think to follow from their observations. Zurif et al. (1989) and ZSF are not defending research that has such modest goals. The examples they cite clearly indicate their belief that syndrome-based studies can be used to inform us about the cognitive mechanisms underlying normal and (various patterns of) impaired performance. Unfortunately, there is nothing in Zurif et al.’s (1989) exposition of the medical analogy that explains how the syndrome-based approach can overcome the impediments we have identified in attempting to draw meaningful inferences about normal cognition from group performance of brain-damaged subjects. The fact that syndrome-based methods can be used with some success to learn *some* things about the members of the clinical group (e.g., the probability of a cooccurring impairment) does not serve as an argument that they can be used to reveal *other* kinds of thing that one might want to know about the group members (e.g., the nature of the cognitive mechanisms that have been damaged in these patients). If there is some feature of the analogy that they have failed to point out that could be offered as a response to the problem that the behavioral signs used for clinical categorizations of aphasias do not establish homogeneity at the level of underlying cognitive deficit, then they should clarify what that feature is. Otherwise, we must conclude that the argument fails to achieve the generality that Zurif et al. (1989) and ZSF wish to ascribe to it; that is, it remains argument by faulty analogy.

THE EXAMPLE FROM LEXICAL PROCESSING

Zurif et al. (1989) cited the results from a study of abnormal lexical processing (Swinney, Zurif, & Nicol, 1989) as a prime example of the sort of findings based on syndrome-inspired research that can provide insights into the cognitive disruptions underlying patients’ deficits and ultimately into the nature of language processing mechanisms. In the view of Zurif et al. (1989) and ZSF, this study demonstrated preserved encapsulation of form-based lexical access in a clinical group. The supposed

⁴ It is not entirely clear to us what ZSF mean when they claim that in observational sciences (such as astronomy) one is not concerned with whether the classifications one uses are valid. Surely, no astronomer would accept a classification scheme that failed to distinguish the northern star (Venus) from real stars!

moral for the single-patient versus syndrome-based research debate is that syndrome-based research does lead to meaningful conclusions about the nature of normal language mechanisms as well as about hypotheses concerning the dysfunction of these mechanisms under conditions of brain damage. It is unfortunate that ZSF should insist on this example since the conclusions reached by Swinney et al. (1989), and repeated by Zurif et al. (1989) and ZSF, are *not* supported by the results that were reported in the original study. In Caramazza and Badecker (1989) we pointed out this limitation in rather general terms. Since ZSF return to this matter, it is useful to consider in detail the Swinney et al. study and the claims it is supposed to support. We will show that ZSF have failed to make the case for their position on syndrome-based research even on their own terms. Contrary to what has been claimed in Zurif et al. (1989) and ZSF, the results from Swinney et al. (1989) do not remotely motivate the claim that brain damage has slowed, but otherwise left intact, the modular component of lexical access in so-called Broca's aphasics.

The modularity hypothesis of lexical access. Experimental evidence with the cross-modal priming paradigm (Swinney, 1979; Tannenhaus et al., 1979) has been interpreted as providing support for the hypothesis that initial access of the meanings of a polysemous word is obligatorily exhaustive—all meanings are accessed, access depends only on information about lexical form, and it is unaffected by context—but that subsequently context determines the final interpretation assigned to the word. In this experimental paradigm, subjects hear sentences, some of which contain ambiguous words (e.g., The gardener was responsible for watering every plant* on the enormous estate). The subjects are instructed to listen and understand the sentences. They are also instructed that they are required to perform a second task which involves deciding whether a visually presented string of letters that appears at some point during the presentation of a sentence forms a word. The experimental words used in this secondary task appear either immediately after the offset of the ambiguous word or after a short delay (200–1000 msec after the point indicated by an asterisk in the example sentence above). Typically, half of the target words are related to the dominant meaning of the ambiguous word (e.g., prime: PLANT—target: TREE); the other half are related to the nondominant meaning (e.g., prime: PLANT—target: FACTORY). Response times are the dependent measure of interest.

