Theoretical and Methodological Considerations in Aphasia Research and
Practice: Valid Inferences About the Structure of Normal Language
Processes from Patterns of Acquired Language Dysfunction
are Only Possible for Single-Patient Studies

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The analysis of acquired language disorders offers a unique opportunity
for exploring the nature of the linguistic and cognitive mechanisms that
subserve normal language performance and for relating these mechanisms to
neural structures.   This view is based on the assumption that the range and
type of possible patterns of language impairment are a direct function of the
structure of the normal language processing system.   By investigating the
forms of language impairment consequent to brain damage, we can reconstruct
the organization and processing structure of the normal language system.
That is, we assume that there is a transparent relationship between impaired
language performance and the structure of the normal language system.   A
pattern of impaired language performance is taken as empirical support for a
model of normal language processing (over some other model) if it is possible
to hypothesize a functional lesion to the system such that the lesioned
system generates the observed pattern of performance.   This methodological
assertion is captured schematically as follows:

$$M + L \rightarrow O,$$

where M, L, and O stand for model of normal language processing, functional
lesion, and performance, respectively.* This much is standard practice in
research on aphasia.

Saying that this is standard practice in our field is not to say, of
course, that there are not major problems of method and theory yet to be
resolved.   Quite to the contrary, there is the issue of what constitutes an
explanatory account of language processing--i.e., what we take to be the
proper form of M; there is the issue of how to constrain the range of
possible functional lesions, L--i.e., what we take to be the possible forms
of L; there is the question of how to specify the range of relevant observa-
tions from brain-damaged patients for a theory of language processing--i.e.,
what aspects of an aphasic patient's performance are relevant to a model of
language processing; and so forth.   These issues are in need of careful
consideration and analysis.   I will not dwell on them except insofar as it is
necessary to set the stage for the two claims under consideration in this
symposium: (1) that valid inferences about the structure of normal cognitive
systems from patterns of impaired performance are only possible for single-
patient studies, and (2) that the classification of patients into categories
such as Wernicke's aphasia, conduction aphasia, expressive aphasia or more
modern categories such as agrammatic aphasia, deep dyslexia, phonological
agraphia, and so forth, is not only useless but positively harmful for
research into the nature of language disturbances and the structure of normal
language processing.   I will defend these two related claims here.

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* The inferential schema for linking performance to a model of a cognitive
system is considerably more complex than indicated here. For a more detailed,
although still incomplete, discussion of this issue, see Caramazza (1986).

But before I begin let me hasten to point out, lest a discussion of these issues be seen as merely an academic exercise, that clinical aphasiology, as any other clinical enterprise, must ultimately rest on sound theoretical foundations. The issues under consideration here are of central importance for basic research in cognitive neuropsychology. Theoretical developments in the nature of language dysfunction are not possible without sound theoretical analyses and empirical investigation. These, in turn, must be based on unimpeachable methodological foundations. A dispassionate look at the current state of research on aphasia more than justifies a detailed consideration of the basic presuppositions that motivate the theoretical and empirical claims in this field of study. It is not an exaggeration to say that over one hundred years of research on aphasia has shed little light on the nature of normal language processes and the form of their dissolution in conditions of brain damage.

There are two culprits contributing to this state of affairs: inadequate theory and inappropriate methodology. There is a pressing need to develop increasingly more detailed models of language processing and attendant methodology for relating patterns of acquired language disorders to models of normal cognitive systems. Conversely, if the analysis of language disorders is to be more than merely an atheoretical description of deviant language use, it must be informed and constrained by a theory of language processing. The crucial point here is that we cannot discuss issues of method outside the context of some or other theoretical framework--there is no meaningful, theory-independent evaluation of method. Accordingly, this discussion of method in cognitive neuropsychology is offered from a specific theoretical vantage point. It is only proper, therefore, that I outline my theoretical perspective at the outset.

