

"CEREBRAL PALSY AND LANGUAGE DISORDER"

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1. INTRODUCTION

Our knowledge of the possible methods to acquire language or causes of failure to do so is largely speculative both at a general and a particular level. It is more so in the case of non-normal (Subnormal / abnormal ?) studies. Moreover, it is also not clear as to the extent to which language disorders are related to a central nervous system etiology. Hence, in this paper an attempt has been made to find out the causes of language disorder which may not be directly related to the pathological investigation of the brain and nervous system but irrevocably responsible for the poor language development of a CP child.

This paper is based on private observation of a CP child for a decade and not on any experiment or research. Hence, no scientific truth is claimed. While living with the child, it is also felt that neurolinguistic study of the language is no less different than psycholinguistic study of the child.

Finally, no attempt has been made to investigate as a result of brain damage which specific development of a non-normal child takes place and at what stage.

2. BRAIN AND NERVOUS SYSTEM :-

Right from the moment of our birth our body is controlled and dominated by a single but complex organ, a library, switchboard, signal-box, computer and many other things all rolled into one - the brain. It matures slowly, unlike other body organs, reaching its peak of efficiency and potential and with remorseless steadiness, it slides into decline. Its proper functioning determines the "normality" of the behavior of the individual and the part he can play in society, its efficiency determines to some extent his intelligence and his ability to compete, its storage capacity and information retrieval system, his potential to learn and remember. In addition its computing power determines his creativity and is

the primary function which distinguishes between the mature human being and the other animals on earth.

In the first place, the brain is probably composed of ten thousand million (10,000,000,000) nerve cells and perhaps four or five times these number of supporting "glial" cells. They work as communication units, receiving electrical signals from other parts of the body. On the whole, messages from the eyes tend to finish up in the back of the brain or occipital lobes; messages from the ears, nose etc. ~~on~~ the side or temporal lobes. In the front exist a large chunk of nervous tissues which seems to be more directly related to the personality and mood of its owner than to any of his senses. There are parts related to motor areas and there are parts principally involved in the storage of information, memory etc.

These areas are known as the cerebral cortex and they are located on the outer surface of the brain itself - i.e. right up against the inside of the skull. This is also known as 'neocortex' tucked in beneath the cerebral cortex, growing out of the spinal cord are the so-called lower centres - the old brain which deals with bodily functions such as cardiac activity, respiration, temperature etc.

Hence, we note that the brain is divided into two major functional areas in evolutionary terms - the old and the new. The former deals with the vital automatic processes and the latter with the receiving and interpreting of sensory input and the decision making processes which ^{guide} our interaction with the outside world.

But most of the functions of the brain are quite unknown and even the ones we know about are very poorly understood. It is assumed to be the organ for higher mental function, of the mind and intellect: but there is surprisingly little evidence for this, and no one has any idea what physical structures or mechanisms subserve these functions.

The brain is known to control all bodily functions by means of motor and other nerves which carry impulses from the brain outward to all parts of the body. Sometimes these are under our voluntary control, mostly they are involuntary, reflex or automatic. Reflex actions are the result of impulses passed inwards from the body towards the brain by means of sensory nerves. Clearly the brain is a very delicately organized piece of machinery and its cells are extremely specialized for their job.

The brain and the nervous system are complicated almost beyond description with much of their mechanism and rules of operation still poorly understood. It is only known that the nervous system consists of the Central Nervous System (CNS) and the Peripheral Nervous System (PNS). The brain and spinal cord make up the former and the latter consists of the nerve fibres by which the Central Nervous System is connected to all parts of the body. Human beings have a more complicated brain than any other animal species and its only ^{is} complexity which makes it different from the brains of other mammals.

