

Developmental (Congenital) Aphasia and Acquired Aphasia and Dysphasia: Identification and Differential Diagnosis*

J. Eisenson

Developmental (Congenital) Aphasia: Identification

Developmental (congenital) aphasic children are the most severely linguistically delayed who are not also mentally retarded or emotionally handicapped. In addition to their language impairment, these children also suffer from problems of identification — really misidentification — by too many names and labels, and consequently from misdiagnosis and inappropriate treatment.† These children may also be labeled as *dysphasic* or *aphasoid* — terms that I recommend be reserved for children with less severe impairments than those designated as aphasic. Some professional colleagues who prefer to reserve the term *aphasic* for acquired linguistic impairments use the general term *severely orally linguistically handicapped*. Among the less fortunate misdiagnostic labels are *mentally retarded*, *autistic*, *childhood schizophrenic*, and, despite evidence of ability to hear, *deaf*. A more recent and appropriate designation is *central auditory disorder*. More of this later.

I recommend the use of the term developmental (congenital) aphasia, or if you wish, central auditory disorder, for the child who, despite conditions and observations I am about to present, is severely delayed in both the comprehension and production of language.

- 1) Based on observation and if possible nonverbal assessment, appears to have adequate intelligence for the acquisition of spoken language.
- 2) Has no abnormalities in the structure of the oral mechanism.
- 3) Shows no evidence of early emotional (relating) problems.

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† I shall use the terms developmental and congenital aphasia synonymously for the population of brain different children who are the subjects of most of this paper.

Jon Eisenson, Ph.D.
Emeritus Professor
Hearing and Speech Sciences
Stanford University

- 4) Has no hearing problems except for spoken language. In this regard, the real problem is for listening rather than for hearing.
- 5) Has parents or other caregivers who are available, willing, and presumably capable to provide normal opportunities and stimulation for learning spoken language.

Acquired Aphasia and Residual Dysphasia in Children

The term *childhood acquired aphasia* should be restricted to those children who had acquired language normally and then, subsequent to identified cerebral pathology suffered through accident or disease, became impaired in language functioning. If the child improves and has only residual language and associated cognitive deficits, the diagnostic term should be *acquired dysphasia*. These children will be considered later.

Following is an expanded consideration of the conditions (criteria) for establishing a diagnosis of developmental aphasia and/or dysphasia.

Adequate intelligence does not imply normal intelligence or normal cognitive functioning. Mildly and even moderately mentally retarded children do learn to understand and in turn to speak the oral system of their environment. As a total population, most retarded children are delayed and slower in their acquisition than the nonretarded. Their speech is more often characterized by defects of articulation, voice, and dysfluencies than for normal children (12% compared with 4.5%). However, their predominant deficiencies are in sparseness of vocabulary and difficulty in the comprehension of abstract meanings. But, as Benton (1978) observes, "There is also evidence that many retardates show pronounced impairment in the development of linguistic function that cannot be accounted for by their low mental age." Such children may be considered to be dysphasic-retarded.

Children with *structural anomalies* of the oral mechanism are not immune from the possibility of brain damage or brain-difference that may also make them aphasic. However, the anomaly per se such as cleft lip and/or cleft palate or deviant dental structures would be much more likely to interfere with intelligible speech production than with spoken language comprehension.

Absence of Emotional (Relating) Problems

The particular concern is with the possibility of infantile autism and early childhood schizophrenia.

There is no evidence that aphasic children do not relate normally to mothers and other family members, at least not until they are at an age when language acquisition is expected. Emotional problems are then more likely a reaction to failure in comprehension and language production than a consequence of initial abnormal relating and language learning conditions.

Hearing and Listening

Some aphasic children do have hearing losses of between 15 and 25 decibels in the speech range. Such losses, ordinarily considered to be "mild", seldom cause language acquisition problems in the vast majority of children. In the special population of aphasic children, this range of loss serves as a compounding factor for processing — listening and decoding — what they hear.

From my point of view, the primary impairment in developmentally aphasic children is a deficiency in *Central Auditory Processing*. The impairment is, in effect, a *Central Auditory Disorder*. The effect of a central auditory disorder is to produce "...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" (Eimas, 1971).