I will assume that answers to such questions as "How do we comprehend or produce sentences?" or "How do we read or write?" will be given in terms of computationally explicit, information-processing accounts of these cognitive abilities--that is, by giving an explicit account of the organization and structure of a set of processing components which together comprise the cognitive system that subserves a specific ability. Thus, for example, a minimal description of the sentence production process will include mechanisms for generating sentence structures, a lexical system sufficiently articulated to specify the semantic, syntactic, morphological, and phonological features of lexical entries, working memory processes, speech production mechanisms, and so forth. The objective, then is to articulate the functional architecture of the cognitive system in enough detail that we may identify the processing modules, their computational burden and internal structure, and their organization in the service of a specific performance such as sentence production. In this view, the production of a sentence is considered to be the end result of a complex set of processing operations involving a complex set of mechanisms (e.g., Garrett, 1984). This much I take to be uncontroversial among cognitive neuropsychologists.

We may now ask "How do we build such models?" There are two major steps, loosely speaking, in this task. The first step is to develop a "sufficient" account of the processing system being considered--that is, an account which specifies the sequence of operations needed to generate the principal features of the performance that characterizes a particular ability. To make this point less abstract let us consider as an example how we might go about developing a model of spelling (or writing) to dictation.

A "sufficient" model of the spelling process must be able to account for the principal features that characterize this process--viz, that we are able

to spell both familiar and unfamiliar words. There are various potential solutions to this problem. One solution is to propose a processing structure such as that schematically represented in Figure 1.

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              I
              ↓
        ┌──────────────┐
        │ Perceptual/  │
        │ Phonological │
        │ Processes    │
        └──────────────┘
              ↓
        ┌──────────────┐
        │   Lexicon    │
        └──────────────┘
              ↓
        ┌──────────────┐
        │ Graphemic    │
        │ Output       │
        │ Processes    │
        └──────────────┘
              ↓
           Spelling
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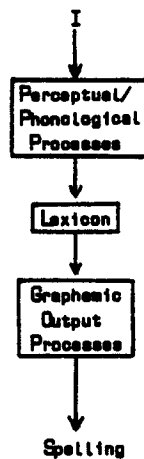
Figure 1. A very simple model of spelling.

In this model a single processing component converts a phonological representation into a graphemic representation, both for familiar and unfamiliar words. A model of spelling such as this would be "sufficient" for spelling in Finnish (a near-perfectly phonemic orthography) or Korean Han'gŭl (a perfectly transparent orthography) but might be inadequate for English, French, and many other languages. In these latter languages the mapping of phonemes onto graphemes is not one-to-one but one-to-many and many-to-one (e.g., /i/ -> ea, ee, ... as in /rid/ -> reed, read, ... and /i/ or /ɛ/... -> ea, as in /lid/ or /lɛd/ -> lead); furthermore, many words have idiosyncratic spellings so that their correct spelling requires word-specific knowledge (e.g., /jɑt/ -> yacht; /so/ -> sew). Two solutions have been proposed for this problem. One is to attempt to increase the computational power of the lexical sound-to-print conversion mechanism so that it can compute the correct spelling for orthographically opaque languages--i.e., both regular and idiosyncratic spellings. The other solution is to propose a functional architecture which contains separate processing mechanisms for spelling familiar and unfamiliar words, respectively. A schematic representation of this latter type of model is shown in Figure 2--the lexical system provides word-specific graphemic information, the phoneme-to-grapheme conversion mechanism computes graphemic representations for unfamiliar words.

The second step in our model building task involves the collection and evaluation of experimental evidence to choose between (or among) competing functional architectures of the cognitive system under consideration or between (or among) alternative hypotheses within a single functional archi-tecture. Continuing with our example of a model of the spelling process, we might wish to devise experimental tasks to help decide which of the two general architectures of the spelling process discussed above--the single- or dual-process model--gives a "correct" characterization of the spelling system.

