Patient Classification in Neuropsychological Research

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In a number of papers we have been concerned with the type of inferences that are legitimate in "experiments of nature" where the experimenter does not and cannot control the modifications to the cognitive system that are introduced by brain damage. We have argued that in such cases very restrictive conditions must be met in order to be able to draw valid inferences about the structure of normal cognitive mechanisms. Two consequences of these conditions are (1) patient classification into syndrome types (e.g., phonological dysgraphia, agrammatism, and so forth) can play no useful role in research concerned with issues about the structure of normal cognitive functioning or its dissolution under conditions of brain damage; and (2) only single-patient studies allow valid inferences about the structure of cognitive mechanisms from the analysis of impaired performance. Zurif, Gardner, and Brownell (1989, Brain and Cognition, 10, 237–255) have taken exception to our conclusions and propose to show the limitations of our arguments. In this paper we respond to their criticisms. © 1989 Academic Press, Inc.

Cognitive neuropsychological research is motivated by the assumption that the analysis of impaired performance in brain-damaged patients provides a window into the structure of normal cognitive systems—that is, by the assumption that the results generated by this research activity will provide insights into the structure of cognitive mechanisms—and that it will ultimately provide important constraints for theories of the neural basis for cognitive functioning.

Along with many others, we share in this conviction. However, it must be emphasized that it is not immediately obvious that a research enterprise based on the analysis of pathological states will in fact generate...
the promised, nontrivial empirical constraints for theories of normal cognitive processing. Whether this research enterprise will live up to its promise is not an issue that can be decided merely by logic and reflection. It is strictly an empirical matter: We will have to see how useful neuropsychological data turn out to be in constraining detailed claims about the nature of cognitive mechanisms. But, while we cannot decide whether, in the long run, cognitive neuropsychology will be scientifically productive, we can evaluate the logic of its practice. In other words, we can evaluate whether, independently of its ultimate usefulness, the practice of cognitive neuropsychology is methodologically coherent. For, unless we can show that the inferences about normal cognitive mechanisms that may be drawn from the analysis of cognitive deficits are, at least on the basis of our current understanding, free of basic fallacies, then there can be no reason to expect that the enterprise could be scientifically productive in the first place. If there are reasons for believing that certain of its practices cannot lead to valid inferences, then we would be well advised to exclude them from the armory of the cognitive neuropsychologist.

In a number of papers we have tried to deal with the issue of the logic of inferences in cognitive neuropsychology. Specifically, we have been concerned with the type of inferences that are legitimate in “experiments of nature” where the experimenter does not and cannot control the modifications to the cognitive system that are introduced by brain damage. The results of these analyses have been quite controversial and have even engendered hostility. We have argued that there are very restrictive conditions that must be met in order to be able to draw valid inferences about the structure of normal cognitive mechanisms from the analysis of impaired performance consequent to brain damage. Two practical consequences of these conditions are that (1) patient classification into syndrome types (e.g., Wernicke’s aphasia, phonological dysgraphia, semantic access dyslexia, optic aphasia, agrammatism, and so forth) plays no useful role (and is harmful) in research concerned with issues about the structure of normal cognitive functioning or its dissolution under conditions of brain damage; and (2) only single-patient studies allow valid inferences about the structure of cognitive mechanisms from the analysis of impaired performance.1 The detailed arguments on which these conclusions depend have been presented in various publications.

1 Note that these are the consequences of having to satisfy a set of conditions which minimally ensure that valid inferences for normal cognition could be drawn from pathological states. These practical consequences have gotten all the attention at the expense of the really interesting issues about the assumptions we must make to be able to draw any inferences at all about normal cognition from pathological states. Of special interest is the assumption of transparency (Caramazza, 1984)—the assumption on which the whole enterprise is based but which has not received serious scrutiny.
NOTES AND DISCUSSION

(Badecker & Caramazza, 1985, 1986; Caramazza, 1984, 1986; Caramazza & McCloskey, 1988; McCloskey and Caramazza, 1988) and will only be very briefly summarized here.

ONCE MORE ON OUR POSITION REGARDING CLINICAL CATEGORIES

As already noted we have argued that studies which seek to identify the characteristics of the normal cognitive processing system cannot legitimately use clinical categories as the basis for their research. The reason for this contention is relatively simple.

We begin with the (uncontested) assumption that brain damage results in a transformation of the normal cognitive system. The nature of the transformation is a function of the intrinsic structure of the cognitive system and the type of damage the system has undergone. If we assume a constant (true) functional architecture across normal individuals (again uncontested—our assumption of universality; Caramazza, 1984, 1986), then the only variable to worry about in cases of brain damage is the transformation of the hypothesized cognitive system. However, the specific form of functional damage to the cognitive system—the functional lesion—in any individual brain-damaged subject is unknown. This fact sets very specific constraints on the range of legitimate inferences one may draw about normal cognition from the study of brain-damaged subjects.

Consider the usual situation we face in trying, through the study of brain-damaged subjects, to determine empirically which of two theories, T1 or T2, is to be preferred. How do we make such evaluations? We can conclude that T1 is to be preferred to T2 if it is possible to specify a specific transformation of T1 such that the pattern of observed performance in a patient may be derived from the “functionally lesioned” T1, but it is not possible to specify a transformation of T2 that would allow us to explain the patient’s impaired performance. Note that this situation is the standard one in evaluating alternative hypotheses in any scientific enterprise—we choose the theory which, everything else being equal, is maximally consistent with experimental evidence. Note further, however, that this situation diverges in a crucial respect from other experimental sciences.

In typical experimental sciences the experimenter performs some manipulation and then observes the effects of the manipulation. The only unknown in the latter situation is the theory. The experimenter knows

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2 The point here is that changes of a system can only result in deformations of the specific mechanisms and knowledge that characterize the premorbid normal cognitive system. To illustrate this point through a *reductio ad absurdum*, brain damage in a monolingual English speaker cannot result in a fluent speaker of French: Damage to this language system can only result in different deformations of the processing system for English.
both the results and the experimental manipulations that led to the obtained results. In research with brain-damaged subjects we have a radically different situation. We may think of brain-damaged subjects as experiments of nature in which the specific experimental manipulations in these experiments—the functional lesions to the cognitive system—are unknown. In order to interpret the results from these experiments we must uncover the experimental conditions (read transformations of cognitive system) that apply in each experiment of nature. In other words, unlike the typical situation in other experimental sciences, our task is complicated by the fact that we do not know the experimental conditions that Nature has set in any particular brain-damaged subject. Instead, we must infer these conditions from the patient's performance.

The situation we have described differs in a fundamental way from the typical experimental procedure in other sciences. Unlike these other sciences we do not start with some theory, make predictions, set up an experiment, get the results, and then on the basis of the results determine whether the theory's predictions were confirmed. In cognitive neuropsychology, a theory is preferred to another theory if on the basis of a patient's performance we can hypothesize a functional lesion to one theory, but not to an alternative theory, such that the functionally lesioned theory can generate the observed performance. There are important implications that follow from the above considerations.

One straightforward implication is that the basic unit of analysis in cognitive neuropsychology must be the individual patient. This follows directly from the observation that brain-damaged subjects are experiments of nature in which we do not know the experimental conditions that were manipulated, but must infer these from the individual patient's performance. In other words, the starting premise is that brain-damaged patients have unknown transformations to the cognitive system which we will attempt to identify through an investigation of individual patients' performances. This conclusion follows logically from the uncontested assumptions discussed above.

A related implication concerns the question of the amount and type of evidence that is deemed necessary for determining the locus (or loci) of a functional lesion(s) in a theory of the structure of the cognitive system. From the preceding it is obvious that there is no stock answer to this question, other than the general statement that the evidence which will be relevant for the latter purpose is a function of the theories one is willing to entertain about the structure of the cognitive system—a trivially true claim. After all, the notion of functional lesion is not independent of particular theories of the structure of cognitive systems: Functional lesions are statements about the possible transformations that a theory of normal cognitive processing may undergo, and as such they cannot be specified independently of the theories which may embody
the hypothesized transformations. It follows logically from the preceding that the a priori classification of patients into clinical categories such as agrammatic aphasic, surface dyslexic, and so forth can only be theoretically arbitrary. The only nonarbitrary classification of patients that is possible is a posteriori, that is, on the basis of the theoretically relevant performance which allows the identification of a functional lesion in a cognitive system. But the latter is equivalent to saying 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.

A methodological corollary of the above considerations is that no valid inferences may be supported when the data tendered for explanation are the average performance of the clinical group on some set of tasks. One reason for this prohibition is quite simple. In order to draw inferences about the normal system from averaged (or grouped) data, one must assume the homogeneity of the group. Such homogeneity cannot be established merely on the basis of an arbitrary set of behavioral features. If it is to be meaningful, it must also be established at the level of the functional lesion to the processing mechanisms that the relevant behaviors are thought to reflect. However, we have seen that a functional lesion can only be inferred from all the theoretically relevant performances of a patient. We must conclude, therefore, that patient-group research is methodologically valid only in the trivial case where it has been established that patients have identical functional lesions (to the limit permitted by current theory), in other words, after a single-patient by single-patient research project. In every other case patient-group research cannot allow meaningful interpretation.

If these arguments are correct, and to our knowledge no one has challenged their correctness, then the methodological conclusions we

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3 We should emphasize that by single-patient methodology we do not mean that we should restrict our research effort to a single patient. It ought to go without saying that our theories, whether restricted to cognitive processing or concerning the relationship between cognitive mechanisms and brain structures, must be motivated by evidence from more than a single patient (or a single experiment). For example, if we had some hypothesis about the brain structures associated with a specific cognitive mechanism, we might wish to evaluate this hypothesis by determining whether lesions to the brain regions in question are associated with functional lesions to the hypothesized cognitive mechanism. In this case, we would have to study various patients with functional lesions to the cognitive mechanism in question and determine whether these patients have damage to just those brain regions hypothesized by the theory. Note that the type of evidence that is relevant here concerns a series of single-patient studies. We should not confuse a series of single-patient studies with patient-group methodology. The former uses the single-patient methodology to infer functional lesions to individual patients, and the latter conflates the performance of individual patients into an average for a preestablished group of patients.
have reached must be accepted, no matter how unpalatable they may be. We are fully aware that acceptance of these conclusions would undermine that tradition in neuropsychology which relies on patient-group research methodology: research carried out in this tradition would not be able to serve as the basis for theoretically defensible claims about cognitive mechanisms or their neurological bases. For this reason, we have encouraged broad discussion of these problems, inviting principled criticism of our arguments and conclusions (see, for example, the special issue of *Cognitive Neuropsychology* (1988) devoted to problems of theory and method in cognitive neuropsychology).

