

DEVELOPMENTAL DYSPHASIA - IDENTIFICATION AND EVALUATION

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DEDICATED TO:

All those affectionate ones who made this study possible

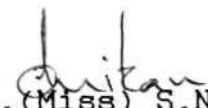
and

to my guide KARANTH MA'AM

CERTIFICATE

This is to certify that this Disertation entitled:
DEVELOPMENTAL DYSPHASIA - IDENTIFICATION AND EVALUATION is
the bonafide work in part fulfilment for the Second year
MSc, (Speech and Hearing) of the student with Reg.No.M9322.

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C E R T I F I C A T E

This is to certify that this Dissertation entitled :
DEVELOPMENTAL DYSPHASIA - IDENTIFICATION AND EVALUATION has
been prepared under my supervision and guidance.



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DECLARATION

I hereby declare that this Disseratation entitled: **DEVELOPMENTAL DYSPHASIA - IDENTIFICATION AND EVALUATION** is the result of my own study under the guidance of **Dr.Pratibha Karanth** Prof. and Head of the Department of Speech Pathology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore
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Reg.No. M9322

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TABLE OF CONTENTS

	Page No
Introduction	1 - 4
Review of Literature	5 - 50
Methodology	51 - 55
Results and Discussion	56 - 71
Summary and Conclusion	72 - 74
Bibliography	75 - 91
Appendix	

INTRODUCTION

Bobby is a child brought for speech and language evaluation. A battery of tests administered on Bobby indicate that he is not performing at the appropriate age level on speech and language tasks. Bobby, as the clinician observed is somewhat delayed in his language development or in other words, he appeared to be somewhat 'slowed down' in his language abilities. On trying to isolate the cause for Bobby's problem, the clinician finds none of the causes of neurologic, cognitive, sensory or emotional appropriate. The diagnosis in this case is hence a big query. Is he a pure case of delayed speech and language development or does he have some finer problems which have just evaded the clinician's eye? The answers to these questions are far from being satisfactory.

Multiple terms and labels have been used to refer to and classify children with language disorders. In addition to their language impairment, these children also suffer from misidentification by too many names and labels assigned to their condition. Among the misdiagnostic labels are 'mentally retarded', 'autistic', 'childhood schizophrenia', 'deaf and 'delayed speech and language'. Thus, there is a waste basketing of terminologic confusion.

Some children with language disorders appear to be developing normally in all other areas of development except in their language abilities. The parents feel that they must be just 'lazy and 'not trying to talk' or that their child has some psychological problem or brain abnormality. The parents are given all sorts of advice 'Do nothing', 'he will surely out grow it' "Einstein was late to talk'.

Clinicians are thus unable to place these children with characteristics as described above under any of the set diagnostic labels. Thus the presence of a discrete diagnostic label is severely lacking. Based on the several researches conducted on the language problem seen in children, clinicians began to widely use the coinage 'developmental dysphasia' for children like Bobby.

The term developmental dysphasia has come into use to denote slow, limited or otherwise faulty development of language in children who do not otherwise give evidence of gross neurological or psychiatric disability. The outstanding handicap of developmental dysphasia is social and educational rather than physical and sensory or motor.

The severely developmentally aphasic child is a rare child indeed. However rare, he does exist and is a puzzle to himself, a source and cause of bewilderment to his parents, and a challenge to the pediatrician, educator, psychologist and language pathologist for understanding, diagnosis and appropriate treatment.

The identification and evaluation of children with developmental dysphasia is universally recognized and is a continuing challenge for clinicians and researchers interested in understanding and helping such children (Lahely, 1988; McCauley and Demetras, 1990). The identification of developmentally dysphasic children is particularly complicated given the heterogeneity of the language problems present in these children (Stark and Tallal, 1981), diversity in the aetiology of developmental dysphasia (Leonard, 1987) and the lack of understanding of the factors affecting prognosis (Bishop and Edmundson, 1987). Still other factors complicating the selection process include the varied operational definitions used by researchers (Tallal, 1987) and the varying performance and adequacy of measures incorporated within those definitions (Demetras, 1984; Fuchs, et al. 1987).

More recently, trend has been to use terms like 'language disorder', (Hughes and Sussman, 1983), 'language delay' (Tallal and Stark, 1978) or specific language impairment (Johnston, et al. 1981).

Inspite of the several characteristics quoted above and despite a lot of research carried out in this area, the differentiation between delayed speech and language development without any associated problems and specific language impairment has not really come about. Hence, the main aim of the present study is to develop a checklist which will aid the clinician in differentially diagnosing delayed speech and language without any organic involvement/problem from developmental dysphasia. The diagnostic tool, it is hoped, will also aid the clinician in successful management of the developmentally dysphasic child.

REVIEW OF LITERATURE

Child language disorders as a speciality in speech-language pathology grew out of three important and divergent sources of information.

1. Adult aphasiology
2. Other medical disciplines
3. The field of deaf education.

Roots in Neurology: Adult aphasia: In the 1800's impetus for the study of the relationship between language behaviour and that part of the brain responsible was sparked by the provocative work of neurologists like Broca (1861) and Wernicke (1874) cited in Aram and Nation (1982). They were later joined by psychologists, linguists and speech-language pathologists interested in brain-behavior relationships.

It was only logical and a matter of time until parallels between adult aphasia and child language disorders were noted. The study of adult aphasia had a major impact on the early work in child language disorders, it served as both the inspiration and experience base from which the pioneers in language pathology launched their work.

Roots in other medical disciplines: A handful of individuals representing various other medical disciplines, notably child neurology, psychiatry and pediatrics, began to present descriptions of children who do not talk who were referred to as aphasic.

Orton (1937) cited in Aram and Nation (1982) was perhaps the first neurologist to become concerned with communication disorders in children. In his book 'Reading, Writing and Speech problems in children', he provided classifications, descriptions and treatment programs for developmental alexia, developmental agraphia, developmental word deafness, developmental motor aphasia, developmental apraxia, stuttering in childhood and a group of mixed or combined syndromes.

Strauss (1954) cited in Aram and Nation (1982) in a paper titled 'Aphasia in children' reflected on his 30 years of experience with aphasic children. He referred to this disorder as oligophasia, signifying a deficit in language or lack of language development rather than a loss of language, he identified three types of oligophasia.

1. Receptive oligophasia : A disturbance in auditory perception
2. Expressive oligophasia : A disturbance recognizing and forming phonemic patterns.
3. Central oligophasia : A disturbance of symbolization

Ingram and Reid (1956) cited in Aram and Nation (1982) provided the most comprehensive information (characteristics and presumed etiology) of 78 developmentally aphasic children.

Roots in education of the deaf: These professionals were experienced in observing and remediating children who did not talk. Therefore they drew attention to children with little or no language and developed techniques for working with such children.

Ewing's (1930) cited in Aram and Nation (1982) contribution is notable as he gave rise to one of the earliest treatments for these children. Other contributors were Myklebust (1954), McGinnis et al. (1956) and Hardy (1965) cited in Aram and Nation (1982), who arrived at the fundamental observation that some children with and without hearing-impairments learned language easier than others.

McGinnis, Myklebust and Morley worked independently but came together in 1950s and gave birth to the field of child language disorders. Along with Kleffner and Goldstein (1956) cited in Aram and Nation (1982), McGinnis provided description, classification and probably most notably a systematic teaching method for aphasic children (Association method). These workers defined aphasia in children as an inability to understand and or express language resulting from a central nervous system dysfunction. From this they described two sub-groups:

1. Expressive or motor aphasia is characterized by
 - a) lack of expressive speech
 - b) adequate understanding of speech, comparable to a normal child
 - c) Vocalizations consisting of patterns of sounds repeated over and over
 - d) a partial or complete inability to imitate actions or positions of the tongue, lip and jaw or of sounds and words.
 - e) adequate control of muscles used in speech and for other acts such as chewing or swallowing.
 - f) adequate hearing
 - g) adequate intelligence.

