THE PROBLEM OF APHASIA IN CHILDREN

H. VENKATAGIRIAPPA

While aphasia in adults has attracted much research and clinical studies, corresponding literature in the field of 'aphasia' in children is wanting. There is the fundamental question as to whether there exists an entity called 'aphasia in children.' Some deny the existence of aphasia in children on neuroanatomical and neurophysiological considerations (Sugar 1952). However, all the workers in the field are unanimous on one point, i.e., there is a category of children whose inability or inefficiency in acquiring language cannot be ascribed to *(a)* mental retardation, *(b)* sensory involvement, (c) motor disability and/or incoordination and *(d)* emotional disturbances and/or (e) lack of environmental stimulation. A majority of the workers are inclined to call these children aphasics. However, there are at least three differences between adult aphasics and these children.

1. It is very well known that aphasia in adults is caused by damage to the brain. However, there is clear evidence that such a localized lesion in the central nervous system (C.N.S.) of children who are not old enough to have developed speech and language does not preclude the acquisition and use of language by these children in the normal course of their development.

Penfield and Roberts (1959) opine that three cortical areas important for speech will be developed in the dominat hemisphere, that is, in the left cerebral hemisphere and rarely in the right cerebral hemisphere irrespective of 'handedness'. The three areas referred to are: *(a)* a large area in the posterior temporal and posterior-inferior parietal regions which has been termed the posterior speech area; *(b)* a small area in the posterior part of the third frontal convolution, termed the anterior speech area, and (c) part of the supplementary motor area within the midsagittal fissure termed superior cortical speech area or the supplementary speech area.

However, a small lesion before the development of speech in the child may produce some displacement of the expected location of these areas within the dominant hemisphere. In other words, localized lesions in the dominant hemisphere before the beginning of acquisition of speech do not result in inability or marked inefficiency in developing speech and language.

There is a great deal of evidence now to prove that in the event of a diffuse lesion in the dominant hemisphere such as due to some progressive brain disease, the entire speech mechanism will be shifted to the other hemisphere.

Penfield and Roberts (1959) write: 'Examples of completely successful transfer of speech mechanism from the left to the right hemisphere in the children

Mr H. Venkatagiriappa is an Internee at the A.I.I.S.H.

under three or four of age are numerous' (p. 240). Roberts (1960) reports a case who was diagnosed as having chronic progressive encephalitis, the onset of which was at the age of 15 months. Following the onset of the disease she began to develop seizures and developed right hemiparesis. Neurological examination revealed diffuse atrophy of the left cerebral hemisphere. During two operations with a time interval of 22 months in between, most of the parietal lobe, the entire posterior half of the left hemisphere as well as the inferior frontal region including the Broca's area were excised. Though she had some initial difficulty in developing language, she learned to speak, to read and to write. It is assumed that this had become possible with the use of the right cerebral hemisphere.

Brain (1964) also indicates that both cerebral hemispheres are *equipotential* for language purposes, and transfer of language function from what would be the dominant hemisphere to the other occurs if the initially dominant hemisphere is damaged before the age of four or five. He reports that 'instances have occurred in which a lesion of the left cerebral hemisphere in a child who has already acquired speech has caused aphasia, recovery has taken place, and later an intercarotid injection of sodium amytal on the right side has caused aphasia, and demonstrated therefore that the right hemisphere was now concerned with speech'. (Brain 1964, p. 8).

Actually, an entire cerebral hemisphere can be removed in a child and cause no 'aphasic' disturbances. Krynauw (as quoted by Sugar 1952) had removed the entire left cerebral hemisphere from a child with infantile right hemiphlegia. The child had learned to speak and had no speech difficulty after the operation. It is assumed that speech had become possible by use of the right hemisphere.

Then, if one were to explain the lack of or retardation in language development of these so called 'aphasic' children in terms of damage to the central nervous system, one has to postulate damage to the speech areas in both the cerebral hemispheres plus other areas which would have normally taken over the language functions when the original speech areas were damaged. In the event of such widespread lesions in both the cerebral hemispheres, it is most likely that the child would suffer from severe intellectual limitations. And hence, the resultant inability to develop language may be due to mental deficiency rather than due to brain damage itself. In other words, inability or inefficiency in acquiring language may be *secondary* to mental retardation which might be the *primary* result of such brain damage.

Some believe that 'aphasia' in children may represent a disturbance of development (of C.N.S.) rather than a brain damage (Hawke, 1965). This is an interesting hypothesis which needs to be tested by autopsy of central nervous systems of children who were supposed (or 'diagnosed') to have had 'developmental aphasia'. Partial support to this hypothesis is to be found in the autopsy study of a child—who was diagnosed as an aphasic—by Landau, Goldstein and Kleffner (as quoted by Kleffner, 1962). Postmortem examination of this child's C.N.S. tissue revealed bilateral deficiencies of cortical tissue in parts of the temporal, parietal and occipetal

lobes, and severely degenerated medial geniculate nuclei. In spite of such extensive pathology in the brain the child had an audiogram within the normal range, had performance IQ within the normal range and had relatively normal social responses. And with special training, he learned to speak, understand speech, read and write. The above hypothesis seems all the more plausible in view of the fact that many children diagnosed as 'developmental aphasics' would not reveal signs of C.N.S. pathology when neurological examinations are done. (Tayler, 1964, Kleffner, 1962).

2. The second important difference between adult aphasics and children diagnosed as 'aphasics' is: Remarkable specificity for various language functions is established in the adults' brain while the child's brain is still 'plastic' in that no area in the C.N.S. will have assumed special importance for a particular language function. The consequences of this difference are two-fold.