Zurif, Gardner, and Brownell (1989, hereafter ZGB) propose to show the limitations of our arguments and conclusions. Their paper consists of two parts. In part one, after a brief summary of our position, they offer a number of observations which are supposed to undermine our arguments for the claim that only single-patient studies allow theoretically meaningful conclusions about cognitive mechanisms. In part two, they discuss the specific case of agrammatism with the objective of showing that, contrary to our position, syndrome-based research can be productive and theoretically coherent. Our response to ZGB’s criticism is closely patterned on the organization of their paper. We will discuss each of the points they raise in the order in which it is made.

**ON REASONING-BY-ANALOGY**

ZGB introduce their criticism by noting that medical scientists regularly and successfully use syndrome-based reasoning but that we have deemed it unacceptable when used by the neuropsychologist. They seem to be saying “How can a form of reasoning be valid in one area of ‘knowledge’ but invalid in another?” In fact, they go on to state that neuropsychological syndromes are no more undermined by the kinds of objections we have raised than would be a disease entity such as Legionnaire’s disease when confronted by analogous objections (see p. 246).

We are unsure about the intended force of the analogy between Legionnaire’s disease and neuropsychological syndromes to which ZGB draw our attention. It could be that the analogy was merely intended to have a rhetorical function: to predispose the unwary into thinking that “good medical practice” cannot be “bad neuropsychology.” Alternatively, ZGB may have intended the analogy to serve as a form of reasoning: If A is good, and B is similar to A, then B is good. This is a notoriously poor form of reasoning.

Surely, ZGB would not attempt to take advantage of the unwary, nor would they engage in sloppy *reasoning-by-analogy*. What, then, are we to make of the analogy? Since under no construal could it be taken to constitute an argument against our position, we will treat it as a stylistic
device intended to set the stage for the promised "particular scrutiny" to which ZGB would subject our position.

ON FRAMING THE QUESTIONS FOR DISCUSSION

Before undertaking the promised "particular scrutiny" of our position, ZGB provide an outline of the questions they will address in their paper. In this context they state that "... [they] feel that the focus on group versus single case studies is in fact the wrong one, and that a more profitable question is exactly how best to carry out theoretically directed research for the purpose of understanding normal cognition" (p. 239).

We wholeheartedly agree with ZGB on this point. In fact, we find discussions of method to be an unnecessary distraction from empirical and theoretical research. Unfortunately, these cannot be divorced from the methods we use to collect and interpret observations. Consequently, issues of methodology, including the issue of single-patient versus patient-group studies, will have to form part of any serious evaluation of a theoretical position or empirical phenomenon; and these evaluations will necessarily involve claims about those observations that can be taken as relevant to a particular theoretical domain. If there is disagreement about which observations will be considered relevant or methodologically untainted, there can hardly be agreement on which theoretical account is to be favored. In a sense, we are always making methodological evaluations in scientific activity. The only difference between the standard, methodological evaluation in everyday scientific activity and the explicit and formal claims we have made is that in the latter case the evaluation does not merely concern a local empirical fact but a whole class of empirical observations, and the practice from which they are derived. We do not see how one can be interested in dealing with issues of "... how best to carry out theoretically directed research ..." without simultaneously being concerned with the issue of what might constitute relevant evidence for a theory and, in the present context, with the issue of whether patient-group studies can, in principle, provide that evidence.4

ON PUTATIVE ARGUMENTS AGAINST THE SINGLE-CASE-ONLY POSITION

In a very brief section titled "Averaging Performance," ZGB present their first argument against what they call the single-case-only position.

4 We should note, once again, that in our various papers on matters of theory and methodology, our interest has not strictly been with the issue of single-patient versus patient-group studies. Rather, this latter issue emerged as a consequence of the more important question of what theoretical conclusions are possible from the analysis of impaired performance consequent to brain damage.
On Averaging Performance

ZGB begin by noting about our position that "... there is nothing very compelling about [these] arguments" (p. 242). They go on to assert that there is nothing the matter with averaging performance in patient-group studies that a little knowledge of statistics cannot quickly cure—things such as considering the distribution of scores. We are at a loss as to how to respond to these assertions. In our papers we have gone to great lengths to present as clearly as we could the arguments for our conclusions. ZGB do not discuss these arguments, nor do they show which part, if any, of our arguments is inadequate, contradictory, fallacious, or simply wrong. Instead, they baldly assert that our arguments are simply not compelling. Once again, as a rhetorical strategy unsupported assertions may have their value. In science, however, this practice cannot take the place of reasoned discussion and argument.

In this section, ZGB also write that "McCloskey and Caramazza (p. 596) seem to imply that using groups requires that a researcher accept the equivalence of data collected during the selection and experimental phases of a study. To us, the asymmetry in the two classes of data ought to be uncontroversial" (p. 242). What ZGB present as a mere "seem to imply" is in fact the heart of a detailed set of arguments which show that a priori classification cannot play a meaningful role in cognitive neuropsychological research. More specifically, we have shown that the criterion of homogeneity of functional lesion can only be "satisfied" a posteriori—that is, by considering all the theoretically significant facts, including what ZGB call independent performance. The consequences of this line of reasoning have been spelled out in great detail in our papers (see Caramazza (1986; pp. 51–59); Caramazza & McCloskey (1988; pp. 523–525); McCloskey & Caramazza (1988; pp. 593–595)). If ZGB find our explicitly stated arguments wanting, they could point out the flaws. The flat assertion that "... the asymmetry in the two classes of data ought to be uncontroversial" cannot be taken as an argument.

On Replication of Findings

ZGB's second argument in support of patient-group studies (or against single-patient studies) concerns the problem of replication of results. They introduce this section by noting that we have argued that "... sin-

5 It is unclear what ZGB mean when they say that our arguments are not compelling. They would seem to leave open the possibility that our arguments are correct, but not sufficiently well presented to make a compelling case for our position. If this is what they meant, then they should agree with our conclusions. Obviously, however, they disagree with our conclusions. So, we must infer that by saying that our arguments are not compelling they mean that these fail to make the case we intended. It is too bad that they have not said how the arguments fail. This would have been the best way to undermine our position.
gle-case methodology does not permit pure replications in a controlled fashion" (p. 242). They note that we have also argued that this situation, while not entirely to our liking, is something we are going to have to learn to live with. We justify our calm in the face of the problem of being unable to have "strict replicability" in cognitive neuropsychology by suggesting that discrepant results will not, in the long run, create irresolvable difficulties. This contention is based on the view that, since in evaluating theoretical claims the weight received by any particular observation depends on the overall context of available evidence, a discrepant result will tend to diverge from other results and thereby diminish its "evidential weight" (Caramazza, 1986).

ZGB approve of this line of reasoning, and they would like us to believe that it can be applied to the problem of patient-group studies. Regrettably, they do not tell us how. We have read and reread the section where ZGB discuss this matter, but we still do not understand how they made the connection. Perhaps it is best to quote them in full.

While agreeing with this general point about converging results, we suggest that it can apply with equal force to dispersion within a group. Assume that we have a hypothesis detailed enough to allow us to set up the conditions for its experimental evaluation. These initial conditions are related to the hypothesis by a series of steps: uncertain as to whether just the relevant factors have been abstracted for analysis, and conversely, uncertain as to whether those left out will not intrude upon the analysis.

To our minds—and this is critical—the selection of subjects from some putatively relevant target population figures unexceptionably in this set of intuitions. So, although we do not have full control over the prior specification of functional lesions, it seems to us that a hypothesis stated within a coherent theoretical framework ought to be able to help us out on this matter, no less so than it helps us to devise other aspects of a proper experiment. And, just as sequences of experiments help us to home in on a theoretical concern, so too ought they to yield a better informed appreciation of patient characteristics that bear on this concern. Also, in most cases, group results will be less prone to idiosyncratic results than will a single case, so that, over the long run, fewer discrepant empirical findings should emerge from analysis of group data. (p. 243)

There are several problems with the paragraphs we have quoted. How does having a hypothesis help with the problem of not having "... full control over the prior specification of functional lesions ..."? What do they mean by this? How could having a hypothesis help if it turned out that the patients included in the group had damage to different cognitive mechanisms? This is all very confusing.

Or consider ZGB’s assertion that "... just as sequences of experiments help us to home in on a theoretical concern, so too ought they to yield better informed appreciation of patient characteristics that bear on this concern." What could they mean by this? They could be extolling the virtue of experimental sciences; but the virtues of scientific practice
need not extend to all putatively scientific activities, and this is precisely what is at issue here. ZGB do not specifically address the question of whether, as we have argued, patient-group methodology would violate basic tenets of sound scientific practice. In other words, experimental sciences are virtuous, if at all, independently of what we decide about the validity of patient-group studies. We are not licensed to accept that patient-group studies are theoretically useful just because we think that a sequence of experiments may allow us to converge on a plausible theoretical claim.

The third point we wish to make in reaction to the long quotation from ZGB concerns their claim that “... group results will be less prone to idiosyncratic results than will a single case [sic], so that, over the long run, fewer discrepant empirical findings should emerge from analysis of group data.” The reasonableness of this view is only apparent. What is meant by “discrepant empirical findings?” Discrepant with respect to what? Is the claim that the results of patient-group studies are less likely to differ one from another than would results of single-patient studies? This may very well be true, but it is equally irrelevant. The results of single-patient studies may differ because the patients differ in the nature of damage to the cognitive system. These differences in performance among patients are the very stuff on which our theories are built. Contrastively, patient-group studies may result in fewer discrepant results, but only because the interesting information in individual patients is averaged away.

But let us consider the problem of potentially idiosyncratic results from a different perspective. Let us accept that by their very nature single-patient studies run the risk of taking seriously the performance of a patient who is in fact “bizarre” in some way or other—perhaps the premorbid abilities of the patient differed in some interesting way from the normal population (the problem of the martians among us). In such a case, we would have results that are at variance with the true pattern that would have been obtained had the patient not been strange to begin with. These cases are a nuisance we would prefer not to have to deal with, but there may be such cases in the pool of patients who come to our attention for study, and we have to devise procedures for discriminating between them and the non-bizarre cases. As mentioned above, we have addressed the problem that this situation creates for single-patient studies in an earlier publication.

