Cognitive neuropsychology generally, and cognitive-theory-driven approaches to treatment specifically, have been getting big press, with everyone from computational modelers to speech-language pathologists participating in the excitement and praise. From the perspective of clinical aphasiology, the source of this excitement is that cognitive neuropsychology provides a principled way of conceptualizing a language disorder, which thus can be related to a theoretical foundation and possibly even be tested by a number of approaches, including computer modeling (Hinton and Shallice, 1991). The processing models of cognitive neuropsychology have also shown clinicians the potential productivity of thinking about language as a number of processes and as a set of components. They have provided a logical way to proceed by allowing hypothesis testing about the way language is processed in the brain. This detailed study of deficits in aphasia can influence ways to treat aphasic and other cognitively impaired individuals. Nevertheless, I believe that the contributions that theory-driven therapy can make to treatment for aphasia are limited in both number and scope.

This limitation is primarily because cognitive neuropsychology has never pretended to be a theory of therapy and in fact has only indirect relationships to therapy. Caramazza (1989) has articulated this point well; he notes that “an informed choice [from among intervention approaches] cannot be made in the absence of an equally rich theory of the modifications that a damaged cognitive system may undergo as a function of different forms of intervention.” What Caramazza is implying is that even after we get the deficit correctly nailed down by using a model that delineates the sources for any given functional impairment, it does not
necessarily follow that we know what to do about it. The theories that drive theory-driven treatment are not theories about how to fix deficits; rather, they are theories about how and why the deficits occur.

This is probably nowhere better illustrated than in Howard and Franklin’s detailed case study, Missing the Meaning? (1988). Described on its cover as “a textbook example of how to do cognitive neuropsychology,” this work concerns the single-word processing of MK, a patient who seems to have Wernicke’s aphasia. Using a box-and-arrow processing model derived from Morton and Patterson (1986), Howard and Franklin present data that they gathered over a 3-year period, delimiting those aspects of the model that have apparently been rendered inaccessible (in MK’s case, as a result of his brain damage) and those that have not. For some individuals, such as Gilbert (1992), this work is taken as a major contribution not only to cognitive neuropsychology but to the clinical enterprise as well.

Howard and Franklin’s interpretations of their results will be of continuing great value to SLP’s [sic] who at times are convinced that a particular patient may have just about everything. Take heart, these authors will show you how to apply a model-based interpretation to aphasic problems, a useful way to approach both diagnosis and treatment. (p. 54)

But in their closing chapter, Howard and Franklin themselves make the following comment concerning therapy:

We are both aphasia therapists, and one of our aims in interacting with MK was to “improve” his language, to try to lessen the limitations that his aphasia imposed on his life. Over the years that we have been seeing him, we have tried a number of different treatment approaches, partly in order to improve his language, but also as an experimental tool in trying to understand his problems. None have been particularly successful, and so no results seem worth reporting. We wonder, sometimes, whether our failures are the inevitable result of multiple and severe processing impairments. The difficulty that is the greatest impediment to communication is probably his severe problem in sentence comprehension. (p. 113)

In terms of clinical remediation, Howard and Franklin’s Missing the Meaning? might be better titled Missing the Boat. But why does their work seem so irrelevant to their therapy? Part of the answer of course is that MK’s major difficulty (i.e., sentence comprehension) was more encompassing than the single-word tasks that were the focus of the study. But there is another, substantially more important reason. Howard and Franklin never intended to deal with treatment in their work. Instead, they were interested in the precise description of a deficit. To make this or any similar aim relevant to treatment, another type of theory must be invoked.
My thesis is that relevant theories for such a goal are not cognitive neuropsychological in nature; rather, they are theories about how one conducts aphasia therapy. The remainder of this paper will explore the theory of therapy and theory-driven treatment models.

WHAT DOES THERAPY DO TO A PATIENT?

An underlying assumption of theory-driven therapy as it relates to the clinical process is that deficits and therapeutic techniques will be straightforwardly and unambiguously related. Isolating the functional deficit will directly entail a therapy technique to repair a broken or destroyed box. This is a theory concerning the nature of the therapeutic process; however, it is only one of many equally viable underlying assumptions and alternatives. It certainly is possible that theory-driven therapy may furnish data that is ultimately relevant to an "exercise the deficit" theory of therapy, but there are a number of other plausible contenders for Caramazza's "rich theory of the modifications that a damaged cognitive system may undergo as a function of different forms of intervention." What follows is a sampling from among them. Before beginning this discussion, however, it is necessary to point out that the experimental evidence for all of them is limited, despite the ease with which some of them are accepted.