Of course, these highly schematic models are gross oversimplifications of the processing structure of the spelling system. A more realistic, if still underdeveloped, functional architecture of the spelling process is given in Figure 3.
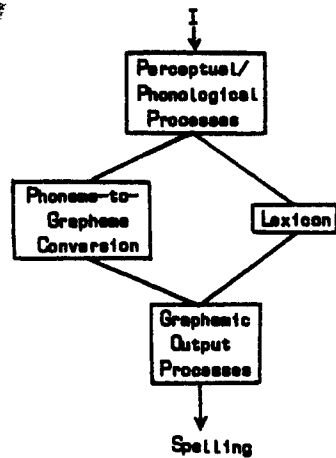
Figure 2. A spelling model that distinguishes the processes for spelling familiar and unfamiliar words.
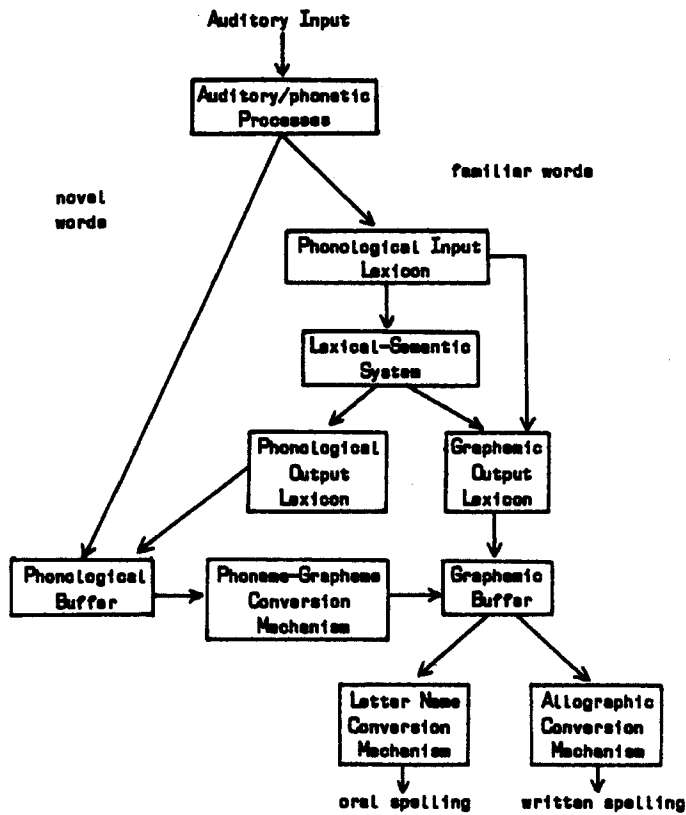
Figure 3. A more realistic model of the functional architecture of the spelling system.

This latter model represents a less incomplete description of the processing components that subserve spelling (see Caramazza, Miceli, and Villa, 1986; Ellis, 1982; Goodman and Caramazza, in press; for further discussion). It should be further emphasized that the explanatory usefulness of these models depends crucially on our ability to provide increasingly detailed descriptions of the internal structure of the components of processing that comprise a given functional architecture. That is, we must articulate the computational burden and structure of each component part of the system as well as its organization. The systematic analysis of patterns of acquired cognitive disorders can play a central role in this effort. It can only do so, however, if the appropriate methodology is used.

I have suggested that even the simplest cognitive performance (e.g., spelling a word) is the result of a complex series of processes involving many processing modules. Damage to any part of this complex process will result in impaired performance--the specific form of impairment being determined by the locus of damage to the cognitive system. For example, in our model of spelling, damage to the phoneme-grapheme conversion mechanism results in a different pattern of spelling impairment from the one we would find were the damage to be to the graphemic output lexicon. In the former case, the patient should be able to spell familiar words but not unfamiliar words; in the latter case, the patient should produce phonologically plausible spellings for familiar and unfamiliar words but not necessarily the orthographically correct spelling (e.g., /brid/ -> bread; /jɑt/ -> yot). Still other patterns of spelling impairment are expected for damage to other components of the spelling system (e.g., damage to the graphemic buffer).

In the ideal case, damage may be restricted to a single component of processing. Most often, however, brain damage affects multiple components of a system, (e.g., the phoneme-grapheme conversion mechanism and the graphemic output lexicon) producing complex patterns of impairment. These latter patients' performance, although considerably more difficult to work with than that of the single-component deficit patients, also constitute, in principle, empirical tests of our model of the spelling process. In other words, each patient's performance should be explicable by hypothesizing functional lesions to the proposed model of spelling.

