Central Auditory Disorders and Developmental Aphasia: Is There a Difference?

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In the first stage of preparation for this paper, I set out to find "hard" definitions for central auditory processing, central auditory disorders, and, in keeping with the title, developmental aphasia. The search led me in several directions, occasionally into cul-de-sac lanes, sometimes along pleasant and nostalgic roads. Every so often, I felt much like the legendary horseman who all at once rode off into all possible directions. But firm definitions were elusive. I found myself involved with points of view, mostly negative, of minimal brain dysfunction, whether or not the damage was minimal and the dysfunction appreciable; and in keeping with my own biases, explanations of problems in reading when the teaching method required auditory intake and auditory processing for the decoding of a visual display. The names of Wernicke and Luria and their assumptions about the auditory area of the cortex and phonetic discrimination were recurrent. More recent references were to psychoneurological learning disabilities as well as to psychological learning deficits. Sometimes the difference between the two escaped me.

For entirely personal reasons, I had little difficulty in arriving at satisfactory definitions of congenital aphasia and developmental aphasia. At the moment I do not raise any question about the possibility of a difference. Personally, I go along with what Eimas (1979) offered as a working (operational) definition of central auditory disorders. Eimas referred to "...deficiencies in the ability to perceive sounds of speech categorically, to analyze and code speech in terms of a phonetic feature code, and to appreciate and utilize contextual information." Such deficiencies, Eimas noted, "...may be functionally related to the development of language disorders other than those associated with reading disabilities." On the positive side, I like to think of central auditory processing much along the line of Lasky & Katz (1983) as the way our central mechanisms receive, perceive, decode and utilize speech-sound signals. It is how and what we do with the speech signals our hearing mechanisms respond to and our central mechanisms select for listening and responding. At this point we have the implied distinction between hearing and listening.

Perhaps we cannot provide a firm definition of what we are talking about any more than we are likely to find a firm definition of cognition. This minor weakness has not prevented the propagation of courses, books, and advanced degrees in cognitive psychology.

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Even in the absence of firm definitions, the members of our related professions — including those of us who are audiologists, hearing and speech scientists, speech/language pathologists (I much prefer speech/language clinicians), and pediatric neurologists — have been neither modest nor negligent in devising tests and procedures for the assessment of central auditory processing deficits in children. Some of these are adaptations of approaches used with adults. Keith (1981), in his edited symposium *Central Auditory and Language Disorders in Children*, included numerous references as well as negative precautions on the evaluation of central auditory disorders. More recently, Berick et al. (1984) reviewed some of the better known tests and procedures. Central auditory function is also a subject of study in dysfluent speech (Hall & Jerger, 1978; Wynne & Boehmler, 1982). I assume that earlier studies of delayed feedback in stutterers and the effect of masking noise on fluency were also concerned with aspects of central auditory functioning and dysfunctioning.

In a chapter on "Neurologically Based Disorders", James Hardy (1978) devoted three pages to discussion that negates notions of minimal brain dysfunction and developmental aphasia as either viable or particularly useful diagnostic entities. Nevertheless, albeit reluctantly, Hardy did consider the possibility that there are children who may have developmental auditory agnosia and perhaps even developmental auditory aphasia. Hardy describes a six-year-old boy who had normal hearing sensitivity when tested with pure tone audiometry, but who could not be tested with speech stimuli. The boy rejected the speech signals and would not respond to them. The rejection itself is diagnostic. The boy's speech output was wholly unintelligible and consisted of combinations of tones and noises. His intonation patterns resembled those of questions and possible attempts at explanations. The child scored no better than chance on language test items presented orally but scored at age level on intelligence test items not calling for oral/aural communication. As a possible partial explanation of the boy's difficulties, Hardy suggested the likelihood of "Difficulty in analysis of the sequencing of the language code."

