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A Theory of the Deficit: A Prerequisite for a Theory of Therapy?

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> *"If we are to serve aphasic patients better . . . and to turn clinical art into clinical science, then we must begin to develop explicit and falsifiable theories of treatment, to test their assumptions and contrast various theoretically-driven forms of treatment."*–Holland (1991)

The relationship between theory and therapy has become a subject of increased discussion in recent years, as volumes of *Clinical Aphasiology* can attest. Nonetheless, are we in agreement about why we need to develop theoretically driven forms of treatment? My assumption is that theories are necessary to relate the nature of the disability or impairment to what we are trying to do about it. This means that we need first to provide a clear statement or hypothesis about the nature of a deficit before we can then develop theories about what forms of therapy or intervention are appropriate and effective and why. Becoming an adult with aphasia produces many and various effects, of course. It therefore follows that, to address a range of effects, we will need to compare not only different theoretical accounts of the same phenomena but also different types of theories.

It does not matter that we may have different theories. What does matter is that we are explicit about them and explicit about why a particular therapy should be appropriate. The only falsifiable theories are explicit ones, as Holland (1991) makes clear: "If clinicians and clinical researchers simply demand from themselves a written justification of why they believe such and such will work in treatment and why something else might not, a major step will be taken in the development of testable hypotheses about the process of treatment in aphasia." In this paper, I suggest one approach to making an explicit hypothesis about why a specific treatment for a specific aspect of the language impairment should or should not

work. This is far from comprehensive; the theoretical basis that I describe here does *not* address many of the aspects or variables involved in providing therapy for someone with aphasia—it deals only with the language impairment itself.

My premise is that any treatment that can address the language deficit in aphasia must begin with a theory about the *nature* of the deficit. Another basic premise, and one on which the previous premise is prefaced, is that the deficit cannot be inferred from observing or examining the surface symptomatology, as is suggested by much current research (Berndt, 1991; Howard & Patterson, 1989). This means that treatment cannot be planned only on the basis of the observed presence or absence of a particular linguistic feature in the language of a person with aphasia. Therefore, any treatment for the language deficit requires a prior hypothesis about the underlying nature of an individual's language impairment that explains why a particular pattern of symptoms might be observed. This hypothesis can be derived from a number of converging sources, for example, from informed observation of the person with aphasia communicating in a variety of contexts or from performance on tests of a range of specific language tasks suggested by the observation (Byng, in press).

The crucial question here is what informs the observations and what motivates the testing that shapes the hypothesis. A current approach is to draw on the basic architecture of the language-processing system offered by cognitive neuropsychological models along with a logical way of thinking about the deficit. In addition to the basic architecture, psycholinguistic concepts about representations and processes that may be involved in comprehending and producing language are used (e.g., Coltheart, Sartori, & Job, 1987).

Toward the end of the 1970s, cognitive experimental psychologists were becoming interested in testing empirically based architectural models of the normal language-processing system by investigating whether damage to one or more of the components of these models could account for different disorders of language processing (Coltheart, Patterson, & Marshall 1980). This methodology has developed into a discipline known as cognitive neuropsychology. The basic approach is to try to identify where the deficit underlying the impaired language arises within these empirically based, multicomponent, modular representations of language processing. Current models usually represent the component modules of the language-processing system through box-and-arrow diagrams, in which the boxes represent stores of information and the arrows represent processes linking them. These models offer a logical, coherent framework with which to approach a basic account of a language impairment. A patient's performance pattern can be elucidated in terms of a pattern of both the impaired and preserved components of the language-processing system.

Using concepts about, and the architecture of, the normal language-processing system has a number of benefits that can affect the design of a therapeutic procedure and the interpretation of the outcome (Howard & Patterson, 1989). One of the benefits of this approach is that it can provide some theoretically motivated predictions of the outcome of therapy. Because the models try to make explicit the relationships between different components of the language system, it is possible to hypothesize about what should happen to one component when a different component is treated. Thinking through the implications of the architecture of the language-processing system can assist in making predictions about the effects of a therapy. This in turn can help to measure the effectiveness of intervention; if we can demonstrate that our therapy has affected those aspects of the language system that we anticipated should be affected, but not those that we did not anticipate should be affected, we are in a stronger position to claim that our intervention was influential in bringing about the change. Alternatively, if the therapy does not produce the expected outcome, then we are in a better position to ask why.

