

Theoretical and Methodological Considerations in Aphasia Research and Practice: Connections Between Linguistics, Neuroscience, and Aphasia

David Caplan
Montreal Neurological Institute, Montreal, Quebec

ABSTRACT

The role of linguistic theory in clarifying aphasic disturbances of language is explored in relationship to disturbances of sentence comprehension. The study of aphasia can also be relevant to the construction of linguistic theory, on the basis of single case studies, series of case studies, and group studies. Deficit-lesion correlations are extremely useful for the development of theories of the neural basis for language but the location of a lesion is not predictive of any particular disturbance of language processing.

In this paper, I shall describe in very broad outline the general relationships between linguistic theory, neuroscience, and aphasiology, as I see them at this point in the development of these sciences. In part, this paper is a response to the paper by Caramazza on this topic in this volume (Caramazza, this volume, a), which deals with one aspect of the relationship between aphasiology and linguistics; namely, the use of data from aphasia to develop theories of language structure and processing. The interested reader may wish to compare some of the statements and analyses in this paper with those in the paper by Caramazza.

Stated in the most general possible way, there are four possible relationships between aphasiology on the one hand, and linguistics and neuroscience, on the other. The study of aphasia could influence our understanding of linguistics and/or neuroscience, or the study of linguistics and/or neuroscience could influence our understanding of aphasic phenomena. In my view, at present, an understanding of linguistic theory and models of psycholinguistic processing can and has greatly aided understanding of aphasic phenomena, and there are several examples of areas in which analyses of language breakdown have added considerably to our understanding of normal language processing. Our understanding of the functional neuroanatomy for language is largely based upon the analysis of the neuropathological correlates of aphasic deficits. Thus, three of the four areas are active profitable areas of research. The fourth area, however -- understanding aphasia on the basis of neuroscience -- will likely have to await a much deeper understanding of the neurological basis for language before it will become a profitable area of interaction. I shall illustrate these interactions in turn, and comment upon a number of methodological and theoretical issues as I proceed.

The Role of Linguistics and Psycholinguistics in Aphasiology

Linguistic theories of language structure and psychological (psycholinguistic) models of language model processing can be of use in aphasiology primarily in providing hypotheses regarding the nature of breakdown of core psycholinguistic processes in aphasic patients. By "core" psycholinguistic processes, I mean those processes devoted to the recovery of the form and literal meaning of words and sentences. Linguistic theory and psycholinguistic models provide detailed, specific categories within which to view aphasic impairments, which have been often attested and justified by

considerable study of the structure of normal language and its processing. These theories, therefore, provide a formal descriptive basis for aphasic deficits which otherwise are only described intuitively, and, in other cases, provide a means for disentangling complex sources of abnormalities in what otherwise would be confusing aphasic performances. Allow me to illustrate these points with an example drawn from the realm of sentence comprehension with which I am most familiar.

Consider the performance of patient KG, illustrated in Table 1.

=====

Table 1. Performance by KG on various sentence types.

SENTENCE TYPES WITH ABOVE-CHANCE PERFORMANCE

<u>Sentence Type</u>	<u>Correct</u>
1. Patrick persuaded a friend of Joe's to wash.	12/12
2. Patrick allowed Joe to hit himself.	10/12
3. Patrick vowed to Joe to pray.	10/12
4. The monkey was pushed by the frog and the rabbit.	12/12
5. The goat that hit the frog kissed the cow.	10/12

SENTENCE TYPES WITH CHANCE PERFORMANCE

<u>Sentence Type</u>	<u>Correct</u>
6. Patrick seems to Joe to be praying.	4/12
7. The monkey was given to the goat by the frog.	8/12
8. The goat that the frog hit kissed the cow.	3/12

=====

Table 1 illustrates a subset of sentences which KG was asked to comprehend. (A more complete description can be found in Hildebrandt, et al., in press). At first glance, there seems to be no particular pattern to the sentences that KG understands correctly, and those which he has difficulty understanding. Sentence types (3) and (6), for instance, have the same number of words and highly similar structures, as do sentence types (4) and (7) or (5) and (8). Without the tools of linguistic theory and models of sentence parsing and interpretation, this pattern of retained and disturbed comprehension of syntactic form appears to be random -- certainly, it is not obviously interpretable.