2. Receptive/sensory aphasia is characterized by
- a) a lack of understanding of speech
 - b) lack of expressive speech that could fall into one of the four categories (little or no vocalizations; scribble speech-jabber or chatter that had considerable inflection and was usually accompanied by facial expression and by gestures; echolalia or appropriate use of a limited number of words or phrases).
 - c) adequate control of muscles used in speech and for other acts such as chewing and swallowing.
 - d) a discrepancy between the ability to hear and ability to understand spoken language.
 - e) a discrepancy between intelligence and ability to understand spoken language.

As a psychologist, Myklebust was interested in why children did not respond to sounds. In 1954 his book, 'Auditory disorders in children', appeared. Here he differentiated four groups of children with auditory disorders caused by (1) peripheral deafness (2) aphasia (3) psychic deafness and (4) mental deficiency.

In her classification of speech disorders in childhood, Morley's (1957) cited in Aram and Nation (1982) first

category was disorders of language that she subclassified into (1) aphasia of two types, mainly receptive and mainly executive (2) alexia (3) agraphia (4) delayed development of speech associated with (a) general mental retardation (b) mental illness (c) hearing deficiency.

The work of the three M's set the stage for the entrenchment of etiologic typologies for classification, diagnosis and treatment of language disordered children in the 1950s and 1960s.

Terminologic confusion:

Language disorders in children were first recognized and described by physicians (Gall, 1825; Vaise, 1866; Wilde, 1853) who noted that there were children in schools for the deaf and the mentally retarded but still could not speak. Their lack of oral language was compared to the loss of language in adult aphasics who had sustained brain injury. The term 'aphasic' was applied to these children even though they, unlike adult aphasics had never spoken and displayed no obvious signs of brain damage.

Landau, Goldstein and Kleffner (1960) gave the term 'congenital aphasia' to refer to those children who fail to

develop normal language functions in the absence of deafness, mental deficiency, motor disability, emotional disturbance or gross neurological disability. The same children were referred by Benton as 'developmentally aphasic'.

In other publication (Eisenson and Ogilvie, 1971), the term 'dyslogia' was used to designate the child who though not deaf, not mentally retarded, nor autistic, nevertheless presented evidence of central nervous system involvement associated with severe language delay.

The term 'developmental dysphasia' was used by Tallal, Stark and Curtiss (1976). In addition to the term congenital or developmental aphasia, these children are also labelled as 'aphasoid'.

The terminology conflict in the area of clinical language disorders was discussed by Fry (1968) and Sprcen (1976). They provided the following instructions for forming diagnostic labels in what was called a 'Terminology Generator'.

Choose any term from column I, combine it with one term from column II and one from column III and you have an accepted diagnostic label. The term appearing in a box in column II may sometimes be used alone.

I	II	III
Primary	language	disorder
Secondary	linguistic	disability
Specific	learning	delay
Minimal	cerebral	deficit
Mild	brain	dysfunction
Congenital	perceptual	impairment
Developmental	visual motor	pathology
Chronic	neurologic	syndrome
Childhood	education	handicap
Psychoneurological	aphasia	problem
Functional	dysphasia	injury
	dyslexia	

-More recently, there has been a trend toward the use of rather neutral terms such as 'language disorder' (Hughes and Sussman, 1983), language delay (Tallal and Stark, 1976) or 'specific language impairment' (Johnston, Stark, Mellits and Tallal, 1981) in order to avoid unintended implications regarding the essential nature of the disability. This

diversity of labels indicate that little is known about its true nature or underlying etiology.

Definitions:

The problem of appropriately defining specific language impairment (SLI) in children has led to difficulties in reliably identifying such impairments for both clinical and research purposes.

The definitions of language-learning disabilities have included the use of discrepancy and/or exclusionary criteria. Discrepancy criteria involves the identification of a disparity between an impaired function, such as language or reading and other non-impaired aspects of cognitive functioning. Exclusionary criteria are used to differentiate those children with specific impairments (eg. reading or language problems) not attributable to known causes from children whose language or learning impairments may be attributed to known factors, such as mental retardation or hearing loss.

Given below are the various definitions put-forward by various authors:

There are children who fail to develop normal language functions in the absence of factors which often provide the general setting in which failure of language development is usually observed viz deafness, mental deficiency, motor disability, emotional disturbance or gross neurological disability. With the exception of language, these children appear to be developing normally. This particular language impairment is called developmental aphasia (Benton, 1964).

According to Ingram (1976), it is a condition in which, inspite of normal intelligence and unexceptional home background, the child is slow to develop speech and such speech as he has acquired is marked by defective articulation of certain groups of speech sounds, in particular consonant sounds. Speech output is commonly limited both in amount and syntactical structure and sense of rhythm is typically poor.

Developmental aphasia is a term applied to children who have never followed the normal developmental a course for speech and language but, rather, at each stage of development have missed the normal language milestones (Devel, 1983).

Eisenson (1986) recommends the term developmental (congenital) aphasia for the child who, despite the conditions about to be listed, is severely delayed in both the comprehension and production of oral language.

1. Based on observation and if possible nonverbal assessment, the child appears to have adequate intelligence for the acquisition of spoken language.
2. The child has no abnormalities in the structure of the oral mechanism.
3. The child shows no evidence of early emotional or relating problems.
4. The child has no hearing problems except for spoken language. In this regard, the real problem involves listening rather than hearing.
5. The child's parents or other caregivers, are available, willing and presumably capable of providing normal opportunities and stimulation for learning spoken language.

Specific language impairment is defined as impaired language development with several factors not considered at present, including the following: not the result of sensory impairment, not the result of emotional and/or behavioral

problems, not associated with global cognitive impairments (Bishop and Rosenbloom, 1987).

Etiology:

The causes of specific language impairment are likely to be multifactorial. Two factors that in isolation have no effect on the verbal development may in combination lead to disruption of language acquisition.

1. Genetic influences: The past 5 years have seen an upsurge of interest in the possibility that genetic factors may play a part in the causation of a range of developmental disorders (Rutter et al. 1990a).

Bishop and Edmundson (1986), Robinson (1987) and Tallal et al. (1989) demonstrated a significantly increased frequency of affected primary and secondary relatives in language-impaired as compared to control children.

Neils and Aram (1986) reported the occurrence of a spectrum of language disorders in the immediate family of 74 children aged 4 and 5 years who were diagnosed as language-impaired. They concluded that a strong family pattern exists in association with developmental language disorder.

Tallal et al. (1989 a, b), using a criterion of self-report of language disorder, reading difficulties, or academic failure to indicate signs of impairment, found that 77% of SLI probands had atleast one impaired relative.

Fathers reported some form of impairment more often than mothers (Neils and Aram, 1986; Tomblin, 1989). However, there is some evidence to suggest that impaired mothers have more impaired probands who have more impaired brothers than sisters (Neils and Aram, 1986). This may be confounded by a skewed sex ratio favouring boys in families of language-impaired children (Tallal et al. 1989 b).

Plante (1991) studied 4 families that included a SLI boy to test the hypothesis that developmental language disorders were biologically transmittable. Atypical perisylvian asymmetries and communication difficulty were documented in a majority of the parents, and in the siblings of SLI boys. These findings suggest that atypical perisylvian symmetries reflect a transmittable, biological factors that places some families at risk for language impairment.

Tallal et al. (1991) found that approximately 70% of language-impaired children met criteria for inclusion as family history positive with father reporting a history of language or learning problems one and a half to two times as frequently as mothers. These subjects were significantly lower in socio-economic status and had attention related behaviour problems. They also performed more poorly on standardized academic tests as well as on tests of auditory processing and attention.

In contrast to the previous studies, Whitehurst et al. (1991) found no strong familial component of expressive language delay. Further, family history was not predictive of later language development in expressive language delayed children.

2. Early brain damage: The majority of children with SLI have no objective evidence of brain damage (Robinson, 1987). But according to Benton (1964), the primary cause is a lesion or maldevelopment of the brain, although he did not localize the neurological abnormality to the left hemisphere. Eisenson (1968) stated that "A majority of the children we regard as developmentally aphasic present neurological findings, EEG and otherwise that implicate the left cerebral hemisphere".

Jernigan et al (1987) and Plante et al. (1989) reported a higher prevalence of atypical cerebral configuration for their language impaired subjects.

A recent case report by Cohen et al. (1988) documented an atypical symmetry of the plana temporale, a region associated with language functioning, along with a single dysplastic abnormality in the left insular cortex.