Are cortical pathways being reorganized? This is an old idea, possibly best exemplified by Luria's approaches to the treatment of aphasia. Techniques such as pairing a relatively undamaged skill in hierarchical manner with a damaged one (e.g., "deblocking") or applying an unusual response form to a well-practiced skill (e.g., writing words in the sand with one's fingers) are said to result in some reorganization of brain function that facilitates the ability to perform deficient behaviors.

Are previously untapped capabilities of the undamaged areas of the brain being trained to take over some new functions (in the case of aphasia, language functions)? Melodic Intonation Therapy (MIT) (Helm-Estabrooks & Albert, 1991), is a pertinent example. It is built on the explicit hypothesis that functions associated with the intact right hemisphere may be exploited for purposes of rehabilitating speech in left-brain-damaged individuals.

Is the spontaneous recovery process being strengthened and sped along? This notion is to some degree answerable by research into the timing of treatment, including when to begin therapy and how frequently to provide it to a patient, regardless of the technique involved. The VA Cooperative study on the efficacy of treatment (Wertz et al., 1986) indicated that aphasic patients who began treatment later did as well as those
who had begun earlier. And most of us have the clinical impression that aphasic patients who begin treatment later do not necessarily do worse, although it is possible that more gain might have been made if their treatment had begun earlier. Nevertheless, we all know patients who improve long after the spontaneous recovery period is over.

Are underlying deficits that block the aphasic patient's ability to communicate being mitigated? Should broader deficits than language problems, such as working memory, attentional deficits, perseveration, and the like, be a focus of management? Some examples of therapy approaches that result from this perspective are Helm-Estabrooks's TAP (Treating Aphasic Perseveration) and VAT (Visual Action Therapy) (Helm-Estabrooks & Albert, 1991). Both of these approaches, although linguistically relevant, carry with them the assumptions that some modifiable aspects of brain-damaged behavior, nonspecific to aphasia but nevertheless modulating it, can have fruitful payoff in language gains.

Are compensations or alternatives for bypassing damaged language components being provided? Just as most individuals with cerebellar disease learn quickly, usually without being told, that they can touch their index fingers to their noses a lot more efficiently if they lodge their elbows in their rib cages, one should expect aphasic patients to make adaptations to their disorders. Some clinicians, myself among them, believe that excellent therapy involves exploiting or training compensatory strategies and capitalizing on intact language components.

Are damaged language components being repaired, usually using a set of tools called language drills? Are these drills directed to the functions previously performed by the damaged components? Many researchers and clinicians share this underlying assumption concerning the nature of therapy for aphasia. In fact, it is a pervasive and almost thoughtlessly accepted belief about treatment. And one can hardly fault the neuropsychologists who are doing theory-driven therapy for buying into it. After all, many clinicians do. In essence, the clinical assumption is often that one practices what is hard to do, or cannot be done, until one gets it right.

Is some combination of the above going on, dependent on the extent and presentation pattern of the aphasia and usually coupled with counseling for the patients and their families as well? This is called the way to hedge one's bets, but in the ideal, it is a deliberate choice, not a default.

Before leaving the topic of how change is affected, which I believe is central to increasing our knowledge about the process of therapy, I wish to emphasize that theory-driven therapy cannot substitute for it. A theory about remediation can be informed by normal language-processing models, to be sure, but its core must be a concern with accomplishing repair and compensation. I would also note that in the ideal, it should contain some sound neurophysiological basis as well.
THE FOCUS OF TREATMENT FOR APHASIA

A second set of questions also impinges on what we can and cannot expect from theory-driven therapy. These questions focus on beliefs about what is the proper focus of the treatment. Unlike concerns with how therapy modifies language behavior, these questions do not particularly depend on other disciplines' contributions to the management of aphasia. Generally, they relate to the focus of clinical encounters in aphasia management. The notion of focus of treatment might seem transparent. An obvious answer is that aphasia management focuses on the aphasic patient and the context in which he or she operates. That is certainly true, but this focus also has subtler aspects that must be studied to develop strong treatment theories. I would like to suggest five of them, although there are probably many more.

Should the focus of treatment be the missing language? I agree with Byng, who elsewhere in this volume says that we need to know a great deal about the missing language (i.e., the deficit) if we intend to conduct knowledgeable aphasia treatment. Elsewhere, Byng (1990) suggests that the account must include information about the nature of the deficit; that is, whether the patient is exhibiting a capacity deficit, a problem in accessing representations, or a problem in inhibiting unwanted representations. Second, the locus of the deficit should be specified, not in neurostructural terms, but within the general architecture of a model or process, and it should be related to some understanding of the way in which that component or process normally functions. Finally, she suggests that some consideration must be given to the relevance of the deficit to the communicative needs of the aphasic person and to the rest of his or her language-processing system. For example, to understand the spelling process better, it is certainly interesting to attempt to remediate spelling deficits in aphasic patients. But because many Americans cannot spell very well in the first place, and most are tolerant of spelling errors, its remediation might be of questionable use to an aphasic person.