Hardy appears to be a spokesman for many of our colleagues who are reluctant to accept either the notion of a central auditory disorder or of developmental aphasia except, possibly, when the evaluation includes clear, "hard" neurological evidence to confirm brain damage. The arguments are coldly logical rather than psychological. Hardy does not accept diagnostic classification of a problem by a process of elimination. He and his colleagues hold that mislearning related to inattention may be basic to communicative disabilities and that "The child

may not have learned to attend well enough to discriminate among the acoustic patterns of speech" (Hardy, 1978). This line of argument has several weaknesses. First, in the early months of life, surely without being taught, normal infants show differential responses to speech and nonspeech signals as well as an ability to respond differentially to phonemes with minimal distinctive feature differences. They need only opportunity and do not require teaching to attend to speech and to show evidence that speech is a human species-specific behavior. The position that, because young brains are endowed with plasticity for control of language functions, only bilateral damage to the auditory centers should produce dysfunctions such as auditory agnosia and congenital aphasia is not persuasive.

My concept of cerebral plasticity explains how a function, once established and to some degree shared by centers in both hemispheres, in the event of damage to one hemisphere center for that particular function, may come under the dominant control of the other hemisphere. Initially, we need all the mechanisms with which we are born to establish a function normally and naturally. Further, it is possible that our newer techniques for determining brain function and/or damage may soon provide us with information as to how individual brains are organized and how disorganization is related to cortical, subcortical, and possibly subcerebral systems. It may turn out that we have been looking where it was easy and convenient to look and our looking and seeing may have been limited to the instruments and prejudgements we chose based on our expectations of what and where we would find what we were looking for.

I believe that not enough of our colleagues have been sufficiently impressed with the reality that almost all hearing children learn to speak without being directly taught. Even children with demonstrated articulatory apraxia, unless turned off and away from speaking persons and speaking situations, are usually able to comprehend spoken language. In fact, later in their lives, if articulatory apraxia persists and if they are taught appropriately, they can also learn to read.

The difficulties of children who are identified as having central auditory disorders and those of adults with acquired Wernicke's aphasia are too much alike to be ignored. The results of many investigations provide potent evidence that the impairments of comprehension seen in persons with acquired auditory aphasia constitute a disability of processing speech at the phonemic decoding level. The literature on the subject includes such terms as *word deafness, auditory imperception, auditory verbal agnosia, sensory aphasia,* and *cortical deafness.* Whatever this aphasic auditory disorder may be called, the primary deficit is in speech sound discrimination for temporally ordered events when the signals are presented at the rate and quantity normally spoken by speakers of that language.

Developmental Aphasia

Is there a difference between congenital central auditory disorder and congenital (developmental) aphasia? If there is, it may only be one of preference for terms or of degree of impairment. My preference is *developmental aphasia.* It is a disorder in which the primary impairment is the processing (decoding and in turn encoding) of oral language when the symbol-signals are presented at a normal rate and not in excessive quantity. If this sounds familiar, it is intentional. Processing impairments are evident in children who are "brain different" either because of pre-natal or congenital brain damage, or because of retarded or asynchronous maturation of the regions of the brain involved in speech processing or association between critical areas. For reasons considered earlier, I do not need to discuss the possibility that developmentally aphasic children may not have learned to be attentive to speaking persons, or worse, somehow learned to be inattentive. The drive to hear and to listen is so powerful that the only possibility we need to entertain is that a rare few children may stop listening because they have learned language and the aversive ways of some speakers in their use and abuse of langauge. Until these rare children learn when and why they should not listen, and conversely elect to be mute, the human species-specific drive for language learning is likely to dominate the behavior of potentially normal children.

Children with developmental aphasia may be presumed to show the following: adequate intelligence when assessed with non-language measures and adequate hearing when assessed, however inadequately, by usual audiometric procedures. They usually *do not* show early evidence of emotional disturbances and of non-relating problems and, therefore, should not be confused with children with primary autism. Developmentally aphasic children are almost always born into families whose members are themselves normal speakers and who try to provide normal stimulation and opportunities for listening and responding to spoken language.