In some instances aphasic children appear to be deaf because they stop listening to spoken language. Speech is to them a nondecodable flow of human sounds. They may generalize this impairment by ceasing to respond to environmental sounds, to animal and mechanical noises and other environmental audible events which they usually are able to decode. This may happen as a result of punishment at the hand of parents who do not understand how it is possible for a child to hear non-human sounds and not respond to their speech*. My article among others in the October 1985 issue of *Human Communication* (Vol. 9, No. 5) considers in detail aspects of central auditory disorders and their implications for developmental aphasia.

Available and Capable Caregivers

Few children are likely to learn to speak unless they are spoken to by parents or other persons who are concerned about the result of the quality of their caregiving. Loving the child goes a long way, but it is not enough to get a neonate involved in language behaviour. Perhaps the best explanation of what a parent or other caregiver must be able and want to do comes from the following quotation from deVilliers and deVilliers (1979, p. 99):

"Mothers (and fathers too ...) tailor the length and complexity of their utterances to the linguistic ability of their children. Mothers' speech to one- and two-year-olds consists of simple, grammatically correct, short sentences that refer to concrete

objects and events. There are few references to the past and almost none to the future. Sentence intonation and stress are greatly exaggerated, and clear pauses appear between sentences."

This type and quality of language production — motherese and fatherese — will not overwhelm a child with a flow on in-comprehensible utterance. Such utterance may be compared with how an adult responds when travelling in a foreign country and is exposed to a spoken language never before heard.

Developmental Assumptions

If we may assume that the parents and other significant caregivers do all they can to provide an appropriate speech environment and acceptable physical and emotional care; if we assume that there is no evidence of hearing loss or of an anomaly of the speech apparatus; if we assume that there was no evidence of serious developmental lag in early motor milestones or of prelingual cognitive development and the child fails to acquire language, we must look for other causes to explain the problem.

Neurological Findings in the Developmentally Aphasic

Brain Difference vs. Brain Pathology

Brains that are different in rates of development, either extensively or in specific areas, are deviant from the assumed normal, but not pathological per se. As Geschwind (1979, p. 148) observed: "... brains which show no pathology in the usual sense of the term may yet deviate from the normal." Such deviations, if they involve the parts of the brain that process language intake and output, may account for some instances of severe language delay in children who are identified as aphasic or dysphasic.

Except for the severity of their language delay, many developmentally aphasic children do not present clear-cut "hard-sign" evidence of central nervous system pathology. I include in "hard-sign" evidence such defects as motor disabilities, sensory dysfunctions, and perceptual-motor delays or integrative impairments. Indicators in these categories are found in about one-third of the population who are behaviourally aphasic. Many more show evidence of at least "minimal brain dysfunction". Signs include delayed laterality, late walking, awkwardness, attention difficulties, and perceptual-motor irregularities. But some aphasic children, except for their severe delay in the comprehension and production of language, show neither the expected "hard signs" of neuropathology or the more frequent "soft signs". However, as Ferry (1981, pp. 5-6) argues:

"Delay of deviation in language development is due to disordered brain function ... speech and language delay or impairment may be the only symptom or sign of neurological impairment. This is a reflection of functional localization in which severe damage to a circumscribed area may occur while

* When I am confronted with this problem I explain to the parent the difference between the two hemispheres in the kind of sounds each hemisphere normally processes. I emphasize that almost always the left hemisphere is the one that deals with spoken language; the right with non-speech sounds.

other areas of the brain remain perfectly intact. Thus, although a child with delayed speech development may have a perfectly normal general neurological examination, this should not rule out the possibility that his delayed speech is due to a neurological problem.”

Electroencephalographic (EEG) and Related Findings and other hard-sign evidence of neuropathologies in children with severe language delay and/or disorder are reviewed in Eisenson (1983, Ch. 5). These include a study by Forrest, Eisenson, and Stark (1967) who found that 37 of 73 children had abnormal EEGs. Rapin and Wilson (1978) found that in a population of 87 children all of whom based on neurological observation were considered to have structural brain damage, 26 showed enlargement of the left temporal horn, 6 of the right, and 14 of both left and right. Rapin and Wilson note that “the lateral temporal cortex is concerned with auditory, and in the left hemisphere with linguistic processing.”