With the tools of linguistic theory and parsing models, however, the pattern is extremely specific. According to Chomsky's theory of syntactic structure (Chomsky, 1981) the sentences in Table 1 all contain empty categories; that is, noun phrases which are phonologically unrealized but which are present at abstract levels of syntactic representation. A depiction of the aspects of syntactic representation critical for our present purposes is illustrated in Table 2.

=====
Table 2. Structures of sentence types reported in Table 1.

SENTENCE TYPES WITH ABOVE-CHANCE PERFORMANCE

Sentence Type

1. Patrick persuaded a friend of Joe's PRO to wash.
2. Patrick allowed Joe PRO to hit himself.
3. Patrick vowed to Joe PRO to pray.
4. The monkey(1) was pushed trace(1) by the frog and the rabbit.
5. The goat(1) that trace(1) hit the frog kissed the cow.

SENTENCE TYPES WITH CHANCE PERFORMANCE

Sentence Type

6. Patrick(1) seems to Joe trace(1) to be praying.
 7. The monkey(1) was given trace(1) to the goat by the frog.
 8. the goat(1) that the frog hit trace(1) kissed the cow.
- =====

As can be seen in Table 2, the abstract empty noun-phrases are of two types -- PRO and TRACE. The reader will notice that KG has difficulty understanding only sentences which contain TRACE, not PRO. The reader will also notice that not all sentences containing TRACE are misunderstood. To discriminate between those sentences which are easily understood and those which are not understood we must have recourse to not only linguistic theory but also to models of parsing. In one model of parsing (Berwick and Weinberg, 1984), the noun-phrase which serves as the antecedent of a TRACE -- illustrated by the subscripts in Table 2 -- receives its thematic role -- Agent, Theme, etc. -- from the TRACE; that is, the TRACE is constructed in the phrase marker and receives a thematic role which it transmits to its antecedent. In parsing terms, the noun-phrase which serves as the antecedent of the TRACE cannot be assigned a thematic role until TRACE is constructed, assigned a thematic role, and co-indexed with its antecedent. This antecedent noun-phrase thus remains in a working memory system while it is missing a critical semantic feature -- its thematic role. This is true for all sentences containing TRACES. In the TRACE-containing sentences that KG understands, this series of operations -- construction of the TRACE, assignment of the TRACE, and transmission of the thematic role to the antecedant noun-phrase -- is carried out without the assignment of a thematic role to a second noun-phrase interrupting the process. The sentences which KG finds difficult are ones in which this process is interrupted by the assignment of a thematic role to another noun-phrase. For instance, in sentence (8), the subject noun-phrase of the relative clause, "the frog," is assigned the thematic role of Agent before the TRACE in object position in the relative clause is constructed. This entails that the matrix subject noun-phrase -- "the goat" -- must be retained in memory before receiving a thematic role while another noun phrase receives its thematic role. The same is true in the passive of the dative, illustrated in Sentence (7) in Tables 1 and 2. Because of the existence of so-called "inner datives" which can be passivized in English (The monkey was given the goat by the frog.), the TRACE cannot be assigned the thematic role of Theme immediately after the verb is presented but must be assigned a Thematic role only after the Thematic role

of Goat is assigned to the object of the preposition "to" in the prepositional phrase "...to the goat."