Note, however, that the existence of these cases is a problem for everybody, and it is not solved by proposing to carry out group-studies instead of single-patient studies. If such strange cases populate our clinics, then they are likely to find their way into patient-group studies as well. If they are included in group studies, the effect of their inclusion would be to distort the true pattern of performance which, presumably,
we would have obtained had we not included them in the group. In other words, strange cases are a problem for all of us. The real difficulties lie elsewhere. How do we tell which of the so-called strange cases are indeed strange and which are just cases that disconfirm our dearly held theories? The group-study approach has two ways of dealing with atypical cases, neither of which is satisfactory.

One "solution" is to treat atypical cases just like all other cases and include them in group studies. As already pointed out this move violates the assumption of homogeneity, resulting in a distorted picture of the true pattern of performance. The other "solution" is to exclude these cases from the group study. This move allows us to get "reliable" results for the group study, but opens the door to a more severe criticism. In fact, both "solutions" are subject to this criticism. Namely, potentially important information is being ignored: In one case—including the atypical patient in the group—the potential counterevidence is smothered in the averaging process; in the other case—excluding the atypical patient from the group—the potential counterevidence is ruled inadmissible by pretheoretical fiat. If the putatively strange cases were not in fact strange, but true (albeit rare) results that are simply inconsistent with our current hypotheses, then the procedures of group studies would ensure that such cases could not constitute counterevidence for our theories, either by averaging them away or by excluding them from being considered relevant evidence. Obviously, a patient should not be considered atypical only because his/her performance does not conform to our hypotheses about the world: A patient may differ from our theoretical expectations without necessarily being strange. In short, patient-group studies have the effect of insulating our theories from falsification from a crucial source of evidence by a methodological pretext—a totally unacceptable practice in any scientific enterprise.

A final word on this problem. In this discussion of the consequences of potentially idiosyncratic results we have seen that one objection to the proposed "solution" of conducting group studies is that it blocks the possibility of using potentially interesting results to reject the hypothesis that led to the classification scheme that was used to group patients in the first place. This objection is quite general and applies not only to putatively atypical cases, but to all cases. The classification scheme used to group patients is, after all, a hypothesis about the nature of the presumed common deficit in the patients grouped together. But the hypothesis does not (or ought not to) concern only those features of a patient's performance that are used for classification. The hypothesis underlying a classification scheme, whether explicitly or implicitly held, concerns claims about the full set of theoretically relevant performances. In other words, the classificatory act presupposes that each patient included in a class has, at least, a deficit to a common mechanism(s) and,
therefore, his/her performance on all tasks that involve the putatively damaged mechanism(s) ought to be affected similarly. Such a hypothesis may be disconfirmed either by finding that the performance features used for classification are dissociable or by finding that predicted performance on tasks other than those used for classification does not obtain. In either case, any patient whose performance does not conform with theoretical expectations may be taken as providing counterevidence to our hypothesis. It would be simply bad science to insulate our hypotheses from falsification either by ruling inadmissible apparent counterexamples through exclusion or by smothering them through averaging (see Caramazza (1986) and McCloskey & Caramazza (1988), for extensive discussion).

ON PATIENT VARIABILITY

Another point introduced by ZGB in this section of their paper concerns the problem of subject variability over time. They point out that if single patients evolve over time, then the analysis of these patients' data will present us with problems similar to those we face when we try to group together different patients. This is a correct observation. For this reason, it may be inadmissible to average the performance of single patients unless it is qualitatively identical over the theoretically relevant range. Note, however, that if we could not find sufficient stability in individual patients' performances, then we would be faced with some very difficult problems concerning the relevance of pathological performance for theories of normal cognitive functioning. In other words, we may find that we are not able to accept the transparency assumption (see Caramazza, 1984, 1986). In any case, if there are major problems of performance evolution in single-patient studies where we have the opportunity to study them in great detail and determine the course of evolution of their performance, then things could only be worse if we were faced with trying to interpret not a single well-studied case but an average of unknown mixture.

But why did ZGB bring up this potentially ugly problem with neuropsychological research in this discussion? What is the intended force of this point? Suppose that performance evolution is, in fact, a major factor to consider in single-patient studies. What should we conclude

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Lest we be misunderstood, we wish to emphasize that we find it dubious that a theory or a well replicated result that is based on sound methods will be rejected because of a single observation. Theories of cognitive processing cannot rely for their support on the evidence from a single patient, any more than they can rely on a single normal-group experiment; and most theories of cognitive processing reflect their dependence on converging sources of evidence. Although it is obvious, we reiterate that in no way does our position imply that one should base one's theories on the evidence from a single patient or a single experiment with a group of normal subjects.
from this observation? There are two possibilities. One option is that we develop a research methodology that can compensate for the problem. We may or may not be successful in this effort. The other option available to us is to throw in the towel and accept the judgment of those who told us all along that we could not do anything interesting with pathological performance. Notice, however, that we cannot take our quandary as a justification for doing patient-group studies: Two wrongs do not make a right. If performance evolution presents difficulties for single-patient studies, where very detailed analyses offer the opportunity to evaluate the changing pattern of performance in a patient, imagine the difficulties it would pose for patient-group studies. What would be the meaning of average performance across a group of patients with different functional lesions at different points of evolution of their disorder? It seems clear that if the problem raised by ZGB is a real one, then we have further reason for not carrying out patient-group studies. So, why did ZGB bring up the problem of patient evolution in the present context? It is not at all clear.

ZGB conclude this section by throwing in another argument by analogy. They note that "[t]hroughout social, cognitive, and medical science, investigators habitually group together subjects for certain experimental purposes," and they go on to point out that "[these investigators] do this without having any assurance that certain apparently irrelevant differences within their subject populations may in fact invalidate or distort the phenomena which they are studying." We have already commented on the value of reasoning-by-analogy as a form of argument. It is very dangerous to say that because something may be fine in one domain (assuming that it is so), it must be fine in another. This way of reasoning is a poor substitute for what is required to motivate the validity of inferences in a given domain of research.

**ON DATA AND THEORY, BOTH GOOD AND BAD**

The section of ZGB's paper titled "Data and Theory" again presents us with a number of different issues. Here too we will respond by taking up each point in turn.

The dominant aspect of this section is a change from a defense of the patient-group methodology to an attack on single-patient studies. ZGB begin by noting that the issue of "... single-case-only position is orthogonal to a more important distinction: that between good and bad science." If by this they mean that research based on single-patient studies can be either good or bad, we have no disagreement. As in all sciences, the quality of scientific activity is a function of its practitioners at determined historical moments. Some of the research produced is good, much of it is bad, most of it is quickly forgotten. This is no less
nor more true in cognitive neuropsychology than in other disciplines. But, once again, the relevance of the issue raised by ZGB is not obvious.

The problem we have raised in various publications does not concern which particular research projects are good or bad; the point is not to grade individual researchers or their projects. The problem concerns whether a particular approach can, in principle, give its practitioners the chance of producing good science. That is, the issue is whether there are any reasonable conditions under which one may expect that patient-group studies can produce results that allow meaningful interpretation. If the type of material we have chosen to work with (the performance of brain-damaged patients) does not allow us to articulate such conditions, then the issue of good or bad science does not even arise: The work is simply irrelevant to the scientific enterprise.

ZGB attempt to side-step the latter issue by criticizing specific research projects based on single-patient studies. The criticisms may be justified. In particular we share with them a concern for the all too common reification of dissociations of performance (see Rapp & Caramazza (in press) for discussion). But, at the price of seeming relentless, we must point out that even if all the criticisms raised by ZGB against various research projects based on single-patient studies were to be correct, that would not add anything to the present discussion. These criticisms do not undermine the possibility of doing meaningful research with single-patient studies, nor do they help dispel the fundamental objections we have raised against patient-group studies.

After their criticism of single-patient studies, ZGB produce a non sequitur in support of patient-group studies. We quote them in full.

Relatedly, it is not at all clear how to stop the proliferation of representation distinctions. Indeed, as typically practiced, the approach [the single-patient studies] seems geared to capturing dissociations. Without details concerning the representational formats of each component, it is very difficult to begin to interpret conjunctions of symptoms. Are they necessarily associated as the consequence of damage to one "module," or are they accidentally associated as the result of damage to two different components? In such an indeterminate situation, dissociations form a slippery slope. Just as the existence of Legionnaire's disease is not undermined because one of its symptoms—fever, say—can occur in isolation, so, too, the fact that single cases show dissociations among symptoms does not make their co-occurrence less interesting. Nor, more pointedly, does it render invalid grouping of patients showing such conjunctions. (p. 245).

There are a number of problems with these comments. We will not dwell overlong on these here other than to point them out. First, it is not clear why ZGB claim that the method of single-patient studies is "geared to capturing dissociations." Single-patient studies are equally appropriate for investigating associations of symptoms (e.g., Caramazza, Miceli, Villa, & Romani, 1987; Sokol & McCloskey, 1988); and, as
we have argued elsewhere (see Caramazza (1986) and McCloskey & Caramazza (1988)), there is nothing special about patterns of dissociation of impairment. Patterns of association of impairments can be equally informative. The method of single-patient studies is indifferent to the issue of association or dissociation of symptoms. Second, we do not understand the leap from the incorrect claim that single-patient studies are "geared to capturing dissociations" to the statements concerning associations of symptoms (see quote above). Not only do we not understand the jump, but we do not see the reason for bringing up the whole issue of the interpretation of association of symptoms. In any case, our position on the problem of how to interpret associations of symptoms is clear. To claim that the association of impairment in two tasks reflects a functionally necessary state of affairs is to claim that the two symptoms result from damage to a component of processing that is common to the two tasks. The latter claim, however, depends on our ability to articulate a theoretical position that explicitly motivates the processing component (and, to the extent that is possible, its internal structure) that is putatively shared in the two tasks in question. Having said as much, we are at a loss to see what this issue has to do with the rest of the argument ZGB are presenting at this point of their paper.

