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Dr G T Spencer also presented a paper.

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Diagnosis, Treatment and Management of Speech Defects in Childhood

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Speech Disorders in Childhood

Prevalence of Speech Defects

A large number of different figures have been given for the prevalence of speech defect in childhood. In the 'thousand family' study in Newcastle, Morley (1957) found that 14% of 5-year-old children had severe defects of articulation and that these were so marked in 4% of them that teachers found that they were unintelligible.

In the 11,000 7-year-old children studied in a national survey by Pringle *et al.* (1966) 4·5% of boys and 2·3% of girls were considered to have 'markedly poor oral ability' and 20·9% of boys and 15% of girls were considered by their teachers to have 'below average oral ability'; 16·2% of boys and 11·4% of girls were not fully intelligible on testing and stammering was found on examination in 1·3% of boys and 0·8% of girls.

It is clear from these and similar statistics that the problem of speech defects in the community is considerable and its importance is increased by the fact that children with significant speech defects very often have difficulty in learning to read and spell. Paradoxically, very little instruction is given about speech disorders to nurses, health visitors, social workers, dentists and medical students in their ordinary curricula.

It is often found that children with speech defects are immediately transferred to the care of the speech therapist for diagnosis as well as treatment without an adequate medical examination having been performed. All too often the speech therapist is newly qualified and relatively inexperienced, and may have to diagnose by 'hunch' and treat by intuition. In contrast, the well-qualified experienced speech therapist will make a meticulous and scientific assessment of a child's speech disorder, using tests of expressive language, comprehension, hearing and articulation.

Classification

The classification of speech disorders (including language and articulatory disorders) poses many problems, partly because the articulatory patterns encountered in children suffering from abnormalities of articulation are only now being studied systematically by speech therapists and phoneticians (Beresford & Grady 1972). Linguists are still very much at the stage of studying the developmental patterns of the syntax of normal children and as yet have hardly begun to study children with deviant language development (Huxley 1969, Klima & Bellugi 1966). It is therefore necessary to classify patients by clinical criteria which are supported by the testing of language, hearing and articulatory functions carried out by the speech therapist (Ingram 1968) (*see* Table 1).

Table 1

Clinical classification of speech disorders in childhood

(1) Disorders of vocalization (dysphonia)
(2) Disorders of respiratory coordination (dysrhythmia)
(3) Disorders of speech sound production with demonstrable dysfunction or structural abnormality of tongue, lips, teeth or palate (dysarthria):
(*a*) Due to neurological abnormalities: upper motor neurone lesions, nuclear agenesis, lower motor neurone lesions, abnormal movement patterns
(*b*) Due to local abnormalities: jaws and teeth, tongue, lips, palate, pharynx, mixed
(4) Disorders of speech sound production not attributable to dysfunction or structural abnormalities of tongue, lips, teeth or palate, but associated with: mental defect, hearing defect, true dysphasia, psychiatric disorders, adverse environmental factors, combination of these
(5) Developmental speech disorder syndrome (specific developmental speech disorder)
(6) Mixed speech disorders, comprising two or more of the above categories

Dysphonia

By dysphonia is implied loss or impairment of voice. Affected children tend to whisper or to speak hoarsely but when they shout the defect becomes less obvious. The commonest causes are chronic or recurrent laryngitis, frequently associated with upper respiratory tract infections, especially if these occur in the first two years of life, and over-use of the voice through excessive shouting. Frequently these factors are combined in children of Social Classes IV and V who come from relatively poor social conditions and often from large families in which respiratory disease in early life is common. Often children in these circumstances have to shout to make themselves heard and it is when they first go out to play with other children that dysphonia becomes apparent. A less common cause of dysphonia is papillomata of the larynx, and a still rarer cause is familial paresis of the vocal cords, which I have only encountered twice.

In view of the possibility that dysphonia may be due to papillomata of the larynx or to paresis of the vocal cords, laryngoscopy should always be performed. This frequently shows chronic inflammation, often associated with nodules on the vocal cords.

Treatment is difficult because it essentially consists of resting the voice for a prolonged period, which is impracticable in children under 8, and usually requires that the child should be in hospital or convalescent home. If there is evidence of persistent infection, appropriate antibiotics should be given.