Let me summarize the main points of my discussion thus far so that I may set the stage for my argument that patient-group studies do not allow valid inferences about normal cognitive systems. I have made three points:
1. Normal cognitive performance (O) is the result of the activity of a set of processing components which together comprise a cognitive system, M.
2. The possible forms of cognitive impairments are constrained by the nature of normal cognitive systems. More specifically, impaired cognitive performance (O) reflects the activity of a functionally lesioned cognitive system, that is M + L -> O.
3. Basic research activity in cognitive neuropsychology involves determining, for any patient (P), whether or not there is an appropriate modification of a cognitive system, by hypothesizing a functional lesion to it, which would account for the observed pattern of impaired cognitive performance (O).
The critical point here is that the functional lesion, L, which is needed for relating a patient's performance to a model of a cognitive system, is not given a priori but must be inferred from the patient's impaired performance. This situation sets precise limits on the type of methodology that allows valid inferences about normal cognition from the analysis of impaired performance. The argument in support of this claim may best be made by analogy to

the reasoning involved in interpreting results in a typical laboratory experiment.

A highly stylized description of the reasoning involved in relating experimental results to theory is as follows. A model of cognitive processes is related to predicted patterns of results through a set of experimental conditions. Thus, given a model, M, we predict a specific pattern of results (Performance O1) under certain exprimental conditions, C1, and a different pattern of results (performance O2) under a different set of conditions, C2. For example, given a particular model of visual search we might predict that reaction time for detecting a specific letter in an array of letters increases linearly with the number of nontarget letters but that reaction time for detecting that same letter in an array of digits is independent of the number of digit distractors. Schematically we can represent these situations as
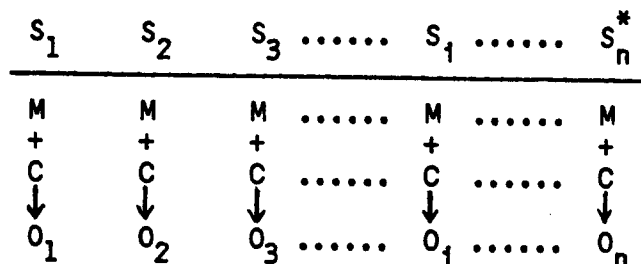
$$M + C1 \rightarrow O1$$
$$M + C2 \rightarrow O2.$$

An important feature of this schema for our present purposes is that a single model (M) predicts different patterns of performance (O1, O2,...) under different experimental conditions (C1, C2,...). If the predicted results obtain, we increase our confidence in the model (over alternative models).

This schema for relating experimental results to models of cognitive systems is strikingly similar to that presented earlier for relating impaired performance to models of normal cognitive systems. Indeed, for illustrative purposes it is useful to consider each patient as an experiment of nature where the functional lesion, L, corresponds to a particular set of experimental conditions in a typical laboratory experiment. The only difference is that in one case we relate results to a model of cognitive processing through a set of experimental conditions while in the other case we relate results to a model of cognitive processing through a set of hypothesized functional lesions. This seemingly minor difference is all-important, however. For, whereas experimental conditions (C) are under the control of the experimenter, and therefore are a known quantity, the functional lesions needed for relating patients' performance to a model of cognitive processing are not under the experimenter's control but must themselves be inferred from the patients' performance. This means that, whereas for a typical laboratory experiment we can duplicate at will the experimental conditions to test any number of normal subjects, we cannot do the same in a clinical setting where the precise nature of a functional lesion is not under the control of the experimenter and therefore may not be duplicated. A direct consequence of the difference between the two situations under consideration is that while, under given conditions, it is legitimate to average the performance of a group of normal subjects, there are no nontrivial cases where averaging patients' performance is justified.

Given the importance of the conclusion we have arrived at--after all, were it to be correct, the vast majority of the results reported in the neuropsychological literature would be uninterpretable--it may be worth the effort to present in a little more detail the arguments that support this conclusion. The principal argument concerns whether or not the conditions for averaging performance across subjects or patients are satisfied in either case. The basic condition to be satisfied is that the objects over which we are averaging are tokens of the same type. Is this condition satisfied in the case of research with normal subjects? Consider the schematic representation of a group of subjects shown in Figure 4. Averaging performance O1 through On is justified if M and C are equivalent in relevant respects. We

know that the Cs are equivalent, since these are under the control of the experimenter, and we assume that Ms are equivalent. It is legitimate, therefore, to average the performance of groups of normal subjects when they are tested in equivalent conditions. Parenthetically, it has often been pointed out to me that we may not assume that Ms are equivalent in relevant respects. If, indeed, we are not justified in making this assumption the consequence is not to legitimize averaging over patients' performance but to make averaging over normal subjects' performance illegitimate (but, see Caramazza (1986) for discussion of this issue).