This suggests that pre- and posttherapy measurement of not only treated but untreated items must be carried out, as has become well established. Additionally, there should be measures of untreated language functions, some of which are related to the function being treated and some of which are not. The relationships between the functions must be specified so that predicted improvements in related but untreated functions can be measured, providing yet more evidence for the efficacy of the therapy. In this paper, I will illustrate this aspect of theoretically driven therapy at length.

In a sequence of studies with a small number of people with agrammatic Broca's aphasia, the sentence processing deficits demonstrated by these people on a number of specific assessments were interpreted to be in line with the "mapping deficit hypothesis" of Schwartz, Linebarger, and Saffran (1985). This hypothesis suggests that patients with symptoms of agrammatism cannot assign, or map, appropriate semantic (or thematic) roles onto Noun Phrases in particular structural positions; that is, they are unable to coordinate sentence form and sentence meaning to interpret even simple sentences. The hypothesis suggests that this might affect not only sentence comprehension but also sentence production. Current linguistic theories suggest that this kind of mapping is not a single process but involves two types of information and procedures: lexical information and general procedures that apply to specific sets of thematic roles. The lexical representation of a lexical item (e.g., a verb) specifies which thematic roles are involved and how they are assigned to particular positions within the sentence structure. In addition, as there are systematic regularities in the assignment of thematic roles (e.g., Agents are usually mapped onto Subject NP positions), mapping may also involve procedures that are not item specific (Byng & Black, 1989).

This hypothesis about our patients was made on the basis of specific features of sentence comprehension and production; namely, that the patients were unable to comprehend reliably simple reversible sentences, either active declarative, passive, or locative sentences. Additionally, their sentence production was structurally limited, with the patients relying on simple utterances, either holophrastic or at least minimally structured. Some of the patients also made errors assigning thematic roles in a comprehension task for single words, suggesting that their problems in comprehending sentences were not because of failure to parse the syntax.

With the first patient, BRB (Byng, 1988), a therapy task was devised that focused only on enhancing the comprehension of one set of thematic roles and on input. We adopted this approach on the premise that if an aspect of the language system is being changed (in this case, we hypothesized this to be the procedure that maps thematic roles and grammatical relations), then the effects of that change should not be confined to items practiced but should be observed in any task involving the changed procedure. Therefore, if a therapy affects a process or a set of processes involved in mapping sentence form and sentence meaning, any language task involving these processes should show benefit, provided there are no other deficits remaining that would further interfere with that process. In this way we can plan for and anticipate theoretically specified generalization. The rationale underlying this therapy, then, was that if the hypothesis about the mapping deficit was correct, then remediation of the mapping deficit in one modality should bring about improvement in the untreated modality and in untreated thematic roles.

The results of the therapy demonstrated that BRB had made a statistically significant improvement in comprehending all sentences where he had to map thematic roles onto syntactic relations to interpret the sentence correctly. This included not just the sentence types used in therapy but also reversible simple active sentences and passive sentences. BRB had therefore learned more than just a strategy for interpretation of sentences, such as that the most agentlike entity will be in the subject NP position, which would facilitate comprehension of active sentences. Because application of that strategy would not have allowed BRB to interpret locative or passive sentences any better, he must have learned some principle about mapping thematic relations.

After the therapy, a statistically significant shift was measured in the production of narrative speech, away from producing single words or phrases and toward combining nouns and verbs to form structured utterances. It therefore seemed that the hypothesis about the deficit underlying his problems in one aspect of both sentence comprehension and production might have been correct—treatment effects had generalized beyond the treated sentence types both to untreated sentence types and to a different modality after treatment of only a limited number of specific

exemplars. Other aspects of BRB's impaired language that were not related to the therapy did not improve, however, so that although there is generalization, it is only a specific one related to the therapy.