The results of such experiments (e.g., Swinney, 1979; Tannenhaus et al., 1979) have shown that at short SOAs (<200 msec) both meanings of the ambiguous word are primed (e.g., both the TREE and the FACTORY senses of PLANT), but that at longer SOAs (>200 msec) only the context-appropriate sense of an ambiguous word is primed (e.g., only the TREE sense of PLANT in the example given above). These results have been interpreted as support for the modularity hypothesis (Fodor, 1983) of

lexical access. On this hypothesis, initial access of the meanings of a polysemous word is obligatorily exhaustive. However, context does play a crucial role in determining the interpretation of a word but only after all senses of a word have been accessed on the basis of lexical form alone. Thus, the fact that at short SOAs all meanings of a word are primed independent of context is taken as support for the hypothesis that lexical access is “cognitively impenetrable”; the fact that at longer SOAs only the contextually appropriate meaning is primed is interpreted as support for the hypothesis that the effect of context on lexical interpretation occurs not at the point of lexical access but in the subsequent selection of one of the obligatorily accessed meanings. Independently of whether one agrees with this interpretation of the results, it is important to note that it depends on two pieces of evidence: (1) all senses of a polysemous word are primed at short SOAs, and (2) only the contextually appropriate sense is primed at longer SOAs.⁵ Both pieces of evidence are needed to support the modularity hypothesis of lexical access.

The Swinney et al. (1989) experiment. Swinney et al. reported an experiment with three groups of subjects: a group of healthy controls, a group of agrammatic Broca’s aphasics, and a group of Wernicke’s aphasics. The experimental procedure was similar to the one described above except that only *one* SOA condition was used—visually presented target words appeared immediately after the offset of the ambiguous word. The relevant results were as follows: Both the healthy controls and the Wernicke’s aphasics showed priming for both senses of the target word independent of context; the Broca’s aphasics, however, showed priming only for the dominant sense of the target word independent of context. Furthermore, the response latencies for the Broca’s and Wernicke’s aphasics were very slow (on the order of 1500 msec compared to the normal subjects’ 900-msec response latencies). Swinney et al. interpreted these results as indicating that the lexical access process in both Wernicke’s and Broca’s aphasics has retained its modular character—i.e., it is cognitively impenetrable—except that it functions abnormally slow in the case of the Broca’s aphasics. They go on to claim that a consequence of this slower but modular functioning of the lexical access process in Broca’s aphasics is that only the dominant sense gets accessed in the time frame within which the lexical decision response is made. However, it should be apparent that the conclusions reached by Swinney et al., and repeated by Zurif et al. (1989) and ZSF in support of syndrome-based research, are unrelated to the results they report.

⁵ This condition is necessary in order to establish that priming is determined by the context-appropriate meaning of the (prime) word that is available to a subject at some point in the course of processing a sentence.

In order to even contemplate the possibility that Broca's aphasics have retained a modular lexical access process it would have to be shown, at the very least, that these patients obligatorily access all senses of a polysemous word at (relatively) short SOAs. Swinney et al. have not shown this. To the contrary, their results show that the Broca's aphasics only access the dominant sense of a polysemous word. The only conclusion that may be sustained by this result is that lexical access in Broca's aphasics is *not* obligatorily exhaustive. However, Swinney et al., Zurif et al., and ZSF conclude that lexical access in Broca's aphasics is, in fact, obligatorily exhaustive but abnormally slow. In support of this possibility they cite the fact that these patients' response times were abnormally slow. They conjectured from this result that lexical access in Broca's aphasics is so slow that at the SOA used in their experiment the patients had only had time to compute one of the two meanings—the more frequent one—but that the secondary meaning would obligatorily be computed in due time. However, the fact that Broca's aphasics had slow response times cannot on its own be used to support the contention that they obligatorily access all meanings of a word. That slow response times cannot on their own be taken as an indication that lexical access would be obligatorily exhaustive is indicated by the fact that Wernicke's aphasics had longer response times than Broca's aphasics but they nonetheless showed priming for both senses of the ambiguous target words. Thus, slow response times are not a reliable indicator of obligatorily exhaustive lexical access.