(a) The adult aphasic may manifest marked difficulty in a specific area of language behaviour such as naming or word finding depending upon the locus of the damage in the brain while the 'aphasic' child only shows a decreased verbal learning ability without specific deficits (Cohn, 1962).

(b) While a lesion involving a definite strategic portion of the brain may cause irreparable disruption of one or more language modalities in the adult aphasic, because of non-specificity for language functions in the C.N.S. of aphasic child, recovery is more complete and wholesome (Dekaban, 1961).

3. The third difference between 'aphasia' in adults and 'aphasia' in children lies in the approach to their rehabilitation. This difference stems from the fact that while the adult aphasic had acquired language which he loses, partially or completely, due to brain damage, the 'aphasic' child is yet to learn the language. As it was rightly pointed out by Schuell, Jenkins and Jimenez-Pabon (1965, p. 338), in the rehabilitation of adult aphasics ' . .. the clinician's role is not that of a teacher. He has nothing to do with teaching the adult aphasic to talk or to read or write. He does not teach the patient sounds, or words or rules for combining words. Rather, he tries to communicate with the patient and to stimulate disrupted processes to function maximally'. But in the case of 'aphasic' children, the problem is one of teaching them sounds and words and grammar and syntax. Hence, the techniques that are used in providing language therapy for adult aphasics cannot be used with 'aphasic' children and vice-versa.

From the above discussion it is evident that there is no similarity between what we know of aphasia in adults and what is referred to as 'aphasia' in children except that both the groups show markedly reduced facility in language usage, the former because of loss of an already acquired language system and the latter because of marked inefficiency in acquiring language. While it is definitely known that aphasia in adults is due to brain damage, it is by no means certain that the

same is true of 'aphasic' children. In fact, the neuroanatomical and neurophysiological evidences cited above contradict such an assumption. Hence, the term 'aphasia' is a misnomer when used for children.

In recent years much interest is being evinced in learning disabilities of children who show normal or above normal intelligence (on performance tests of intelligence), who are not deaf or blind, who do not show any motor disability or incoordination and who are not grossly emotionally disturbed. This condition in children is referred to by Johnson and Myklebust (1967, pp. 7-10) as 'psychoneurological learning disability'. This condition may be evident in both verbal as well as non-verbal learning though verbal learning disabilities are more predominant and are easily identified. Implicit in this term are three assumptions: *(a)* Deficit in learning is due to *disability* and not due to *incapacity;* that is, the child has the necessary equipment to learn but is obstructed in some way in the process of learning; *(b)* that this obstruction has its basis in as yet obscure neurological involvement; (c) the observable symptoms of the condition are behavioural rather than neurological. It is suggested that instead of the term 'aphasia', the term 'psychoneurological learning disability' may be used for the condition discussed in this paper.

However, a caution about the diagnosis of children with psychoneurological learning disability (that is, childhood aphasia) is in order. As it was pointed out earlier, the diagnosis of the condition is made by a method of exclusion; that is, when the child's inability or inefficiency In verbal learning cannot be accounted for adequately in terms of such factors as *(a)* mental retardation, *(b)* sensory involvement, (c) motor disability and/or incoordination and *(d)* emotional disturbances and/or lack of environmental stimulation. Obviously such an approach to diagnosis can be erroneous. In fact Bender (1960), holds that childhood aphasia cannot be differentiated from childhood schizophrenia and the greatest number of aphasic anomalies, in early childhood, are found in schizophrenic children. Eisenson (1960) reports that 90 per cent of children earlier diagnosed as 'aphasics' turn out to be either mentally retarded or deaf, or emotionally disturbed. Whetnall (1961), while cautioning about the harm that can be done by indiscrete labelling describes eight children who had been diagnosed as aphasics. It later turned out that three of them were 'late developers' who began to develop speech on their own, without any special training between 4 and 4½ years. Another was mentally retarded with an IQ of 52. Still another was severely deaf with a 90-dB (I.S.O.) hearing level, combined with cerebral palsy. One more child was premature, less than three pounds at birth who presented general problems of development. The last child was both deaf and very retarded. A study made by Goldstein, Landau and Kleffner in 1958 (as quoted by Kleffner 1962) indicates that exhaustive investigations including *(a)* detailed developmental and medical histories, *(b)* skull X-rays, *(c)* clinical neurological tests *(d)* vestibular tests and (e) complete audiometric tests failed to differentiate conclusively, a group of children diagnosed as 'aphasic' (N=69) from a group of children diagnosed as deaf (N=114).

Also, the children do not fit into neat categories. Children with learning disabilities may also have minor motor incoordinations, as well as some degree of emotional disturbance. A child may have learning disabilities superimposed on deafness; mental retardation or any one or more of other handicaps. The possibility of multiple handicaps being present in a child should make one all the more cautious in categorizing children.

From the above discussion the following conclusions may be drawn: (I) There is a group of children who are confronted with a special difficulty in verbal learning (and to a lesser extent in nonverbal learning) including speaking, reading and writing. And this difficulty cannot be ascribed to *(a)* mental retardation, *(b)* sensory involvement, *(c)* motor disability, and *(d)* emotional disturbances. Presumably, this condition is due to neurological involvement, the exact nature of which is yet to be discovered. (2) The use of the term aphasia to denote this condition is misleading and hence may be dispensed with. Instead, the condition may be referred to as 'psychoneurological' learning disability. (3) Caution may be exercised in diagnosing children with psychoneurological learning disability.

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