Plante et al.(1991) found that atypical perisylvian asymmetries were linked to language-disorder. Measurements (MRI) of other brain areas revealed that extraperisylvian areas were occasionally deviant in individual SLI subjects, but no one region was consistently deviant across the SLI group. Thus a prenatal alteration of brain development underlies SLI.

Jernigan et al. (1992) found no evidence of structural brain damage, but found some differences from control children interms of the relative size of different brain areas.

Aram and Eisele (1994) suggest that the models that involve bilateral or more diffuse areas of the brain, particularly the developmental relationship between more widespread brain systems, would appear to be more adequate.

3.Recurrent Otitis Media: A history of severe recurrent otitis media has been linked recently to language - learning disability. Data from Tonini (1983) do not indicate that such a history accounts for children with SLI. If however, a child has a pre-existing language problem, otitis media with SLI may well complicate the child's progress by interacting with perinatal risk factors (Bishop and Edmundson, 1986).

Silva et al. (1986) concluded that children who experience bilateral otitis media with effusion tend to remain disadvantaged developmentally through the mid-childhood years as indicated by language problems, speech articulation problems, reading problems and behaviour problems.

In contrast, Roberts et al. (1991) found no reliable relationship between early otitis media with effusion experience and receptive and expressive language scores and

measures of semantic syntactic competence between the ages 4.5 and 6 years.

Grievink et al. (1994) indicated that a history of otitis media with effusion even upto 9 instances did not have negative influences for language performance at age 7. Intermittent as opposed to more continuous otitis media with effusion was found to affect language ability negatively.

4. Auditory perceptual deficit: Eisenson and Ingram (1972) proposed that the language disordered child's inability to process and produce language had its etiology in auditory perceptual dysfunction. This theory has stimulated considerable research into the abilities of children with language disorders, particularly, abilities in the areas of temporal ordering, auditory discrimination and auditory memory.

In learning language, the order of phonemes is crucial in distinguishing words. Thus a deficit causing communication problem is malfunction of temporal ordering. Monsees (1968) concurred that language-disordered children were impaired in their ability to report the temporal order of auditory stimuli presented to them. According to Tallal and Piercy (1978), these difficulties represent a failure to

discriminate the sound quality of stimuli when the stimuli are presented in rapid succession. This discrimination problem occurred when the interval between the tones was short. The subjects also displayed difficulty in discriminating speech sounds that incorporated rapidly changing acoustic spectra. However, these children were able to understand single words presented in isolation. The ability to attend selectively to a particular stimulus in the presence of other auditory signals (figure-ground discrimination) has its importance in language-learning. Keir (1977) found that normal children could understand words when the background noise was as loud as the word, themselves, but the language-learning disabled children performed well only when the background noise was 10-15 dB below, the level of the words. Keir also said that the language learning disabled children performed well on the standard discrimination tests only if the testing conditions were quiet. There is a strong possibility that the language disordered children had deficit in auditory memory. Menyuk (1964 a) found that language disordered children (between 2 and 7 years of age) made a considerable number of omissions in recalling sentences and were not able to repeat sentences between 3 and 5 words in length.

Stark et al. (1967) concluded that the dysphasics have impaired auditory memory for sequences and tended to forget the first item in a sequence. Eisenson (1968) postulated that they also have defective storage systems for speech signals.

Keir (1977) found that a high percentage (63%) of his subjects had significant short-term memory problems. The striking feature of his test results was the very sharp cut-off point between success and failure in these children. For eg. they would repeat 3 digits quickly and confidently, but when an extra digit was added they would be unable to remember any of the digits.

According to Eisenson, the primary impairment in developmentally dysphasic children is a deficiency in central auditory processing. The impairment is, in effect, a central auditory disorder, which produces 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, 1979).

5. A symbolic defect: A cognitive deficit could be considered as a cause for SLI. Weiner (1969), using

Wechsler Intelligence scale for children found that both language-deficient and their controls had performance IQs of 90 (+/- 5 points). He also found that the experimental group had significant deficiencies on all tasks related to the auditory modality and that they functioned in a less integrated manner than the controls.

Morehead and Ingram (1973) and Johnston (1978) observed that the language-impaired children may have deficits in representational abilities including symbolic play and mental imagery as well as language. Bartak and Rutter (1975) felt that the dysphasic child lacked the imagination of the normal child.

Inhelder (1976), using nonverbal tests of operativity and verbal concepts, found that the dysphasic child was capable of solving problems despite his inadequate expressive vocabulary. She also found that the development of the figurative aspects of thought was impaired in the dysphasic children.

Stark and Tallal (1981) administered the Wechsler Intelligence Scale for children and found that of the 132

language-impaired children, 50 had performance IQs below 85 and a few had performance IQs of 50 or less. It was suggested that these low scores were an artifact of the verbal directions inherent even on the performance items of the test. Consequently, 10 of these children were given the nonverbal Hiskey-Nebraska Test of Learning Ability. They were still found to have IQ, in the retarded range.

6. An attentional deficit: The children with language-disorders display an attentional defect, which may be exhibited as impulsive behaviors and are called as hyperactive children. The classic description includes distractibility, lack of proper inhibition, overly intense responses and perseverative or compulsive behaviors. Such children appear to be 'always on the go' and display a low tolerance for frustration, to which they respond with emotional lability and a tendency toward temper tantrums.

These problems have long been associated with children with language learning problems (Strauss and Lehtinen, 1947; Strauss and Kephart, 1955). Efforts to establish a cause have indicated that these children may lack normal cerebral inhibition (Ong, 1968) or may have problems in selective attention and focussed arousal (Sheer, 1976). Some researchers suggest that these patterns of behavior may be

inherited or that they may represent a brain damage (Eisenson, 1972), post natal disease, food allergy (Crook, 1975) or a selective developmental lag in maturation of relevant areas of the brain (Kinsbourne and Caplan, 1979; Safer and Allen, 1976).

7. Social deprivation: There is some evidence that parents of children with SLI show less accommodation to their needs. For eg. Kriegsmann et al. (1975) found that the mothers of language disordered children were more restrictive and punitive and less responsive than those of normal children.

Horsborough et al. (1985) found that the mothers initiated more (used more interrogatives especially wh and quiz questions) and responded or commented less to the children (with respect to the description of objects). Moreover they used less numbers of utterances/turn, more non-informative or no responses, and less expansions and initiations. The authors interpret that the mothers of atypical language learners are influenced by the characteristics of their children and specifically that the formal linguistic characteristics of the children in terms of expressive language stage and language comprehension levels appeared to be more important for maternal

adjustments than the functional conversational abilities of the children.

Yoder (1989) found that the mothers of specific language impaired children who used proportionately more information seeking questions had children who showed greater mastery of auxiliary use twelve months later.

Thus these are the various etiological factors that could result in specific language impairment.

Classification:

Whereas some consensus exists with respect to the types of aphasia in adult patients, the classification of developmental language disorders is still in the process of being elaborated and validated. A number of classifications have been proposed.

Karlin (1962) divided aphasias in children into acquired and congenital. He further divided the congenital aphasias into:

- a) Speech aphasias :(i) verbal-auditory agnosia (word deafness).
 :(ii) motor aphasia (dysphasia).
- b) Visual aphasias :(i) alexia (word blindness)
 :(ii) agraphia

Aram and Nation (1975) divided the developmental language disorders into the following groups:

- a) Repetition strength
- b) Non-specific information-repetition deficit
- c) Generalized low performance
- d) Phonological comprehension-formulation-repetition deficit.
- e) Comprehension deficit
- f) Formulation-repetition deficit.

Wolfus et al. (1980) divided developmental dysphasia into two categories.

1. Expressive: Characterized by deficits in the production of syntax and phonology, but not in the comprehension of syntax or in semantic ability.
2. Expression-receptive characterized by greater impairment on measures of phonological discrimination, digit span, semantic ability and linguistic tasks in addition to showing global syntactic deficit.