The implications of these findings are consistent with those of Luria (1982) and Geschwind (1979) and the general agreement among neuropsychologists and neurolinguists that the left temporal cortex has a special responsibility for the processing of speech signals. An impairment in this processing results in a disability to decode spoken language at the rate and in the quantity at which speech is usually presented. This, I believe, is the essence of developmental aphasia. Evidence to support this position follows.

Perceptual Dysfunctions in Aphasic and Dysphasic Children

During the mid-1960s to early 1970s Eisenson and several colleagues conducted a series of studies on perceptual functioning in which they compared aphasic and post-aphasic (dysphasic) children with normal age peers. These studies are reviewed by Eisenson (1983, Ch. 5). In the late 1970's and the 1980's Tallal and her associates in the United States and England conducted a series of studies on the discriminative and sequencing abilities of dysphasic children. These studies are reviewed by Eisenson (1983, Ch. 5, Tallal and Piercy, 1978, Stark, Mellits, and Tallal, 1983, and Tallal, 1985). Following is a summary of the findings:

- 1) Defects (errors and delays) in the discrimination and sequencing of auditory events occur when the inter-stimulus interval between events is less than 150 milliseconds.
- 2) Discrimination problems appear when the auditory events have rapidly changing features and are of short duration. (These are characteristic of speech events.)
- 3) Production errors in speech in imitative tasks are related to errors in perceptual functioning.
- 4) Errors for non-verbal events usually carried out in experimental laboratories are similar to those for speech events.
- 5) Discriminative judgements are generally more accu-

rate when subjects are permitted to make immediate responses than when short delays are introduced. The short delay periods presented no problems for normal speaking age peers. This finding is interpreted as indicting defective storage and/or poor strategies for the retrieval of auditory (speech) events.

Oral (Articulatory) Apraxia and Dyspraxia

A small percentage of children with developmental aphasia who have made considerable progress in language comprehension may continue to have difficulties in oral language production. Some may, in fact, be suffering from oral (articulatory) dyspraxia rather than aphasia. This impairment may also be congenital and occur as an associated problem. Less frequently, the dyspraxia and the more severe apraxia may be present as a discrete impairment and be confused with expressive aphasia.

Congenital oral dyspraxias are impairments in the ability to produce voluntary movements of the muscles of the larynx, pharynx, tongue, lips, palate, and cheeks that are required for an intended sequence for a speech act. Oral apraxia implies severe impairment. If the comprehension of oral language is not initially impaired, then the child has potentially good capacity for the intake and decoding of spoken language but is impaired in the “mechanics” of production — the expression of encoding. It is not surprising to find that some children will reduce or completely turn away from linguistic communication because for them normal communicative interchange is not possible. Because of this, it is important to make a distinction between congenital aphasic impairments that produce a primary difficulty in language decoding and motor (productive) impairments that may occur in association with the aphasia or as a separate disability.

Developmental Aphasia and Childhood Autism

Is childhood autism an extreme form of developmental aphasia or is it a separate syndrome with overlapping features but a different etiology? May some children suffer from both pathologies, as Cohen, Capurulo, and Schaywitz (1976) believe? Is it more tenable to view the two as syndromes that are related and thus permit designations such as “aphasia with secondary autistic reactions”, or “autism with aphasic components”? Because of our limited knowledge of the etiology of childhood autism, it is not possible to make a firm differential diagnosis based on organic causes. However, if we emphasize the cognitive and linguistic behaviours as the essential differential factors, we could make a case for a continuum of degrees of severity, or for overlapping aberrant behaviours.

My own observations and reviews of clinical histories also suggest the possibility that misdiagnoses and consequent inappropriate treatment of children with developmental aphasia are conducive to producing withdrawal and other autistic expressions. In effect, the

diagnosis produces the behaviours which in turn "justify" the diagnosis. In comparing developmentally aphasic with autistic children we need also take into account the presence in some autistic children of exceptional skills such as verbal reproduction, arithmetic computation, "calendar minds", musical ability, and often remarkably proficient visuospatial orientation.

Childhood Acquired Aphasia

As indicated earlier in this paper, the term *childhood acquired aphasia* should be restricted to children who had acquired language normally and then, as a result of identified cerebral pathology suffered through accident or disease, became impaired in previously established language functioning. For the sake of the present discussion, age 12 will be considered the upper limit of childhood impairment. *Acquired dysphasia* is suggested as the appropriate term for residual or maintained deficits following a period of recovery.

