When is Aphasia, Aphasia?

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In 1861 Paul Broca wrote one of his most famous papers on aphasia and the pony express expired. In 1982 researchers (or some of us) are considerably less sanguine than was Broca about what aphasia is and what it is not, and probably we are also less sure of when our mail will arrive. I can do nothing about the mail service except tell the truth about when I put a manuscript in the mail. Nor can I settle the issue of when aphasia is aphasia, but I can review and evaluate others' answers. I will.

How people respond when asked, "When is aphasia, aphasia?" depends on their definition of the disorder; on their attitudes about the nature of ordered and disordered behavior, including language behavior; on how they were trained and who they admire; and, I suspect (although I cannot prove it) on how many aphasic speakers they have seen and for how long. I will review selected of those responses, beginning with a discussion of several definitions of aphasia, for it is in those that writers are usually most cogent about what aphasia is for them.

When is Aphasia, Aphasia?
What the Definitions Say

Benson (1979) says, "Rather than discussed, viewpoints on aphasia have often been argued. Much of the controversy can be traced directly to the definition of aphasia..." (P.1). It is impossible to know whether he intended to fan or douse the flames of controversy with his own definition. Here it is: "Aphasia is the loss or impairment of language caused by brain damage" (P.5). (Does it seem warmer in this room already?) Clearly this definition is inadequate for it fails to distinguish aphasia from other acquired language disorders.

Darley's definition (1982) is more complete, and it may satisfy a greater number of aphasiologists. He defines aphasia as:

> Impairment, as a result of brain damage, of the capacity for interpretation and formulation of language symbols; multimodality loss or reduction in efficiency of the ability to decode and encode conventional meaningful linguistic elements (morphemes and larger syntactic units); disproportionate to impairment of other intellective functions; not attributable to dementia, confusion, sensory loss, or motor dysfunction; and manifested in reduced availability of vocabulary, reduced efficiency in application of syntactic rules, reduced auditory retention span, and impaired efficiency in input and output channel selection (P.42).

This definition is firmly in the Schuellian tradition (Schuell, Jenkins, and Jiménez-Pabón, 1964). It requires that aphasia be reserved for a language disorder crossing all modalities - reading, writing, listening, speaking, and gesturing. Subscribing to this definition means denying that syndromes involving a limited number of modalities such as alexia with or without agraphia can be called aphasic.

A definition with different implications for deciding when aphasia is aphasia is offered by Goodglass and Kaplan (1972). I have included their discussion of normal language so as to clarify their definition of aphasia.

> Normal language may be regarded as depending on a complex interaction between sensory-motor skills, symbolic associations and habituated syntactic patterns, all at the service of the speaker's intent to communicate, and subject to the intellectual capacity which he brings to the task of manipulating them so as to carry out his intent. Aphasia refers to the disturbance of any or all of the skills, association and habits of spoken or written language, produced by injury to certain brain areas which are specialized for these functions (P.5).

They label as aphasic a variety of limited disorders such as "selective disorders of auditory comprehension, object-naming, articulation, reading or repetition..." (P.5), and they would classify alexia with or without agraphia among the aphasias.

So would Albert, Goodglass, Helm, Rubens, and Alexander (1981) and Damasio (1981). Albert and his colleagues, for example, say that "The assessment of language function must deal with this aspect of dysphasia--its possible selectivity for particular modalities of input or output and--in some instances--for specific input-output combinations" (P.12). Damasio adds, "Aphasia is a disturbance of one or more aspects of the complex process of comprehending and formulating verbal messages that results from newly acquired disease of the central nervous system" (P.51).

The first issue is whether disturbed language to be aphasia must cross all modalities. Darley (1982) says yes; others say no.

Definitions by other authors make trenchant other issues. McNeil's (1982) definition, for example, suggests that aphasia may not be primarily a language disorder at all. McNeil defines aphasia this way:

> Aphasia is a multimodality physiological inefficiency with, greater than loss of, verbal symbolic manipulations (e.g., association, storage, retrieval, and rule implementation). In isolated form it is caused by focal damage to cortical and/or subcortical structures of the hemisphere(s) dominant for such symbolic manipulations. It is affected by and affects other physiological information processing and cognitive processes to the degree that they support, interact with, or are supported by the sumbolic deficits. (P.693).