3. CEREBRAL PALSY CAUSES AND CLASSIFICATION

3.1 CEREBRAL PALSY. What is it ?

Cerebral palsy is a part of ^a continuum of dysfunction which at one end merges into the field of mental sub[^]normality and at the other end into that of 'minimum brain dysfunction'. As a result, neurological mechanisms of posture balance and movement are disorganized. The muscles which are activated for maintaining posture, balance and movement become uncoordinated or weak. Sometimes brain damage also results in sense defects of vision and hearing, abnormalities in speech and language and aberrations of perception. 'Agnosia', 'apraxias', 'hyperkinesia', distractibility may also be the result of organic brain damage. All these defects lead to various learning problems

and difficulties in communication.

It is not evident that every cerebral palsied child has all of these problems or some of these associated handicaps. But it is definite that even if the CP child is only physically handicapped, due to paucity of movement, the child cannot explore the environment fully which results in lacking everyday experiences and retards the development of language and affects the child's speech. And as his general understanding suffers, it appears that the child is mentally retarded. This can go so far that normal intelligence has been camouflaged by severe physical handicap. Furthermore, the lack of movement can affect the general behavior of the child. Thus the same abnormal behaviour may be due to lack of satisfying emotional or social experiences for which movement is necessary.

3.2 Causes of CP :-

There are many causes of brain damage including abnormal development of the brain. Anoxia, intracranial bleeding, excessive neonatal jaundice, trauma, infection, lack of oxygen in the mother's womb are some of them. Causes of CP take place in the prenatal, perinatal and postnatal periods. In all cases, it is an immature nervous system which afterwards continues to develop in the presence of the damage.

3.3 Types of CP :-

CP may be classified as quadriplegia, diplegia, paraplegia, triplegia, hemiplegia, monoplegia and so on. And they may be spastic, athetoid or ataxic. Though these types or classifications are not clear cut but for clinical reasons, these classifications are done.

CP consists of both motor delay and motor disorder. All these conditions are also called the 'Developmental' Disabilities'. They may be due to

(1) Mental Subnormality which is caused by various metabolic disorders, chromosome

anomalies, lencodystrophies, microcephaly and other abnormalities of the skull and brain.

(2) Deprivation of normal stimulation associated with social, economic and emotional problems.

(3) Malnutrition alone, but usually together with deprived environments, cause the developmental delay. Once malnutrition is treated, lack of normal stimulation may still retard the child's development.

(4) The presence of non-motor handicaps which may lead to motor delay e.g. blindness, severe perceptual defects, apraxias as well as mentally handicapped children with delay in any developmental area may have an associated delay in motor development.

(5) Finally, presence of motor handicaps e.g. Spinabifida, the myopathies, myopathies and various progressive neurological diseases and congenital deformities may obviously delay development.

4. NEUROLINGUISTICS AND LANGUAGE DISORDER

Neurolinguistics seeks to understand the form of representation of language in the human brain; the neurological basis for language and speech and the nature of the mechanisms and processes implicated in language use. It is a broad field of study necessitating a multidisciplinary approach to language and brain function. Other areas of clinical importance subsumed within neurolinguistics include language disability in children, dyslexia etc.

Very few neuroanatomical and neuro-histological studies of the language areas of the brain have been conducted. It is believed that in 90% of the right-handers, the left hemisphere is dominant for language and certain portions of telencehalic cortex

including some sub cortical structures are responsible for processing of any language.

Language disorder among children has not been the object of linguistic research to the same degree as adult aphasic patients. Such research is relevant to a number of questions concerning the language acquisition process. For instance the nature of the relationship between the brain and knowledge of language; if the proposed neurological substrate is damaged the consequences thereof for acquisition / knowledge of key linguistic principles. The ways in which a developing grammar can be damaged and ^{what} the long term linguistic consequences of a specific language impairment might be, are as yet unknown.

Secondly, the research is important because approximately 8.4% of the world's children population (Silva 1980) and nearly 15% (?) of India's has been suffering from language disorder in some form or the other.

Few details are available about the actual character of the linguistic impairment as is the case with the adult population. Language impaired children appear to acquire grammatical structures in roughly the same development sequence as non- impaired children but tend to have a protracted rate of acquisition, a disparity in their performance on comprehension versus production tasks and persistence of early-learned forms alongside later mastered forms (Johnston and Schery 1976; Leonard 1982).

