When is Aphasia Aphasia?
The Problem of Closed Head Injury

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The most recent comprehensive statistics regarding the incidence and prevalence of head injury were the result of an NINCDS survey published in 1980(Kalsbeek, McLaurin, Harris and Miller, 1980). The survey suggested that the incidence of head injury was 200/100,000 people; corresponding prevalence figures are 450/100,000 people. Nine hundred and twenty-six thousand Americans who suffered head injury between 1970 and 1974 were estimated to be still exhibiting sequelae in 1974. Head injury is the major cause of death in persons under age 35 in this country. All estimates of the extent of the problem are considered to be underestimates, and the statistical trend suggests that these numbers are growing.

Head injury has been called "the invisible epidemic." Given the magnitude of the problem it behooves the clinical aphasiologist to consider the nature of head injury. Three features distinguish head injury from the stroke population that is the more traditional concern of the aphasiologist. The first distinction is that the overwhelming majority of head injury is of the closed-head type, rather than the penetrating head injuries upon which American aphasiology first learned its trade. Closed head injury differs from open head injury primarily in that the cognitive/behavioral result is much more likely to be a generalized one instead of the focal problems that the profession is used to dealing with.

The second distinguishing feature follows logically from this fact. While the ideal treatment program has yet to be defined, it is clear that as a result of the multiplicity of cognitive and psychiatric sequelae, rehabilitation following closed head injury is a much more exquisitely interdisciplinary enterprise than is aphasia rehabilitation, at least as it is presently practiced in this country. The third distinguishing feature stems from the statistics presented a moment ago; the typical head injured individual is much younger than is the post-stroke aphasic patient, an aspect of considerable importance in the overall planning picture.

It seems obvious that all three of these aspects of head injury have serious implications for the aphasiologist in relation to prognosis, to goals of treatment, and to approaches to treatment. However, given the topic of the panel discussion, I will concentrate my remarks on a single aspect of the first feature, the one that has to do with the differences between language problems in head injuries of the closed type, and the focally-produced aphasias as we have come to know them. I hope that these remarks will provide some grounds for the serious reconsideration of the role of the clinical aphasiologist in the rehabilitation of patients who have suffered closed head injury.

In the interests of pure contentiousness, I would like to state my answers to the question of when is aphasia aphasia in their most blatant forms first, and then to argue the reasons for them. So here they are.

If the language problems seen in closed head injured patients don't look like aphasia, sound like aphasia, act like aphasia, feel, smell or taste like aphasia, then they aren't aphasia. Further, they will not be terribly responsive to the traditional methods by which we have come to treat aphasia.

Heilman, Safran and Geschwind (1971) have suggested that the incidence of frank aphasia in blunt head injury is about 2%. Sarno (1980) has suggested that the incidence may be as high as 32%. This means, of course, that aphasic patients do exist in the head injured population. But the more impressive data are that language is disturbed in 75% or more of patients who have had head injury (McKinley, 1981). The paradox is that language is not disturbed in typically aphasic ways. In essence, aphasia is probably NOT aphasia in the vast majority of the head injured.

My bias is that, rather than aphasia, the language problems found in closed head injury are much more likely to be the manifestations of more general and more pervasive memory and cognitive deficits, and need to be treated in the context of these memory and cognitive deficits. Language and communication problems DO exist in the head injured. But if we were to view them or to treat them as we have come to view and treat aphasia it would mean that we would have to rely upon memory abilities, cognitive capacities and communicative competencies that are usually reliable in aphasia, but are shifting sands in the head injured.

I'd like to spend the next few minutes describing a few of the commonalities and differences between aphasia, which results from focal (or perhaps disproportionately emphatic) injury to the speech and language areas of the brain, and the language deficits that most typically result from closed head injury. I'll begin with areas of overlap.

SIMILARITIES

In both the aphasic and the head-injured, the most prominent area of overlap lies in the problem of anomia. The centrality of word-retrieval problems in aphasia, and the frequency with which a variety of aphasic syndromes evolve into anomic problems is well known, and does not require much further discussion. This feature is shared by the language deficits of closed head injury, where a similar frequency of anomic problems is encountered, and maintained with similar persistence and tenacity.

Yet in the retrieval problems of aphasic and head injured patients, there is a qualitative difference that leads some investigators to note that "nonaphasic" naming errors are a feature of head injury. In addition to the circumlocutions, paraphasias, and reduced fluency (as measured by categorial generation of words) typical of aphasia, the head injured exhibit additional naming disabilities. Specifically, head injured patients make naming errors that are related to their personal situations, to their stimulus-boundness, (e.g., the opposite of good-bye: bad-bye), or make errors of confabulation (Q - "what do you like on your hamburgers? A - "mosquitos"). One plausible explanation for this qualitative difference in naming errors is simply that there are only a limited number of ways to err in naming. The aphasic patient tends to limit his errors to the smaller, more appropriate and even more predictable subset. The head injured patient, who if he is not dysarthric, is highly likely to be very verbal, is not limited so extensively by what he knows about his language, and/or is impulsive and perceptually disinhibited enough to act in disregard of it.

A second area of overlap between the two groups is that both initially tend to exhibit problems in auditory comprehension, and in both cases, comprehension abilities are likely to improve first. In both cases, too, problems in comprehension of complex material are likely to persist well

into recovery. Further, both groups are likely to exhibit associated reading and writing deficits. Finally, spontaneous recovery occurs in both groups, although in the case of the head injured, the length of coma and of post traumatic amnesia is more predictive of extent of recovery than is the analogous measure, severity of stroke, predictive of recovery from aphasia.