Dysphonia occurs in 3–5% of patients referred to hospital speech clinics (Greene 1964, Birrell 1954).

Speech Dysrhythmia

The smooth modulation of speech and the intactness of its rhythm depend on the accurate coordination of respiratory and articulatory mechanisms. When this fails, the rhythm of speech is interfered with and dysrhythmia is said to be present. It may show itself in the undue prolongation of word sounds, the arrest of speech which occurs most often at the beginning of sentences or phrases, or in the repetition of syllables at the beginning of words and at consonants. The latter is loosely referred to as 'stammer'.

Speech dysrhythmia occurs commonly. In their classic study of stammering in Newcastle, Andrews & Harris (1964) reported that the incidence of stammering in the children studied was between 3 and 4%. Pringle *et al.* (1966) give a rather higher figure in the 11,000 children aged 7 whom they studied; boys were found to have stammered in 7·9% of cases and girls in 4·5%. On examination, Andrews & Harris found that about 1% of children whom they studied stammered, and Pringle *et al.* found that 1·1% of 7-year-olds stammered. It is interesting to note that in spite of its high incidence very little is known about its causes. It is clear, however, that in the majority of cases the disorder is transient and clears up without specific treatment.

Stammering has been attributed to genetic factors though Andrews & Harris (1964) thought that hereditary factors were more likely to be contributory than causal. Johnson (1959) made the suggestion that the concern shown by parents when their children 'cluttered' (i.e. showed slurring, hesitations and repetition of syllables) at the age of 2–4, which is a normal phenomenon, tended to focus the child's attention upon his disability and thus encouraged him to perpetuate it. He emphasized the importance of 'the negative evaluation of the child's non-fluencies

by the parents and identification of these as stuttering'. Speech dysrhythmia has often been regarded as a manifestation of maladjustment and regarded as a psychiatric symptom, but Andrews & Harris (1964) found no significant excess of psychiatric symptoms in the children studied who suffered from speech dysrhythmia. They felt that any excess could well be secondary to the effects of the abnormality of speech. They summarized their point of view as follows: 'Stuttering can be regarded as the product of certain adverse environmental factors acting upon the genetic matrix. From the pathologic point of view there is reason to believe that these causal factors operate by distorting the pattern of learning of speech.'

Various attempts to classify the different types of speech dysrhythmia and the stages of dysrhythmia which occur as children grow older have been made. Andrews & Harris, for example, recognize a condition of 'transient developmental stuttering' which tends to occur between the ages of 2 and 4 and disappear after a period of months or perhaps a year. They also recognize a second group of 'benign stutterers' in whom dysrhythmia occurs between the ages of 3 and 11 and disappears after a lapse of between six months and six years.

Only about 1% of the child population show persistent stuttering and it is this group which has been studied in particular detail by Bloodstein (1949, 1950, 1960*a*, 1960*b*), whose account of the development of speech dysrhythmia is perhaps the most coherent yet produced. In the first phase, most often found in children before they go to school, speech sounds and syllables tend to be repeated, most often at the beginning of phrases or sentences. There is considerable variation in the severity of the dysrhythmia from time to time but speech tends to be intelligible and the child is not particularly concerned about his symptoms. In the second phase repetitions become more frequent, and prolongations, hesitations and blocking occur. Associated movements of the limbs and body, often accompanied by grimacing, are noted within a period of weeks or months and the child appears to 'struggle to get his words out'. In the third stage the hesitations and blockings become more frequent and repetitive stuttering or stammering less so. A child 'gets stuck' more often and is increasingly aware of his speech defect. In the fourth phase the pattern is fully developed. The child avoids words which he thinks are difficult to articulate, and to escape the need to talk becomes a major preoccupation in life.

Therapy for speech dysrhythmia: Many different forms of treatment have been suggested, varying from hypnotism to psychoanalysis, and from breathing exercises to delayed auditory feedback. In general, treatment until the child is 7–8 must be by modification of the environment. The doctor and therapist should ensure as far as possible that the child is treated normally, that his speech symptoms are disregarded to the maximum extent and that he is encouraged to communicate.