The term adequate intelligence does not necessarily imply normal or higher intellectual ability or potential. Generally, the acquisition of spoken language is denied only to severely mentally retarded children. It is possible, as Benton (1978) noted, that "There is also evidence that many retardates show pronounced impairments in the development of linguistic function that cannot be accounted for by their low mental age." Relatively few children in my investigations of developmentally aphasic children fall into the range of the mentally retarded. A few we studied at the Stanford Institute of Childhood Aphasia were well above average in intelligence even when assessed by verbal items. These, of course, were children who had been in therapy for two to three years and were successful in academic studies. One made it to High School Valedictorian.

Differential Features

Developmentally aphasic children cannot encode speech because of a primary deficit in decoding oral language. In contrast, children with oral (articulatory) *apraxia* may have little or no decoding problems unless they are also aphasic. Just as normal children decode

(comprehend) speech before they begin to speak, children with articulatory apraxia may be competent decoders until the limitations of a one-way communication system turn them off and away from speech and speakers. Those who are taught to sign do much better.

At this point we need to differentiate between developmental aphasia and early acquired (childhood) aphasia. Children may become aphasic as a result of cerebral lesion after they have learned to understand and express themselves through speech. If the damage is limited to one hemisphere, more likely as a result of external trauma than disease, recovery of most language functions is usually rapid. An interesting early feature of acquired aphasia in children is a period of mutism, an apparent "loss of ability to initiate speech" (Hecaen, 1976) — Hecaen also noted that, in the early stages, "Disorders of auditory verbal comprehension appeared in more than a third of the cases." Residual impairments include disturbances in naming and serious cognitive and academic problems (Satz & Bullard-Bates, 1981). For example, Cooper & Ferry (1978) described a syndrome of auditory agnosia associated with acquired aphasia for a group of children who were also prone to seizures. Persistent problems included "some degree of dysphasia that may range from auditory verbal agnosia and no functional verbal communicaton to less severe difficulties in...academic areas such as reading and spelling...many of the children required special education."

The literature on early acquired asphasia is burgeoning (Eisenson, 1984; Satz & Bullard-Bates, 1981). Contrary to earlier impressions, there is mounting evidence that most children who have incurred aphasic impairments in childhood do not recover completely, and they are likely to be left with residual deficits which demand continued attention and treatment. Descriptions of the deficits include the presence of impairments strongly suggestive of central auditory disorders.

Primary autism, developmental aphasia, and central auditory disorders.

Is there a possible relationship among primary autism, developmental aphasia, and central auditory disorders? In the 1950's, Dr. Loretta Bender and I frequently appeared on the same panel at professional society programs. Dr. Bender took the position, from which she did not vary, that, if there were such an impairment as congenital or developmental aphasia, it was best regarded as a subtype of childhood autism. I took the position that the two syndromes were separate, but that if pushed I could make a case that primary autism was in fact a severe form of congenital aphasia. I think that I can still argue that such is the case. I can also be persuaded that the presence of echolalia, mutism, and other indications of auditory deficits together suggest that we are dealing with variations of central auditory disorders. I have not gone into detail on the more recent studies of developmental aphasia. However, that information seems to support the view, at least in keeping with my perceptions and prejudices, that the underlying impairment in developmental aphasia is for the processing of acoustic events which are rapidly changing and which require phonemic and temporal order resolution.

Stark, Mellits, & Tallal (1983) reported that language delayed children are less proficient than normal speaking children on tests of visual sequencing. Do children who are, or were, aphasic have parallel and comparable difficulties for the processing of visual events? They probably do, or would, if the visual events were to be presented so that they are as rapidly changing, as transitory, as instantly fading as are auditory signals, and especially those signals that constitute the symbols of speech. In the real, non-experimental world, this is not so. Only speech events fade into the past at the very moment they are presented. Visual events are almost always available for re-viewing, for second and third looks, without our needing to call upon short term memory to recall and process the content. Normally, what we look at, even should we be flying at the speed of the fastest moving jet plane, can be looked at and re-viewed before it is out of sight. What we hear and try to listen to and decode for meaning can be reconstructed only from memory, a memory that requires a knowledge of language to make the guesses reasonably correct and confirmable. Lacking this knowledge, developmentally aphasic children are very poor at guessing. They are weak at dealing with the assumptions and anticipations inherent in the game of linguistic probability. Perhaps, in Chomsky's constructs, developmentally aphasic children lack or are retarded in the establishment of a "Language Acquisition Device" (LAD). They are also slow in establishing a Speech Auditory Device (SAD). This may very well be their primary retardation. Thus, they start out as SAD LADs. Fortunately, with appropriate training and with maturation, both of these early deprivations can be overcome.

