

The Logic of Neuropsychological Research and the Problem of Patient Classification in Aphasia

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A critical analysis is presented of the assumptions that must be made to use the data from aphasia to constrain models of normal language processing. The implication of these assumptions for patient classification and research methodology in aphasia is considered.

Patient classification plays a crucial role in aphasia research. The nature of the classification scheme one adopts, however, is not arbitrary but depends on the goals of the investigator. In this paper I will argue that *if* one studies aphasia with the goal of developing a theory of the structure of the processing components that define normal cognition and the neuroanatomical and neurophysiological substrates of the processes in the normal brain *then* the classical typology of aphasia does not constitute a theoretically defensible classification scheme. Parenthetically, although I state the claim of the theoretical indefensibility of the classical classification scheme in this relatively weak form it may be worth considering whether any other reasonable goal in investigations of aphasia may not lead to similar conclusions to those we arrive at when considering the goal I have stated. Leaving aside this issue, I will propose that, given the stated goal, we must develop a new, theoretically motivated typology of aphasia based on psycholinguistic principles.

Cognitive neuropsychology is the study of the relationship between the brain and cognitive processes. The goal is to propose a set of statements about the processing components that define normal cognition and the

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neuroanatomical (and neurophysiological) substrates of these processes in the normal brain. The most direct way to get at this relationship would be to study the workings of the normal brain. Unfortunately, methodologies have not yet been developed that are sufficiently powerful to study directly the workings of the normal brain as it performs complex cognitive functions. Consequently, the study of brain-damaged populations has provided, and continues to provide, the major source of information on which to base a neurocognitive theory.

The assumptions that must be made to motivate the use of data from pathological populations to develop theories of the neuroanatomical realization of cognitive processes in the normal brain logically subsumes those needed to motivate the weaker goal of using these data to develop theories of cognitive processing. Thus, with the caveats to be discussed below, we can use the analysis of performance of brain-damaged individuals just as we would that of normal individuals to develop and test theories of cognitive processing independently of claims concerning brain-cognitive process relationships. The primary focus of the paper will be on the justification of the use of data from pathological populations to constrain models of normal cognition. I will not consider the issue of cognitive process-brain relationship since the arguments I wish to develop can be made independently of this latter issue.

The most fundamental assumption of cognitive neuropsychology is the *fractionation assumption*—the belief that brain damage can result in the selective impairment of components of cognitive processing. The fractionation assumption is a complex supposition that requires, at least, that two basic conditions be met for its practical application. First, the fractionation assumption requires that a complex psychological function can be represented in terms of more basic components of processing, or modules. Second, the fractionation assumption also depends on a strongly construed belief that the pathological performance observed will provide a basis for discerning which component or module of the system is disrupted (I will call this the “transparency condition”).

The notion of modularity is the most basic assumption of the Information Processing approach in cognition. It states, essentially, that cognition consists of the functioning of a number of independent processing units. Complex psychological functions (e.g., sentence comprehension) are expressible in terms of more basic processing units. Processing units are typically defined in terms of two dimensions: a set of procedures or operations and a code over which the operations are applied. Accepting the modularity condition does not entail endorsement of any particular information processing model; it commits one only to the general information processing metaphor of cognition. This componential view of cognitive processing allows complex functions to be expressed in terms of basic processing components; e.g., function X may be expressed in terms of the components a , b , c , and d .

Brain damage may selectively impair one processing component, e.g., component *d*, such that function *X* cannot be realized in terms of the normal processing components. The critical question with which the transparency condition is concerned is the interpretation of performance on function *X* when one of the components needed for its execution is impaired.

A less than optimistic view of the interpretation of pathological performance is that even if the fractionation assumption were to be true, the remaining, unimpaired processes will work differently when one component is not functioning normally. That is, the position could be taken that there is a *de novo* organization of the remaining components such that the processes involved in the original complex function now work differently. In this case, the pathological performance would not have a transparent relation to the working of the normal system and would make the analysis of pathological cases irrelevant for the understanding of normal cognition. For obvious reasons I will assume that there is a transparent relation between pathological performance and normal cognition.