Thus, a combination of linguistic theory -- specifying the existence of different types of empty categories -- and models of parsing -- specifying the memory requirements involved in assignment of thematic roles to noun-phrases containing different types of empty categories -- combine to describe and explain the otherwise confusing pattern of sentence comprehension found in this patient. In general, linguistic theory and psycholinguistic models are extremely useful in delineating specific patterns of performance in the primary tasks of language use -- speaking, auditory comprehension, reading, and writing. The example I have chosen illustrates the utility of highly abstract aspects of linguistic and parsing theory in describing and explaining one small aspect of these deficits, but it should be noted that linguistic analyses of much more concrete aspects of word and sentence form and meaning also are relevant to aphasic disturbances. In our own work on syntactic comprehension, we have found double dissociations between patients' abilities to understand pronouns and reflexives, to understand the thematic structure of verbs and to co-index noun phrases, and many other dissociations related to overt aspects of syntactic structure. I have argued elsewhere (Caplan, in press a) that categories historically derived from clinical intuitions -- such as agrammatism -- can receive specific descriptions and explanations in linguistic and processing terms. The utility of linguistic theory also is not restricted to syntax and sentence structure but extends to the specification of the phonological form of words, and, therefore, to all of those processes, including reading and writing, which are related directly or indirectly to the phonological forms of words (see for instance Caplan et al., 1986, for some discussion of such relationships). Linguistics and psycholinguistics provide a large number of extremely useful, well-defined, and well-attested concepts for use in the description of aphasic phenomena.

The Utility of Aphasiology in the Construction of Linguistic Theory and Psycholinguistic Processing Models

In turn, the study of aphasic breakdown of language can and has influenced theories of language structure and functioning. For instance, Jakobson's (1941) notions of the relationship between phonemes and of the relative complexity of phonemes in the phonemic inventories of natural languages were in part developed on the basis of patterns of retention and disturbance of phonemes in aphasia. More recently, the study of acquired disorders of reading has reintroduced and provides important support for a view of reading which postulates both phonological mediation and direct visual whole-word recognition as separate means of accessing the mental lexicon from print (Coltheart, et al., 1980; Patterson et al., 1985). Rather than concentrate on the details of these analyses, however, I would like to focus in this section of this paper upon some theoretical and methodological aspects of the process of inferring normal function from the normal study of pathological performance in brain-damaged subjects. My comments are in part a response to papers by Caramazza (1986; this volume a), and shall be organized around his claim that inferences regarding the normal language processing system can only be made from pathological performances on the basis of single case analyses. The virtues of single case analysis are amply illustrated in the work reported by Caramazza in this volume and I shall not restate them here in detail. Single case analyses provide the best opportunity for studying language pathology in detail, and there is no replacement for detailed single case analysis if one's goal is to document a

particular deficit in an individual. On the other hand, though single case analyses are necessary to document an aphasic deficit, they are frequently not sufficient for this purpose or for the purpose of inferring some aspect of the normal language processing system from pathological performances. There are several reasons for their insufficiency.

The first is that, despite the detail in which single cases can be studied, there are frequently questions outstanding regarding the analysis of the deficit in a single case which cannot be empirically resolved, often for practical reasons pertaining to patient availability at the time when these secondary questions arise. Thus, many analyses of deficits in individual patients rest partially upon empirically unverified premises. For instance, the analysis of KG's performance discussed above assumes that KG has a single deficit affecting sentences containing TRACE in certain circumstances, as described, and not a series of deficits affecting different sentence types. This application of a simplicity argument -- a form of Occam's Razor -- is countenanced by the form of arguments in many sciences, but does not ensure that an analysis is in fact correct. These caveats do not and should not lead to the rejection of analyses based upon single cases, but they do and should lead to a conservative approach to acceptance of a theory of normal function based upon single case analyses. For instance, in the case of KG, an advocate of a theory of syntactic representations which did not specify TRACES (e.g., Bresnan, 1982, or Gazdar et al., 1985) would note the existence of case KG and the support it gives Chomsky's theory of syntactic representations without abandoning their own approach to linguistic structures on the basis of this single case.

A much more convincing approach is a series of analyses of individual cases all of which coalesce upon a particular aspect of theory of normal language theory and function. Caramazza (this volume b) presents such a series of case analyses, all converging upon a single model of the lexical semantic system.

Caramazza (1986; this volume a) makes a distinction between the study of a series of cases and the study of what he calls "groups." By "groups" he has in mind the averaging together of performance data from several patients. In brief, his argument is that because the functional deficits differ in each patient in a group, the functional system underlying performance on a given task is different in each member of the group. Only a careful case by case analysis can allow an investigator to be certain that his patients are functionally homogeneous; that is, that they all have the same functional deficits. If this analysis is performed on each of the tasks on which each patient is tested, there is no need for averaging group data. If this analysis is not performed, averaging together group data is illegitimate because of the likelihood of the heterogeneity of the functional systems underlying performance in individual patients.