The most troubling point in the cited paragraph concerns the non sequitur regarding whether dissociations recorded for single-patients "render invalid groupings of patients." We confess immediately that we do not understand the train of thought that is supposed to link the several sentences leading to the conclusion at the end of the quoted paragraph. While we may agree that the dissociation of symptoms "does not make their co-occurrence less interesting," nothing follows from this about whether patient-group studies allow valid inferences about normal cognition. In any case, we have never claimed that the possibility of doing meaningful research with patient-groups is excluded merely by the fact that symptoms dissociate. The principal objection we have raised against patient-group studies concerns the fact that a priori classification of patients is theoretically arbitrary; and hence we cannot assume homogeneity of functional lesions in the group.

ON DIFFERENT FORMS OF DAMAGE—ANOTHER NON-ISSUE

ZGB next introduce the issue of partial vs. complete damage to cognitive mechanisms. They claim that "... proponents of the information-processing—single-case-only position appear not to distinguish failures of processing from partial or complete damage to stored representations" (p. 246). Besides being irrelevant to the current debate, this is a false claim. What could have led ZGB to make such a claim? What is there either about the information processing approach in cognitive neuropsychology or about the methods of single-patient studies that would
predispose those who adopt these positions (what ZGB have labeled the information-processing–single-case-only position) to making the error of not distinguishing the possibility of damage to processes from damage to representations?\(^7\) ZGB’s bald assertion is once again unsupported by argument. In this case, too, it is more than evident that ZGB’s claim not only does not correspond to the actual practice in cognitive neuropsychology, but that even if it were to do so, it would be irrelevant to the issue of whether patient-group studies do or do not provide a valid methodology for inferring the structure of normal cognitive mechanisms.

AN INTERIM CONCLUSION

In the first half of their paper, ZGB discuss the position we have expressed in several publications regarding the use of a priori classification schemes as a means of studying cognitive mechanisms. We have argued that it is not possible to frame meaningful theoretical or empirical questions about normal cognitive processing by reference to clinical types. A methodological implication of our analysis is that only single-patient studies allow valid inferences about normal cognitive mechanisms from patterns of impaired performance. ZGB have expressed their disagreement with our position. Regrettably, however, this expression of disagreement is not supported by detailed argument and analysis. As we have seen, no well-formed arguments were presented either (1) against our contention that, in principle, single-patient studies allow valid inferences about the structure of normal cognitive mechanisms, or (2) in favor of their position that patient-group studies allow valid inferences about normal cognitive mechanisms.

ZGB’s failure to articulate a general counterargument to our position does not exclude the possibility that they might be right in claiming that patient-group studies do allow valid inferences about normal cognitive mechanisms. Nor is it the case that the only way in which our position could be undermined is by a general counterargument. Our position would be undermined if it could be shown that there is at least one instance of a priori-classification-based research programs which allows valid inferences and meaningful theoretical claims about normal cognitive mechanisms. Since ZGB claim that research on “agrammatism” provides just such an instance, it is important to determine whether this is in fact the case. Hence, in the next section of our response we examine this claim and its supporting arguments.

\(^7\) It should not go unsaid here that distinguishing between damage to representations and damage to processing of representations is much less simple a matter than would appear from ZGB’s discussion of this issue.
ONCE AGAIN ON AGRAMMATISM

For the sake of clarity, and at the price of being repetitious, we begin by restating our position concerning the use of agrammatism in cognitive neuropsychology and neurolinguistics. For a more detailed statement of our position, the reader is referred to previous studies by Badecker and Caramazza (1985, 1986).

ON THE CO-OCCURRENCE OF SYMPTOMS

One criticism we leveled against the use of clinical categories such as agrammatism concerned what we described as the arbitrary grouping of symptoms. We described two scenarios by means of which two symptoms of agrammatism (such as function-word omission and reduction of phrasal complexity in sentence production) might co-occur. One is that both symptoms result from impairment to a common set of components. In this case, we may properly assume that there is a necessary link between these two symptoms: They co-occur because they both result from impairment to a common set of processing components. On the other hand, they might arise from impairments to different sets of components, in which case the link between the two symptoms is accidental. Under such conditions, the symptoms may be entirely dissociable. But if the link between these symptoms is indeed accidental, then defining a clinical category in terms of their co-occurrence entails that no unitary account of the clinical category is correct (because the clinical grouping so defined merely represents the intersection of two functionally independent deficits). The trouble that such arbitrary symptom combinations pose is that, unless their independence is recognized, the conjunction of these symptoms is commonly taken as inviting a unifying account. That is, syndromes tend to get elevated to the status of theoretical entities, and the conjunctions of symptoms by which these syndromes are defined tend to be transformed into evidence for theories of processing and representation which treat the conjunctions as necessary. The situation is further complicated by the possibility that the group of agrammatics identified in terms of these two symptoms is heterogeneous with respect to the symptoms' causes. That is, when the two symptoms are found to co-occur, in some patients, the two symptoms derive from a common origin; while in others, the link between the two is accidental. Under this condition, limiting group membership only to those who show both symptoms would still not guarantee the homogeneity of the group.

How does one determine for any particular agrammatic subject whether the two symptoms co-occur accidentally, or by necessity? There is no stock answer that we are aware of. However, one bit of evidence concerning this issue would have to be the dissociability of the two symptoms. If one has evidence that they dissociate in some cases, then the
burden of proof lies with the researcher who wishes to maintain that a particular instance where they co-occur reflects a necessary link of the sort we have described. Consider as an example the case of two symptoms standardly associated with the clinical category "agrammatism": reduced phrase length and the omission/substitution of function words.

There is a significant body of literature which documents their dissociability. Studies of cases clinically categorized as agrammatic include patients with severe phrase-length reduction in the presence of only mildly affected performance in terms of function-word omissions or substitutions, as well as patients whose phrase length is well within the normal range but who present with a significant impairment in function-word production (Kolk, van Grunsven, & Keyser, 1985; Miceli, Mazzucchi, Menn, & Goodglass, 1982; Miceli, Silveri, Romani, & Caramazza, 1989). Table 1, adapted from Miceli et al. (1989) indicates the degree to which these two symptoms fail to correlate among four so-called agrammatic patients.

Two of the patients that we have excerpted from this study were within the normal range on the measure of utterance length (T.F. and M.L.), while two were only moderately impaired on the measure of function-word omission (F.B. and M.L.). The feature of this pattern that is noteworthy is that the low measure of fluency can co-occur with both a low and a high function-word omission rate (F.B. and G.F., respectively), just as the high fluency measure co-occurs with both a low and a high omission rate (M.L. and T.F., respectively). This pattern provides strong evidence for the independence of the two symptoms. It is quite clear, then, that we cannot simply assume that the co-occurrence of these two symptoms in some patients reflects a causal link between these signs. Nor, for that matter, can we simply assume that no such link exists in

<table>
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<tr>
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<th>Mean length of utterance</th>
<th>Function-word omissions</th>
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<td>Normal controls</td>
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<tr>
<td>Range</td>
<td>6.70–13.01</td>
<td>0–1.6%</td>
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<tr>
<td>Average</td>
<td>8.39</td>
<td>0.27%</td>
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<td>Patients</td>
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<tr>
<td>F.B.</td>
<td>4.15</td>
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<tr>
<td>G.F.</td>
<td>4.55</td>
<td>49.6%</td>
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<tr>
<td>T.F.</td>
<td>10.50</td>
<td>50.6%</td>
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<tr>
<td>M.L.</td>
<td>7.44</td>
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*Mean number of syntactically appropriate, consecutively produced words in a sentence in spontaneous speech, ignoring morphological and function-word errors.
any particular case. If data are to be grouped according to this co-occurrence, this grouping must be motivated by more than a pretheoretic intuition about the relationship between the two symptoms. Furthermore, this grouping must be accomplished in such a fashion that group homogeneity, at the functional level, is assured: We have argued that neither of these requirements has been met in any study based on the clinical category of agrammatism (Badecker & Caramazza, 1985).

THE PROBLEM OF IDENTITY

In Badecker & Caramazza (1985) we argued that the characteristics of patients clinically lumped together under the category agrammatism give us little confidence that the group could have the status of a natural category defined in terms of a specific functional lesion to the language processing system (c.f., Miceli et al., 1989). So while we admit that such natural categories exist in principle, the categories of patients based on clinical impression provide no help in identifying them (and can in fact be seen as a hindrance). The problem of identity is the problem of identifying patients whose impairments are identical up to the limit mandated by current theoretical understanding and empirical evidence.

ZGB confuse this problem with the problem of clinical classification. They suggest that the problem of identity cannot be so difficult if, in arguing that agrammatism does not constitute a natural category of impairment, we were able to identify patients as agrammatic. We did not argue that agrammatism cannot be a clinical category—we argued that there is no natural category corresponding to this clinical category. That is, agrammatism has no theoretical status. When would we be willing to grant that a particular grouping of patients constituted a syndrome with a theoretical basis? When the grouping picks out those individuals who have suffered loss or impairment to the same components of the language processing system. What we did by identifying significant patterns of variation within the clinical category was to show that these observations offer little hope that the clinical group represents such a natural category of impairment. We did not question anyone’s ability to use vague and atheoretic principles in order to lump patients together (as is done in clinical grouping)—we simply argued that the grouping of data based on such principles had no value with respect to the goals of neurolinguistics and cognitive neuropsychology. Indeed, we argued that grouping data on the basis of such principles was counterproductive and inimical to the enterprise because, without motivation, it elevates an idealized version of the clinical group to the status of a natural category of impairment to the normal processing system.
ZGB state that the focus of their complaint concerning our arguments against group studies of so-called agrammatic aphasics is that we limited our attention to an impairment that is defined solely in terms of speech production. Since many group studies based on this clinical category address issues concerning the sentence comprehension of so-called agrammatic patients, it is suggested that our arguments have no force with respect to these studies. Do our arguments concerning agrammatism hinge in any way on the inclusion or exclusion of comprehension disorder as a defining characteristic of the syndrome? As a matter of fact, they do not. To make this point, we will begin with a discussion of why we chose the version of agrammatism that is more faithful to the principles of clinical categorization. We will then address the question of whether matters improve for the group-study approach if the list of criteria for grouping patients is expanded to include comprehension impairments.