From the age of 8 to 9 direct therapy becomes possible and increasingly therapy takes the form of training in so-called 'syllable-timed speech'. Patients are taught to speak syllable by syllable with regular unaltering rhythm so that their speech pulses tend to be evenly spaced. It takes considerable concentration and effort on the part of the patient to learn to speak in this way and it is of great importance that once he has learned to speak with 'syllable-timed speech' he should continue to do so at home and at school in spite of the fact that his speech sounds to him to be somewhat 'abnormal'. The results of this form of treatment have been most encouraging but there is a tendency to relapse; it is necessary to see children following the initial period of intensive therapy in which 'syllable-timed speech' has been established, in order to ensure that they are kept up to the mark (Andrews & Harris 1964).

Dysarthria

By dysarthria is understood defective articulation due to neurological or anatomical abnormalities of the lips, tongue or palate or their related structures, e.g. cleft palate, hypoglossia, severe malocclusion of the jaws, palatal disproportion or obstruction of the nasal airway. The simplest neurological disorder in anterior protrusion of the tongue during sucking and speech which produces 'lisping' or difficulties with the *s* and *s*-combination sounds. This is often an hereditary trait. In other cases there may be paresis of the palate due to lower motor neurone disease, or to upper motor neurone disease originating in the brain stem or cerebral hemispheres, as in hemiplegic or diplegic cerebral palsy in which there is paresis of the limbs. In ataxic cerebral palsy the same weakness and incoordination of voluntary movement seen in the limbs may also be found in the lips, tongue and palate. In dyskinetic cerebral palsy there may be involuntary movements of the lips, tongue and palate similar to the involuntary movements found in the limbs; in addition in this disorder there is frequently dysrhythmia due to the lack of co-ordination of respiratory activity and the movements of the articulatory muscles. In myasthenia gravis the same tendency to fatigue on voluntary movement which is so evident in the limbs is also present in the lips, tongue and palate, and

speech tends to deteriorate towards the ends of sentences, especially towards the end of the day.

In paretic, ataxic and dyskinetic cerebral palsy, as well as in congenital dystrophia myotonica and myasthenia gravis, there is a tendency for spontaneous swallowing to be less frequent than normal so that drooling from the mouth may be a marked feature. In these circumstances, the speech therapist's first aim may be, somewhat paradoxically, to teach the child to keep his mouth shut. It may also be held open if the nasal airway is inadequate, most often because of adenoidal enlargement. The resulting syndrome of an open mouth, excessive drooling and hyporhinophonia is a common one, and a good example of the way in which associated organs may interfere with the function of the tongue, lips and/or palate. The errors in articulation which patients make when they suffer from dysarthria may be characteristic. For example, in cleft palate or palatal disproportion syndrome, persistent nasal escape is typical; the 'lisping' of tongue-thrust is easily recognized. Patients who suffer from spastic paresis of the lips, tongue and palate tend to speak slowly with apparent effort, whilst when there is choreoathetosis the articulatory errors are bizarre and inconsistent and often associated with dysrhythmia and sometimes dysphonia.

Tongue-tie is a very uncommon cause of speech disorder, though commonly blamed by relatives for a variety of types of defective speech including slow speech development.

Developmental Articulatory Apraxia

This was the name given by Morley (1957) to a rare disorder in which voluntary control of the lips, tongue and palate was impaired during speech. Involuntary movements of the organs might be within normal limits. Frequently there was a history of feeding difficulties and drooling in infancy and later in weaning, but most children appeared normal until it was observed that they were slow to speak and apparently unable to reproduce speech sounds, particularly sequences of speech sounds correctly. There was a tendency to improvement as affected children grew older but the condition was frequently a major problem so far as treatment is concerned. More boys than girls were affected.

Secondary Speech Disorders

In secondary speech disorders the motor and sensory functions of the lips, tongue and palate and associated structures appear to be normal, but speech is impaired because of disease which does not directly affect the articulatory or respiratory apparatus. The majority of affected children show slow speech development but there may be characteristic features of articulation which give clear indications of this particular diagnosis. For example, children who suffer from hearing impairment, particularly when it is severe in the higher frequencies, are characteristically slower to learn to speak than normal children and particularly slow to learn to articulate *s*, *sh*, *p* and *th* sounds correctly.