Though Caramazza is correct that measures of central tendency and deviation from central tendencies (the means, medians, and standard deviations which enter into most statistical analyses) derived from performance of groups of patients on a particular task cannot delineate the functional deficits of each of the patients on that test, I believe his argument that single cases and series of cases are the only source of information pertaining to aphasia from which one can infer aspects of normal function is overstated and erroneous. There are at least two circumstances in which what he calls "group data" -- measures of central tendency (his "averaging") and deviation from central tendency -- of groups of aphasic patients are useful in the validation of aspects of normal function.

The first is when we can predict that one aphasic deficit entails a second abnormality in performance, because the first function which is deficient is needed for the accomplishment of the second. For instance, I have argued (Caplan, in press a) that the omission of function words and inflectional morphemes in expressive agrammatism arises at a stage of sentence planning that involves the creation of syntactic structure and that, accordingly, expressive agrammatism (defined in terms of the omission of these vocabulary elements) is necessarily accompanied by the simplification of syntactic structures (above and beyond the obligatory simplification of structure that would follow from the omission of these vocabulary elements). Let me construct a scenario in which the use of group data is a legitimate, necessary and useful tool to be used in the validation of this hypothesis.

Suppose that we have some quantified measure of the omission of function words and inflectional morphemes, and, similarly, of the simplification of syntactic form. In Caramazza's (1986, this volume a) terms, we have observed some quantifiable performance, O(1), relating to the omission of function words and inflectional morphemes, and a second quantifiable performance, O(2), related to the simplification of syntactic structure. We predict that, because of the psychopathogenesis of O(1) in relationship to a model of sentence production, every individual who manifests O(1) will manifest O(2). Now, suppose that every individual who manifests O(1) does, in fact, manifest O(2), but to such a slight degree that each individual patient's performance is not distinguishable from that of normal subjects with respect to O(2). How can we ascertain that the prediction made by our theory is validated? Simply examining each individual subject will not tell us, nor will, in the absence of some measure of consistency and magnitude of the performance of each aphasic subject with respect to normal performance in the domain O(2), the analysis of the entire series of patients on a case-by-case basis. Statistical treatment of the data, on the other hand, can clearly establish whether the repeated observance of a difference between normal and aphasic subjects of small degree of magnitude is statistically reliable and significant, and therefore will be invaluable in validating the hypothesis that the two behaviors co-occur, and, thereby, the hypothesis that they are functionally related.

More complex circumstances arise when a minority of patients do not show the expected co-occurrence of pathological performances. These circumstances raise the question of the interpretation of unpredicted and apparently disconfirming data regarding a performance in the face of statistically significant patterns of data that conform to the hypothesis. These are extremely difficult questions of interpretation, but there is nothing special about the case of aphasia regarding these problems. Experiments with normal subjects frequently show that a minority of subjects and/or items do not produce performances in the same direction as the statistically significant majority performances, and we do not abandon theories of normal performance based upon the statistically significant majority performances because of these unpredicted apparently disconfirming minority instances of behavior. The question of what significance these unpredicted minority performances have is difficult. They point to the need for a deeper level of theory construction which will tolerate, and even predict, such variation in performance patterns. However, the need for deeper theories does not invalidate the interpretation of statistically significant group data in these circumstances. Indeed, the question of the significance of a minority performance only arises when statistical analysis of the entire group performance shows that the majority performance is statistically reliable.