Disorders in language are mainly of two types. a) Primary disorders - refer to those who have 'Specific language impairment' 'developmental aphasia' (Benton '64') or simply a 'language disorder (Rees '73) and b) Secondary disorder which denotes total retardation, autism or hearing impairment.

A child with 'specific language impairment' suggests impairment of one or more areas of linguistic development in absence of other impairments including any impairment of non-

verbal intelligence. (American Psychiatric Association 1980). Most of these children perform below-age expectation in non- linguistic areas too, on Piagetian tasks of symbolic abilities' (e.g Kanhi 1981) in measures of motor development and coordination (King, Jones and Lasky 1982) and in measures of auditory, processing skills (Tallal & Piercy 1973, '75).

Developmental aphasia generally means an impairment in the production of spoken language or development of phonological disorders. In fact developmental aphasia, a deficit in the volitional control of the articulators in producing speech movements (Williams, Ingham and Sosenenthal 1981), developmental dyslexia and developmental aphasia are not mutually exclusive categories and further a child's diagnosis may change overtime (Wetherby 1985). These phonological disorders or functional articulation disorders are described by Shriberg and Kwiatkowski (1982) as impairment in knowledge and use of phonological rules.

4.1 Adult Aphasia and language disorder :-

It is believed that adult aphasia faces the problem of language impairment which was premorbidly normal with a sound cognitive system. In other words the organization of language, cognition and the interaction of these two systems are believed to be similar to normal adult prior to brain damage. Common clinical attributes in aphasia is a naming deficit - an inability or inconsistency in giving the appropriate name for an object an action and so on, upon confirmation. It may manifest itself in word difficulties (i.e. groping for the appropriate word) Semantic paraphasias (i.e. Word Substitute) or empty speech (i.e. Use of such non-specified words as 'thing' 'be') in the patients spontaneous speech output.

Nearly all aphasiacs manifest some phonological difficulties in speech output with a

broad array of phonological errors. These can be reduced to four descriptive categories (Blumstein '73); phoneme substitute errors in which one phoneme is substituted for another e.g. teams - (Kimz); Simplification errors in which a phoneme or syllable is deleted - e.g. green - (gin); Addition errors in which an extra phoneme or syllable is added e.g. See (Sti) and environment errors in which a particular phoneme occurs that can be accounted for by the influence of the surrounding phonological context. The environment errors include metathesis e.g. degrees (qedriz) and progressive and regressive assimilation e.g. championships (ixeinsips) and crete (trit) respectively. The study of aphasia reveals that the processing operations which affect the access to the linguistic components are impaired but whether the structure of the same is also affected or not is still questionable. The study also reveals that language is independent (within limits) of cognition and organized into sub- systems which have their own vocabulary, structure and operating characteristics but probably they do not have localized areas of the brain.

4.2 CP and Language Disorder :-

It is highly debatable whether the present neurophysiology is at all capable of bringing out enough information about the damaged nervous system. Because normal child psychology and normal physiology are used as a prism through which people tend to judge subnormal children and form hypothesis which are unproven. Neuropsychology has rarely been used as basis for these sub-normal children. Mental sub normality has a very large number of different causes and conditions associated with it. The majority of *being subnormal in terms of family/social* causes are not scientifically proved because the consequences of *life* and experiences of the people are likely to differ widely because of its diversity of causes.

Hence, knowledge of the possible 'causes' of a CP child's failure to acquire language is largely speculative and no clear- cut concept in this matter has been evolved both in

general and in particular cases, CPs are just labeled as 'low-grade Subnormal' without any extensive neurological investigation.