Agrammatism is identified clinically in terms of a set of output characteristics (omission and/or misselection of function words and inflections; omission or nominalization of main verbs; and, commonly, reduced phrase length), and these criteria were at one time taken to include preserved comprehension as well. Among others, Zurif, Caramazza, and Meyerson (1972) observed that patients they characterized as agrammatics showed signs of grammatical impairment in input tasks (judgments of relationships among words in a sentence); and Caramazza and Zurif (1976), Heilman and Scholes (1976), and Schwartz, Saffran, and Marin (1980) reported that subjects in this clinical category showed asyntactic comprehension as well. However, the empirical claim that the two types of impaired performance constitute a necessary co-occurrence has not withstood the test of observation. Miceli et al. (1982) report two patients in whom the pattern of agrammatic production occurs in the absence of asyntactic comprehension. Kolk et al. (1985) report a patient who pre-

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8 Asyntactic comprehension is defined operationally as impaired performance on sentence tasks when the thematic relations are, in terms of the NP referents, reversible, and "normal" performance when, for example, pragmatic considerations would suggest that the thematic relations are not reversible. Thus, the correct interpretation of locative sentences like The square is above the triangle, and active and passive sentences like The boy kissed the girl and The boy was chased by the girl requires that one employ the syntactic encoding of this thematic information. When only pragmatically plausible sentences are employed, though, real-world knowledge may suffice (as in The boy was throwing the rock and The house was painted by the boy). Similarly, if the measure of performance is a sentence-picture matching task, the response options may allow a subject to perform correctly without using the syntactic structure of the sentence (as when the sentence The girl kissed the boy is paired with two pictures, one of a girl kissing boy and the other of a girl kissing a toy). For expository purposes we will refer to the two latter cases as instances of nonreversible sentences.
sents with speech that is "classically" agrammatic (frequent omission of function words, inflections, and main verbs), yet whose comprehension remains intact on a variety of stringent measures. At best, then, all one can claim from available evidence is that quite often, perhaps even most of the time, (clinically) agrammatic production is accompanied (by clinically) asyntactic comprehension, but the conjunction of agrammatic production and asyntactic comprehension is not a necessary one. It is on the basis of this dissociation that we did not consider asyntactic comprehension as a necessary component of the clinical category of agrammatism. Founding a category of data on the co-occurrence of agrammatic production and asyntactic comprehension would only amount to an arbitrary lumping together of symptoms.

This assessment has important consequences regarding the significance of such group data. Any attempt to explain data from a grouping based on the arbitrary association of asyntactic comprehension and agrammatic production is obliged to address the full range of relevant data—and this must include data from the dissociation cases as well. That is, if the functional impairment which results in (a variety of) asyntactic comprehension may be independent of the functional impairment that is responsible for (a variety of) agrammatic production, then we cannot seek to explain their co-occurrence by appeal to a theory that does not even speak to the same impairments when they occur in isolation. For all its

9 Other case studies which document this dissociation include Nespoulous et al. (1988) and Caramazza and Hillis (1989). Howard (1985) describes a case study which suggests that the co-occurrence of these production and comprehension impairments may be limited to a particular modality: the patient’s spontaneous speech and auditory comprehension showed no evidence of agrammatism or asyntactic comprehension; yet sentence level performance was agrammatic in writing tasks, and asyntactic in reading comprehension.

10 As Berndt and Caramazza (1980) and Howard (1985) point out, asyntactic comprehension operationally defined occurs independently of agrammatic production (e.g., Caramazza & Zurif, 1976; Caramazza, Basili, Koller, & Berndt, 1981; Heilman & Scholes, 1976). Since the defining characteristics of so-called agrammatic patients relate to their speech, though, the performance of such asyntactic comprehenders with nonagrammatic speech is seldom taken as relevant to the characterization of agrammatism.

11 It is important to note, in this regard, that our conclusions are in no way dependent on the proportion of cases that show the dissociation or association. Whether the dissociation between the agrammatic production and asyntactic comprehension is rare can only be understood in one of two ways. Either its (apparent) rarity is due to the functionally anomalous organization of the language system in the rare cases, or it is due to matters that are entirely irrelevant to the functional architecture of the normal processing system (e.g., the anatomical proximity of the neural substrate of two functionally distinct processing mechanisms, the dependence of these neural tissues on the same vascular structure, etc.). The suggestion that one can comfortably ignore the reported dissociation because it might only reflect a premorbidly abnormal system has become increasingly less appealing as more and more cases of this sort have been identified (Miceli et al., 1982; Kolk et al., 1985; Nespoulous et al., 1988; Caramazza & Hillis, 1989). We argue that this suggestion ought no longer to be taken seriously.
obviousness this point seems to have been missed. It may be worth the effort to elaborate on it briefly.

For the sake of exposition let us accept ZGB’s contention that the relevant category of analysis ought to be “. . . the type of aphasia usually referred to as agrammatic.” In other words, let us focus on those brain-damaged subjects who present with both agrammatic production and asyntactic comprehension. A particular theory of language processing is supported (over an alternative theory) by this pattern of language impairment if it is possible to functionally lesion it (but not the alternative theory) so as to derive the observed pattern of performance. Note that this theory necessarily includes a set of processing components that are common to the production and comprehension of language. Damage to these (common) processing components results in the co-occurrence of agrammatic production and asyntactic comprehension.¹²

But, what about the attested double dissociation of agrammatic production from asyntactic comprehension? Are these cases not clear counterevidence to the theory which postulates common mechanisms for sentence production and comprehension? Not necessarily. These cases of dissociation merely falsify the empirical claim that the two symptoms must necessarily co-occur under all conditions. One could argue that the cases of dissociation arise from damage at levels other than that which when damaged results in the co-occurrence of asyntactic comprehension and agrammatic production (e.g., Caramazza et al., 1981). In other words, the theory is not falsified just in case it is structured in such a way that functional lesions at one level of the language processing system result in agrammatic production with asyntactic comprehension, while functional lesions at other levels of the language processing system result in asyntactic comprehension without agrammatic production or agrammatic production without asyntactic comprehension. However, and this is crucial, a theory of language processing which can account for the co-occurring pattern of agrammatic production and asyntactic comprehension but cannot account for the isolated occurrence of agrammatic production or asyntactic comprehension is falsified by the dissociation cases. Obviously, then, any serious research program concerned with elucidating the structure of language processing mechanisms through the analysis of language disorders cannot arbitrarily exclude the dissociation cases from consideration. We cannot simply set aside the dissociation cases and suppose that an account that seeks to explain only the conjunction of the production and comprehension deficits will say anything about the functional nature of the patients’ impairments (or about the

¹² We leave aside for the moment the fact that, to our knowledge, no one has proposed a nontrivial processing theory of sentence production and comprehension that assumes common language-specific mechanisms.
normal processing system). Such an approach only amounts to the position that, in reference to an explanation of asyntactic comprehension (or agrammatic production) in one group of subjects, one can stipulate in advance that the asyntactic comprehension (respectively, agrammatic production) impairments of another group of subjects is irrelevant to how subjects will perform on a range of tasks. We find such a priori reasoning to be indefensible.

There is, however, a reading of ZGB’s claim that is not so peremptorily dismissive of potentially important empirical evidence. This position goes something like the following. Let us for the moment (and only for the moment) put aside those cases of dissociation of agrammatic production and asyntactic comprehension and focus on those where the two symptoms co-occur. Once we have made progress in understanding the “typical” cases of agrammatism (agrammatic production-with-asyntactic-comprehension), we will then consider the “atypical” cases of dissociation of the two symptoms. But note that in the absence of strong theoretical motivation for treating the combinations of asyntactic comprehension and agrammatic production as a natural category of deficit, there is no reason why this group should be more relevant to the study of the comprehension (or production) deficit than some other group (say, asyntactic comprehension with deep dyslexia or asyntactic comprehension with hemiplegia). Still, for the sake of completeness we will ignore the fact that ZGB do not provide any theoretical motivation for this choice, and we will go on to consider what might be gained by basing research on the average performance of the putatively “typical” agrammatic group. Does adopting this position overcome the objections we have raised against classification-based research? Not at all.

Suppose for the moment that the clinical patterns labeled, respectively, agrammatic production and asyntactic comprehension could individually serve as the basis for theoretically meaningful groupings of patients. We would then have two populations whose members are identified, respectively, by a particular form of production impairment and

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13 Drawing a distinction between typical and atypical cases of agrammatism presupposes a natural category of deficit which is characterized by damage to a common set of language processing mechanisms implicated in sentence production and comprehension. Given this presupposition, those cases in whom agrammatic production and asyntactic comprehension are found to co-occur will be considered typical. As we have repeatedly pointed out, the distinction between typical and atypical forms of impairment has the “merit” of prejudging the very issue that is supposed to be addressed in our research. If the issue of how the language processing system is organized is an empirical matter, then it should not be prejudged by excluding from consideration (the so-called atypical cases) just the evidence that would disconfirm our preconception about the structure of the language processing system.
a particular form of comprehension impairment.\textsuperscript{14} We already know that the two populations are not coextensive—there is ample evidence for the dissociability of asyntactic comprehension from agrammatic production. But there is also evidence that some (perhaps many) of the patients who present with agrammatic production also present with asyntactic comprehension. Consequently, among the population of brain-damaged patients we will find some who only present with agrammatic production, some who only present with asyntactic comprehension, and some who present with both agrammatic production and asyntactic comprehension. Even if we were to assume that among the latter group there are some patients whose co-occurring impairments in language production and comprehension arise from damage to a common set of processing mechanisms, we cannot exclude that there are some in whom the two symptoms co-occur accidentally (as the result of damage to unrelated processing mechanisms). That is, given the empirically established independence (dissociation) of the clinical patterns identified as agrammatic production and asyntactic comprehension, their joint appearance in a patient could equally signal damage to independent processing mechanisms or damage to a common set of mechanisms involved in sentence production and comprehension. This much follows logically from the preceding considerations. And, since the premises on which this argument is constructed—the empirically established dissociation of agrammatic production and asyntactic comprehension, and the possibility that brain damage may affect any of the hypothesized components of the language processing system—are uncontested, the inescapable conclusion must be that we cannot assume that patient groups formed on the basis of the co-occurrence of agrammatic production and asyntactic comprehension are homogeneous with respect to the underlying cause of their impairment. Consequently, and once again, we find no reason for thinking that ZGB's assertion that including asyntactic comprehension as one of the clinical features for patient classification will somehow obviate the criticisms we have directed against patient-group studies.