$$
\begin{array}{cccccc}
S_1 & S_2 & S_3 & \cdots & S_i & \cdots & S_n^{*} \\
\hline
M & M & M & \cdots & M & \cdots & M \\
+ & + & + & & + & & + \\
C & C & C & \cdots & C & \cdots & C \\
\downarrow & \downarrow & \downarrow & & \downarrow & & \downarrow \\
O_1 & O_2 & O_3 & \cdots & O_i & \cdots & O_n
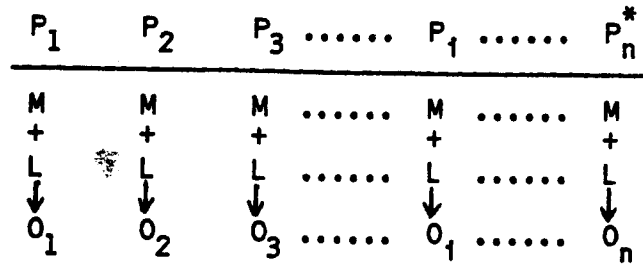\end{array}
$$

* S = subject; M = model of cognitive system; C = experimental
conditions; O = performance.

Figure 4. Schematic representation of the logic for relating the performance of normal subjects to a model of a cognitive system.

Consider now the case for research with brain-damaged patients. (Of course this schema should also contain the variable C, for experimental conditions to indicate that the patients' performance was obtained under a particular set of conditions. However, as in the typical, laboratory-experiment situation these conditions are under the control of the experimenter and, therefore, can be made equal across patients. They are not listed here to simplify exposition.) Averaging performance O_1 through O_n would be justified if we could assume that Ms and Ls are equivalent in relevant respects for patients P_1 through P_n. We have already expressed our willingness to accept the assumption that Ms are equivalent in relevant respects. We cannot do the same, however, for the Ls--these are not under the control of the experimenter. It is an empirical matter to be decided by careful analysis whether or not two functional lesions are equivalent in relevant respects. Therefore, averaging of patients' performance is only legitimate if and only if we have empirically demonstrated that the patients have equivalent functional lesions.

Although we have opened the door to the possibility that averaging patients' performance may be justified under certain conditions, there is little to be cheerful about since the situation in which averaging patients' performance is justified is a trivial one. Let me elaborate.

I have argued that averaging patients' performance is legitimate only in the case where we satisfy the homogeneity condition--i.e., where we have empirically demonstrated that the functional lesions in a group of patients are equivalent in relevant respects. This means that for each patient we

$$P_1 \quad P_2 \quad P_3 \ldots\ldots \quad P_i \ldots\ldots \quad P_n^*$$

$$
\begin{array}{ccccc}
M & M & M \ldots\ldots & M \ldots\ldots & M \\
+ & + & + & + & + \\
L & L & L \ldots\ldots & L \ldots\ldots & L \\
\downarrow & \downarrow & \downarrow & \downarrow & \downarrow \\
O_1 & O_2 & O_3 \ldots\ldots & O_i \ldots\ldots & O_n
\end{array}
$$

* P = patients; M = model of cognitive system; L = functional lesion; O = performance.

Figure 5. Schematic representation of the logic for relating the performance of brain-damaged patients to a model of a cognitive system.

must identify the locus of functional damage in a cognitive system and that the damage is equivalent for the patients we wish to group together. But, how might we go about doing this? To infer the locus of functional damage to a cognitive system we must analyze a patient's performance over a theoretically determined range; that is, over that range of performance which unambiguously allows us to hypothesize one functional lesion as opposed to another in a given model of a cognitive system. For example, in the model of spelling I presented earlier, the performance that is (minimally) relevant for uniquely determining a functional lesion to the graphemic buffer is the following:

- impaired spelling performance in all tasks
- absence of any lexical effects on spelling performance; i.e., no effects of word frequency, form class, or concreteness
- an effect of word length on spelling performance
- spelling errors consisting of letter substitutions, additions, deletions, and transpositions, i.e., letter-based transformations of target response
- qualitatively equal spelling performance for familiar and unfamiliar words (or nonwords)

For the model of spelling entertained here, this pattern of performance is only possible if the graphemic buffer were damaged (Caramazza, Miceli, Villa and Romani, 1986).

