Specialty Recognition in Neurogenic Speech, Language and Cognitive Disorders: Right Hemisphere Communicative Disorders

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What are the credentials needed to assess and treat right hemisphere (RH) communication disorders? In an ideal world we would be able to specify with precision the knowledge base, the diagnostic tools and the therapy techniques required to work with this population. The world is far from ideal, but it seems to me that we can identify two prerequesites for effective treatment by speech-language pathologists. The first is a thorough command of the symptoms. The second is a theory about their cause. Both are equally important to the task. Without the former we cannot know what we are treating. Without the latter we cannot begin to know how.

The symptoms fall into two major categories: those that affect communication directly, and those with an indirect effect. The former category consists of linguistic, extralinguistic, pragmatic and affective disorders. The latter includes left-sided neglect and the various visual-perceptual deficits commonly associated with RH damage. The knowledge base must include these latter deficits. To operate without an understanding of them is to make an a priori decision that perceptual impairments are independent of all other RH communication deficits. Such a decision may be valid, but the clinician must be able to support it with theory.

No one would deny the role of theory in guiding our approach to communication disorders of every stripe, from dysfluency to dysarthria. We cannot jump inside peoples' brains and, consequently, we cannot deal in absolutes. Absolutes may not exist, but best guesses do. To identify the credentials needed to work with this population, it is vital to specify just what clinicians need to have best guesses about. To that end, I will outline what I believe are the central questions about which clinicians should have working hypotheses.

First, clinicians must decide whether they are dealing with multiple discrete and independent deficits, or with one or several common underlying processing disorders. This requires examination of the ways and degree to which the symptoms in each area are related to one another and to deficits in other areas. Beginning with the extralinguistic impairments, one must ask if there are common features in the failure to integrate story elements into an overall theme, in the failure to appreciate contextual cues and to use them adequately to apprehend implicit or intended meaning, in the failure to distinguish the important from the trivial, and in deficits in what is referred to as "problem solving" and "abstract reasoning." Are these impairments expressions of an underlying disorder in recognizing, using and integrating significant contextual cues? Does a similar processing deficit figure in the symptoms of pragmatic, affective, and perceptual impairments as well?

Are affective disorders -- poor motivation, reduced responsiveness to the emotional aspects of facial expression and situational setting, deficits in prosodic processing and production, and an aberrant sense of humor -- the result of some sort of emotional disorder and thus distinct from deficits in other areas? Or do they represent a form of hypoarousal linking them to an attentional impairment? Or are they, too, symptoms of a failure to account

for and integrate significant contextual information? Or, finally, could both of these last causal factors be operating so that failure to utilize contextual cues represents the cognitive outcome of a particular type of attentional deficit? One's position on these issues determines whether one treats affective disorders by referral to psychiatry or by work on attention and/or cognitive processing.

The search for underlying processing deficits must include an exploration of ways in which pragmatic disturbances relate to affective and extralinguistic disorders. Again, one must begin by questioning if there is a common theme in the tendency to overpersonalize, the failure to take into account the listener's needs, the tendency to digress and interrupt, the failure to read body language and maintain eye contact, the apparent lack of interest in the impact or quality of a response, and the general lack of sensitivity to rules governing conversational exchange. If so, does that theme resonate with the processing impairments listed above in attention to stimulus significance and the integration of contextual cues? Is there a fundamental process or set of processes that enables us to distinguish the crucial from the irrelevant and to draw pattern and form from the chaos and cacophony of sensory input? If one assumes that such a mechanism exists and that it may be implicated in RH communication disorders, one must then question whether the damage occurs in primary or later stage processing. That is, does the problem occur at the level of stimulus awareness, in an initial transformation, or at some later representational stage?

Questions such as these raise the issues of perception and left neglect -- those deficits that affect communication only indirectly. Again, one must begin by asking if there is a common link to disturbances in visual integration, figure-ground, scanning, tracking, the tendency to visually fixate and perseverate, problems in line orientation, in visuomotor sequencing, and in the construction of overall form in visuoconstructive tasks. Then one must theorize about their effect or lack of it on the group of direct communication disorders. Does the failure to adequately respond to visual input, whether in facial expression or in complex pictures, represent a deficit in visual perception, a cognitive deficit, or possibly an emotional one? To what extent are visual perceptual deficits themselves cognitive? Do they reflect a deficit in "seeing" or in "seeing as"? If the patient has difficulty in naming the items in a fragmented picture task, is this the result of a problem in seeing all the features, or in recognizing and integrating the key features? If the latter, is this an early stage effect of damage to the same mechanism that at a later stage enables us to integrate input on the more symbolic level of narratives and complex scenes? Does the patient fail on figure-ground tasks because he somehow cannot "see" -- that is, distinguish, surfaces one from another in the visual array? Or does he fail because he cannot go beyond the absolute surfaces in such a way that enables him to suspend belief and construct or "see" a whole fish or bottle when only a piece of it appears? Is this a perceptual literalness or are his perceptual problems somehow cognitive in nature?