It must be stressed that the modularity and transparency conditions are *paradigm* assumptions that are not subject to empirical refutation in any simple sense of this term; it is unclear what empirical evidence would constitute sufficient grounds for rejecting the conditions of modularity and transparency.

The acceptance of the fractionation assumption as I have stated it leads to a *theoretical typology of aphasia syndromes*. That is, given an explicit theory of language processing we can deduce the possible patterns of language deficits that *can* be observed. Unfortunately we do not have a sufficiently well-worked out theory of language processing and, consequently we must be satisfied with the normal scientific situation of having to determine a typology by a mixture of deductive and inductive criteria. Nonetheless, an empirically defensible typology of this type places strong constraints on theories of language processing.

The logical plausibility of the fractionation assumption and the two conditions it subsumes, the modularity and transparency condition, does not imply that it is empirically reasonable, or that we should accept it without question. On the contrary, given its central status in modern neuropsychology, the fractionation assumption should be subjected to close scrutiny in an effort to ascertain that it is pragmatically defensible and, if so, what conditions must be satisfied for its reasonable application. In other words, we must consider both the empirical motivations that can be adduced to support the fractionation assumption and the methodological restrictions that must be observed for its proper application.

The major empirical justification for adopting the fractionation assumption is the classically established view that language functions are dissociable in aphasia. Over a century of research in aphasia has documented, if

nothing else, that brain damage can result in differentiated patterns of language disturbances. Some patients present with marked difficulties in one aspect of language in the context of relatively spared language functions in other areas, while other patients present a different constellation of spared and impaired language functions. The existence of differential patterns of language impairments is *prima facie* motivation for some form of the fractionation assumption. A note of caution must be entered here, however. The dissociations described in the classical literature (as well as most of the recent literature) have concerned complex psychological functions—language comprehension, language production, reading, etc. If we adopt the current reductionist view of cognition which assume that such complex language functions are reducible to more basic psychological processes, then the issue becomes how to establish that a particular processing component(s) is disrupted when a complex function is impaired. My initial discussion of this issue is to consider some rather simple, idealized cases but I will go on to consider more realistic situations in a later section of this paper.

The basic data from which we draw inferences about the structure of cognitive processes are the patterns of cooccurrences and dissociation of symptoms that result from brain damage.

Consider the idealized case of a patient with a deficit to just *one* processing component—this patient should show impaired performance in all tasks that implicate that processing component and normal performance in all other tasks. We thus have a patterned dissociation in which there is a set of cooccurring symptoms with the presumption that all result from a disruption to a single processing mechanism. One example might be the case proposed by Caramazza and Berndt (1978) in which various symptoms associated with the clinical category of agrammatic Broca's aphasia are all assumed to result from a disruption to a syntactic processing component. In this case the agrammatism in production and the asyntactic comprehension disorders in these patients are assumed to reflect the disruption of a syntactic processing mechanism.

It is possible, however, to have situations where more than one processing component is disrupted. Consider a case where two processing components are disrupted. This patient should present with a set of symptoms due to the disruption of component 1, a set of symptoms due to the disruption of component 2, and a set of symptoms that result from the interaction of the disruption of components 1 and 2.

An example for this latter case might be a patient who has both a syntactic mechanism impairment as well as an impairment to a working memory system. In this case we would expect to find symptoms like those observed earlier for the syntactic mechanism impairment plus symptoms associated with the memory disorder. In addition we should observe symptoms that result from the interaction of the two disrupted

mechanisms; for example, particular difficulties with certain types of sentences.