4.3 Problem of Diagnosis :-

In spite of the physiological basis of labeling children as CP cases it is very difficult to predict the degree of their sub-normality in the true sense of the term. The main problems that a doctor faces are :

- 1) Babies with marked early neurological signs may later prove to be only mildly affected or even normal, whereas apparently mildly affected ones may become progressively worse with the years.
- 2) Normal child development is the basis on which the abnormal development is appreciated. Assessment and treatment should not, however, rely upon a strict adherence to normal development schedules. Even 'normal' children show many variations from the 'normal' development sequences and desired patterns of development. A CP child has frequently achieved abilities at one level of development, omitted abilities at another level and only partially achieved motor abilities at still other levels. *There is thus a scatter of abilities in the case of a CP child.*
- 3) Speech and language, social and emotional and intellectual levels may all be at different ages. None of these ages may coincide with the child's chronological age. This is more so in case of the CP child.

4.4 General causes of Language Abnormalities of a CP Child :-

Failure of language development in many subnormal is that the causes must be many and various, both between and within individuals.

1) There is increasing evidence suggesting that mothers or parents of 'normal' children adjust their speech in accordance with the age and advancement of their children's verbal abilities (Ryan 1974) but the exact way in which this is done in relation to the children's growing competence is not really clear. However there are some indications that with sub-normal children this normally useful process may be taken to disadvantageous lengths.

2) There is also a suggestion (Jeffrey and Cashdan 1971) that parents tend to talk to their subnormal children in a way that only requires single word answer which are often the names of subjects. Casual observation in many subnormal schools suggests this may happen fairly often, even under the guise of stimulating language.

3) People sometimes talk too simply to subnormal individuals and thus do not provide them with a verbal environment from which they can learn. Normal people talk down to them, by talking too loudly with exaggerated slowness and emphasis and as though the recipient will not understand.

4) Another reason for the possible dysfunction of a subnormal child's language development is that at the age when words are first produced the 'normal' child is still in frequent physical contact with the mother, with much picking up on her part, and face-to-face interaction of various kinds. But such physical contact and the associated kinds of interaction are less likely to occur at a time when the child is starting to talk. This in itself may further retard the child's development. Since there will be fewer useful opportunities for the extensive verbal, semi-verbal and non-verbal dialogues' and interchanges carried on with a sub-normal child.

5) Parents of subnormal children welcome any form of speech rather than none, however unproductive conversationally. This may give rise to the persistence of some

form of speech'.

6) Any error of a normal child is generally welcome^d by his parents and is enjoyed but any mistake of a sub-normal child is heavily corrected. Hence, the child's competence in the development of language is checked.

7) Finally, there is a strong cultural tendency to judge people by how they talk. Such general cultural assumptions may lead people, including subnormal people, to expect that subnormal people will speak badly and understand little. This attitude of the society is held responsible for their poor performance in language too.

5. CONCLUSION :-

'Amae haraar dawle boshia dile Jitbo Kamon Kore ?' Rabindranath Tagore, (If you place me in the team of losers how can I win ?)

In studying or treating subnormal children we see only the child's defect and not our own involvement. 'To wish to treat the symptom is to reject the child himself' (Mannoni 1964). In reality, the language skills of subnormal people cannot be considered in isolation from their social situations and life experiences. It is often found that CP children have difficulty with verbal skills because adults do pay very little attention or do not listen to them at all. The role of the adults in the CP child's environment in maintaining defective articulation has not been investigated for various reasons either lack of attention or acceptance of partial intelligibility and a reluctance to correct it. Hence, it is found that 'Subnormal children spoke less, but their mothers spoke more' (Marshall et al 1973) than was the case with the comparison normal group.

Children who are labeled as sub-normal generally determine the expectations of behaviour of both the children's and the adults. As they are categorized as deficient in

respect of language, society does not bother to know the complexity of processes involved in it and label them as subnormals .

In Special Schools too, teachers or guides become so particular with 'helping' the child that the behaviour of teachers towards subnormal children has not been investigated. Teacher's role within segregated schools may be evaluated properly so that they can be termed a real help to the child.

Finally, we have to remember that language programs are only a small part of the extensive literature on behaviour modification - this may be because it is more difficult to carry out successfully or because it is more difficult to train nurses and other personnel to do so or because it is low priority in hospital settings.