Answering this question requires a position on the nature of visual perceptual processing. Clinicians must delve into the area of information processing and explore the issue of "top down" and "bottom up." They must ask if the perceptual deficits associated with RH damage are the result of a deficit in creating representations about the visual world, or if they are the result of a failure to adequately recognize the surfaces and edges contained in the visual array. That is, are RH perceptual problems primarily a deficit in seeing and noting what is there, or in attending to what is

important and in integrating those significant features into a coherent pattern?

Finally, because we know that the presence or absence of left-sided neglect exacerbates not only the perceptual disorders, but the group of direct communication disorders as well, clinicians must adopt a theory about the nature and cause of neglect. Several theories are available. There is the Heilman theory of hypoarousal, the Kinsbourne theory of an innate rightward attentional bias, the Bisiach theory of a disturbance in the inner representation of external space, and the Mesulam theory of a deficit in directed attention and recognition of stimulus significance in relevant contralateral and ipsilateral space. Most of these theories implicate attention -- both tonic and phasic. Some suggest it plays a crucial role in determining what we should attend to as well. Clinicians must take a close look at attentional deficits implicated in neglect and explore their potential impact on RH communication disorders.

One cannot weigh the relative value of theories of neglect and attention without some knowledge of current theories on the neuroanatomy of the RH -- the interhemispheric and limbic connections that seem to characterize its layout. The knowledge base should also include some sense of the wealth of multimodal association areas in right cerebral cortex, some idea of where these areas are, and some sense of the effect of lesion site on behavior.

Neuroanatomical knowledge, of course, affects every aspect of evaluation and treatment. If, for example, one supports the current theory that prosodic disturbances differ in type according to an anterior versus posterior site of lesion, one is in effect also supporting the theory that prosodic problems are not emotionally based. If they were, we would expect a more generalized effect on prosody, rather than one that differentially affects comprehension versus production according to locus of lesion. Such knowledge helps determine whether or not to treat a disorder.

For those disorders one does decide to treat, the central question becomes whether or not to stimulate recovery of function or to teach the patient to compensate for his deficits. In either case, clinicians must have a rationale for their decision. Compensation techniques usually involve the use of verbal cuing and teaching analytic strategies. If the patient does not look to the left margin or maintain eye contact, he is given a verbal cue or trained to verbally self-cue. If he cannot solve a problem, organize sentences into a paragraph, or recognize the theme of a story, he is given less complex forms of the same task along with various analytic and linear strategies or rules for approaching it. This is compensation -- using the left hemisphere (LH) to overcome the deficits associated with RH damage.

Stimulation of underlying processes, on the other hand, may not involve teaching a set of rules at all, but rather working in novel ways that challenge the limits of the patient's capacity in attention, perception, recognition of stimulus significance, association and integration. Just as they do in aphasia treatment, clinicians must be able to support their choice between stimulating recovery or bypassing that process in favor of teaching the patient to compensate for his deficits.

In summary, the credentials for working with RH patients should include the ability to answer these questions. What are the symptoms? How would you evaluate and treat them? How would you evaluate the effectiveness of your treatment? Addressing questions of diagnosis and treatment requires not so much that clinicians know the right answers, as that they have defensible theories. I have tried to outline some of the types of questions necessary to developing those theories. Lest it appear that the credentials should

also include degrees in neuroanatomy and cognitive psychology, remember that the literature is available just as it is in linguistics and other fields that intersect with the study of aphasia. Not to be forgotten as well is the crucial role played by clinical insight in developing hypotheses.

Clinicians cannot hope to stimulate recovery without some idea about the processes they are trying to help the patient recover. If they don't, they will develop diagnostic tools that are merely symptom checklists, and therapy that is based only on compensatory techniques rather than on recovery of function. Overdependence on the verbal and analytic skills of the LH in treating RH disorders is a way of dodging our responsibility to take the risk of making "best guesses." It spells the difference between a technician and a therapist, and we cannot afford to allow speech-language clinicians to become right hemisphere technicians.