I have presented these two ideal cases to highlight what is probably a rather simple point: The cooccurrence of symptoms in the first case (single processing component impairment) is of a different nature from that in the second case (multiple processing component impairment.) In the latter the symptoms are dissociable albeit with the constraint that the dissociation occurs along the boundaries determined by the two processing components—symptom set 1 can be dissociated from symptom set 2. A dissociation of the symptoms in the first case would constitute evidence against the theory that postulated the hypothesized processing component as responsible for the observed symptom complex. (This last claim is actually too strong as it requires that individual symptoms can result only from the disruption of the hypothesized processing component and not from any other impaired processing component. In any case, it should be clear that the study of the dissociation and cooccurrence of symptoms can be used to both test and formulate hypotheses about the structure of psycholinguistic processes—the point I am trying to make is that the mapping of symptoms to processing components can be a mapping of symptoms to one or many processing components.

In the discussion so far I have simplified too much. I have been talking of symptoms defined as poor performance on a task as if it were clear what that meant. Poor performance on a task could result for many different reasons. Any cognitive task will involve a complex set of processing components and, consequently, poor performance on such a task could be due to disruption to any one of the processing components that define normal processing on that task. Actually what I should have said, then, is not “poor performance” but rather “particular pattern of poor performance.” Thus, for example, we would not simply say poor comprehension but a particular pattern of poor comprehension.

Let me *summarize* the two main points I have tried to make. First, we must assume that brain damage can *on occasion* result in the fractionation of the normal cognitive system along theoretically significant lines and that the resulting performance in such cases bears a transparent relation to the functioning of the components of processing that define normal cognitive processing. Second, I have argued that symptom complexes can be of two types: Those where all the observed symptoms result from a disruption to a single processing component and those where the observed symptoms result from disruptions to independent processing components. The first type requires that symptoms necessarily cooccur and a dissociation of the symptom complex is grounds for rejecting the model that predicted the cooccurrence of symptoms. The second type requires that symptoms dissociate along the boundaries set by the

symptom subcomplexes for each processing component (see Caramazza & Berndt, in press, for a discussion of these issues applied to the case of agrammatic Broca's aphasia).

In practice however, brain damage rarely affects a single processing component totally while completely sparing other processing components. The typical situations we are confronted with are most often rather complex. In fact, even in those relatively clear cases of selective impairment to a single processing component our expectation should be that performance in such cases will reflect the contribution of four factors.

1. The contribution attributable to the "true" effects of the hypothesized disruption of a processing component.
2. "Normal" individual variation in performance.
3. The effects of "compensatory" operations.
4. Effects that result from disruptions to other processing mechanisms besides the hypothesized component.

I will assume that factors 2 and 3 do not pose insurmountable difficulties. Two can be handled statistically (although I must point out that it is not clear exactly how this is to be done—What is the appropriate control group? (See Shallice, 1979, for further discussion.) Three can be handled through the use of converging operations. We have assumed that pathology does not create *de novo* systems and thus compensatory operations will be alternative strategies presumably available to normal subjects.

It is the fourth factor that raises special difficulties. There are at least two basic difficulties: First, because it is not obvious which symptoms or performance features indicate the disruption of a processing component and, second, because in practice we either fail to test patients to insure that no other mechanism besides the one of interest is impaired in the patient(s) we are studying or, if we have data which indicate that some other processing component is disrupted, we ignore it either because we think that it does not contribute significantly to the critical performances or because the impairment is only a mild one relative to that of the target processing component. The first difficulty is a theoretical one and hopefully we can resolve it as we develop more explicit and detailed theories of cognitive processing. The second difficulty is both a theoretical and methodological one and must be addressed directly in our research if we are to progress in our efforts to understand language processing through the study of aphasia. Indeed, my claim is that unless we address this issue directly our research efforts will be hopelessly doomed. What I am proposing is that we adopt a methodological principle—which I will call the *sufficiency condition*—which states that the use of pathological cases to study normal processing requires an "exhaustive" analysis of their performance. The justification for proposing this methodological constraint is that the interpretation of cooccurrence and dissociation of symptoms as reflecting impairments of specific processing components,

as well as the interpretation of individual symptoms themselves, requires that we have sufficient information to unambiguously (or relatively unambiguously) determine that the observed performance can be assumed to result from a disruption of the hypothesized processing component.