If one were to ignore this problem and grant for the sake of argument that the Broca's aphasics might in fact obligatorily access all meanings of a word but more slowly than normal subjects, Swinney et al. and ZSF would have had to have provided positive evidence for it, and not simply assume that obligatorily exhaustive access would have obtained if the proper experimental conditions—testing for priming at longer SOAs than those used in their experiment—had been included in the experiment. In the context of the present discussion this would require showing that at longer SOAs than those tested in the Swinney et al. experiment both senses of the ambiguous target words would be primed independent of context. This experimental condition was not tested, nor was any other evidence obtained for context-independent priming for the target's secondary meaning. Consequently, we must conclude that the claim in Swinney et al., Zurif et al., and ZSF concerning slowed but modular lexical access in Broca's aphasics is only speculation.

The Swinney et al. story faces other difficulties. In order to maintain that lexical access is obligatorily exhaustive independent of context, it would have to be shown that context *can* affect the interpretation of words in the lexical decision task. Thus, for all one knows, it is possible that Broca's and Wernicke's aphasics would *not* show an effect of context on

lexical priming. This expectation is not implausible given that such patients are supposed to have comprehension difficulties.⁶ In the original experiments with normal subjects (Swinney, 1979; Tannenhaus et al., 1979) the effectiveness of context was demonstrated by including an experimental condition in which target words were primed at relatively long SOAs, and showing that in this condition only the contextually appropriate sense was primed. However, this condition was not included in the Swinney et al. experiment. Consequently, we have no evidence that Wernicke's and Broca's aphasics show the pattern of priming effects that would be expected on the basis of the modularity hypothesis of lexical access (i.e., only priming for the contextually appropriate sense at long SOAs). In the absence of such positive evidence no conclusion about the possible effects of context on lexical access in Broca's and Wernicke's aphasics is possible.

Since Swinney et al. (1989) did not provide positive evidence for context effects on lexical priming in their experiment, ZSF offer a curious argument in order to motivate their claim that sentence context would affect the interpretation of word meanings in Broca's aphasics. Their argument is as follows: "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 [sic] effect and that . . . the patients' abnormality is to be stated in terms of a delay in providing lexical information to the sentence processing constituents" (ZSF, 1991). There are two parts to this argument. Part one asserts that the agrammatic Broca's aphasics who were tested in the Swinney et al. (1989) experiment did in fact understand the sentences that were used to disambiguate the polysemous prime words. The evidence cited in support of this claim is not particularly convincing: apparently patients were able to provide adequate paraphrases of the context sentences when probed *twice* (or *four* times, depending on which paper one reads, Swinney et al. or ZSF, respectively) during the experiment—that is, on 2 or 4% of trials. It is unclear to us that this procedure adequately assesses the patients' ability to comprehend the context sentences (especially in light of the fact that the patients were supposed to have shown comprehension difficulties in clinical evaluation for classification as Broca's or Wernicke's aphasics). Part two of the argument is to build on this assumption by adding the further assumption that since patients were supposed to have understood the context sentences, these would eventually exert an effect on lexical priming. Note that this "eventual effect" is merely assumed—

⁶ Furthermore, it is entirely possible that one consequence of brain damage (in some patients, including some who are classified as Broca's or Wernicke's aphasics) might be to damage those mechanisms that exploit context in order to restrict the meaning of ambiguous words.

it was not shown experimentally. From these *assumed* effects ZSF draw the conclusion that the Broca's aphasics' abnormality in the priming study is to "be stated in terms of a delay in providing lexical information to the sentence processing constituents." Thus, it would seem that the "empirical" basis for ZSF's thesis about the nature of the lexical processing deficit in agrammatic Broca's aphasics is provided by a series of *assumed* effects. Consequently, this thesis should be given as much consideration as any other unsupported hypothesis.