These, along with suddenness of onset, are the features that relate the language of the head injured patient to aphasia. And while they are compelling, they by no means tell the whole tale. Let us turn now to differences between the two groups.

DIFFERENCES

Halpern, Darley and Brown (1973), and later Wertz (1978) describe "the language of confusion" in such a way as to identify the head injured as its major speakers. Darley more recently summarizes the pattern as follows:

> It is this pattern of a high degree of irrelevance of content, coupled with paradoxically adequate syntax and fluency that differentiates the language performance of confused patients from that of aphasic patients. (Darley, 1982, P. 25)

This is a good starting place. But it is necessary for the aphasiologist to describe the pattern as fully as possible if appropriate treatment is a goal, and to use the resultant description to begin the process of relating the pattern to its neurologic substrate.

The system of Bloom and Lahey (1978) has been designed to classify children's language disorders. Two of its categories, however, capture the essential difference between aphasia and head-injured language. Extending their system to adults, aphasic language could be considered to be a disorder of form; head injured language could be considered to be a disorder of use. The vocabulary used to describe head injured language, some features of which persist well into late stages of recovery, is instructive in this regard. The descriptions include the aforementioned "nonaphasic" naming errors and irrelevance. Also included are such terms as digressiveness, difficulty in self-monitoring that includes impetuousness and disinhibition, difficulty in attending to topic, disorganization, difficulty in initiating speech, and its converse problem--once initiated, speech is difficult to stop, and difficulty in changing topic.

It is in the area of language pragmatics that aphasia and head injured language most vividly contrast. Consider the following example from a CADL protocol: Asked whether he had ever experienced Clasmapsia Dostinnia, the CADL item intended to assess if patients can comprehend that they <u>don't</u> comprehend, a head injured patient first sat silently for five seconds. That achieved for him the direct probe "Do you know what Clasmapsia Dostinnia is?" He replied, "Doesn't that mean an abnormal fear of dust?" No aphasic patient would simultaneously break so many pragmatic rules.

Particularly in the area of communicative competence, where the emerging data consistently demonstrate surviving ability in the aphasic patient, the linguistic and communicative behavior of the head injured patient appears to be deteriorated. The reasons for this deterioration are as yet unclear, but they probably will be shown to relate to the consistent cognitive and memory disorders that are displayed by head injured patients, quite often in conjunction with a language facility that appears to have been unaffected by the injury. (For a very thorough discussion of the memory and cognitive

aftereffects of head injury, the reader is referred to Levin, Benton and Grossman, 1982.) It is also conceivable that the psychiatric sequelae that so uniformly affect the head injured also play a role. In this regard, recent estimates of personality change following head injury approach 70% (Jennett, Snoek, Bond, and Brooks, 1981). It is perhaps the case that psychiatric, cognitive, memory and language difficulties coalesce to create "communicative incompetence."

These matters are badly in need of further research. But whatever the contributing interaction turns out to be, it will surely be related to generalized neural effects of head injury. For example, even in cases in which minimal microscopic changes were identified before death, Levin, Benton and Grossman (1982) refer the reader to the instructive examples provided by Stritch (1956, 1970) who observed in such patients extensive and systemic microscopic degeneration of neural tissue at autopsy. The extent of such change allows one to consider that the appropriate comparison is not between aphasic and head injured language, but perhaps between the language of the head injured and the demented.

THE DEFINITIONAL PROBLEM

I want to conclude with a reference to the definitional problem of aphasia in head injury. It would perhaps be quibbling to argue whether aphasia exists in the head injured, unless the definition of aphasia is explicit enough to let us recognize aphasia when we see it, and exclusive enough so that every aberration of language is not consumed by it. I believe both common usage of the term and research on aphasic language behavior has made the definition tight enough to make the argument considerably more consequential than mere quibbling.

As I wrote these remarks, I found myself wondering why nobody has ever raisedsuch questions concerning the language problems of mental retardation. I doubt seriously if anyone ever considered the aberrant language of adult retarded citizens as "aphasic" or even that the difficulties that retarded children have in learning language were the result of some variant of "childhood aphasia." I think that those problems were avoided by common sense--that the centrality of cognitive deficits in such folks overshadowed the language deficits so obviously that the "proper" perspective was inadvertantly achieved. But "aphasia" seemed to have been there before the similar centrality of the unique cognitive/psychiatric/memory problems of the head injured (or the demented for that matter) was recognized.

I don't think it does a lot of good to say that head injury produces aphasia just because the head injured patient performs poorly on some aphasia tests. So would the retarded, the demented, and the psychiatrically disturbed. The QUALITATIVE differences in performance of each of these groups should take precedence. Nor does it do a lot of good to describe the problems of language in the head injured, as Sarno does, as "subclinical aphasia" (1980). This approach essentially begs the question.

If the language disorder is aphasia, then regardless of the etiology, it should be so labeled and most importantly, so treated. If it is NOT aphasia, and I would argue that in the great majority of the head injured, the language problem is not aphasia, then inappropriate treatment may occur as a result of inappropriate labeling. In far more general terms, it is important that future research be directed to disentangling the cognitive

and language problems of the head injured, and that this research not necessarily be bound by aphasic/nonaphasic comparisons.

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