Wilson and Risucci (1986) classified them in the following manner:

Developmental language disorders	Sub-type description of neuropsychological profiles
1. Receptive-Expressive	a) Auditory semantic comprehension disorders. b) Auditory and visual semantic comprehension disorder.
2. Expressive-receptive	a) Auditory semantic comprehension and auditory and visual short-term memory disorder. b) Expressive and/or receptive disorder. c) Global language and memory disorder - deficits in both auditory and visual cognitive and memory factors.
3. Expressive	a) Auditory memory and retrieval disorder - deficits in various aspects of auditory memory and semantic retrieval b) Expressive disorder } primary deficit c) Expressive disorder } involve organization & retrieval d) No deficits.

DSM III-R (American Psychiatric Association, 1987) classified developmental language disorder in the following way:

1. Developmental articulation disorder
2. Developmental expressive language disorder
3. Developmental receptive language disorder

Rapin and Allen (1987) classified in the following manner:

<u>Disorder sub-type</u>	<u>Comment</u>
a. Verbal auditory	Also called word deafness. There is no auditory verbal comprehension. The problem is thought to have a poor prognosis and children need to be taught to understand language through the visual channel.
b. Semantic- pragmatic deficit	: Fluent and well formed and articulated speech which initially is echolalic and delayed echolalic, progressing into well meant monologues. Auditory verbal comprehension is literal and the child often responds to keywords in the sentence (tangential responses).

Other features of expressive language include verbal stereotypes, perseveration and circumlocutions; said to have features of transcortical sensory aphasia. They are pragmatically impaired in their ability to take turns and to maintain a topic in discourse.

d) Phonological : Speech is dysfluent in short syntactic deficit utterances, usually with morphological errors. Comprehension may be impaired but less so than expression and phonological contrasts are reduced, said to be reminiscent of Broca's aphasia.

e) Phonological programming deficit : Utterances are longer but there is a moderate severe problem of speech intelligibility. Speech sound contrasts are severely reduced.

f) Verbal dyspraxia : Speech is dysfluent and severely unintelligible motor planning deficit is present and other general motor deficits also present.

Sub-groups of SLI children have been identified which may be differentiated by different linguistic characteristics.

1. Semantic-pragmatic SLI (Bishop and Adams, 1989).
2. Phonological SLI ('speech' and 'speech plus', Haynes, 1992).
3. Grammatical SLI ('classic SLI' Haynes, 1992)
4. Familial aggregation (Gopnik and Crago, 1991) - genetic basis present.

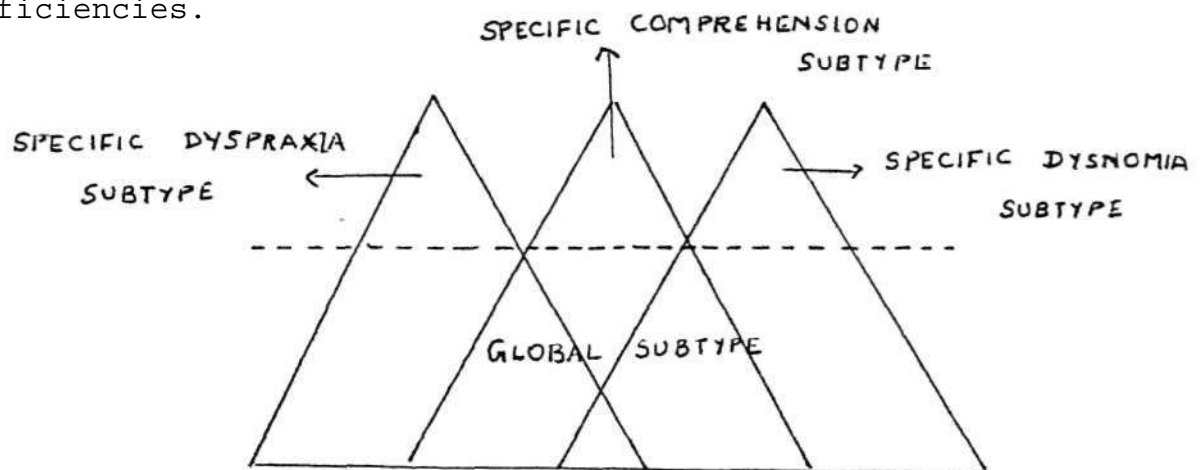
Korkman and Hakkinen-Rihu (1994) used NEPSY (Neuropsychological Investigation) for children to divide the developmental language disorders into the following sub-groups .

1. Global subtype- This category contained children with extensive receptive and naming deficiencies, with or without verbal dyspraxia. It was predicted that spelling problems would occur in this group.
2. Specific comprehension subtype - Spelling problems would occur. Less impairment in the auditory perceptual domain

but still some impairment in the comprehension of complex verbal instructions and/or concepts.

3. Specific verbal dyspraxia subtype - Characterized by deficits in the execution of oral motor sequences and the repetition of long and unfamiliar words, without language level deficiencies. No spelling problems present. No concomittant receptive deficiencies.
4. Specific dysnomia subtype - consists of children with specific problems in name retrieval.

This classification does not include an 'expressive subtype'. The subgroups should be looked upon as dimensions of disordered language development, rather than as discrete syndromes. Three of the subtypes (1, 2 and 3) represent impairments that are more or less restricted to one domain, whereas one subtype, 'the Global subtype' represents a combination of severe receptive and naming deficiencies.



This figure illustrates the view that in milder cases one dimension may be affected alone, but with increasing degree of impairment the likelihood increases that more than one dimension is affected.

CHARACTERISTICS

1 - Perceptual deficits:

A number of studies have presented evidence that auditory and speech perception abilities of some children with language impairments are significantly poorer than those of their age matched peers (eg. Elliot and Hammer, 1988). These deficits may be in the form of :

a) Difficulty in temporal sequencing: Sequencing is the ability to hold a series of events in mind and to respond to an on going event in the light of immediately past events. But this ability is affected in the specifically language impaired children and consequently they will not be able to understand speech (Stark, 1967).

b) Difficulty in processing- It has been reported in the literature that development dysphasic children take longer time to process non-linguistic information (Lowe and

Campbell, 1965) and that they had impaired capacity to process rapidly changing acoustic information (Tallal et al. 1981) which leads to the faulty perception of sounds.

c) Difficulty in discrimination- The developmental dysphasic children exhibit impairment for speech-sound discrimination for instance /ba/ vs /da/ (Tallal and Piercy, 1974) and /i/ and /u/ when embedded in multisyllabic (Leonard et al. 1992) but the perception of non-speech environmental sounds may not be impaired. According to Cohen et al. (1991), the language impaired children had more difficulty than controls in discriminating place of articulation contrasts only when they were presented to left ear as well as a difficulty in discriminating voice contrasts selective to the right ear which suggests a bihemispheric dysfunction as a basis of SLI.

d) Memory deficits- It has been reported that SLI children have some sort of memory problem that may underlie their linguistic impairment (Griffiths, 1972; Graham, 1980, Kirchner and Klatzky, 1985). There deficits have been in the form of inability to store and recall word strings (Ceci et al. 1981; Kail et al. 1984), inability to recall the first word in a series (Stark, et al. 1967), inability to recall the tone sequences (Lincoln, et al. 1992), and the inability

to verbally repeat single non-words of one-four syllables (Gather Cole and Baddeley, 1980).

However, Vanderlely and Howard (1993) reported that there was no significant differences between the performance of SLI children and the controls on short-term memory tasks.

Thus, the presence of short-term memory problems is still a controversy.