The second instance in which averaging is useful is in evaluating claims related to what linguists term "markedness." Markedness refers to the relative complexity of two structures. The notion of markedness may also be applied to processing. In cases of what we may term a "strong" markedness relationship, one processing operation is said to be an integral and necessary part of a second. In these cases, impairment of the first operation would necessarily entail a disturbance of a second processing operation. Predictions of this sort hold regardless of the functional deficit a patient has, since there is no conceivable functional deficit which could affect the first processing operation and leave the second intact. In terms of Caramazza's (1986, this volume a) formulation, the functional deficits (his Ls) in each patient are irrelevant to the predictions made by markedness theory with respect to behavior of individuals and groups of individuals. Just as in the case of the association of deficits, using measures of central tendency and of dispersion of individual subjects' performances is legitimate and revealing with respect to how aphasic performances bear on this aspect of theories of normal performance (for an example of such an analysis in terms of markedness of syntactic structure see Caplan et al., 1985).

In short, there are a number of approaches to analysis of data pertaining to pathological performances in brain-damaged subjects which are useful in inferring aspects of normal language and processing from these pathological performances.

The Relationship Between Neural Sciences and Aphasiology

The study of aphasic deficits in conjunction with measures of organic lesions has been the most profitable source of information pertaining to the neurological basis for language. These deficit-lesion correlates have led to virtually all of our present theories regarding the locations in the brain in which language is stored and processed, and there is every reason to believe that such correlates will continue to be important sources of theory in the future. Indeed, given our increasing ability to delineate the functional deficits in individual patients and to measure a wide variety of aspects of lesions using new imaging techniques such as PET and SPECT scanning, this database is likely to expand considerably in the immediate future and to lead to new and interesting theories of the neural basis for language.

On the other hand, given present limitations regarding our understanding of the neural basis of language, it seems to me that knowledge of the neuropathological features of a lesion gives almost no information about the language functions which are retained and disturbed in an individual patient. There is, admittedly, a statistically reliable correlation between lesion site as measured on CT scans and the classical aphasic syndromes (Nasser and Hayward, 1978; Basso et al., 1986), but these correlates are extremely general and admit of numerous exceptions. Moreover, the classical aphasic syndromes themselves are heterogeneous with respect to the functional deficits in individual patients (Schwartz, 1984), and the ability to predict (with some 85% certainty) that a patient will have a particular syndrome but does not tell us what particular form of a syndrome he will have. For instance, though we can predict that a patient with a primarily frontal lesion is likely to have "Broca's aphasia," we cannot predict whether the particular form of aphasia will consist of apraxia of speech, dysarthria, some degree of anomia, stereotypic utterances, repetitive utterances, retention of automatic sequences, agrammatism, other forms of nonfluency, an associated dysgraphia, dyslexia, or other pathological features. The

prediction that a patient is likely to have "Broca's aphasia" on the basis of his lesion site is, therefore, of little use in establishing the exact deficits a patient has, though it may delineate a broad range of possible deficits with some accuracy. This restriction on the predictions based upon neuropathological features of lesions seems to me to be principled: that is, it follows from basic aspects of the relationship between neurological structure and language function.

Though we may accept that language is related to the brain because of particular features of neural elements and their organization, we do not now know what those features are. Recent studies of histological features which seem to distinguish neurons in the traditional language areas from those in adjacent areas of association cortex, though a first step towards the identification of the cells which support language, do not clearly indicate what features of the cells are relevant to the support of language. For instance, Galaburda (1982) has shown that certain cells in Broca's area stain with Braak's stain, a marker of lysosomal contents. Lysosomal contents, however, reflect the excretory products of cell metabolism, and their relationship to the features of cell structure and function which are relevant to the support of language is far from clear. In the absence of knowledge about which neurons and which features of neurons are relevant to language processing, we are restricted to hypotheses regarding the general localization of areas of the brain in which language functions are carried out. Recent studies of my own (Caplan et al., 1985) and a review of the literature on localization of specific language function (Caplan in press b; in press c; submitted), show that individual components of the system devoted to core psycholinguistic processing, though all localized within the association cortex of the perisylvian region, are highly variable with respect to the exact locale and extent of tissue devoted to each subcomponent. If this is the case, knowledge of lesion site in an individual would, in principle, be inadequate to predict the particular deficits a patient has in this sphere of language.