Note that we have not suggested that there is any a priori basis for concluding that all patterns of asyntactic comprehension must be functionally independent from production impairments. We have not argued against a unitary account of asyntactic comprehension plus agrammatic production for every case. Such unitary deficits may in fact exist. But given that we do have evidence that they are independent in some cases,\textsuperscript{14} We have just seen that the features that define agrammatic production dissociate. As we have argued repeatedly, there is no reason to expect that patients defined by an arbitrary set of production parameters constitute a theoretically homogeneous group. That is, there is no reason to suppose that the patients thus grouped would all present with a common set of underlying deficits.
instances of the putatively unitary deficit are, at least at this stage, indistinct from instances of the mixed deficit. With that in mind, though, it makes even less sense to employ a method whereby these two functionally distinct deficits would be analyzed in terms of the average performance of the heterogeneous group. In other words, there is no reason to believe that data from the group we might designate as agrammatic-with-comprehension-deficit would constitute a suitable basis for evaluating claims about the normal processing system, since the average performance of this group might be compatible with accounts of the impairment that would not be compatible with either of the two "subdeficits" (the mixed deficit and the unitary deficit). The complexity of the situation is not miraculously simplified by patient grouping.\textsuperscript{15}

**THE RELEVANCE OF THE GROUP STUDIES ZGB DISCUSS**

ZGB discuss two sorts of group studies of agrammatic subjects which are meant to exemplify the usefulness of the clinical grouping for neurolinguistics and cognitive neuropsychology. In the first case they discuss Grodzinsky (1986a, 1986b), and in the second, Swinney, Zurif, and Nicol (1989). On our reading, the putative significance of the Grodzinsky studies is twofold. On the one hand it is offered as providing a theoretical basis for the clinical grouping agrammatic-with-comprehension-deficit, and on the other, as providing the explanatory link between a particular pattern of comprehension deficit and the output disorder. The discussion of Swinney et al. (1989) suggests that the clinical group can be useful as a means for examining issues of language processing (as opposed to language representation).

**GRODZINSKY AND OTHERS**

ZGB suggest that Grodzinsky's group studies of agrammatism are important because they provide a theoretical basis for (1) the assertion that there is homogeneity in the clinical category with respect to comprehension impairments, and (2) the assertion that the comprehension impairments and the agrammatic production deficit may be the result of a common underlying impairment. That is, Grodzinsky's group studies are supposed to have demonstrated that agrammatism-with-comprehension-impairment specifies a homogeneous group in that members of this group will exhibit difficulty in comprehending sentences whose syntactic representations involve traces. This would, among other cases, include simple passive sentences, but not simple active sentences. This hypothesized processing/representational impairment will be reflected in com-

\textsuperscript{15} The situation is even more complicated than this since, as we have argued, agrammatic production and asyntactic comprehension do not provide the basis for homogeneous patient groupings themselves.
prehension tasks whenever successful performance requires a subject to recognize (and take advantage of) the syntactic distinction between active and passive sentences. Thus, Grodzinsky’s theory suggests that, regardless of the variation that one sees in the production or comprehension of these patients, those classified as agrammatic-with-comprehension-impairment will do worse on reversible passive sentences than on reversible active sentences in, say, a sentence–picture matching task. This hypothesis is judged relevant to the characterization of agrammatic production because traces, like function words, are considered part of the nonlexical vocabulary of a language. So, the story goes, if the theory about the comprehension impairment is correct, then there may be a basis for a unitary account of agrammatic production and asyntactic comprehension.

The problem with Grodzinsky’s hypothesis is that it is either false or vacuous. Let us assume for a moment that the hypothesis is meant to explain the comprehension impairment that ZGB describe as one criterion in patient selection in group studies of so-called agrammatics. We quote their statement in full:

Another factor in patient selection is that these patients must also have a comprehension limitation. Usually this limitation is operationalized as below normal comprehension of reversible passive constructions (e.g., the boy was chased by the girl) in the face of near normal comprehension of reversible active sentences (e.g., the girl chased the boy). (p. 248)

In an apparent reference to Grodzinsky’s work, ZGB write that:

With respect to grammatical representation, studies have applied fairly far reaching principles to account for a nonintuitive collection of sentences that agrammatic patients—as chosen in the above manner [sic]—have difficulty understanding. (p. 248)

If the agrammatic patients were indeed chosen in the above manner, then it is not at all surprising that the performance of the group of patients classified as agrammatic-with-comprehension-impairment was homogeneous under Grodzinsky’s characterization. The characterization of the comprehension impairment in terms of syntactic structures and traces is a simple restatement of the patient selection criterion. How could patients grouped in this way fail to show the homogeneity? After all, they are said to have been selected for study on the basis of the putative explanatory principle: selective impairment with passives.

The alternative interpretation of Grodzinsky’s theory of agrammatism is that ZGB were mistaken about the selectional criteria and that there is actually an empirical claim being made here. That is, cases of agrammatism-with-comprehension-impairment are to be identified in any in-
stance where agrammatic production co-occurs with asyntactic comprehension. Again, by asyntactic comprehension we mean worse performance on sentences like reversible actives and passives, reversible locative sentences, etc., than on so-called nonreversible sentences (like *The boy threw the rock* and *The rock was thrown by the boy*). Under this construal of the intended selectional criteria, if there are subpatterns in the patients’ performance on reversible sentences, these are not employed to exclude one case but not another. In this case, one of the empirical predictions that Grodzinsky’s theory makes is that, regardless of whatever variation exists in this patient group, one thing should be true in every case. Performance must be worse for reversible passive sentences than for reversible active sentences. In fact, when single-case studies are used to evaluate this claim, it turns out that each of the three possible orderings are found: Passives worse than actives; passives and actives the same; and passives better than actives. For example, in one case that we have seen in our own laboratory, the patient (F.M.) was at chance for reversible actives (41% correct; $p > .20$) but above chance for reversible passives (65% correct; $p < .05$), and his performance on the two categories of reversible sentences was statistically different as well ($\chi^2 = 4.91, df = 1, p < .05$). This pattern may, in fact, be rare; but there is no a priori reason to suggest that the rarity of this pattern is due to something that is less relevant to the theory of linguistic processing or representation than the comparatively more common pattern involving better performance on actives than passives.

Even if cases such as F.M. did not exist, though, there are numerous other common performance patterns on comprehension tasks which demonstrate that Grodzinsky’s theory fails to establish the homogeneity of the clinical category. For example, it is not evident that Grodzinsky’s account of agrammatism is consistent with any case where performance on active reversible sentences is significantly different from performance on active nonreversible sentences (since only passive sentences should be affected by the impairment that Grodzinsky hypothesizes). This pattern is evident in the performance of patient V.S., as well as patients reported in Caramazza and Zurif (1976), and Martin, Wetzel, Blossom-Stach, and Feher (in press). Grodzinsky’s account is also not consistent with cases where performance on both actives and passives is impaired equally (e.g., patients J.R. and H.T. reported in Schwartz et al., 1980). Finally, Grodzinsky’s theory of how passive sentences should be processed by so-called agrammatists predicts that truncated passives (e.g., *The girl was hit*) should induce performance significantly below chance levels, while full passives (e.g., *The girl was hit by the boy*) should

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16 V.S. scored correctly on 76% of the reversible actives, but 100% on the nonreversible active sentences ($\chi^2 = 6.19, df = 1, p < .02$).
induce chance level performance. Martin et al. (in press) examine just this prediction in a study of four patients classified as agrammatics-with-comprehension-impairment. Each of these four patients showed worse performance on passive sentences than on active sentences. However, none of these patients scored significantly lower on the truncated passives than on the full passives—and in the case with the greatest difference between the two passive constructions, performance was better on the truncated passives. Thus, the various orderings of difficulty in processing actives and passives have been observed and reported; and this observation ought to serve as prima facie evidence that Grodzinsky’s account cannot serve to demonstrate the homogeneity of the clinical category agrammatism-with-comprehension-impairment.

Given that Grodzinsky’s theory is confronted with counterexamples on virtually every prediction regarding agrammatism-with-comprehension-impairment and the active/passive distinction, it simply cannot be true that this theory establishes the homogeneity of the patient category that ZGB wish to submit to group-study analysis. Note that we have not attempted to argue that Grodzinsky’s theory cannot be true for specific subjects.\(^{17}\) That is not the issue here. If it is true of specific subjects, though, it must be established individually by detailed tests of all of the theory’s predictions for each of those subjects. To our knowledge this has not been done. What has been done instead is to evaluate the theory in terms of the performance of a heterogeneous group, and it is irrelevant whether that group does or does not conform to some of the theory’s predictions.

**ON-LINE STUDIES**

Before we address the Swinney et al. (1989) results in detail, we feel compelled to examine two of the points that ZGB make with respect to the sorts of group study their work exemplifies. The first concerns the relationship between the type of study (i.e., group vs. single-case) and the variety of performance measure that is employed. ZGB make the point that on-line measures of subjects’ performance on certain tasks provide an important means for examining processing failures (as opposed to “representational failures”), and that, for various reasons, the use of on-line tasks requires that one does group studies. We will not dispute the first point here.\(^{18}\) On the issue of whether a particular experimental method mandates the averaging of data from a number of

\(^{17}\) We have made this point repeatedly. The issue of whether a particular theoretical claim is or is not worthwhile is independent of the means one uses (or misuses) in its evaluation (see Caramazza, 1988).

\(^{18}\) We suspect that the intended message in ZGB’s discussion is that so-called on-line measures are, in principle, the only means for examining processing failures, or the processing mechanisms of the normal system. We do not see why this should be the case.
different subjects, though, this cannot be true. We take it, for example, that single-case studies such as those of Tyler (1985, 1988) fall squarely within the paradigm of "on-line" processing studies. Without evaluating any other aspect of such studies one way or the other, the assumption of homogeneity was addressed by averaging the single subject's performance across a number of testing sessions. Now it is true that averaging a single patient's performance across testing sessions requires that one have some confidence that the patient has not changed qualitatively over time. If one has reason to believe that such changes have taken place, then the averaging procedure is obviously inappropriate. (See our discussion of patient variability, earlier in this article). One may mistakenly assume that no such changes have occurred in a particular subject, but this is surely not a problem limited to the single-case study. The group study too must assume that the modicum of similarity across test subjects continues to hold at the moment of the experiment. Unlike the group study, though, the single-case experiment does not make the additional and unwarranted assumption of across-subject homogeneity. Thus, in the absence of evidence of within-subject variation, the individual patient is the limiting case. If multiple tests are mandated by some technique in order to derive the statistical power needed to test a particular hypothesis, one can do no more to guarantee subject homogeneity than to study the same subject. In the case of experiments on non-normal subjects, the one thing one can do to virtually guarantee theoretically significant heterogeneity is to average the data of different patients.