The doctor's contribution to the diagnosis of the cause of secondary speech disorders is of particular importance. The suspicion of a hearing defect may be aroused by the mother's history of prenatal rubella or of familial deafness, by a history of hyperbilirubinæmia or meningitis in the neonatal period, or by a story that the child had repeated upper respiratory tract infections in infancy associated with middle ear disease. A history of the child's motor, linguistic, adaptive and social milestones of behaviour all being retarded generally suggests the presence of underlying mental retardation. Failure of the child to make the normal eye-to-eye contact with his mother during feeding and a tendency later to be more interested in objects than people and to 'live in a world of his own' raises a suspicion of infantile autism with associated retardation of speech development. Children with elective mutism usually babble normally, but though they may speak normally at home, they often fail to communicate with neighbours and remain silent in school for months and even years.

Children with acquired dysphasia usually have a history of antecedent head injury or disease affecting the brain, such as meningitis, cerebral thrombophlebitis or profound hypoxia. Characteristically, after the acute cerebral episode the amount of utterance is reduced, and they use simple grammatical structures, omitting prepositions, auxiliary verbs, articles and conjunctions; they have associated difficulties later on in learning to read and spell.

Severe social deprivation may be recognized almost immediately when the history is taken but complex parental deprivation, particularly when it occurs in materially well-endowed homes, may be much more difficult to recognize. The effects of poor social conditions on language development have been particularly well studied by Bernstein (1965) and Hardy (1970).

On examination it is important that the various syndromes associated with mental defect should be recognized. In the speech clinic in the Royal Hospital for Sick Children, Edinburgh, in recent years, atypical mongols, gargoyles, cretins and children suffering from phenylketonuria have been identified. In addition, full audiometric, psychiatric or psychological assess-

ments have been found necessary in a high proportion of suspected mentally retarded patients.

It is unrealistic to expect the speech therapist to diagnose rare neurological conditions such as congenital dystrophia myotonica, nuclear agenesis, pontine tumours, mild hemiplegic, diplegic, ataxic or dyskinetic cerebral palsy, or progressive brain diseases such as Friedreich's ataxia, Huntington's chorea or Wilson's disease, all of which have been encountered in the clinic. On the other hand, the characteristics of the language used by children suffering from secondary speech disorders may give a clue to the nature of the underlying diagnosis; the characteristic features of the language used by children suffering from infantile autism have been well described by Sutherland (1964) and Wolff & Chess (1964). Similarly the difficulties of children with acquired dysphasia in word-finding and in the construction of grammatical sequences have been well described by Alajouanine & L'Hermitte (1965). An experienced speech therapist can often point out these characteristic speech anomalies and assist the doctor in the diagnosis of the underlying cause of the secondary speech disorder. As noted above, further investigations by echoencephalography, brain scan, electroencephalography, electromyography and biochemical tests may be required, as may further assessment by an otolaryngologist, psychologist, psychiatrist or social worker.

Developmental Speech Disorder Syndrome

This is the name given to the retardation of speech development which occurs in healthy children of average or superior intelligence, coming from normal home backgrounds, who show no evidence of dysarthria or secondary speech disorders. For the purposes of description the child is considered to be retarded in speech development if he has no words by the age of 18 months and no phrases at the age of 30 months. This disorder is 2–3 times more common in boys than girls (Ingram *et al.* 1971). In a high proportion of patients, relatives have a similar history of slow speech development or of difficulty in learning to read and spell; there is also an excess of sinistral ambidextrous relatives in some families and an unexplained excess of twins. There can be weak lateralization in relation to hands, feet, eyes and visual fields, a finding which has been recorded in series after series of patients for over forty years (McCready 1926, Zangwill 1960, Brain 1965). The great majority of patients have normal motor, adaptive and social milestones.

Patients are mildly affected when their language development appears to have been within normal limits but they have been slow to learn to produce word sounds correctly. Mothers often say in these circumstances, 'he has the words but he can't say them'. On examination, such children are found to substitute or omit consonants and consonant clusters which are normally late to be acquired. The errors made are similar to those of normal younger children, and can be demonstrated by using a chart of the sounds the child produces and comparing it with the sounds of a normal healthy younger child (Table 2).

