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Model-Driven Treatment: Promises and Problems

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THE "DECADE OF THE BRAIN"

Neuroscientists like to refer to the 1990s as the "Decade of the Brain." One television discussant recently described brain research in the 1990s as "the greatest intellectual guest of all time" because it promises to change the way we think about perception, learning, intelligence, and communication in the normal and damaged nervous system. Certainly, we can expect the next decade to bring continued advances in both physiologic imaging and models of brain activity. The technologies that enable us to see the brain in action, along with the application of nonlinear cognitive models, ought to lead to a more holistic and dynamic view of various cognitive processes.

In their comments at the 1991 Clinical Aphasiology Conference, McNeil, Odell, and Tseng noted the tendency for cognitive research to stay stuck within the functional architecture of the Wernicke-Lichthiem model. Cognitive-linguistic research generated in the previous two decades was preoccupied with linguistic validation for centers and pathways of the brain (McNeil, Odell, & Tseng, 1991). Those were the decades of CT scans, artificial intelligence, and neuropsychology. Cognitive-linguistic research reflected the technology and attitudes of the time—the brain was viewed with fixed images, and mental processes were discussed through analogies to computers. Psychological and neurolinguistic experimentation largely was applied to validate long-held, conventional notions about the brain's architecture.

We are now on the threshold of revolutionary changes in the neurosciences that will cause us to think differently about the brain. We are able to observe mass activity in the nervous system through computerized imaging of metabolic functions, neuroelectrical activity, blood flow, and other physiologic markers. Physiologic studies examining the brain when

involved in exposure to different stimuli or involved in activities that require adaptation to experience now allow us to envision better the physical bases for cognition. Additionally, hypotheses are emerging to account for interactions in complex, dynamic brain systems using models that have what Gleick has referred to as the "right features" (Gleick, 1987, p. 299), that is, where the organizational structure itself has both stable and unstable features, and regions can be structured to have changeable boundaries.

In the February 1991 issue of *Scientific American*, Freeman illustrated neuroelectrical brain activity with what he called "phase portraits," based on computer replications of EEGs taken from a rabbit brain during the perception of odors. Freeman believed these electrical patterns demonstrated that brain activity, like other events in nature, can be studied as a system having *chaotic* properties. In science, *chaos* refers to complex behaviors or phenomena that seem random but actually have order. The formalisms of chaos theory, originated in physics and mathematics, have come to be applied to analyses of a full spectrum of natural events, from arrhythmic activity of heartbeats to changes in weather patterns. Chaos theory proposes that nature is organized with a dynamic, fluid geometry formed by the influences of "basins of attractions" (Gleick, 1987). Changeability is one of the prime characteristics of a chaotic system (Freeman, 1991). In brain research *changeability* has been demonstrated in neural patterns as a function of learning, in response to new inputs, and whenever the tendency for vast collections of neurons to shift abruptly and simultaneously from one complex activity pattern to another is observed (Freeman, 1991). The contour patterns described by Freeman demonstrate how one stimulus condition is changed following exposure to an intervening condition, thus providing evidence for how new experiences or contexts can affect subsequent perceptions. Freeman and his coworkers (Freeman, 1991; Skarda & Freeman, 1987) have suggested that chaos is what makes the brain different from artificial-intelligence machines.

If brain activity is indeed chaotic, then it is conceivable that brain damage disrupts the neurophysiologic balances, or basins of attraction, that need to be in place to maintain chaos. In a damaged state, regions that are normally active would have diminished reactivity once their interactions were disconnected, resulting in truly random, inefficient nervous system processes.

Along with the application of chaos theory to mental models, new hypotheses about the psychological side of cognition may also emerge from what Shallice (1989) called the "ultra-cognitive researchers," that is, research that has little interest in defining the neural basis of cognition (Margolin, 1992). One of the prime characteristics of the ultra-cognitive models is their proponents' stated neutrality on the issue of neuroanatomical architecture.

PROMISES

Each new piece added to the puzzle of human cognition contributes to the emerging picture of brain processes as dynamic activations that flow and change within and between multiple mental networks. We see a picture of the brain in which preserved processors, or attractors, could be manipulated and the brain "rehabilitated." Dynamic imaging and dynamic models of brain activities promise to provide both a picture and a schema of brain damage as a condition that compels rather than defies therapy.

TESTING THE MODELS

The Decade of the Brain is going to give us models of cognition and brain physiology that may or may not fit with the experiences of clinical aphasiology. Aphasia therapists need to conduct their tests or to challenge, defend, or explain treatment rationales within the context of one or another model. We are going to have to participate in shaping the framework if linguistic-cognitive models are going to come close to representing our experiences and our conceptions of aphasia and its treatment.

In his remarks in the *Archives of Neurology* regarding the future of cognitive neuropsychology, Margolin seemed to suggest that computational models, or computerized "lesion simulations," ultimately might provide a basis for cognitive rehabilitation techniques (Margolin, 1991). It is encouraging to see that a neurologist could perceive cognition to be treatable. However, theoretically-based therapeutic approaches that are equally valid and tested might be viewed as inadequate, or poor practice, if they could not be predicted by a computational model. There is a substantial amount of literature describing individuals with aphasia who improved their language performance as a function of treatment that was not model-driven (Loverso & Horner, 1991).

Fortunately, we have clinicians here and in Europe who are contributing a clinical perspective in cognitive research. At this juncture we have some hypotheses about how single words are processed, but there is a great deal of work to be done before we understand what the brain does when we speak, listen, read, and write to communicate our ideas with others.