The general point that we have to make regarding these studies is quite simple. If these on-line methods are superior to others for addressing certain types of issues, then so be it. The assumptions that such methods require for use in group studies are no different from the corresponding assumptions relevant to so-called off-line methods. Averaging performance across subjects does not provide an automatic means for guaranteeing homogeneity among the subjects. Nor does it address the issue of arbitrariness. These issues are not addressed in ZGB's discussion of on-line measurement, so it is not clear what the relevance of these studies are on the matter of the theoretical basis of agrammatism.

The second point that we feel would be advisable to raise again concerns what ZGB identify as a failure of information-processing accounts to "distinguish failures of processing from partial or complete damage to stored representations" (p. 246). They go on to say that:

Without providing an independent theory of the processor, or without seeking to provide evidence for processing modules in terms of real-time operating characteristics, a hypothesized functional lesion is indistinguishably a hypothesis about the processing unit and/or about the knowledge representation it operates on. Moreover, it is a claim that begs a fundamental question: Even if a particular process is said to be disrupted, is it so disrupted that it makes no contribution
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If this is meant to serve as an argument concerning the inherent limitations of single-case studies, then our only response is that ZGB have muddled the issue by suggesting that single-case methods contrast with group-study-based methods just as theories which make an all-or-none statement about the effect of damage to a component contrast with those theories which entertain degrees (or types) of impairment to a component in a modular system. However, the all-or-none character that ZGB attribute to information-processing theories and their single-case methodologies is simply not accurate. The same thing goes for the representation/process distinction. There are often inherent difficulties in distinguishing a representational account of a phenomenon from a processing account (if only because "processing" is short for "processing of representations"); but the theoretical distinction is no more problematic for single-case methodologies than it is for group-study methodologies. ZGB have simply set up two false dichotomies that should be ignored.

LEXICAL PRIMING STUDIES

In a study of so-called agrammatic and Wernicke's aphasics, Swinney et al. (1989) applied an experimental method known as cross-modal priming that has been used previously with normal subjects (e.g., Swinney, Onifer, Prather, & Herskowitz, 1979; Seidenberg, Tanenhaus, Leiman, & Bienkowski, 1982) to demonstrate that lexical access is a form-based procedure that is functionally autonomous with respect to the mechanisms of word-sense disambiguation. The pattern that has been consistently replicated for normal subjects (including by Swinney et al., 1989) is that when a word like plant is presented in a spoken sentence context, a lexical decision for a visually presented, semantically related word like tree will be facilitated (in comparison to a decision for a matched item page when preceded by plant). The particular pattern of priming with normals is important: When the off-set of an ambiguous prime like plant is separated by 0 msec from a target like tree or factory, facilitation will be found for both items, irrespective of whether the related sense was appropriate to the sentence context that the prime occurs in. With longer prime-target asynchronies, though, priming can be measured only for the target that is semantically related to the contextually appropriate sense of the prime. It is the nonselective nature of the priming found at the 0-msec delay which presumably indicates that the lexical access procedure is isolated from the process of word-sense disambiguation.

Swinney et al.'s (1989) results with regard to the four subjects in their study who were clinically categorized as agrammatic was that the patients...
showed cross-modal priming for targets related to the primary reading of an ambiguous prime, but not for targets related to its secondary reading. Also unlike the results for normals, an ambiguous prime like plant failed to facilitate decisions for factory, which is semantically related to the secondary reading of plant, even when this secondary reading is uniquely appropriate to the sentence context (as in The Ford Motor Company has an assembly plant just outside of metropolitan Boston). Swinney et al. (1989) hypothesize that these patterns are the consequence of a slowing of the "module-internal operations" of lexical access, and they conjecture that for agrammatic patients "the facilitation for all senses of an ambiguous word should be shown, but at a point in a sentence that is noticeably later than that which is shown normally" (p. 32).19

Let us accept that the priming patterns that held for the groups of patients in this study also hold in truth for each group member that Swinney et al. tested. What do these data speak to? To questions regarding the modularity of lexical access? It is difficult to see how. ZGB (like Swinney et al., 1989) conclude that their data demonstrate that "the brain damage that underlies agrammatism does not seem to destroy the modular basis for lexical access" (p. 251). Surely, this possibility was a straw man to begin with. If information from sentence context cannot influence the module-internal processes of access in the normal case (i.e., if lexical access is encapsulated), then demodularizing these processes would require that brain damage can result in the reorganization of the functional system.20 Suppose, though, that the agrammatics in their study only showed priming for the contextually appropriate meaning. Would it force us to abandon the modularity theory for normal access? Since this priming pattern is also found in the normal case (with sufficient prime-target asynchronies), it is unlikely that even Swinney et al. (1989) would take such a pattern as evidence that the modularity argued for by Swinney et al. (1979) and Seidenberg et al. (1982) was an illusion. There is very little reason to believe that the reported patient data obtained with the use of "on-line" methods contributed to the theory of the normal processing system.

More to the point, the only sorts of conclusions that ZGB (or Swinney et al., 1989) draw from these results are those regarding the consequences of patient group membership. We have repeatedly acknowledged that clinical data can obviously speak to questions like: What can be found

19 Given that semantic priming effects are notoriously fleeting in the case of normals, this conjecture seems a bit curious to us. Nevertheless, we will accept the possibility of its correctness for the sake of argument.

20 Perhaps this could be true, but if so it seriously undermines the logic of all neuropsychological research and may well rule out the possibility that data from brain-damaged patients could shed light on the normal system (see Caramazza, 1984).
in members of one clinical category versus another? That is, what are the probable consequences (with respect to some performance measure or another) of presenting with agrammatism-with-comprehension-deficit? We have only argued that patient-group data such as those reported by Swinney et al. (1989) cannot speak to questions about the normal processing system. Nor, for that matter, can they be used to illuminate a particular case of impairment, since there is no evidence that agrammatists as a clinical category are homogeneous with respect to the priming patterns that Swinney et al. (1989) report.

We can illustrate the latter point by considering Swinney et al.'s own results. These investigators found that, irrespective of biasing sentence context, ambiguous primes (plant) facilitated lexical decisions for words related to the primary sense (tree) of the priming stimulus but not for words related to the secondary sense (factory) of the priming stimulus. But suppose that one of the four patients tested by Swinney et al. had shown a different pattern of performance. For example, suppose that one of the four patients did not show any priming at all (or showed some altogether different pattern). What would be the implications of this discrepancy in the pattern of priming results? One could try to dismiss the noncompliant case as a statistical anomaly. Alternatively, one would have to conclude that the patient classification scheme failed to pick just those cases with a common underlying deficit. That is, the category of agrammatism would be heterogeneous at the level where significant generalization can be stated: in terms of the functional lesion to the language processing system. But we don't have to discuss this problem in hypothetical terms. There is evidence which, in crucial respects, is at variance with the results reported by Swinney et al. That is, there are reports of patient-group studies which presumably show that so-called agrammatic Broca's aphasics do not show lexical priming.

Milberg and Blumstein (1981) administered a lexical decision task (in the visual modality) to groups of aphasics and found that the Broca's and Conduction aphasics in their study did not show facilitation for items primed by semantically related words. Milberg, Blumstein, and Dworetzky (1987) administered a lexical decision task to patients they classify as Wernicke's and Broca's aphasics. These subjects had to determine whether the third item in an auditorily presented triplet was a word. The second word in each triplet was polysemous, and the first and third words of each triplet were semantically related to one, both, or neither meaning of the ambiguous item. Unlike the normal controls and the Wernicke's patients in this study, the patients grouped as agrammatic Broca's aphasics showed no semantic facilitation in any priming condition. Clearly, there is a discrepancy between these results and those of Swinney et al. (1989). In a footnote, Swinney et al. (1989) address these contradictions by suggesting that the difference might be attrib-
utable to the fact that "these studies employed an isolated word paradigm, while [Swinney et al.] assessed priming in the context of sentence processing" (p. 36). Inasmuch as the central result of the Swinney et al. (1989) study is that the so-called agrammatic aphasics were facilitated only by the primary interpretation of an ambiguous word and that they showed absolutely no effect for sentence context, such an explanation is curious. Why should the sentence context matter if Swinney et al.'s own patients show the same pattern of priming regardless of the sentence context? That is, there is no evidence from the pattern of priming that the sentence makes any difference. One less taxing account of the discrepancy is obvious: The patients differ in their impairments. Membership in the clinical category does not produce homogeneity.

Another problem with the reported study concerns the results for the Wernicke's aphasics. These patients, unlike the normal control group or the agrammatic Broca's aphasics, showed priming in all conditions, regardless of biasing sentence context or whether the target stimulus was related to the primary or secondary sense of the priming stimulus. Furthermore, as Swinney et al. point out in a footnote, these patients presented with asyntactic comprehension. By their own account, then, asyntactic comprehension dissociates from agrammatic production. More important in the present context, asyntactic comprehension is associated with, at least, three different forms of (disordered) lexical priming patterns: (1) those cases where priming only occurs for the dominant sense of an ambiguous word, regardless of sentence biasing context; (2) those cases where priming occurs for all senses of an ambiguous word, regardless of sentence biasing context; and (3) those cases where no priming at all is obtained (Milberg et al., 1987). Given this surplus of patterns of relationship between asyntactic comprehension and lexical priming deficits, what compelling theoretical reason led ZGB (and Swinney et al.) to assume that the pattern they chose to discuss, but not other patterns obtained by themselves or by other investigators, reflects a causal relationship between the two deficits? None was provided. The arbitrariness with which particular patterns of results are deemed important (or less so) is more than apparent. This arbitrariness is intrinsic to classification-based research and patient-group studies. No meaningful scientific enterprise can be built on such arbitrariness.