A Linguistic Profile of An Aphasic Child

What are developmentally aphasic and dysphasic children like in their linguistic acquisitions and productions when they do begin to decode and encode language? Are the differences between these children and normal peers quantitative or qualitative? What are the likely "residual" deficits often translated and manifested into learning disabilities and slow academic achievement? An important precautionary observation is that children who are labelled as aphasic or dysphasic are members of a hetergeneous population. This may be a result of initial mislabeling or a failure to appreciate that these children change in their linguistic profiles as they mature. In part, these changes may be an expression of the training and education to which they are exposed once a diagnosis is made.

Our own findings support the observation of variability among children whom we diagnosed and treated as aphasic from 1963-1973. For example, Stark, Poppen, & May (1967) found that 5 of 8 aphasic children had difficulty in a sequencing task (impaired memory for sequencing), but 3 children did as well as the control subjects. Part of the problem may be that children with a history of aphasic impairments continue to express uncertainty and anxiety

in experimental situations and perform at a less than optimal level. This behavior may also account for the difference in expressive language usage reported by Morehead & Ingram (1973). Normal preschool and aphasic children were compared on the basis of Mean Length of Utterance (MLU) according to Roger Brown's stages rather than on the basis of age, I.Q., or parents' socioeconomic level. In the early stages, there were few differences between the experimental group (age range from 3 years to 9:6 years) and the controls (age range from 1:7 years to 3 years). Differences increased at the upper stage-levels, permitting an observation that, even with training, aphasic children tended to use shorter utterances, grammatically less complex constructions, and fewer transformations than did normal children. The investigators projected that, at about age ten, aphasic children — really postaphasic or dysphasic children — would be at about the level of normal 3-year-olds. Morehead and Ingram observed that their linguistically deviant children, despite increases in vocabulary, failed to develop — or at least ot use — the complex structures that are there as tokens but which are less frequently used by younger normal children at the same mean length of utterance (MLU) levels. They stated: "Clearly, the major differences between normal and linguistically deviant children at comparable linguistic levels were not in the organization of specific subcomponents of their base syntactic systems. Rather, the significant differences were found in the onset and acquisition time necessary for learning base syntax and the use of aspects of that system, once acquired, for producing major lexical items in a variety of utterances."

As a general observation, relevant to Morehead & Ingram and other investigations conducted at Stanford, we found that severely linguistically delayed children designated as aphasic are quantitatively rather than qualitatively different from normal children in their linguistic acquisitions and productions. What they acquire, at least in relationship to MLU, is essentially the same as acquisitions of normal children. However, the linguistically delayed (post-aphasic) children do not use what they acquire as creatively as normal children for producing varied utterances. This difference, we speculated then may represent a cognitive deficit in representational behavior, at least as these data are related to findings in experimental studies. I suggest that this difference is at least in part an expression of apprehension, of fear of committing an error, rather than of an irreversible deficit. In several studies, we found that aphasic children, as well as those who had progressed enough to be considered post-aphasic, took twice as long as linguistically normal children to indicate choices in test situations.

I wish it were possible to consider recent studies on pragmatics and their implications for aphasic children. My conjecture is that most aphasic children will be delayed shadows of normal ones in becoming linguistic pragmatists. In this respect they will probably require more direct teaching to understand the subtleties of pragmatics, and the differences in how to say what needs to be said in order to enhance the likelihood that the intentions and purposes of their utterances will be achieved. Prutting and Kirchner (1983) are optimistic that it is possible to "teach" linguistic pragmatics to children who are retarded in language acquisition. At this time I share that optimism.

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