As a result of a deficit analysis, such as that which precedes theory-driven therapy, we should have a better idea of what variables need to be controlled, what procedures cannot be used, and what kind of information the therapy needs to convey. However, what should occur in treating these deficits is not necessarily clarified by such models. In fact, it is often not easy to see from the analysis how treatment should progress, or even that a direct attack on damaged or missing functions is preferable to an attempt to bolster intact ones. There is still room for clinical ingenuity. For example, some of Byng's very interesting earlier work on sentence processing in aphasia (1988) resulted from an initial deficit analysis. The treatment that was derived, however, was a creative approach to active
linguistic problem solving centered on the patients’ deficits. It is possible that this therapy’s success resulted from what these tasks permitted the nondamaged parts of these patients’ brains to do. In any event, once the deficit is clearly analyzed, it can be the focus of treatment, possibly by directly attacking it, but also possibly by devising provocative and engaging language activities in which the rest of the brain can become involved.

**Should the focus of treatment be on the language that is preserved?** Most clinicians use preserved language skills as the *background* for their clinical interventions with disordered aspects of language. Some smaller number put preserved language and communication in the *foreground*, focusing treatment there and exploiting what skills are left. Just as explicit analysis of the deficit should precede loss-centered approaches, specific inventories both of preserved processes of language and communication and of the contexts that maximize good performance should precede the treatment. It strikes me as entirely possible to derive a box-and-arrow model for treatment that parallels the processing models for deficits.

Potential treatments range from deblocking drills to coaching aphasic patients in the use of strategies that serve to minimize or even mask the extent of their deficits in language. It is interesting that positive changes occur in the deficits despite a focus on the preserved skills. There is practically no explanatory research on why this is so. One possibility is that such a focus reduces the amount of effortful processing, which itself often directly results from a deficit-centered struggle on the part of the patient. This in turn allows more automatic access to intact components of the language processor. For treatments of this genre, it seems particularly important to seek alternative and competing explanations of effects.

**Should the focus be on the tasks that are used in treatment?** One of the thorniest of issues in aphasia treatment is what appears to be a tendency on the part of many practitioners to confuse the *tasks* of treatment with the *process* of treatment. Thus, as Byng (1990) points out in a recent lecture, “PACE is viewed as a therapy technique, rather than the medium” through which the therapist teaches the patients a socially acceptable end run on a communication problem and gives them opportunities to communicate in a minimally complicated microcosm of communication. Davis (1983) refers to this tendency to treat tasks rather than patients as “pure task blindness.” To legitimize the study of how good therapists go about doing what they do is a critical step in specifying adequate treatment for aphasia.

**Should the focus be on the interaction between the person with aphasia and his or her environment?** The patient with aphasia is not an invariant language deficit on (one or) two legs. The deficit almost always varies as a consequence of context, both linguistic and interpersonal. This variation must be accounted for in developing falsifiable theories of treatment, for one cannot escape expectations, setting, interpersonal skills, that enhance or impede communication. These are overarching concerns,
affecting everything from generalizing treatment effects to modifying communication behaviors of others in the aphasic individual's environment. Not only language but also sociolinguistic and interpersonal skills should be legitimate concerns for treatment theory.

**Should the focus reflect some balance of the above?** In a recent paper, Byng (1990) defined the scope of aphasia treatment to include at least the following:

1. an account of the uses of language made by the aphasic patient before becoming aphasic;
2. a delineation of the nature and effects of the language deficit on the whole language system;
3. an attempt to remediate the deficit;
4. an attempt to increase the use of all other potential means of communication to support, facilitate, and compensate for the impaired language;
5. an enhancement of the remaining language;
6. an opportunity to use newly acquired and emerging language skills not only in a clinical environment but also in more natural communication situations;
7. the facilitation of adjustment to the loss of communication skills; and
8. an attempt to change the communication skills of those around the aphasic patient to accommodate the aphasia.

This is perhaps a disturbingly broad definition of aphasia treatment. Yet, if aphasic patients are to be served appropriately, each feature of this "work scope" needs to be accounted for. To do this, I have been trying to suggest that clinical intervention in aphasia will require a theory of therapy. The principles of **language therapy**, just as much as the principles of **language processing**, need to be delimited and clinically tested. Until that occurs, the motion of using a theory of the deficit to dictate the aphasia therapy offers many hints but few palliatives. No one can do this particular theoretical work for us. We have to do it for ourselves.

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