Table 2
Classification of the developmental speech disorder syndrome

Severity	*Description*	*Other terms*	
Mild	Retardation of acquisition of word sounds. Language normal	Dyslalia	
Moderate	More severe retardation of word sound acquisition and retarded spoken language development. Comprehension normal	Developmental expressive dysphasia	Developmental mutism
Severe	Still more severe retardation of word sound acquisition and spoken language development. Impaired comprehension of speech.	Developmental receptive dysphasia. Word deafness. Auditory imperception	Developmental mutism
Very severe	Gross failure of speech development. Impaired comprehension of language and significance of other sounds. Often apparent deafness	Auditory imperception. Central deafness	Developmental mutism

Children classified as being moderately severely affected show significant backwardness in uttering language, have restricted vocabularies and use simple immature grammatical forms but have no difficulty in the comprehension of speech. More word sounds will be defective than in mildly affected children, and in general their 'phonetic age' or 'articulatory age' will be less than in mildly affected patients whose language, though not their articulation, is intact. There is always a history in moderately severely affected patients of retardation of speech development. The child may not babble until the age of about 18 months or say 'Ma' and 'Da' until the age of 2, and he may not say his first intelligible words until the age of 2½ or later.

Speech development not only occurs late in such patients but is slower to develop once it begins, so that the progress that a normal child would make in a six-month period between 2 and 2½ years takes the child moderately affected by the developmental speech disorder syndrome one or even two years to achieve.

Severely affected patients not only have severe retardation of the ability to utter word sounds correctly and in acquiring vocabulary and grammatical forms, but also have difficulties in comprehending spoken language. In many instances the parents note that the child was slow even to recognize his own name and to comprehend spoken commands and questions. Many

children have been noted to lip read before they seem to understand by using their hearing. Children of this type have been said to suffer from 'developmental receptive dysphasia' or 'auditory imperception'. They tend to be severely affected and to have very slow speech development, and a high proportion never develop verbal fluency even when adult Their word-sound difficulties are even more marked than in those with moderate degrees of the syndrome.

A minority of the most severely affected children suffering from the syndrome not only have difficulties in comprehending speech but also in recognizing the nature and significance of non-speech noises in their environment and in localizing them. They may appear to be deaf for months or even years after birth. Characteristically, they appear to hear intermittently and may respond to quite quiet sounds whereas formal testing using loud sounds has resulted in no response. I remember one child who appeared to be deaf after a wide variety of formal tests had been given and who then responded to the click of a ball point pen being retracted at the end of the interview. Such children have been said to suffer from central deafness and have been called noncommunicators by Murphy (1964). It is hardly surprising in these circumstances that a significant proportion of very severely affected patients find their way into schools for the deaf.

The prognosis for mildly affected children with normal language development who are slow to acquire word sounds is good, and the majority speak normally by the age of 7 at the latest. The prognosis for children who are moderately severely affected is rather less good, but the majority, though they may have later educational difficulties, progress and have relatively normal speech. The prognosis for children with comprehension difficulties and difficulties in perceiving the significance of sounds other than speech is much less good and a small proportion of those who are severely affected, and a large proportion of those very severely affected, never use spoken language as a primary means of communication.

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Orthodontic Aspects of the Diagnosis and Management of Speech Defects in Children

The orthodontist is not usually primarily involved in the diagnosis of speech defects except in the case of lisping associated with malocclusion of the anterior teeth. His assistance may be sought in cases in which malrelationship of the jaws and malocclusion of the teeth are thought to be possible etiological factors in speech defects.

Since 80% of specific speech movements are made in the anterior part of the mouth (Kimball & Muyskens 1937) it is not surprising that a causal relationship between speech defects and malocclusion has long been assumed to exist.

Narrow high palates, incisal irregularities, spaced or absent teeth, open bite and antero-posterior arch malrelationships (Angle's Class II and III malocclusions) are commonly listed as the malocclusions chiefly associated with defective speech (Farrer 1888, Hellman 1917, Kessler 1954). Speech sounds said to be affected are the bilabial, labiodental, linguodental and linguo-alveolar consonants in the anteroposterior anomalies, the palatal consonants in cases of high palate, and the very common defect, the defective *s* sound or sigmatism associated with irregular incisors and anterior open bite.

The correlation between defective speech and malocclusion is not, however, absolute. Several studies such as those of Van Thal (1935), Hopkin & McEwen (1956) and Lubit (1967) have shown that malocclusions are not in general the primary