PROBLEMS IN THE APPLICATION OF MODELS

In the past couple of years I have attempted to incorporate existing theoretical models of linguistic-cognitive processes into my clinical practice

because the treatment studies built on these models are convincing. Processes and associations in theoretical models, such as those suggested by Patterson and Shewell (1987), have been helpful to me in focusing certain aspects of treatment with certain patients at certain times.

There is resistance to the use of group designs in model-based treatment reports; thus, information about some of the effects of variables one could typically examine from a large data base drawn from a heterogeneous sample is not available in their work. Additionally, the evidence for the modules and processes contained in models like Patterson and Shewell's is more substantive in some areas than in others; some parts of the map may be more accurate than other parts. Furthermore, having a map can help you find your way, but it cannot help you drive the car. Any positive outcome in aphasia treatment results partly from a fruitful focus of therapy and partly from the confidence and skills of the clinician who implements the therapy. The best outcomes probably come from clinicians who have confidence that they are doing the right things. They are sure that they are going to make a difference, and so they do.

A direct *frontal attack* on linguistic-cognitive deficits is only one aspect of the speech-language pathologist's therapy with a person with aphasia. In treatment studies we often ignore the simple truth that aphasia therapy, like aphasia, is multifaceted and has aspects that might more accurately be called psychotherapy and metacognitive or metalinguistic therapy. Treatment tasks, even those that are intended to develop automatic mental processes, frequently require getting the patients to understand why they are having the difficulties they are having, why they need to participate in what are sometimes tedious exercises, what they can consciously and routinely do to communicate better, and then getting them to do it. Also, at some point, with most patients with aphasia a prominent facet of therapy involves helping the patient work through the psychosocial adjustments necessary when living with a chronic communication impairment.

Model-driven therapy, as envisioned by its proponents, requires extensive *definitive assessment* across tasks that are balanced for control of certain variables, such as the frequency of occurrence of a given word in the language, the "wordness" of the word, the part of speech it represents, its regularity, and its concreteness. The assessment rarely uses published tests, so most of the tasks lack performance norms. Processing deficits are identified by computing the number or percent correct and then considering what chance performance might be for a given task. With the exception of rare agnosias, deficits tend to be partial losses. Consequently, this so-called definitive testing is open to a fair amount of subjective interpretation. Additionally, reading, writing, listening to, or speaking these long, balanced word lists, as well as undergoing assessment across multiple domains, can constitute an exhausting amount of testing. It is

not easily or appropriately accomplished with all patients, nor is it within the constraints of some reimbursing agents.

Although I recognize that it is sometimes necessary, I find even cursory testing with an aphasic patient during the acute or early postacute period of recovery to be uncomfortable. Causing patients to focus too closely on their language deficits right after they have stared into the wide maw of their own mortality seems to be missing the point of the moment. Definitive testing may not be reasonable or appropriate in the early recovery stages.

Because processing deficits are usually partial and require an analysis of *relative* differences between areas of performance, we cannot ignore fatigue, order effects, or any other factor that might affect *reliability.* Reliability of performance and stability of the tests used should receive far more attention in the literature.

The timing of intensive, deficit-directed therapy also needs to be investigated, especially with the more severely impaired patient. Efficacy has been demonstrated with the stable, chronically aphasic patient, who may be several years post injury, but access to treatment is often determined by financial constraints, and for the majority of patients, access can diminish with chronicity, when there is a reduction of third-party payment for treatment once the patient has *recovered* from the neurological insult.

CONCLUSION

There is nothing novel about the idea that language therapy should be driven by theories about cognition. The writings and work of Head, Wepman, Goldstein, Schuell, and others support the notion that linguistic impairment results secondarily from a processing impairment (Martin, 1981). What we are discovering is evidence for what those impairments might be and how cognitive resources, or, perhaps chaos within the nervous system might serve to allow us and our patients to comprehend and express messages. In this decade and into the next millennium the best ideas coming from an array of neurosciences might ultimately map brain physiology to mental processes.

Over a decade ago, A. Damien Martin asked us to consider the role of theory in our therapy. In the September 1981 issue of *Topics in Language Disorders,* Martin said that language behavior can be described with reference to cognitive processing. In the model he proposed, cognition has an integrated, hierarchical organization that is cybernetic and interrelated in its nature and contains processes that occur in parallel with one another. He described how interrelated and parallel scanning processes might be involved in word retrieval. He suggested that we consider, for example,

the ease or difficulty of lexical access when we design articulatory tasks for the patient with a phonological expressive disorder.

Martin said that language as a *code* is an artifact, and therefore, the code *itself* is not impaired by damage to the processing organism. He reminded us that aphasic speakers use language pretty much the same way as normal speakers do but in a reduced and less efficient manner. Finally, he suggested that the more detailed and explicit the model of cognitive processing, the more detailed and explicit the diagnostic hypotheses and the therapeutic goals may be. My own ideas and loosely conceived theory for aphasia treatment originally came in part from the writings and presentations made over a decade ago by Dr. A. Damien Martin. I continue to appreciate both his insight and his foresight. He, like Loverso and Horner (1991), pointed out that an absence of overt theory in our work and in our research is, at the very least, scientifically undesirable. Suggested models for aphasia treatment that are based on prevailing theories of linguistic-cognitive processes can provide an overt rationale for certain aspects of therapy. Treatment theories will always benefit from more clinical trials, more discussion and elaboration, and from constant input from clinicians. Treatment models should, and will, be drawn and redrawn in response to new information and influences so that we can continue to structure aphasia therapy for the best possible outcome.

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