I will develop further the issues I have raised thus far by considering briefly case-study and patient-group research methodologies and relate them to the problem of patient classification.

1. Case-study methodology. The single-case-study approach has the obvious advantage that if a patient is studied in sufficient detail we will have satisfied the sufficiency condition and thus provide considerable constraints on the interpretation of the pattern of impairments. In this case the performance on a specific task is less important than the overall pattern of performance on a set of tasks that jointly relate to various processing mechanisms. That is, we are not simply interpreting a single performance but a set of performances that mutually constrain the interpretation of any single performance—converging operations minimize the risk of overinterpretation of the performance in any single task.

However, even with these virtues, the case-study methodology is not problem free. Problems arise when we consider the issues of replication and extension for testing alternative interpretations of the symptom complex identified in the single case.

Thus suppose that we have studied case A who presents a pattern of symptoms X which we have interpreted as resulting from an impairment to processing component *a*. We could be concerned with two kinds of issues. One is simply replication. Here the basic problem consists in determining that we have in fact replicated the results in case A by finding case B who presents all of the “relevant” features found in A. Two problems may arise: (1) We may find that case B does not have all of the symptoms found in A or, (2) we may find that B has some additional symptoms to those found in A. The first situation most likely would not be called a replication and may be used to argue for the dissociability of the symptoms found in A and thus as a potential disconfirmation of the hypothesis that component of processing *a* was disrupted in A. The second situation, finding additional symptoms, could constitute a replication if we could motivate independently the claim that the additional symptom(s) in B result from a disruption to some processing mechanism other than X.

The second issue we could be concerned with regards extensions designed to test alternative interpretations for symptoms complex X. Extensions logically subsume replications and thus implicitly involve classification. An acceptable extension is only one which has established a clear replication. However, if we have a situation as the one we just described where case B has additional symptoms to those found in A the status of the presumed extension (and in a sense any extension could be interpreted

as a case B situation) is problematical. I don't have the space to develop these issues here and I am not sure that if we took the time to develop them that we would arrive at satisfactory analyses. However, I have developed the analysis sufficiently to allow me to make a critical point. Replications and extensions in single-case studies implicitly assume a typology of aphasia (classification). That is, replications and extensions are intended to generalize to particular types of aphasia. The generalization is not, strictly speaking, to a particular pattern of symptoms but to a patient type defined in terms of a disruption to some processing component. This nuance is not unimportant as the distinction we have drawn has important consequences for the definition of syndromes.

2. *The patient-group research methodology.* Unlike case-study research which can, in the extreme case, simply consist of a list of well studied patients making minimal assumptions about the relationship among these patients, the patient-group research method cannot proceed without some rather strong assumptions.

Group research is motivated by the assumption that the members that comprise the group show an important set of common characteristics. Patient-group research requires a slightly different assumption from that made for normal subjects; specifically, it must be assumed that the patients in a group all show a deficit to the same processing mechanism(s) *and* that patients do not have deficits to other processing mechanisms. This assumption of homogeneity is critical to group research. The problem is a practical one—group-study research typically involves grouping patients on the basis of classification criteria that are not sufficiently detailed and theoretically motivated to insure that the patients in the group are homogeneous in terms of the components of processing that are impaired in these patients.

Patient grouping is normally accomplished on the basis of classical syndrome type. (There is also the case of grouping by lesion site but this procedure will not be considered here. See Caramazza & Martin, 1983, for discussion.) The issue to be considered is whether grouping by classical syndrome type satisfies the requirement of group homogeneity for group research. Satisfaction of the homogeneity assumption depends to a large extent on the degree to which classical syndrome classification is based on the logic of fractionation of a modular cognitive system. Thus, a consideration of the appropriateness of patient group research as a means of explicating the structure of normal cognitive processes requires that we analyze the theoretical and empirical status of classical syndrome types.