The same therapy was attempted unsuccessfully with a second patient, JG (Byng, 1988), which then prompted the use of a **further** therapy procedure. This therapy was then replicated with another severely agrammatic patient, AER (Nickels, Byng, & Black, 1991), to investigate its effects in more detail and to reexamine the hypothesis about the underlying disorder in more detail. The basic purpose of the therapy task was to increase the patient's awareness of the roles that the different entities involved in an event play and how those roles are represented in the structure of the sentence. The therapy was designed not to get the patients to practice comprehending and producing sentences of various types but rather to convey to them some aspects of predicate-argument structure (Nickels, Byng, & Black, 1991; Byng, 1992).

Both JG and AER demonstrated statistically significant gains both in comprehension of simple active agentive sentences and in production of structured utterances. This improvement in production was not confined to picture description tasks but also included gains in production of narrative speech and, according to observations and reports from relatives, in spontaneous day-to-day speech. After the therapy both AER and JG were able to produce utterances with more structure and greater content, showing a particular improvement in producing verbs. They were not just producing sentences practiced in therapy better than previously, but they were also generating their own language more effectively. Their ability to comprehend specific sentences did not show such marked improvement, however, as BRB's had; improvement was confined to the sentence type used in therapy, but not just to the lexical items used in therapy. It seems that they had learned a principle about how to interpret that type of sentence. Additional tasks were performed pre- and post-therapy using processes that were predicted not to be involved in therapy and therefore served to act as controls for those aspects of the language that were predicted to be positively affected. In both patients, these control measures showed that the improvements were specific to those aspects involved in the therapy and not to other, unrelated processes. This result was taken to reinforce the specific effects of the therapy.

Because the therapy focused on a procedure rather than on a set of structures or items, we planned for generalization through types of stimuli other than those being worked on in therapy. Because the patients were learning something about structuring sentences involving a specific type of verb, it should not matter which specific verbs within that class were being used, because they were learning not item-specific information but rather some procedures about combining nouns and verbs. We measured generalization of this kind. For example, in naming of action pictures, a subset of

which had been used in therapy (not the specific pictures, but other pictures depicting the same actions as those in the assessment set), JG and AER showed improvement in naming all the pictures, and there was no difference between the treated and untreated items.

The predictions made about patterns of improvement again held good in these patients. Although we were working on aspects of sentence production in our patients, the therapy focused only on revealing to the patients aspects of predicate-argument structure, so we did not anticipate that there would be any increase in other aspects of production, such as the ability to produce closed-class items or to use obligatory determiners, for example. Indeed, these aspects of production did not improve as a result of therapy. This kind of finding strengthens the case for the efficacy of a specific therapy: If the positive outcome was a result of spontaneous or nonspecific improvement, then it is difficult to explain how predicate-argument structure could have improved spontaneously when closed-class items showed no improvement.

The premise of this paper is that theories about the nature of language deficits are a necessary precursor to developing therapies specifically addressed to a given language impairment. However, simply having a detailed analysis of the deficit does **not** by itself suggest the formulation of specific theraputic procedures to effect change. That is the province of theories of therapy. Three recent studies of therapy for people with naming problems show that knowing what the deficit is does not always entail knowing what to do about it. These three studies (Hillis, 1989; Jones, 1989; Marshall, 1989) describe three patients who had poor naming ability, despite having good comprehension of semantically related items as measured on picture-word matching tasks. Table 1 shows some features of the performance of the three people, with hypotheses about the locus of the naming deficit and the aim of the therapy in each case. Although the three studies employ slightly different descriptions, all three people have some deficit between the semantic system and the phonological output system. Given the inexplicit nature of current models, it is difficult to characterize the specific form that the deficits take, but the deficits seem approximate.

However, even the small amount of additional data provided in Table 1 makes clear that these three patients are not the same in many other respects. PC is more like a Wernicke's aphasic, whereas RS is more like a Broca's aphasic. These differences aside, however, one might suppose that *if* there is some direct link from the analysis of the naming deficit to therapeutic intervention, similar therapies might have been generated; this was not the case. On the basis of their spontaneous speech, Jones's and Hillis's patients sound like each other more than either sounds like Marshall's patient, yet the intervention strategies employed were quite different for all three.