In short, then, we find little basis for ZSF's position that Swinney et al.'s (1989) research can inform hypotheses about the structure of normal language processing mechanisms and how these mechanisms may be damaged in conditions of brain pathology. Thus, we see no basis for ZSF's claim that ". . . the Swinney et al. (1989) study has provided an analysis of some relevance to current theorizing concerning modularity" (ZSF, 1991). Swinney et al. (1989) did not show that so-called agrammatic Broca's aphasics obligatorily access all meanings of polysemous words; that is, no evidence was provided for the hypothesis that Broca's aphasics show facilitation, regardless of context, for the less frequent reading of an ambiguous target (it was only conjectured in the original study); nor did they show selective priming for the contextually appropriate meaning at long SOAs (it, too, was only conjectured in the original study). Hence, the priming results that *were* obtained do not provide the kind of evidence that Zurif et al. (1989) and ZSF ascribe to it. Once again, then, we must consider Swinney et al.'s, Zurif et al.'s, and ZSF's claims in regard to the language processing abilities of so-called Broca's aphasics to be unsupported by fact.

On saving appearances. The problems we have raised with the Swinney et al. (1989) study are independent of the single-patient vs. patient-group methodology issue. Still, if the inferences do not go through for any of the individual subjects in the patient group, it would be unreasonable to suggest that the logic of the argument could be improved by pooling their data. Thus, contrary to ZSF's assertion, the Swinney et al. (1989) study does not serve as an existence proof of usefulness of patient-group research for informing theories of normal language processing. Nonetheless, if one were to set aside the problems with this study, one might wish, for the sake of returning to the original purpose of our discussion, to consider the other points raised by these authors with regard to the punitive value of syndrome-based research. Several issues will be considered here.

One issue concerns the role of evidence other than that provided by Swinney et al. (1989) regarding the functioning of the lexical access mechanism in so-called Broca's aphasics. One criticism leveled in Caramazza and Badecker (1989) against Zurif et al.'s (1989) supposed demonstration that Swinney et al.'s study could be taken as an existence proof of the value of syndrome-based research was that this study did not have any

bearing on the problem of how to overcome the problem of patient heterogeneity in syndrome-based research. The question we asked concerned the interpretation of a patient's results if he/she failed to exhibit all the details of the performance pattern hypothesized for a particular syndrome type. Caramazza and Badecker (1989) noted that this is not an idle question in that putative members of the same clinical category invoked by Zurif et al. (1989) failed to exhibit any priming effects for semantically related items in other lexical decision tasks (Milberg & Blumstein, 1981; Milberg, Blumstein, & Dworetzky, 1987). This result could be interpreted as indicating that, for at least some Broca's aphasics, semantic context does not seem to be an effective prime in lexical decision, thus undermining Swinney et al.'s claim that semantic context would affect priming at long SOAs. ZSF suggest that this evidence of variation can be ignored entirely because the primes in the Swinney et al. (1989) study occurred in sentence contexts, whereas the primes in the Milberg and Blumstein (1981) and Milberg et al. (1987) studies did not. This dismissal of seemingly pertinent evidence is presumably based on the belief that lexical access operates differently in sentence and single-word contexts. The latter belief would be justified if one could assume that lexical access procedures can be modified by the context in which they are applied. However, adoption of such a view would leave a conspicuous gap in ZSF's argument: how can access mechanisms that are postulated to be isolated from any information other than the formal characteristics of a lexical stimulus (the modularity thesis defended by ZSF) at the same time be affected by the type of context that the word form occurs in?⁷ That is, it seems contradictory to assume, on the one hand, that lexical access is encapsulated and not subject to context effects and, on the other hand, that context does determine the way lexical access operates. Thus, apart from unsubstantiated conjecture, we know of no basis for discounting the evidence for patient variation revealed by comparison of the results reported by Swinney et al. (1989) with those reported in the studies by Milberg and Blumstein (1981) and Milberg et al. (1987).

In Caramazza and Badecker (1989) we raised the objection that Zurif et al. (1989) had failed to explain their reported dissociation between, on the one hand, the presumably similar comprehension failure in Wernicke's and Broca's aphasics and, on the other hand, the different patterns of cross-modal priming in these two patient types. We argued that since both

⁷ If the reasoning used by ZSF to explain the "recalcitrant" observation in the Milberg and Blumstein (1981) and Milberg et al. (1987) represents an example of what they call "saving appearances," then it is surprising that they should both wish to dismiss "recalcitrant" observations obtained outside the context of sentence processing (those cited above) as relevant to their claim about the modularity of the lexical access mechanism and, at the same time, consider "non-recalcitrant" single-word priming studies when these are needed to make their case (see footnote 5 in ZSF).