THE THEORETICAL STATUS OF CLASSICAL SYNDROME TYPES IN APHASIA

The concept of syndromes plays a critical role in neuropsychological research. I would like to distinguish between a *psychologically weak and*

strong sense of the notion of syndrome. The weak sense simply defines a syndrome as the statistically reliable cooccurrence of a set of symptoms. (See Schwartz, 1982, for further discussion of this issue.) In this sense of syndrome the specific symptoms that are included in a set is determined on strictly empirical grounds. Thus, if it is repeatedly observed that a set of symptoms tend to cooccur, perhaps in relation to damage to a particular region of the brain, then we may wish to assign syndrome status to this constellation of symptoms. In this view it is not necessary that the symptoms included in a set be mutually exclusive; we can have the same symptom appear as part of two different syndromes. Furthermore, there is no strong restriction on the dissociability of the symptoms that define a syndrome, any symptom can be dissociated from the other symptoms in the set. Thus, for example poor comprehension can cooccur with or without poor repetition ability; there is no restriction on which symptoms need cooccur necessarily.

This notion of a syndrome does not assume that the cooccurring symptoms result from a disruption to a single processing mechanism; the symptoms may cooccur because of a disruption to several processing mechanisms that are distributed in neuroanatomically adjacent areas. A lesion in one part of the brain may result in the impairment of several processing mechanisms and give rise with some regularity to a psychologically accidental pattern of symptoms. The dissociation of the symptoms that make up such a syndrome is not theoretically independent. The study of syndromes defined in this manner would seem to be especially fruitful in mapping brain/function relationships. However, we must still deal with the problem of interpreting the psychological mechanisms responsible for the syndrome.

The psychologically strong sense of syndrome makes different assumptions about the cooccurrence of symptoms. A psychologically defined uni-component deficit syndrome assumes that symptoms cooccur because of an impairment to a single processing mechanism that is implicated in the normal execution of the processes that characterize the impaired functions that make up the syndrome. In this sense of syndrome, a set of symptoms *cooccurs necessarily*, and the dissociability of the symptom complex can be a problem for the status of the syndrome. In other words, what is asserted here is that if a processing component is assumed to be implicated in a set of language functions, then a disruption of this processing component will result in an impairment in all the functions that implicate that processing component in their normal execution.

Within this view we can also have multi-component deficit syndromes. This type of syndrome occurs when more than one psychologically independent processing component or module is disrupted. In this case the syndrome consists of a constellation of subcomplexes of symptoms that is dissociable *only* along the lines defined by homogeneous subcomplexes of symptoms. In other words, if the particular theory on which

the multi-component deficit syndrome is based is correct, then, dissociations of this syndrome should reflect the internal homogeneity of the subcomplexes—a subcomplex as a whole should dissociate and not just parts of the subcomplexes. (See Shallice and Warrington, 1980, for a practical application of this view to the case of acquired dyslexia.)

The two notions of syndrome presented here differ with respect to the role they assign to the fractionation assumption in the definition of syndromes. The psychologically weak notion of a syndrome does *not* require that the fractionation assumption be true: all that is required in this view is that symptoms cooccur in some statistically regular fashion. Furthermore, the level of analysis at which symptoms are defined is psycholinguistically arbitrary—grossly defined functions as comprehension, expression, repetition, reading, and writing constitute the defining dimensions of syndromes. In contrast, the psychologically strong sense of syndrome requires that the fractionation assumption be true and, consequently, that symptoms be defined at a psycholinguistically appropriate level.

The classical aphasia syndromes are of the psychologically weak type. Syndromes such as Wernicke's, Conduction, and Broca's aphasia are defined loosely as the cooccurrence of impairments to grossly defined functions.

Thus, quite independently of the empirical status of the classical syndromes, which we don't have the space to discuss here, we should consider whether on strictly theoretical grounds the syndromes defined in psychologically weak terms offer a reasonable basis for patient grouping in psycholinguistic research. The answer is an unequivocal no! Patients in groups formed on the basis of the classical syndrome types could have impairments to different psychological mechanisms and, thus, violate the requirements of homogeneity in group research. Research with groups constituted on the basis of classical typology is *not theoretically defensible*.