Table 1. Comparison of Language Deficits of Three Aphasics Who Received Therapy for Naming Deficits

Descriptive and Evaluative Data	*Subject/Study*		
	Pt. 2 (Hillis, 1989)	*PC (Jones, 1989)*	*RS (Marshall, 1989)*
Speech characteristics	Fluent speech minus content words	Empty speech with omission of content words	Nonfluent, Broca's type aphasia
Word-picture matching	100%	94%	98%
Oral naming	66% (25% semantic errors)	0% (60% no response; 40% neologisms)	32% (few semantic errors)
Written naming	11%	0%	44%
Oral reading	67%	0%	good
Repetition	profoundly impaired	unable	probably good
Hypothesised mechanism of naming problems	an inability to retrieve the correct phonologic representation of the word oral naming	some of his problems in accessing the output form of words possibly arose because of weak semantic drive	a breakdown in the route linking semantics and phonology
Aim of therapy	to improve frequency and accuracy of their writing attempts	to improve picture naming as a measure of increased access to the phonological form due to enhanced access to semantic information	to rebuild the communication between RS's good semantic knowledge and his intact word phonology

Jones used a predominantly semantic strategy based on the idea of judging the relatedness of pictures or words to a central target item. After the judgment task, the patient ordered lettered tiles to form the target word; then, if he could say the name easily, he was encouraged to produce it, but no emphasis was laid on doing this. Hillis's therapy, which involved a cueing hierarchy to facilitate written naming, was devised by noting the sorts of stimuli that sometimes elicited correct names. After this therapy, a hierarchy for facilitating oral naming was introduced. Marshall's therapy for RS consisted of a three-stage procedure in which the patient first identified from a list of five related words one of the items

that was being described. Then he "brainstormed" around a target picture, miming, drawing and giving as many words or ideas as he could related to the picture. Finally, he matched a printed word to the picture from a choice of five and read it aloud.

These three therapy procedures have some similarities, but it is hard to compare them directly because even the two semantically based procedures of Marshall and Jones have very different task demands. The precise formulation of each seems to result from the constellation of observed performance features, not just the identification of the deficit's approximate locus. For example, because her patient (RS) could read aloud well, Marshall incorporated production of the phonological form into the treatment of oral reading with a semantic task (matching of the printed word to a picture) to try to activate the impaired link between semantics and output phonology. Jones's therapy was designed to avoid as much auditory input as possible because single word auditory input was severely impaired for PC. In addition, the lettered tile component to the therapy was introduced to enhance the concept of phonological segmentation; this indirect introduction to the idea of a word being segmented into sounds served as a precursor to future work on phonological segmentation. Hillis's therapy derived from the observation that her patient was frequently able to write picture names when given scrambled anagrams.

The outcome of each of these therapies was, not surprisingly, quite different. The studies raise a number of questions about both the nature of the patients' deficits and the nature of the therapies, such as what it means to locate the naming deficit in the same place when each of the three patients studied clearly differs from the others in terms of many other aspects of language processing. Are the two semantically based therapies the same? Were the patients learning the same information from the therapies? Can we *assume* that the patients are getting the same information from similar therapies? If I were to try these therapies, would the way I implemented them be the same as the way they were implemented by these therapists?

It is clear that deficit analysis does not *by itself* suggest the formulation of specific therapeutic procedures. Because this is what therapy aims to do, theories about how to effect change through therapy must be derived from sources other than the analysis of the deficit. My position is that an analysis of the nature of the language impairment, rooted in a relationship to the normal language-processing system, should provide the most informative way for us to describe the deficit as a basis for therapy. We need to know more precisely how the language system is impaired, what impairments can be treated, what psycholinguistic variables should be controlled in therapy, and what we can predict the effects of the treatment to be. The decision about whether these impairments should be treated and how represents a different set of questions not addressed by the

account of the deficit; that issue rests on the development of a theory of therapy. I consider what I have been describing to be just one of the prerequisites toward developing such a theory of therapy for language deficits, but it is clear that theories for therapy are as crucial as theories of the deficit if our treatments are to progress.

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