patient types presented with supposedly similar comprehension difficulties, it was not obvious what reasoning led these authors to propose a causal link between the lexical access performance in one group (Broca's aphasics) but not the other (Wernicke's aphasics). ZSF respond to this objection by saying that "... the point of the Swinney et al. (1989) finding in this argument [the one concerning the link between comprehension failure and slowed lexical access in Broca's aphasics] 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" (ZSF, 1991). Now, on one reading of this claim it is straightforwardly unproblematic. That is, if the claim were to be simply that the comprehension failure in different patients may *arbitrarily* be associated with different types of lexical access deficits or, for that matter, different types of calculation impairments, motor impairments, and so forth, then we see no difficulty with their conclusion. Such a claim would correspond to a simple affirmation of observed associations of symptoms. But, apparently, this is not the intended sense of the claim being put forth by ZSF. It seems, instead, that they are arguing for a causal link between lexical decision and comprehension performance patterns in Broca's aphasics, but *not* for a similarly causal link between lexical decision and comprehension performance patterns in Wernicke's aphasics.

Leaving aside the fact that, as argued above, no meaningful conclusion is possible about lexical access mechanisms on the basis of the results reported by Swinney et al., it remains unclear to us why a causal link is assumed in one case (Broca's aphasia) and not in the other (Wernicke's aphasia). It was in reaction to this seemingly arbitrary selection of which putative facts need explaining that our objection was raised. Furthermore, our objection was intended to highlight, more generally, this seeming arbitrariness in deciding which constellation of performance features are considered to be theoretically important and which are considered to be theoretically irrelevant. Thus, given that agrammatic production is a complex disorder consisting of dissociable performance features, the fact that not all so-called agrammatic Broca's aphasics show comprehension difficulties, and the fact that for those that do there appear to be different patterns of failure (see Berndt, 1991, for review), we may ask: Which specific features of the performance of so-called agrammatic Broca's aphasics are to be explained by a deficit in lexical access? Is the omission of verbs one such feature? Is the omission/substitution of grammatical morphemes one such feature? Are noun phrase reversal errors to be explained by a lexical access deficit? And, if not, why? Which forms of sentence comprehension failure are to be explicated by appeal to lexical access deficit? Is the relative sparing of grammatically judgment perfor-

mance in many such patients explicable by appeal to the lexical access deficit? And so forth and so on. Since ZSF do not even attempt to address these "recalcitrant" observations, the asserted link between lexical access performance and the features of so-called agrammatic Broca's aphasia is of dubious value.⁸

CONCLUSION

In this response to ZSF's criticism of Caramazza and Badecker (1989), we have argued that they have not provided convincing arguments in favor of syndrome-based, patient-group methodology. We have also argued that they have failed to show that the specific example intended to illustrate the value of syndrome-based research—the Swinney et al. (1989) study—does in fact serve this purpose. Contrary to ZSF's contention, we have not attempted to erect insurmountable barriers between cognitive neuropsychology and other areas of the cognitive sciences and the neurosciences. We have only insisted on a point that ought to be quite uncontroversial: methodology must be at the service of theory and must be appropriate to the specific domain of inquiry. Our analysis (see, e.g., Caramazza, 1986) has led us to argue that the use of impaired performance to constrain cognitive theory can only be meaningful in the setting of single-patient methodology. The types of considerations we have brought to bear on this issue are not different from those used in other areas of the cognitive sciences and the neurosciences to motivate experimental practice.

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⁸ Although in Caramazza and Badecker we also raised the objection that Zurif et al. had failed to link their claims about lexical access deficits in Broca's aphasics with a different account of these patients' language disorder which they also consider in their paper (that of Grodzinsky, 1986), there is no point in pursuing this issue further since not only is the Swinney et al. story devoid of any empirical support but so is Grodzinsky's claim (see Badecker, Nathan & Caramazza, 1991; Caramazza & Miceli, 1990; Martin, Wetzel, Blossom-Stach, & Feher, 1989).

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