2. Cognition:

In general, children with language disorders typically perform within normal limits on formal tests such as the Leiter International Performance Scale (Leiter, 1980), Test of non-verbal Intelligence (Brown, et al. 1982), Weschler Intelligence Scale for Children - Revised (Weschler, 1963) and Columbia Mental Maturity Scale (Burgemcister et al. 1972). But there have been contradictory findings too, Stark and Tallal (1981) administered the Wechsler Intelligence Scale for children or the Wechsler preschool and primary scale of Intelligence (WPPSI) to all language deficient children in their experiment. Of the 132 children, 50 had performance IQs below 85, and a few had

performance IQs of 50 or less. It was suggested that these low scores were an artifact of the verbal directions inherent even on the performance items of the WISC and WPPSI. Consequently, ten of these children were given the nonverbal Hiskey-Nebraska Test of Learning Ability. They were still found to have IQs in the retarded range. Moreover they have difficulty across a variety of non-standardized cognitive measures like the following:

a) Anticipatory imagery: It has been suggested that language impaired children may have deficits in representational abilities, including imagery and language (Morehead and Ingram, 1972; Bartak and Rutter, 1975; Johnston, 1978).

b) Mental rotation: According to Johnston and Weismer (1983), the SLI children are very slow at responding to the mental rotation tasks than the controls but there was no difference in the accuracy of judgements.

c) Haptic recognition: Many studies (Johnston and Ramstead, 1983; Kamhi et al, 1981; Montgomery, 1993) have indicated that children with SLI score poorly on the haptic recognition tasks than their normally developing counterparts. This may be because of deficient cross-modal

processing and limited capacity processing (Montgomery, 1993).

d) Symbolic functioning: The SLI children suffer from a pervasive symbolic representational deficits for instance the ability to mentally generate and manipulate visual images (Terrell et al. 1984; Roth and Clark, 1987). This deficit might underlie both their nonverbal cognitive and linguistic deficits.

e) Hypothesis testing ability: Nelson et al. (1987) and Ellis Weismer (1981) determined that children with language disorders performed more poorly than did mental age matched peers on a hypothesis testing task. They attributed their findings to the language disordered group's difficulty in encoding information for storage in short term memory.

f) Reasoning: It has been reported in the literature that the language impaired children had difficulty with the analogical reasoning tasks (Nippold et al. 1988; Masterson, 1993). According to Ellis Weismer (1985) and Crais and Chapman (1987) the SLI children have general difficulties in constructing integrated representations of information.

g) Fast mapping skills: Fast mapping is a hypothesized process enabling children to create lexical representations for new words after as little as a single exposure. According to Dollaghan (1987), the SLI children could comprehend new words and recall non-linguistic but could not produce the new word.

h) Phonological processing ability: According to Kamhi et al. (1988), the SLI children performed poorly on tasks like 4 word repetition (monosyllabic, monosyllabic presented in noise, 3-item and multisyllabic), rapid naming, syllable segmentations, paper folding and form completion,

i) Counting abilities: According to Fazio (1994), the SLI children had difficulty with rote counting, displayed a limited repertoire of number terms and miscounted sets of objects. But they did not have problems in gestural counting tasks.

These are the various cognitive deficits seen in SLI children.

3. Comprehension:

There is evidence to indicate that the comprehension abilities might be affected in SLI children which could be

in the form of delayed acquisition of sentence comprehension strategies (Vander Lely and Deward, 1986; Precious and Conti Ramsden, 1988), poor comprehension of humor elements and the inability to grasp the nature of multimeaning words (Spector, 1990) and poor comprehension of items which require inferential skills (Bishop and Adams, 1992).

4. Learning abilities:

The general conclusion from many studies conducted to investigate learning patterns in SLI children is that they exhibit a unique learning pattern. The children with SLI learn rules less easily than their peers. When these children are merely asked to observe instances of rule usage, their learning appears to be impaired. However, when they are required to imitate the target rule examples, their learning appears to be more comparable to that of their peers (Connel, 1987; Connel and Stone, 1992, 1993, 1994). Moreover Weismer and Hesketh (1993) indicated that acquisition of novel words was affected by alterations in speaking rate and use of gestures.

5. Linguistic Correlates;

a) Morphology - The language of SLI children does not match that of normally developing children at any point in development. The area of verb morphology stands out as a particular area of weakness in these children (Albertini, 1980; Khan and James, 1983). These children have difficulty in using copula, auxiliary verb inflections and grammatical morphemes (Johnston and Scherry, 1976), have a metalinguistic deficit (Kamhi et al. 1985), are weak in case marking (Lee, 1966; Menyuk, 1964) have difficulty with the function words, articles and pronouns (Leonard, 1982) and difficulty with the acquisition of plurals (Johnston and Scherry, 1976; Gopnik and Crago, 1991; Oetting and Rice, 1993).

b) Syntax: The general conclusion from many studies is that the sentence structure produced by the dysphasic group is representative of that produced by younger children. Moreover they produce well-formed sentences less frequently than normal children (Klee, 1989; Grimm and Weinert, 1990). According to Terrel and Schwartz (1988), SLI children produce lesser number of object transformations in their play.

c) Narration: Liles (1987) compared the language-disordered children with normal language children in their ability to cohere episode units in verbally produced narratives and found that the language-impaired children had more incomplete and fewer number of episodes and omitted more story grammar elements.

d) Conversation: An interesting amount of research indicates that the SLI child's conversational skills are not the same as those of children developing language normally. The conversational partners (Brinton and Fujiki, 1982), linking successive messages in the multiutterance turns of narrative discourse (Johnston, 1982; Liles, 1985) and modifying the form of their messages in response to a partner request for clarification (Gallagher and Dorton, 1978).

6. Articulation:

Many children pass through a period when the articulation is defective, but there is rapid and spontaneous improvement towards normal articulation. This frequently occurs in the early stages of speech development. The child with developmental dysphasia may also pass through such a phase in the early stages of the use of expressive

speech, but at a later age than the normal child. A small percentage of children with developmental aphasia who have made considerable progress in language comprehension may continue to have difficulties in language production. Some may, in fact, be suffering from oral (articulatory) dyspraxia. They do not have an execution or motor impairment severe enough to qualify as apractic or dysarthric. An articulation disorder, however, is not uncommon (Affolter et al. 1994; Stark and Tallal, 1981).

7. Cerebral dominance:

Over the past 50 years researchers have given more importance to handedness because it has been found that left handed children have speech disorders or are backward in reading. Weak, mixed or inconsistent lateral preferences are the most frequent finding. Ingram and Reid (1954) directed attention to this feature in 71% of a large group of patients diagnosed as a case of developmental aphasia.

This finding led Orton (1937) to postulate that developmental language disability is caused by a lack of clearcut cerebral dominance ie. a failure to lateralize language exclusively to one or other hemisphere.

Neils and Aram (1986) studied the handedness and sex of 4-5 years old children with developmental language disorders. Differences between the handedness of a generalized language-disordered group and a normal control group were not found, possibly due to the multiple etiologies of developmental language disorders. Children with severe language disorders, however, were non-right handed more often than children with mild language disorders. Further more, certain types of linguistic deficits were associated with non-right-handedness, whereas age and cognitive abilities were not. Males were more often language disordered than females, however, sex ratios did not significantly differ among the subgroups.

8. Neurological findings:

Many developmentally aphasic children do not present clear cut 'hard' sign evidence of central nervous system pathology. Hard sign includes defects such as motor disabilities, sensory distinctions and perceptual motor delays or integrative impairments. Indicators in these categories are found in about one-third of the population who are behaviorally aphasic. Many more show evidence of atleast 'MBD' signs which include delayed laterality, late

reached a certain level of complexity or involved more than one modality.

12. Socio-emotional problems:

It is unrealistic to expect a child who is experiencing difficulty communicating with others to have social behavior clearly within normal limits. King et al. (1982) reported that families noted problems in social and interpersonal relationships for 4 of 18 children initially diagnosed as language disordered. One child was reported to experience difficulty in relationships with family and peers and to have received professional help. Another child was reported to have problems in sibling relationships and the remaining two had difficulty in peer relationships.

Roth and Clark (1987) studied the symbolic play and social participation behaviours of 6 language-impaired and a normal language learning children on 3 measures of play:

a) The symbolic Play Test (Lowe and Costello, 1976) (b) the Brown-Lunzer. Scale (Brown et al. 1975) and (c) the scale of social participation in Play (Tizard et al. 1976). The results indicated that the language impaired subjects demonstrated significant deficits in symbolic, adaptive and integrative play behaviors in comparison with the

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 nor the more frequent 'soft signs'.

As Geschwind (1979) observed "brain which shows no pathology in the usual sense of the term may yet deviate from the normal. These brains differ in roles of development, either throughout the brain or in specific areas only. Such deviations, if they involve the part 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.

9. Electroencephalographic findings:

Several investigators indicate that abnormal EEG findings occur in a higher incidence among congenitally aphasic children than in children in their age range who are not aphasic. Goldstein et al. (1958) report that about 40% of the 69 aphasic children showed abnormal EEG findings. The aphasic children (14.5%) had a higher incidence of focal abnormalities. Forest et al. (1967) found that 3% of the 73 children' studied had abnormal EEG findings.