How much impairment a given child experiences following the onset of involvement will vary considerably according to several factors that include:

- 1) Site and amount of brain lesion. Acquired aphasia in children is more often associated with bilateral cerebral pathology than is the case with adults. Aphasic impairments also occur more often with right cerebral lesion than with adults.‡ However, "The risk of aphasia with right brain injury, while higher in children than in adults, is still lower than the risk of aphasia following left-sided injury, regardless of age" (Satz and Bullard-Bates, 1981, p. 401).
- 2) The degree of language at the onset of involvement. This will vary considerably from child to child according to age at onset of impairment. We are more likely to find greater linguistic variability in 3 year olds than in those who are 10 or 11. What may constitute recovery for a 10 year old may be initial language acquisition for a normal 3 year old. Precocious children may have more to lose and more to regain than a slow or even normal 3 year old child.
- 3) Intelligence, sex, motivation and stimulation are additional factors that may account for recovery and variability in early stages of recovery.

Etiology

The most frequent cause of acquired aphasia in children is likely to be associated with head injury. Though rare, vascular pathologies also occur which are comparable to those found in adults. Neoplasms are the least likely cause of cerebral pathologies associated with early acquired aphasia. The literature on this subject is reviewed by Satz and Bullard-Bates (1981, p. 400).

Patterns of Early Language Impairments

A striking and frequent feature of the early stage of acquired aphasia is *mutism* "... loss of initiation of speech

‡ This may be so because in young children, especially those who are below age 5, cerebral dominance for language may not be completely lateralized.

or more generally of the inability to communicate" (Hécaen, 1976). Many children, especially in the early acute stage, have great difficulty in auditory (verbal comprehension). This impairment is, fortunately, of short duration. Hécaen (1976) also observes that "Disturbances of naming have a still greater frequency and tend to persist, the lexical poverty being noted at later stages and even mentioned in school reports." On the motor-expressive side, articulatory impairments — dysarthrias — are frequently present in early stages of involvement and are sometimes persistent deficits. In Hécaen's longitudinal study, 4 of 12 cases had chronic dysarthrias.

Written Language Impairments

In the school age population of children with acquired aphasia we are likely to find disturbances in previously established abilities for reading and writing. Contrary to earlier impressions — before the mid-1970s — most of these children have a considerable way to go beyond spontaneous improvement toward a "complete" recovery. In his published 1976 longitudinal study, Hécaen reported that of 15 children, 3 had persistent deficits in reading and 7 in writing. In addition, 11 of the children had new difficulties with arithmetic.

Based on a review of the literature, Satz and Bullard-Bates (1981, p. 421) summarize the usual findings of investigators of acquired aphasia in children: "Even in cases of recovery of aphasia, serious cognitive and academic sequelae were found."

Therapeutic Approaches for Developmentally Aphasic Children

The content that follows is in no way intended to be an adequate treatment of the subject. However, I would feel remiss if I closed this presentation without a statement on therapy.

For those children who are appropriately diagnosed as developmentally aphasic, or, if you prefer, as having central auditory processing disorders, approaches vary with how the clinician views the problem and individual philosophy of therapy. My own approach (Eisenson, 1983, Ch. 9) emphasizes a semantic-syntactic program that is based on our knowledge of normal language acquisition in preschool children. Sloan (1986) has a detailed developmental program that is directed to overcoming the child's central auditory deficiencies.

Of necessity, I believe that a clinician must undertake direct intervention as opposed to a "naturalistic" approach, or one that at the outset teaches a parent to do the teaching. The language content and the materials should be selected for the individual child and should provide maximum opportunity for carryover from the laboratory to the home and other "natural" settings.

Pragmatic Implications

Whatever the immediate goal might be in establishing conventional language, we do not lose sight of the

pragmatic reality that language is a social tool. When used appropriately in content, form, and setting, the language a child uses influences a caring listener in attaining presumably common interests and goals. Normal speaking children, aside from motherese and fatherese, learn how to say what they are able to say to enhance the likelihood that their intentions will be carried out. Aphasic children need direct instruction and frequent opportunity to put the instruction into use. Thus, like normal speaking children, they can be reinforced by success and satisfaction. Environmental manipulation, such as suggested by Prutting and Kirchner (1983) should help in establishing the language of everyday use.

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