Again, we have not argued that the account that Swinney et al. (1989) propose is not true of some cases (indeed, of the four patients they discuss). We simply observe that, with respect to the issue of possible patterns of impairment, the grouping of data in this experiment adds nothing to the single-patient results. (It could not, because the assumption of the homogeneity of these patients has no basis apart from the specific results for these subjects on these tasks.) Furthermore, the different question of whether the pattern of priming is true of all so-called agram-
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mathematics (or all members of any other clinical category) is not adequately tested by group data, since the average performance of the group may be more a product of the relative frequency of one functionally distinct pattern versus another than of, say, different levels of impairment within a functionally homogeneous group. Since the functional homogeneity of the clinical group is just the point in question in this case, one simply prejudges the issue by grouping the data. At best, the grouped data can only bear on the relatively uninteresting question of the statistical reliability of the co-occurrence of the priming pattern and clinical category. At the risk of repetitiveness, though, one must keep in mind that group data concerning the consequences of membership in the clinical category of agrammatism (the only type of question that can be addressed by group studies of agrammatism) are entirely irrelevant to the task of using neuropsychological evidence to motivate and constrain theories of normal language processing or representation. Why, then, do ZGB advance Swinney et al. (1989) as a prime example of the sort of contribution to neuropsychology that exemplifies the need to do group studies in this domain? We have not been able to divine an adequate response to this question.

ON PROCESS AND REPRESENTATIONS

ZGB conclude the section of their paper concerning agrammatism by introducing a further consideration in favor of their view that patient-group studies provide a necessary source of information in constraining theories of language processing. However, in developing these considerations, ZGB are unconstrained by the very "facts" which they themselves have generated or which they cite from other publications. In this paragraph they also introduce the issue of brain/cognition relationship as a further motivation for the need to do patient-group studies. This argument, however, is a simple non sequitur. In commenting on this paragraph, it is useful to quote it in full in order to avoid misunderstandings.

A final consideration: To the extent that agrammatic patients cohere as a group in terms of an underlying disruption to fast-acting, automatic processing modules, it is quite possible that the coherence is in some, at present, indeterminate fashion brought about by the dependence of such modular routines on elementary motor and sensory-motor systems—in the sense that these elementary systems can serve

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21 Uninteresting, that is, from the perspective of cognitive neuropsychology or neurolinguistics. The question is on a par with: What is the degree of correlation between being classified as an agrammatic Broca's aphasic and presenting with right hemiparesis? The possible utility to the clinician of information of this sort does not in any sense render it relevant to the task of identifying the representational and processing characteristics of the language faculty.
as a resource for the language-processing modules. If so, we will have returned to the Wernicke–Lichtheim formulations in which language processes in the brain were seen as an extension of sensory–motor operations that had obvious cerebral localizing value. In effect, should such relations obtain, we will have started to close the distance between cognitive and neuroscience—on the one hand, evaluating and constraining theories containing abstract linguistic representational formats (sic), and on the other, developing a theory of some of the component processors that implement these formats, but yet are rooted to sensory–motor activities with discrete cortical locations. It is a possibility that will require for its exploration well-articulated theories from cognitive science and a willingness to use group-based analyses, incorporating lesion as well as behavioral features in patient selection. (p. 252).

Because there are a number of points we wish to make about this paragraph we will not dwell overlong on any of these. Instead, we will mention each point and only very briefly discuss it here.

In speculating on the possibility that agrammatic aphasics have an "underlying disruption to fast-acting, automatic processing modules," ZGB fail to mention that the putative evidence in support of this contention is anything but unambiguous in this regard. As we have shown in our discussion of the Swinney et al. results, these authors are aware that there are several different patterns of lexical priming deficits associated with asyntactic comprehension (in fact, they are responsible for documenting two of them). This information about the heterogeneity of co-occurring patterns of deficits did not find its way into the conclusion concerning the possible underlying cause of agrammatic aphasia.

ZGB attempt to link deficits in "modular routines" to "elementary motor and sensory–motor systems" by assuming that "these elementary systems serve as a resource for the language-processing modules." Regrettably, they provide no indication of what all this means. In what sense do motor and sensory–motor systems serve as "a resource" for the language processing modules? What architecture of the language processing system could accommodate the authors' claim? Unless ZGB are using the standard terminology of cognitive neuropsychology (e.g., resource) in an idiosyncratic way, we must conclude that the claim is a confused one. In any case, without considerable elaboration, ZGB's comments remain far too underspecified to constitute a serious empirical claim.

ZGB go on to suggest that the supposition of an unspecified language-processing modules' dependence on elementary sensory–motor systems constitutes a return to the Wernicke–Lichtheim hypothesis, which considers language processing as an "extension of sensory–motor operations." Although it is unclear to us, it may very well be true that ZGB's position represents a return to the Wernicke–Lichtheim model. But, if so, it would also represent an inexplicable abdication of scientific responsibility. Surely, since the good old days we have learned enough
about language processing to appreciate that invoking impoverished notions about sensory and motor images and their associations does not help us very much in understanding normal linguistic capacity nor the forms of language disorders that result from brain damage. The picture is not improved by their appeal to brain mechanisms. ZGB tell us that if we accept the line of reasoning that took us back to Wernicke–Lichtheim, then we should see that, in fact, we are making progress in “close[ing] the distance between cognitive and neuroscience.” How did they come to this conclusion? The connection is supposedly provided by the fact that “sensory–motor activities” are associated with “discrete cortical locations.” Still, how does adopting an impoverished theory of language processing help reduce the distance between cognitive science and neuroscience? ZGB further conclude that if we want to participate in these important developments it will require a “... willingness to use group-based analyses, incorporating lesion as well as behavioral features in patient selection.” How did ZGB arrive at the conclusion that, if we wish to investigate issues concerning the neural bases for language processing, we should do patient-group research? We are afraid that we simply have one more case of an unmotivated assertion offered as a valid conclusion.

Finally, we note that one of the recurring themes in ZGB’s article is their criticism of the information-processing approach in cognitive neuropsychology, which they fault for supposedly failing to distinguish between deficits of representation and process. We have already noted that this criticism should not be taken seriously as it is simply inaccurate. Nevertheless, we have also already noted that ZGB are correct in pointing out that there is much confusion about matters of representation and process in the cognitive neuropsychological literature (but not more than in the cognitive psychology literature in general).22 It is ironic, therefore, that ZGB should discuss two very different conceptions of the underlying deficit in agrammatic aphasia—one couched in terms of a deficit to linguistic representations, the other in terms of a processing deficit—but completely fail to discuss the relationship that should (!) obtain between these two different accounts of what is, on their view, the same disorder.

The problem is not that ZGB have failed to discuss an interesting but unnecessary detail about the relationship between two alternative accounts of agrammatic aphasia. The problem goes much deeper. It concerns the issue of whether the representational and processing hypotheses discussed by ZGB meet the minimum standards of explanatory adequacy. Specifically, do these hypotheses about the underlying deficit of agrammat-
matic aphasia account for those performance features that define this patient category? Furthermore, if the two hypotheses are, as ZGB argue, noncompeting accounts of the underlying deficit of the independently identifiable natural category of agrammatic aphasia, then there must be a necessary relationship between the processing account offered by Swinney et al. and the representational account offered by Grodzinsky. However, ZGB fail to discuss either of these issues in any detail, despite the fact that these are crucial to their argument.

Consider first the “process deficit” hypothesis offered by Swinney et al. How does this hypothesis account for the particular behavioral features used to identify agrammatic aphasics? How, for example, does a deficit to “fast-acting, automatic processing modules” lead to the omission of bound morphemes? It is not obvious to us that ZGB have a concrete proposal to offer regarding the relationship between the hypothesized processing deficit and those performance features that define the clinical category of agrammatism (or, if they have a proposal, they have not told us what it is).

Consider now the relationship between the two hypotheses of agrammatism—the representational and the process deficit hypothesis—discussed by ZGB as putative examples of coherent theoretical positions based on patient-group research programs. Since the two hypotheses are offered as noncompeting accounts of agrammatic aphasia, not only should these explain the same range of facts but they should be explicitly (and transparently) related. That is, ZGB should show how one can get from one account of agrammatism to the other—they should show how one gets from a deficit to “fast-acting, automatic processing modules” (Swinney et al.) to an inability “to interpret surface structures that contain . . . traces” (Grodzinsky), or vice versa. Unless the link between these claims is made explicit, ZGB’s pronouncements on these matters amount to no more than bald assertions.

CONCLUDING REMARKS

We have argued that a priori patient classification cannot guarantee that the members of such classes will be (nontrivially) homogeneous with respect to the functional lesion(s) presumed responsible for the patients’ cognitive/linguistic impairments. This observation has led us to conclude that valid inferences about normal cognitive processes from the analysis of the performance of brain-damaged subjects cannot be based on patient-group studies. We recognize that this position strikes some as troubling and that some may even suspect us of propounding a view based on logical fallacy. However, no well-formed argument has appeared showing that our position is, in fact, based on logical fallacy. Instead, the typical response, very well exemplified by ZGB consists of assertions to the effect that since group-based research is legitimate in various other en-
deavors it ought to be similarly acceptable in the domain of neuropsychology. Furthermore, it has been argued that there are research programs which amply demonstrate—an existence proof, as it were—that patient-group research is a valid and productive enterprise. It is extremely difficult to respond to arguments-by-analogy, other than to identify them as such. It is also very difficult to respond to claims about the successful implementation of a research program, other than by a detailed rejection of each of its claims. This is what we have tried to do in response to ZGB’s criticism of our position. We have identified pseudo-arguments as such, and we have shown that the putatively successful research programs on agrammatic aphasia are less-than theoretically or methodologically adequate.

We recognize, of course, that single-patient research has its own intrinsic limitations (see Caramazza, 1986; Shallice, 1979). But, one of these is not that (given various uncontested assumptions) it does not allow valid inferences about normal cognition. Nor is it the case that a cognitive neuropsychology based on (and limited to) single-patient research methodology would somehow find itself at odds with other developments in cognitive science or neuroscience. Thus, despite comments to the effect that our arguments regarding group studies amount to the claim that “neuropsychological inquiry ought . . . to be set apart from other forms of cognitive science” (Swinney et al., 1989, p. 36), we believe that our proposals should be seen as an attempt to do just the opposite—to pursue issues of processing and representation as is validly done in science. All we have done is to argue that methodology should be at the service of theory, experiment, and observation, and not vice versa. A crucial aspect of science is that its methods are tailor-made to deal with the specific puzzles and problems that nature sets for us. It is not that there is a fixed, cook-book procedure for doing science. The only absolute requirement is that the methodology one uses must be one that allows valid theoretical conclusions from experiment or observation. Thus, when ZGB suggest that we propose diminishing “that plurality of approaches which has yielded scientific progress in the past” (p. 253), our response is simply that we have only proposed diminishing the plurality of methods to just those that allow valid theoretical inferences.

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