10. Behavioural problems:

Froschels notes that many of the children who are mute have 'wild behaviour' ie. hyperactivity, impulsivity and distractibility. The behavioural aberrations are same as in brain damaged adults. Most common are the symptoms which have become known as the 'strauss' syndrome (ie. distractibility, hyper activity, impulsivity). Aphasic children manifest a high degree of behavior problems, possibly because of their frustration in being unable to comprehend or produce language or for any number of other reasons or may be because of hormonal, metabolic or electrochemical factors (Stark, 1980).

11. Motor skills:

There is some evidence that children with SLI, can be somewhat clumsy. Affolter et al. (1974) studied the fine and gross motor skills in their clinical population of language-disordered children. They observed hand-eye coordination on tasks such as climbing, inserting a key in a block, building simple block constructions and doing intricate close-fitting puzzles. These children appeared to lose hand-eye coordination whenever a problem or situation

linguistically equivalent normal subjects. The language-impaired group also evidenced significantly more non-play and significantly less solitary and parallel play than their normal peers.

13. Sensory deficits:

Ewing (1930) showed that 6 of 10 developmentally dysphasic children had raised thresholds for certain high frequencies. Other sensory deficits have been noticed in children with language disorders. For eg. abnormal auditory temporal summation (Rosenthal, 1971, and masking level differences (Rosenthal and Wohlert, 1973). But the results of these and other psychoacoustic studies which use puretone or other simple nonverbal acoustic stimuli; have not been related directly to speech processing.

Thus from the review of literature it is evident that developmental dysphasia or specific language impairment could be caused by many factors or combination of factors like genetic predisposition, early brain damage, recurrent otitis media, auditory perceptual deficit, symbolic deficit, attentional deficit and social deprivation.

It is also clear from the review of literature that the developmental dysphasic children or the specific language impaired children have characteristics like perceptual deficits which could be in the form of temporal sequencing, processing, discrimination or memory deficits, cognitive deficits, comprehension deficits, learning problems expressive difficulties, articulation problems, lack of clear cut cerebral dominance, neurological impairments like late walking, awkwardness and perceptual motor irregularities, electroencephalographic abnormalities, behavioral deviations like distractibility, hyperactivity, temper tantrums, impulsivity, perseverative or compulsive behaviours, always on the go, lack of proper inhibition and low tolerance for frustration, motor problems like poor eye hand coordination, clumsy; social problems like difficulty with sibling, peer and interpersonal relationship, exhibiting non-play behaviors; and sensory deficits like slightly raised thresholds for certain high frequencies, abnormal auditory temporal summation and masking level differences.

Traditionally, the diagnostic description of delayed speech and language is used for all the children who have delayed speech and language but without any clear signs of

organic pathology or sensory deficits like hearing loss or mental retardation. Given that the characteristics of developmental dysphasia or specific language impairment are not reported by parents until carefully elicited or observed by the clinicians, it is possible that several of them will be grouped within the looser diagnostic label of delayed speech and language.

Hence, in order to aid the clinician in differential diagnosis of delayed speech and language with no associated deficits from developmental dysphasia or specific language impairment, a diagnostic tool has to be developed. This is also necessary since it helps in proper planning of the treatment strategies.

Thus, this study aims at developing a checklist by assessing children with delayed speech and language and comparing the causes and characteristics present in these children with some of the causes and characteristics reported in the literature. The methodology is presented in the next chapter.

METHODOLOGY

From the review of literature, it is evident that there are a variety of factors which could lead to developmental dysphasia or specific language impairment and there are several features which characterize individual cases. There is very little literature on the process of identification of such children. Neither are there diagnostic tools which can aid the clinician in reliably identifying such children. Hence the main aim of the present study was to develop a checklist to help identify children with developmental dysphasia or specific language impairment and thus differentially diagnose these children from children with delayed speech and language due to purely environmental or social causes with no other associated problems. This differentiation is necessary because the management strategies are different for both the groups of children.

Criteria for selection: The criterion for subject inclusion in this study was a diagnosis of 'delayed speech and language with *no* other associated problems' by a speech-language pathologist in the Department of Speech Pathology. Additionally only children whose mother-tongue was Kannada were considered.

Subjects: Ten subjects (3 females and 7 males) in the age range of 2.3-6 years were selected for the present study. They had reported to All India Institute of Speech and Hearing with a complaint of inadequate speech and language with no other associated sensory, motor or intellectual problems.

The following causes and characteristics of developmental dysphasia short listed were considered for further investigation.

<u>Cause</u>	<u>Characteristics</u>
1.Genetic influence	1. Developmental milestones
2.Early brain damage	2. Handedness
3.Consanguinity	3. Intelligence
4.Social deprivation	4. Social behavior
5.Multilingualism	5. Behavioural problems
6.Recurrent otitis media	6. Hearing abilities
7.Attentional deficit	7. Language abilities
	8. Learning abilities.

These causes and characteristics were studied because these were the major differentiating factors between developmental dysphasia and delayed speech and language due to environmental or social causes and the information pertaining to this could be elicited from the parents

easily. Consanguinity and multi lingualism was also studied even though it was not reported in literature because these 2 factors are considered to cause a variety of disorders with reference to the Indian population.

Tools: Attributes like intelligence, hearing abilities, language abilities and neurologic skills were assessed with the help of certain tests.

The assessment of intelligence was accomplished with the help of Developmental Screening Test (DST) developed by Bharathraj (1977). The purpose of the test is to measure mental development of children from birth to 15 years of age. There are 88 items, distributed according to the age scales 3 months, 6 months, 9 months, 1 year, 1 1/2 years, 2-13 years at yearly intervals and finally at 15 years. The number of item varyfrom 3 at age 12 years to 13 at 3 months level. Appraisal canbe done in a semi-structured interview with the child and parent or a person well acquainted with the child. The test has good validity.

The assessment of hearing abilities were carried out with the help of Behavioural Observation Audiometry (BOA) and Brainstem Evoked Response Audiometry (BSERA) for some

children and with the help of pure tone audiometry (PTA) for some other children BOA is a screening procedure to estimate hearing abilities with the help of behavioral responses for those children who are unable to give conditioned responses. BSERA is an objective procedure in which the hearing sensitivity is estimated with the help of neuro-electrical potentials recorded from the scalp for those children who do not give conditioned responses. PTA is a method of obtaining threshold of hearing with the help of conditioned responses.

The language abilities of the children were assessed with the help of 3 dimensional Language Acquisition Test (3D LAT) developed by Geetha (1984). The purpose of the test is to evaluate the language acquisition in young children between the age of 9 months to 3 years of age. The test provides normative data for language acquisition in children based on an informant interview approach. The items in the test are grouped in to receptive, expressive and cognitive section. Nine age groups were made between 9 and 30 months, the test includes 21 items under each section with 3 item for each age group.

The use of this test was backed by the Language test Kannada for children with language age beyond 3 years.

This test was primarily designed to measure a subject's receptive and expressive language. It consists of two parts. Part-I Semantics which is a major branch of linguistics devoted to the study of meaning in languages and Part II - syntax which is a branch of linguistics which studies the word structure.

Test environment: Testing was carried out in a well lit room free of distractions and the required information was collected with the help of a detailed case history.

Procedure: A detailed case history was taken which provided information on the causative factor. It also revealed some of the typical characteristics and problems present in these children. The case history proforma is shown in the Appendix. Thus, the information regarding the receptive and expressive language level was elicited with the help of three dimensional language acquisition test and Kannada Language Test, the information regarding hearing abilities was elicited with the help of puretone audiometry, BSERA and behavioral observation audiometry and the information regarding IQ was elicited with the help of Developmental Screening Test. The data thus collected is represented in the following Chapter.

RESULTS AND DISCUSSION

The aim of the present study was to probe more thoroughly the possible causes and characteristics seen in children diagnosed as delayed speech and language with reference to the causes and characteristics associated with developmental dysphasia or specific language impairment as reported in the literature and if possible arrive at a checklist to identify the latter as a specific subgroup requiring specific therapeutic management.

Table-I: shows the percentage of causes and characteristics prevalent, in the children considered for the study. It also given the amount of language delay in each child separated for reception and expression along with mean and standrd deviation separately.