Conclusion

I have discussed some of the relationships between aphasia, linguistic and psycholinguistic theories, and the neural sciences. Linguistic theory and aphasiology are mutually interactive, and are likely to exert more mutual influence as studies of language pathology become more detailed. Linguistic and psycholinguistic characterizations of aphasic deficits, coupled with lesion parameters, have been and are likely to continue to be a critical source of data for the construction of neurolinguistic theory. I have argued that, at present, knowledge of neuropathological features of lesions alone tells us very little about the language deficits a patient has. The development of a deeper and a more explanatory neural science, which would relate cellular elements and organization of neurons to specific language functions, would allow this aspect of the interaction between the neural sciences and linguistic and psycholinguistic sciences, and of that between neuropathology and linguistic aphasiology, to develop.

ACKNOWLEDGMENTS

This paper was written while the author was supported by a Chercheur Boursier Senior I Award from the FRSQ (Quebec). Research undertaken in this paper was partially supported by the Medical Research Council of Canada (Operating Grant MA 8602) and a grant from the Sloan Foundation.

REFERENCES

- Basso, A., Lecours, A.R., Moraschini, S. and Vanier, M. Anatomicoclinical correlations of the aphasias as defined through computerized tomography: Exceptions. Brain and Language, 16, 201-230, 1986.
- Berwick, R. and Weinberg, A. The Grammatical Basis of Linguistic Performance. Cambridge, Mass.: MIT Press, 1984.
- Bresnan, J. The passive in lexical theory. In J. Bresnan (Ed.), The Mental Representation of Grammatical Relations. Cambridge, Mass.: MIT Press, 1982.
- Caplan, D. In Defence of Agrammatism. Cognition, (in press a).
- Caplan, D. The biological basis for language. In F. Newmeyer (Ed.), Linguistics: The Cambridge Survey. Cambridge, UK: Cambridge University Press, (in press b).
- Caplan, D. Neurolinguistics and Linguistic Aphasiology: An Introduction. Cambridge, UK: Cambridge University Press, (in press c).
- Caplan, D. On the cerebral localization of language functions. Brain, (submitted).
- Caplan, D., Baker, C. and Dehaut, F. Syntactic determinants of sentence comprehension in aphasia. Cognition, 21, 117-175, 1985.
- Caplan, D., Vanier, M. and Baker C. A case study of reproduction conduction aphasia I: Word Production. Cognitive Neuropsychology, 3, 99-128, 1986.
- Caramazza, A. On drawing inferences about the structure of normal cognitive systems from the analysis of patterns of impaired performance: the case for single-patient studies. Brain and Cognition, 5, 41-66, 1986.
- Caramazza, A. Valid inferences about the structure of normal language processes from patterns of language dysfunction are only possible for single patient studies. (this volume a)
- Caramazza, A. The structure of the lexical system: Evidence from acquired language disorders. (this volume b)
- Chomsky, N. Lectures on Government and Binding. Foris, Dordrecht, 1981.
- Coltheart, M., Marshall, J.C. and Patterson, K, (Eds.), Deep Dyslexia. London: Routledge, 1980.
- Galaburda, A. Histology, architectonics and asymmetry of language areas. In M.A. Arbib, D. Caplan and J.C. Marshall (Eds.), Neurological Models of Language Processes. New York: Academic Press, 1982.
- Gazdar, G.E., Klein, E., Pullum, G. and Sag, I. Generalized Phrase Structure Grammar. Cambridge, Mass.: Harvard University Press, 1985.
- Hildebrandt, N., Caplan, D. and Evans, K. The man(i) left t(i) without a trace: A case study of aphasic processing of empty categories. Cognitive Neuropsychology, (in press).
- Jakobson, R. Child Language, Aphasia and Linguistic Universals. Mouton, The Hague, published in translation, 1941.
- Naessar, M. and Haywood, R.E. Lesion localization in aphasia with cranial computed tomography and the Boston Diagnostic Aphasia Examination. Neurology, 28, 545-551, 1978.
- Patterson, K., Marshall, J.C. and Coltheart, M. Surface Dyslexia. London: Erlbaum, 1985.
- Schwartz, M. What the classical aphasic syndromes don't tell us, and why. Brain and Language, 21, 1-8, 1984.