BIBLIOGRAPHY

1. **Benton, A 1964** :- Developmental aphasia and brain damage. Cortex 1 : 40 - 52
2. **Blumstein, Sheila E. 1989** :- Neurolinguistics: an overview of language - brain relations in aphasia' in Linguists, The Cambridge Survey III Language : Psychological and Biological Aspects. By Frederick J. Newmeyer (ed). Cambridge University Press. Cambridge 1989.
3. **Caplan, David; 1989** :- The Biological basis Language, in The Cambridge Survey III. Cambridge University Press. Cambridge.
4. **Jackson, C.A. 1989** :- Linguistics and Speech - Language Pathology in Linguistics The Cambridge Survey III. Cambridge University Press. Cambridge.
5. **Jeffree, D.M and Cashdan, A. 1971** :- Severely Subnormal children and their parents - an experiment in language improvement. British Journal of Educational Psychology 41:184 London.
6. **Johnston, J. and Schery, T. 1976 : Leonard'82** :- The use of grammatical morphemes by children with communication disorders in D. Morehead and A Morehead (ed). Normal and deficient child language. University Park Press. Baltimore.
7. **Kanhi, A. 1981** :- Nonlinguistic Symbolic and Conceptual abilities of language impaired and normally developing Children. Journal of Speech and Hearing Research 24:446-53.

8. **King, R.; Jones, C. and Lasky, E. 1982** :- In retrospect: a fifteen year follow-up report of Speech - language disordered children. Language, Speech and Hearing Services in Schools 13:24-32
9. **Levitt, Sophie; 1981**:- Treatment of Cerebral Palsy and Motor Delay. Blackwell Scientific Publication. London.
10. **Ludlow, C. 1980** : Children's Language Disorders : recent research advances. Annals of Neurology 7:497-507
11. **Luria, A.R. 1959** :- The directive function of speech in development and dissolution. Part 1. Development of the directive function of speech in early childhood, World P.P 341 - 352. 12. **Lyle, J.G. and Ryan Joanna. 1961** :- A Comparison of the language of normal and imbecile Children. Journal of Mentally Deficiency Research. 5:40-51
13. **Mannoni, M 1964** : The backward Child and his mother, Trans. Pantheon NY.
14. **Marshall, J.C. et al. 1977** : Disorders in expression of language in Psycholinguistics Development and Pathological by John Morton and J.C. Marshall, Cornell University Press. Ithaca. NY.
15. **Paul Fletcher and Michael Garman 1988** : Language Acquisition. Cambridge University Press. Cambridge.
16. **Piaget, J. 1959** :- The Language and Thought of the Child Routledge and Kegan Paul, London.
17. **Rees, N. 1973** :- Auditory Processing factors in language disorders: the view from Procrustes' bed. Journal of Speech and Hearing Disorders 38:304 - 15.

18. **Ryan, J. 1977** :- The Silence of Stupidity in Psycho linguistics, Development and Pathological by John Morton and John C. Marshall. Cornell University Press. Ithaca NY.
19. **Silva, P. 1980** :- The Prevalence, stability and significance of developmental language delay in preschool Children. Developmental Medicine and Child Neurology 22:768-77.
20. **Shriberb, L. and Kwiatkowki, J. 1982** :- Phonological disorders I; a diagnostic classification system. Journal of Speech and Hearing Disorders. 47:226-41
21. **Tallal, P. and Piercy, M. 1975** :- Developmental aphasia: impaired rate of nonverbal processing as a function of sensory modality. Neuropsychologia. 11:389 - 98.
22. **Vygotsy, L.S. 1962** :- Thought and Language Translated by E. Hanfmann and G. Vakar M.I.T, Press. Cambridge.
23. **Wetherby, A.M. 1985** :- Speech and Language disorders in Children - an overview in J.K. Darby (ed). Speech and Language Evaluation in Neurology : Childhood disorders. Grune and Stratton. New York.
24. **Williams, R; Lughan, R; and Rosenthal J. 1981** : A further analysis for development apraxia of speech in Children with defective articulation Journal of Speech and Hearing Research. 244:496 - 505.