From this table it is clear that early brain damage, attentional deficit, behavioral problems, and a language delay have a high percentage of occurrence. For instance, behaviour problems occurred in 90% of the children, early brain damage and language delay occurred in 60% of the children. These causes and characteristics are present at a significant level in these children.

Sl.No.	1	2	3	4	5	6	7	8	9	10		
Case Name	S	D	RP	V	H	R	B	VK	S	BR		
Case No.	81512	78926	76345	92605	89352	96684	91953	97768	86793	98092		
Age/Sex	4.6M	4.10M	6.0M	3.4M	3.9M	2.6M	5.0M	2.3M	4.6M	4.1M		
Family History	-	-	+	+	-	-	-	-	-	-		20%
Early brain damage	+	-	+	+	-	-	+	+	+	-		60%
Consanguinity	-	-	-	+	-	-	+	-	-	-		20%
Social deprivation	-	+	-	+	-	-	-	-	-	-		20%
Multilingualism	-	-	-	-	-	-	-	-	-	-		0%
Recurrent Otitis Media	-	-	-	-	-	+	-	-	-	-		10%
Attentional deficit	+	+	+	+	+	-	+	+	+	-		80%
Developmental milestones delayed	+	-	+	+	+	-	+	-	-	-		50%
Handedness(Lt)	+	-	-	+	-	-	-	-	+	+		40%
Intellectual (Subnormal)	+	+	+	-	-	-	-	-	-	+		40%
	<i>mild</i>	<i>mild</i>	<i>mod</i>							<i>mild</i>		
Social problems	-	-	-	-	-	-	+	-	-	-		10%
Behavior problems	-	+	+	+	+	+	+	+	+	+		90%
Hearing problems	-	-	-	-	-	-	-	-	-	-		0%
Language delay	R=12m E=15m	R=26m E=33m	R=40m E=55m	R=26m E=30m	R=26m E=28m	R=0m E=17m	R=25m E=41m	R=0m E=17m	R=22m E=35m	R=30m E=32m	M=20.9 SD=12.9	M=30.3 SD=12.2
Learning abilities	+	+	+	+	-	-	-	-	-	+		50%

The next set of less frequent causes and characteristics are delayed developmental milestones which occurred in 50% of the children, learning problems which again occurred in 40% of the children and subnormal intellectual abilities which occurred in 40% of the children. These causes and characteristics are present to a lesser degree in these children.

The least frequently occurring factors are positive family history and social deprivation which occurred in 20% of the children, recurrent otitis media and social problems which occurred in 10% of the children. Multilingualism and hearing problems also come under this category since they were not present in any of the children. Thus they are the least significant factors.

Generally, we arrive at a diagnosis of delayed speech and language only if all the obvious causative factors of delayed speech and language like hearing loss or mental retardation are ruled out and there is evidence for lack of environmental stimulation or multilingualism. However in this group of children diagnosed as delayed speech and language there is a high incidence of behavior problems, attentional deficit, early brain damage and a language

delay. In contrast there is no evidence for lack of environmental stimulation and multilingualism. A majority of the children considered in the present study should probably ought to be diagnosed as developmental dysphasia or specific language impairment, rather than delayed speech and language.

In the present study, behavioral problems are the most prevalent characteristic as it occurred in 90% of the children. The problems were in the form of impulsivity, hyperactivity, distractibility, temper tantrums and stubborn behavior. Behavioral problems have been reported to be associated with developmental dysphasia. According to some researchers (Stark, 1980) developmentally dysphasic children manifest a high degree of behavioral problems including distractibility, hyperactivity and impulsivity.

Attentional deficit was exhibited by 80% of these children. There are several reports in the literature emphasizing the fact that an attentional deficit is present in developmental dysphasia and that this could lead to language problems. These children may lack normal cerebral inhibition (Ong, 1968) or may have problem in selective attention and focussed arousal (Sheer, 1974).

History of early brain damage was positive in 60% of the children. The most common perinatal factors reported were caesarian delivery, prolonged and induced labour birth asphyxia and delayed birth cry. The common postnatal factors reported were high fever with convulsion and viral infections like chicken pox.

In the literature, there are quite a few reports on brain abnormality in children with developmental dysphasia. But their reports have been focussed on developmental dysphasic children with prenatal abnormalities like atypical cerebral configuration (Jernigan et al. 1987 and Plante et al. 1989), atypical asymmetry of the plana temporale along with single dysplastic abnormality in the left insular cortex (Cohen et al. 1988), atypical perisylvian asymmetries (Plante et al. 1991) and differences in the relative size of different brain areas (Jernigan et al. 1992).

All the children in the study exhibited a language delay. Two children had a delay in expressive language only. All the other children had both reception and expression affected. Further, there was a wide gap between their receptive and expressive skills i.e. their

expressive abilities were poorer than their receptive abilities. The average delay in reception was 20.9 while the average delay in expression was 30.3. The gap between expression and reception ranged from 8-17 months.

Research has indicated that the language of specifically language impaired children does not match with that of the normally developing children at any point in development. Their mean length of utterance is shorter or lesser than that of the normally developing children.

Delayed developmental milestones were seen in 50% of the children who had a 6 month lag in their developmental motor milestones in terms of head control, sitting and walking. This is in concurrence with the evidence in the literature citing that the developmentally dysphasia children have delayed motor milestones especially late walking.

Learning problems were also present in 50% of these children. There is evidence in the literature to state that developmentally dysphasic children have learning problems and have a unique learning pattern (Connel and Stone, 1992, 1993; Weismer and Hesketh, 1993).

Left handedness was present in 40% of these children. There is evidence in the literature stating that developmental dysphasic children are either left handed or have weak, mixed or inconsistent laterality (Zangwill, 1962 and Neils and Aram, 1986). According to Ingram and Reid, 71% of the developmentally dysphasic children were left handed.

When measured on IQ tests subnormal intelligence was found in 40% of these children. Among these, 30% of the children were mildly retarded and 10% of the children were moderately retarded. While the remaining 60% of the children had normal intelligence. According to Stark and Tallal (1981), out of the 132 children investigated on Weschler Intelligence Scale for the children or the Weschler Preschool and Primary scale of Intelligence (WPPSI), 50 children had performance IQs below 85 and a few had performance IQs of 50 or less.

Family history was reported to be positive only in 20% of these children. There are many reports in the literature indicating that a positive family history could be a causative factor of developmental dysphasia or specific language impairment (Bishop and Edmundson, 1986; Robinson,

1987; Tallal et al. 1981; Plante, 1991). According to Tallal et al. 1991, approximately 70% of the language impaired children met the criteria for inclusion. as family history positive. In contrast Whitehurst et al. (1991) found no strong familial component for expressive language delay. the findings in the present study are in agreement with the study by Whitehurst et al. (1991) but is in contradiction with the study by Tallal et al. 1991).

History of social deprivation was reported in 20% of these children. The nature of the social deprivation was the mother's absence during the child's developmental period and their lack of responsiveness to the children. It has been reported in literature then mothers of developmentally dysphasic children are less responsive and do not accommodate to their child's needs (Friegsmanr., et al. 1975).

Consanguinity also emerged as one of the least significant causative factors since only 20% if the children had parents who reported of consanguinous marriages. While there are no reports in the literature indicating consanguinity as a causative factor of developmental dysphasia, in India it has often been considered as a causative factor in several congenital abnormalities.

Recurrent otitis media was present in only one child and hence the factor did not emerge as a significant factor. There is evidence in literature indicating that children who experience bilateral otitis media with effusion tend to have language problems, speech and articulation problems, reading and behavioral problems. In contrast, Robert et al. 1991 and Grievink et al. (1994) found that there is no reliable relationship between early otitis media with effusion and the language performance.

Social problems occurred only in one of the ten children studied and hence it is one of the lesser significant factors. This was in the form of inability to establish relationship with peers and family members. According to King et al. (1992) and Roth and Clark (1987), it is unrealistic to expect a child who is experiencing difficulty in communication with others to have social behaviour within normal limits. They have difficulty in establishing relationship with family members and peers.