For McNeil the aphasic speaker's linguistic and communicative impairments are secondary. He identifies as primary impairments "such factors as increased fatiguability, increased sensory thresholds, decreased speed of reaction, fluctuation of attention and effort allocation, and inertia of neurophysiological excitation and inhibition" (P.701).

McNeil is not by himself. Kreindler and Fradis (1968) identify six defects they call fundamental and common to all aphasic speakers. These include some of those identified by McNeil, such as "inertness of excitation and inhibition processes" (P.64) and fatiguability, but they also add "blockage within the functional system of the word" (P.64). They hypothesize that these six defects explain aphasic language deficits. They say, for example, that "inertness of excitation and inhibition...(may)...underlie the aphasic patient's difficulty to differentiate stimuli" (P.68). Lesser (1978) and Albert, Goodglass, Helm, Rubens, and Alexander (1981) are among those who also believe some form of McNeil's story.

Advocates of the position that aphasia is a unitary disorder crossing all modalities assume, with Brown (1968), that there exists a central language factor, that performance in all modalities draws upon this central factor, and that aphasia-causing lesions affect those performances more or less equally. They believe this so staunchly that they assume that all limited impairments such as alexia with agraphia must be modality specific transmissive disorders. They also appear to have adopted Schuell's notion (Schuell, Jenkins, Jiménez-Pabón, 1964) that the cause of aphasia is a deficit in the auditory modality, perhaps a deficit in reauditorization.

Those such as Goodglass and Kaplan (1972) who say that aphasia can be limited to one or more modalities believe something else. Lesser (1978) speaks for them. She says, "It can...be argued that differences of language behaviour in the modalities are such that they relate not to performance or transmissive malfunctions but that there are different competences for speech and comprehension" (P.52). Many would agree that speech as a sensorimotor act can be impaired independently of comprehension, as when dysarthria or apraxia of speech disrupts speech movements. Lesser, however, was - or seemed to be - talking about <u>oral language</u>.

The group believing in a unitary concept of aphasia have not only ignored the idea of separate competencies for the modalities, they seem to have ignored the possibility that phonology, syntax, and semantics can be dissociated, especially after brain damage, and that these dissociations can result in discrete aphasic syndromes. On the other hand, Lesser (1978) and others are convinced of such dissociations. She says, "There is evidence which suggests that the levels of linguistic processing represent to some degree psychological processes, and perhaps even brain functions which have some degree of autonomy, although they operate (in normal) within an interacting whole" (P.186).

Thus far no popular writer appears to have said that a language disorder to be aphasia must cross all levels of linguistic processing - phonologic, semantic, syntactic. Probably most writers, especially those with a modality rather than linguistic orientation, assume that it does. It may not. Differences among Broca's and Wernicke's aphasic speakers - which the advocates of a single aphasia would like to ignore - may result from differences in level of linguistic processing that is disrupted, being related to semantics in Wernicke's aphasia and to syntax in Broca's aphasia. I hope it never occurs to some aphasiologist to dictate that aphasia must cross all levels of linguistic processing. But it probably will.

McNeil's assumption that most aphasic symptoms can be accounted for by primary deficits in attention and effort allocation, and his apparent subsequent rejection of aphasia as a primary language deficit, create yet another issue. I think we should not drop "primary language deficit" from our concept of when aphasia is aphasia, however. It seems to me that it is the interaction of variables such as effort and attention with language variables that creates aphasic symptomatology (in fairness, McNeil probably also believes this). Without granting this status to language, we are forced to accept as aphasic the impaired performance of right-hemisphere and brain-stem damaged patients on speech and language tests. And while we may want eventually to include right-hemisphere communication deficits within aphasia's rubric, it seems nonsensical to also include the impaired test performance of the dysarthric speaker whose equilibrium has been

disturbed by trauma and a protracted stay in the total care unit. I like Kreindler and Fradis' apparent compromise (1968). They feature "the functional system of the word" as well as conditions such as impaired effort and fatigue. In other words they feature language as well as non-specific effects of brain damage.