Let me arbitrarily call the range of performance that uniquely determines a particular locus of damage (Li) in a cognitive system O1 through On. Now suppose that we find two patients who satisfy the criteria for hypothesizing Li in a cognitive system--that is, the two patients present with performance O1 through On. In this case we may legitimately average the performance of the two patients over the range O1 through On. This step, while unimpeachable, is also trivial since it provides no information beyond what was available for the two individual cases. Furthermore, notice that we are not justified in averaging the two patients' performance outside the range of performance used to "fix" the functional lesion in a cognitive system. We cannot, in other words, test the two patients on a new task and average their performance in this task unless these are qualitatively identical, in which case this step is merely gratuitous since it provides no additional information beyond that given by the two individual patterns of

performance. Violation of this methodological constraint leads to unpleasant consequences.

Consider the following situation. Suppose we find two patients, P1 and P2, whose performance on a set of tasks is sufficiently similar, in relevant respects, that we can confidently conclude that they have damage to the same component of a given cognitive system. Suppose further that we now test these patients on a new task and their performance on this task differs in interesting ways. If we were to average their performance on this task, it would be tantamount to asserting that the noted variation in performance is theoretically insignificant. But, what is the justification for this step? Notice that the decision to average the performance of the two patients is not theory neutral; that is, it is not merely a methodological expediency. If it were to be such, we would be able to devise a mechanical procedure which allowed us to decide to average performance on any new task once certain antecedent conditions, short of equivalent performance on the new task, were satisfied. But, this is equivalent to saying that there is no possible outcome on this new task that would make a theoretical difference. In other words, performance on the set of tasks used to determine the locus of damage exhausts the types of observations that would provide theoretically significant information. In this case it is not clear why one would want to test the patients on the new task--it has been stipulated that no matter what the outcome, the patients' performance does not have theoretical import. Surely, this is not a position we want to find ourselves in.

The alternative is that performance on the new task could make a significant theoretical difference. If such were the case, then it is not immaterial whether or not the patients' performance on the new task is different in interesting ways. I have already discussed the case where performance on the new task is "equivalent."

When patients' performance on the new task is different there are two possible courses of action open to us. One possibility arises when we are willing to consider the difference in performance as reflecting either that we were mistaken in concluding that they have damage to the same component(s) of processing or that one of the two patients has an additional deficit that affects performance on the new task. In this case, we are not justified in averaging the patients' performance. The other possibility arises when, despite the observed difference in performance on the new task, we are convinced that it is theoretically of little import and, therefore, we feel justified in averaging the patients' performance. This move, although not inconceivable, requires reasonable argument to support it. I confess, however, that I cannot imagine what argument would be offered nor what would be gained by averaging manifestly different patterns of performance. In any case, both situations discussed here require a patient-by-patient analysis, informed by theory, before one may even consider the possibility of grouping patients' performance.

The arguments I have presented here naturally lead to the conclusion that patient-group studies are methodologically unsound and that only single-patient studies allow valid inferences about the structure of normal cognitive systems from patterns of cognitively impaired performance. I consider still valid a conclusion I stated in an earlier paper (Caramazza, 1986) on the issue of patient-group studies: "...either we satisfy the homogeneity assumption by carrying out a series of single-case studies, which makes the concept of patient-group study vacuous, or we fail to carry out a patient-by-patient analysis, in which case we cannot assume homogeneity over the relevant experimental observations" (p.59).

The arguments I have offered in favor of the view that only single-patient studies are methodologically legitimate have clear-cut implications for the issue of patient classification in aphasia. The major implication for our present purposes is that patient classification cannot play any useful role in research which addresses questions concerning the nature of normal language processes through the analysis of acquired language disorders nor those questions concerned with elucidating the structure of language disorders themselves. This conclusion is based on the following arguments.

I have insisted that there is no theory-independent means for selecting a subset of performance as more basic or meaningful than some other subset. I have also insisted that in cognitive neuropsychology the basic unit of analysis is the single patient--one of an indefinitely large number of experiments of nature. Each experiment offers the opportunity to test specific hypotheses about the structure of normal language processes. However, the crucial point here is that there is no independent means for deciding which subset of these experiments of nature constitute tokens of the same type, short of the very theories they are supposed to evaluate. This objection holds whether one attempts to give an empirical or a theoretical justification for patient classification schemes (see Badecker and Caramazza, 1985; Caramazza, 1984; Schwartz, 1984; for further discussion).