None of these children reported of hearing loss. According to Eisenson (1986) and Bishop and Rosenblom 1987, the developmentally dysphasic children have no hearing abnormalities. But Ewing (1930) showed that 6 out of 10

developmentally dysphasic children had raised thresholds for certain high frequencies.

In India, multilingualism is often considered to be related to delayed acquisition of speech and language in children. However, it has not been reported in any of these children. Hence, multilingualism could not a causative factor in these children.

On the basis of the results obtained the children considered in this study could be categorized into three groups.

1. Children with a positive history of early brain damage and the presence of 3 or more of the following 7 characteristics - attentional deficit, behavioral problems, language problems, delayed developmental milestones, left handedness, subnormal intelligence and learning problems. The children who fall under this category could be diagnosed as developmental dysphasia or specific language impairment on the basis of the language delay coupled with a high incidence of causative factors and associated characteristic of developmental dysphasia being present in them. The following (Table-II) provides details on this group of children.

Sl.No.	1	2	3	4	5	6
Case Name	S	RP	V	B	VK	S
Case No.	81512	76345	92605	91953	97768	86793
Age/Sex	4.6M	6 M	3.4M	5 F	2.3 M	4.6 F
Early brain damage	+	+	+	+	+	+
Attentional deficit	+	+	+	+	+	+
Behavior problems	-	+	+	+	+	+
Language problems	+	+	+	+	+	+
Delayed developmental milestones	+	+	+	+	-	-
Left handedness	+	-	+	-	-	-
Subnormal Intelligence	+	+	-	-	-	-
Learning problems	+	+	+	-	-	+

Table-II: Depicts details on group-1.

2. The children with a negative history of brain damage but with the presence of 3 or more of the above 7 characteristics. This group of children would come in the suspicious category ie. they may or may not be developmentally dysphasic children and hence require

extensive neurological and speech and language evaluation. The following table (Table-III) depicts this category of children.

Sl.No.	1	2	3
Case Name	D	H	B
Case No.	78926	89352	98092
Age/Sex	4.10M	3.9M	4.11M
Early brain damage	-	-	-
Attentional deficit	+	+	-
Behavior problems	+	+	+
Language problems	+	+	+
Delayed developmental milestones	-	+	-
Left handedness	-	-	+
Subnormal Intelligence	+	-	+
Learning problems	+	-	+

Table-III Depicts details on group-2.

3) The children in this group have a negative history of brain damage and less than 3 of the 7 characteristics. The children under this category could be diagnosed as delayed-speech and language. This table (Table-IV) depicts this.

Sl.No.	1
Case Name	R
Case No.	96684
Age/Sex	2.6 F
Early brain damage	-
Attentional deficit	-
Behavior problems	+
Language problems	+ Expressive language delay
Delayed developmental milestones	-
Left handedness	-
Subnormal IQ	-
Learning Problems.	-

Table-IV : Depicts details on group 3.

Thus, it is clear from the 3 tables that (2, 3, and 4; that 6 children have a high positive (50% score) in the characteristics ie. attentional deficit, behavior problem, language problems, delayed developmental milestones, left handedness, subnormal intelligence, and learning problems and a positive history of brain damage while 3 children had a negative history of brain damage with 50% score on the 7 characteristics listed. Thus the first group of children

ought to be diagnosed as developmental dysphasia or specific language impairment rather than just delayed speech and language. While the last category could be diagnosed as delayed speech and language.

There is considerable individual variation within these children, for instance while positive scores on most of the causes and characteristics investigated, like early brain damage, consanguinity, attentional deficit, delayed developmental milestones, left handedness, learning and behavioral problem and a language delay, seems to have a negative on all the above causes and characteristics with the exception of the presence of language and behavioral problem.

If the checklist described above is applied 'V' will be diagnosed as developmental dysphasic or a specifically language impaired child while 'R' will remain in the broader category of delayed speech and language.

The term delayed speech and language has been used as a waste paper basket term because many of the characteristics which don't fall under any of the other labels seem to be put under the loose diagnostic category of delayed speech and language. Taking this particular study into

consideration all the children were diagnosed as delayed speech and language but as the results revealed 6 children along with delayed speech and language had several characteristics of developmental dysphasics, while 3 children fall in to the suspicious category. Only one child could be definitely labelled as delayed speech and language with no signs of organic involvement. Thus it is important that before placing any child in a particular diagnostic category extensive and thorough investigation of all the characteristics and causes has to be accomplished. Even though the regular case histories contain this information, the clinician has to go back and look through all of the information obtained so that he could identify the above causes and characteristics and thus diagnose the children appropriately.

The groups I and II in this study have to therefore be followed by more extensive neurological and speech and language investigation. In order to get a better diagnostic picture. This also helps in planning appropriate intervention strategies for instance if delayed speech and language is caused by either multilingualism or social deprivation, the therapeutic intervention strategies should aim at counselling the parents if multilingualism is the

causative factor and provide adequate speech stimulation if social deprivation is the causative factor. However, in the case of developmental dysphasia, the management procedures would aim at specific approaches to reduce hyperactivity and to increase the attention span and hence the need for a more specific diagnosis.

SUMMARY AND CONCLUSION

There has been a lot of research carried out in the area of developmental dysphasia or specific language impairment in terms of the causes and characteristics present in such children. In spite of the existence of vast amount of literature, confusion still exists in identifying these children due to the lack of diagnostic tools to identify such children.

The main aim of the present study was to develop an informal checklist to reliably identify children with developmental dysphasia or specific language impairment and differentiate them from children with delayed speech and language with *no* other associated problems. This is essential as this would not only aid the speech language pathologist in diagnosis but more so in the selection of the appropriate management strategies.

Ten children (7 males and 3 females) in the age range of 2.3 - 6 years were considered for the present study. The criterion for their inclusion in the study was a diagnosis of delayed speech and language with no other associated problems. The mother tongue of the children and selected was Kannada.

A few major causes and characteristics of developmental dysphasia were studied in these children. Information on this was elicited with the help of a detailed case history. Hearing abilities, intelligence and language abilities were assessed with individual tests.

The results indicated a significant proportion of causes and characteristics of developmental dysphasia such as behavioral problems, attentional deficit, early brain damage and a language delay was found to be present in this group of children diagnosed as delayed speech and language.

Delayed developmental milestones, learning problems, left handedness and subnormal intellectual abilities were also present to a lesser degree.

Causes and characteristics such as family history, social deprivation, consanguinity, recurrent otitis media, hearing problems, social problems and multilingualism were either absent or positive in a very small proportion of this clinical population.

Based on these findings a short checklist of 8 features was drawn to help identify the subgroup of developmental

dysphasia and those falling under the suspicious category from the most general group of delayed speech and language. The importance of following these children up with more extensive neurological and speech language investigations was stressed. Interestingly though delayed speech and language is most often considered to be due to lack of environmental stimulation or due to multilingualism, results of this study reveal that social deprivation and multilingualism were the least significant factors in these children.

Apart from differential diagnosis, this check list would also aid the clinician in choosing the appropriate management strategies.

It is important that all the causes and characteristics described in this study be incorporated in to the regular case history proforma so that the children with developmental dysphasia will not be missed out or will be and followed up with more extensive investigations. This check list is an earnest attempt to aid the speech language pathologist in the reliable identification and evaluation of children with developmental dysphasia.

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APPENDIX

Case Name	Case	Age/Sex
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Mother Tongue:

CAUSES

1. Any evidence of brain damage?

Prenatal history

Perinatal history

Postnatal history

2. Does any one in the entire family has a similar problem?

3. Did the child receive adequate speech stimulation during his developmental period?

4. Did the child have repeated ear discharge?

5. Does the child have any attentional problems?

6. Did the parents have a consanguinous marriage?

CHARACTERISTICS

1. Handedness

	Right	Left	Mixed	Laterality
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Grasping

Eating

Playing

2. Social maturity

Recognizes parents

Refuses to go to strangers

Prefers to play by himself

Socializes easily with peers and elders

3. Behaviour History

Distractible

Hyperactive

Impulsive

Always on the go

Tempertantrums

Low tolerance for frustration

Perseverative or compulsive behaviors

4. Learning skills

Is the child's learning ability a quick process or does he require constant coaching?

5. Developmental milestones

Head control

Sitting

Walking

Bowel and bladder control.