THE LOGIC OF NEUROCOGNITIVE RESEARCH

To conclude, I have argued that the classical syndrome typology in aphasia is theoretically indefensible. The rejection of the classical typology is not to be taken to mean that I am opposed to typologies, *tout court*. On the contrary, I have tried to suggest that a necessary part of the theoretical and empirical development of cognitive neuropsychology is the formulation of theoretically and empirically coherent aphasia categories—categories that are defined in a theoretically coherent vocabulary. This entails that aphasic symptoms be defined not in terms of the "pre-theoretical" symptoms such as comprehension and repetition disorder, or with a mixed vocabulary such as "posterior aphasic with good comprehension," but in terms of categories that are derived from a psychologically defensible model of language processing. In other words, I am suggesting that a neurocognitive research program requires that syndromes be of the psychologically strong type.

The approach advocated here has clear methodological implications for aphasia research. An important implication is that patient-group research in which groups are defined by classical syndrome types is methodologically unacceptable. These classical syndrome types do not have a clear psychological status, and the internal heterogeneity in these types violates the homogeneity assumption for group research. Thus, for example, averaging the performance of a group of fluent aphasics makes little sense since these patients may differ considerably in terms of the mechanisms that underlie their poor language performance. We must conclude, then, that patient-group research based on classical syndrome types should not be carried out if the goal of the research is to address issues concerning the structure of cognitive processes.

Do we conclude further from this discussion that patient group research in general should not be carried out? Are single case studies the only methodologically defensible research approach we can use with patient populations? I do not think that this discussion leads to the conclusion that all forms of patient group research are methodologically unacceptable. On the contrary, the discussion of the logic of the fractionation assumption and the definition of syndromes in psychologically strong terms supports a motivation for patient-group research. However, there are certain methodological and theoretical precautions that must be taken in carrying out a patient-group research program. First, the group must be defined by reference to psychologically defensible dimensions and, second, the fractionation assumption must be supported empirically to insure, within practical limits, that the group is homogeneous with respect to the component(s) of processing that might be impaired in each member of the group.

The first of these two constraints is theoretically motivated. In effect, what we are proposing is that the dimensions selected for defining groups are those that are considered on the basis of some theoretical framework to reflect the disruption of a processing component. Thus, suppose that we were to consider agrammatism as a reflection of the disruption of a syntactic processing component. We could, then, use agrammatism as a criterial dimension to define a group for research. However, it is immediately apparent that this theoretically motivated precaution is not sufficient to safeguard against violating the homogeneity assumption for group research. This problem could be best illustrated by considering another example. Suppose that instead of agrammatism we were to take asyntactic comprehension as the defining dimension for grouping patients. One can certainly make the argument that asyntactic comprehension could be a reflection of the disruption of a syntactic processing device. However, asyntactic comprehension could result from disruption to several different mechanisms. Consequently grouping patients on this criterion would not guarantee a homogeneous group with respect to the fractionation of homogeneous processing components. This latter observation motivates

the proposal of a second constraint in group research, viz., the empirical justification of the fractionation assumption.

In practical terms, the empirical justification of the fractionation assumption translates into a control of two types of factors: an empirical check that the group-defining behaviour is explicable in terms of the same processing component deficit in each patient in the group; and, an empirical check that no other processing components that might contribute importantly to the issues investigated are impaired in any of the patients in the group. To carry out these checks, what is required is an extensive and detailed analysis of each patient's performance on a wide range of tasks that are selected because of the *a priori* expectation that performance on these tasks would allow us to detect impairments to other processing components in addition to the one that serves as the group-defining criterion. Furthermore, patient's performance variability in the tasks that constitute the focus of the investigations cannot simply be assumed to reflect random variation. Attempts must be made to consider the possibility of disruptions to other processing components—group heterogeneity. Special tasks should be devised to insure that the variability can reasonably be considered as "random," or at least as unimportant, variation. We have called this approach, in which a group of patients is studied extensively and individual differences are pursued, the "group/case study approach" (Caramazza & Martin, 1983).

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