As far as I can tell there are two types of arguments that have been given to motivate patient classification--one empirically based, the other theoretically-based. The empirical argument goes as follows. There exist clinically identifiable patterns of deficits; i.e., we find that certain kinds of deficits tend to cluster together. For example, the omission of function words and inflectional affixes in spontaneous speech and effortful speech frequently co-occur. Given this fact we might want to consider patients who present with these two symptoms as constituting a natural category. But, what kind of a category is it? Is the co-occurrence of these deficits cognitively based? If our objective is to understand the structure of language processing, then the only significant criterion for relating symptoms is a cognitive one. That is, we must be able to show that there is a theoretically (cognitive) motivated basis for wanting to treat patients with these two deficits as members of the same category--perhaps because these two deficits jointly define functional damage to a specific component of processing. Stated differently, the co-occurrence of the two deficits is predicted on the basis of a theory of language processing. If this criterion cannot be satisfied, then, there is no more reason for grouping patients on the basis of the co-occurrence of these two deficits than on the basis of any other set of features. Thus, for example, we might just as well have defined a category as comprising those patients who present with the omission of function words in spontaneous speech and right hemiparesis. These two deficits also co-occur frequently but, to my knowleddg, no one has proposed that their co-occurrence has any import for a theory of language processing. Clearly, the empirically-based justification for patient classification is unimpeachable on theoretical grounds, but it is equally theoretically vacuous.

Attempts have also been made to offer theoretically-based criteria for patient classification in aphasia. Berndt and Caramazza's (1980) "A redefinition of the syndrome of Broca's aphasia: Implications for a neuropsychological model of language" is a good example of such efforts, although as I have argued elsewhere (Badecker and Caramazza, 1985) it achieves its end by invoking such an abstract model of language processing that it fails to account for any of the significant details of patients' performance. And, in

any case, it is not immune to the criticisms raised earlier in this paper as will become obvious shortly. A more recent attempt by Caplan (1986) focuses more narrowly on agrammatism in its effort to motivate a theoretical basis for patient classification. If I understood the argument, it goes as follows. Linguistic theory provides the motivation for distinguishing grammatical morphemes from lexical morphemes. There exist patients whose spontaneous speech is characterized by difficulties in producing one of these two classes of morphemes--the grammatical morphemes. We may use this performance feature to define a class of patients with impairment to a well-defined level of linguistic theory and, therefore, we are justified in averaging their performance. Unfortunately, this will not do. It will not do because the impairment could result from damage to distinct components of the language processing system and because even if damage were to be located to a common mechanism it does not preclude damage to other relevant components of processing.

The reason for rejecting these apparently more sophisticated attempts at providing a sound methodological basis for patient classification (and patient-group studies) is that they require giving precedence to a subset of a patient's performance over other, theoretically significant aspects of performance even though both sets of performance are needed to decide issues of theory. I have already given what I consider to be compelling arguments against this position. Briefly, this approach either insulates its definitionally-based categories from reasonable empirical test, making the whole exercise less than useful, or it leads to the violation of the homogeneity condition for group studies. Just because the performance feature used to define group membership is theoretically salient in some linguistic theory, it does not guarantee homogeneity over performance in tasks other than the one chosen as the basis for the initial classification. I see no more merit to these theoretically-based attempts at providing a methodologically sound basis for patient grouping than to the empirically-based attempts.

To conclude, I have argued that the only valid methodology for research with brain-damaged patients is one based on single-patient studies. I have also argued that patient classification as a basis for research is not only a useless exercise but a positively harmful one.* These negative conclusions do not undermine the possibility for developing serious programs of research in aphasia. To the contrary, dispensing with theoretically useless classification schemes and invalid methodologies allows us to focus on theoretically significant issues with a sound methodology.

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* Shallice (1979), in an otherwise clear and strong defense of the single-patient methodology in cognitive neuropsychology, does not find reasons for rejecting patient classification as a basis for research. To the contrary, he claims that patient classification must form the basis for generalization in neuropsychological research. This is surprising given that with similar premises I reached the opposite conclusion. Although in Caramazza (1986 I do not explicitly deal with this discrepancy, I do present arguments against generalization to patient types. This issue is in need of further analysis, however.

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