McNeil is not done stirring the caldron. McNeil's definition implies that aphasia is a performance rather than a competence deficit. I'm uncomfortable with this view. First, it is impossible to test competence independently of performance, thereby making McNeil's position practically impossible to test. Second, in protracted treatment with each patient, the clinician usually ends up having used a prodigious number of different stimuli and procedures. And while it can be argued that even hundreds of hours of treatment do not give one an insight into a patient's competence, it is my consistent experience that most aphasic speakers can be made to do some things but they cannot be made to do everything. And global, severe Wernicke's and severe Broca's aphasic speakers never approach normal reading, writing, listening, speaking, or gesturing under even the most sedulous and enlightened clinician.

Some clinicians perhaps feel more comfortable using phrases such as "loss" and "impaired access" rather than competence and performance. I do not know how much these alternatives help, or if they help at all. I do know that many severely involved patients seem to have lost words and rules. And unless one wants to deny that severe aphasia is aphasia, the impaired access hypothesis is inadequate to explain aphasic symptomatology, at least in my opinion. It seems to me, therefore, that we have to implicate impaired competence in the definition of aphasia, if for no other reason than to discourage clinicians from thinking that if they just try hard enough something good will happen.

When is Aphasia, Aphasia?
The Importance of Cognition

Thus far I have said nothing about cognition. It is now time to do so by introducing additional definitions of aphasia. Martin (1981) is among those who would have us believe that aphasia is a cognitive deficit. This is his definition: "The disorder (aphasia) is the reduction, because of brain damage, of the efficiency of action and interaction of the cognitive processes that support language behavior" (P.310). For him cognition means "all the processes by which sensory information is transformed, reduced, elaborated, stored, recovered, and used" (P.311). Therefore, aphasia "is not a 'syntactic' nor 'lexical' impairment. Rather the difficulty lies in the uses of those processes which handle that particular type of information" (P.311).

And while Brown's (1972) definition of aphasia is standard and limited, his view of the relationship of language, cognition and affect (Brown, 1977) leads him (1981) to argue that the aphasic patient, in particular that one with Wernicke's aphasia, will have "changes in affect, behavior, and awareness that are a dramatic - and, in my view, an intrinsic - part of the aphasic disorder" (P.6). And because he describes cognition as meaning "not just ideation but the whole matrix of perceptual, motoric, affective, and linguistic contents that enter into every mental act" (1977, P.IX), he, too implicates cognition in aphasia. He says (1977) "...every disorder of

language also incorporates aspects of a corresponding level in cognition. A change in awareness, an alteration of mood, the presence or absence of delusional or hallucinatory phenomena, these are not additions to the clinical picture but have an inner bond with the aphasia form" (P.27).

Most traditional aphasiologists would like to separate cognition and language and leave cognitive deficit out of the definition of when aphasia is aphasia. Wertz, for example, will tell us (see this volume) that it does no good to talk about aphasia in dementia. Probably not. It strikes me, however, that both Martin and Brown define cognition in ways that tie it inextricably to language and that implicate cognitive deficits in aphasia. They also define cognition differently from traditional aphasiologists, who seem to assume that cognition is only that which goes awry in dementia.

Brown even goes so far with his view of the interactions of language, cognition, and affect as to suggest that the language of delusion may be a "high-level" aphasia. I can hear the groans. What, it will be argued, does it accomplish to call the delusional language of the schizophrenic an aphasia? Certainly I would not want to if I thought speech pathologists would then assume they could treat it. Speech therapy for a schizophrenic is like putting a band aid on a goiter. The matter can be brought closer to our clinical homes, however.

The relationship of language, cognition, and affect is of immediate clinical importance especially when one's patient is a florid, fluent Wernicke's aphasic speaker. At least I know my colleague, Dr. Collins, and I often puzzle over such patients, especially in the acute stage. We wonder aloud if they are not bilaterally involved and confused. There is evidence (Rubens, 1977) that bilateral disruption of blood flow immediately after a stroke is possible, but this does not explain why it is the Wernicke's aphasic person alone who causes us to staff for longer than we ordinarily do. I wonder if such a patient's agnosognosia, irrelevance, and occasional irritability might not imply cognitive and affective deficits. So does Lesser (1978). She says "It is a moot point whether the semantic disruption which appears to exist at a central level in...posterior aphasias must necessarily implicate conceptual thinking or not" (P.187). Perhaps we ought to wonder aloud with Rochford who asked readers of the British Journal of Communication Disorders (1974), "Are jargon dysphasics dysphasic?" A companion question is whether we should admit certain kinds of cognitive deficits into aphasic symptomatology thereby influencing even further what we reply when asked, "When is aphasia, aphasia?"

When is Aphasia, Aphasia?
Contribution of Symptoms

Accompanying most definitions of aphasia are descriptions of aphasic symptoms. Schuell (Schuell, Jenkins, Jiménez-Pabón, 1964), for example, highlights "reduction of available vocabulary, impaired verbal retention span, and impaired perception and production of messages, perhaps secondary to impairment of the first two dimensions" (P.113). Darley's list (1982) is well known and resembles Schuell's. Damasio (1982) mentions two interesting and controversial symptoms. He says, "Paraphasia is the central sign of aphasia" (P.55). And, "A failure to repeat words or sentences is a hallmark of aphasia" (P.56).

I think it is often difficult to differentially diagnose aphasia by listening to a patient speak and read and by watching him write despite the many lists of aphasia symptoms that have been published. The problem with trying to decide when aphasia is aphasia by looking and listening is that there exist no speech-language symptom(s) that are pathognomonic of aphasia. Altered brains have a limited repertoire of speech and language mistakes that they can make regardless of whether their alteration results from fatigue, depression, stroke, trauma, or aging and regardless of whether the damage is focal or diffuse. Granted, if one elicits hundreds of responses from all modalities with a wide variety of stimuli, a hypothesis usually emerges about the presence or absence of aphasia. I would like to suggest, however, that neuroradiologic findings and a variety of nonlinguistic behaviors are as crucial as speech and language ones in diagnosing aphasia. A language disorder is aphasia if the person having it:

1) Has a lesion in the distribution of the middle cerebral artery of the major hemisphere or its borderland which is identifiable on CT scan, if that scan is appropriately timed.
2) Is "reasonable, responsive, and well-oriented...stable, highly motivated, able to work persistently and hard, and to withstand more than ordinary amounts of frustration and discouragement in a manner that commands respect and admiration" (Schuell, Jenkins, Jiménez-Pabón, 1964; P.118).
3) Has good long term memory (McNeil, 1982).
4) Retains premorbid interests such as music if those interests are not primarily linguistic.
5) Makes eye contact.
6) Is aware of what communication is to accomplish even if he cannot accomplish it.
7) Is a good person, if he was good before becoming aphasic.
8) Says he is aphasic (Duffy, 1981).

Conclusion

Decartes in his Discourse on Method, (Sutcliffe, 1968), says, "Good sense is the most evenly shared thing in the world...the diversity of our opinions does not spring from some of us being more able to reason than others, but only from our conducting our thoughts along different lines and not examining the same things" (P.27). Those who think about modalities and a central language factor reason one way. Those who think about types and levels of physiological and linguistic activity reason a variety of different ways. Those who think primarily about underlying mechanisms talk about aphasia in still different ways. All have pieces of the truth. No one is yet among us whose intellectual ladder is tall enough to look down on the parts - or the pieces - and see how they fit. Until that one comes along, we are all free to hug the ground and press on with our individual hypotheses. Our next speakers may not have the ladders but they see clearly and for long distances by merely standing on their tippie toes.

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