4.1.2 Speech and language disorders of childhood in children of average intelligence

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A common question in general practice and paediatrics concerns the long-term consequences of delayed development of speech and language in the early years of life (Fundudis *et al.*, 1979). Formerly, the most frequently held view was that most young children with speech and language problems would eventually develop normally. Recent research reveals that this may be a rather optimistic view – it is more likely to be true of children without major mental and physical handicaps. Two allied clinical questions remain: first, which children will eventually develop normally; second, what is the differential diagnosis in the case of those who do not?

In a brief chapter there is no space to discuss theoretical aspects of the development of speech and language, particularly new psycholinguistic concepts or the rules of grammar and language competence (Chomsky, 1957, 1965; Menyuk & Looney, 1972) which are competently surveyed elsewhere in the psycholinguistic literature. The focus will be on those types of disorders which present in clinical settings, such as deafness, specific developmental speech/language disorders, and elective mutism. Apart from considerations of classification and prevalence, it will not include those disorders associated with mental handicap, brain damage, or infantile autism which are fully dealt with in chapter 4, and in volumes 2 and 3.

Classification

One of the more useful functional clinical classifications is that of Ingram (1959a, 1959b, and 1972) who describes three main categories of speech and language disorders: primary, secondary, and developmental. Modifications of this classification have been developed by other workers (Fundudis *et al.*, 1979).

Primary. These are disorders of speech sound production which occur in dysarthria or with cleft palate. In these conditions there is either demonstrable dysfunction or structural abnormality of tongue, lips, teeth, or palate.

Secondary. These are disorders of speech sound production secondary to other diseases or environmental factors. These include: marked intellectual impairment; demonstrable neurological disorders, and cerebral palsy; deafness; specific psychiatric syndromes, such as infantile autism and elective mutism; acquired dysphasia and the effects of an adverse psychosocial environment.

Developmental speech/language disorders (syndrome). This is a descriptive label for retardation of speech/language development in children who are otherwise apparently normal.

In addition to the above classification a distinction needs to be made between speech and language. For the purposes of a brief chapter, speech can be simply defined as the articulatory skill associated with the production of word sound and spoken language.

Language is a cognitive skill which utilizes a system of symbols (Lewis, 1968) and whose main purpose is communication; it has various forms such as spoken, written, gesture and sign (Rutter, 1972).

Prevalence

The rates of retardation of speech and language reported in population studies are a function of the definition employed. In Newcastle upon Tyne, Morley (1965) reported that 5.7 per cent of 944 children were not using two- to three-word phrases at three years; according to Fundudis and co-workers (1979), 4 per cent of 3300 three-year-old children failed to use three or more words strung together to make some sort of sense. Stevenson and Richman (1976) studied expressive language delay at the same age, but their criterion when standardized tests could not be used

consisted of inability to speak in three-word phrases. They report a rate of 3.1 per cent of 705 children.

It is impossible to provide an accurate estimate of prevalence of rarer conditions when studying a relatively small population of children (Fundudis *et al.*, 1979). The following, therefore, is only a rough guide to the prevalence rate of the different types of conditions associated with speech and language disorders.

Mental handicap. Ingram (1972) reports that the single most common cause of slow speech development in paediatric clinics is mental handicap. If this is defined as an IQ of less than 70, then over 2.5 per cent of the child population is likely to be affected, with over half of these children showing severe language or articulation defects, or both (Rutter & Mittler, 1972).

Deafness. This is one of the major causes of delay in speech and language development (Morley, 1965). About 2 in 1000 children have deafness severe enough to merit the use of hearing aids (Reed, 1970).

Infantile autism. A rate of 2–4 in 10000 children has been described by Lotter (1966) in his Middlesex survey.

Elective mutism. In their population study, Fundudis and colleagues (1979) report a prevalence rate of 0.8 in 1000 at the age of 7–8 years, and therefore conclude that it is as rare a syndrome as infantile autism. However, the frequency is dependent on the age under scrutiny and whether the condition is broadly or narrowly defined.

Severe dysphasia. Serious and persisting language disorder has been estimated as occurring in 0.7 in 1000 children in Scotland (Ingram, 1963) and in 0.4 in 1000 in Newcastle (Fundudis *et al.*, 1979). Again, the condition would seem to be as rare as infantile autism.

Dysarthria. The rate is approximately 1 in 1000 births (Morley, 1965).

Developmental speech/language disorder syndrome. Newcastle research shows that this occurs in 33 in 1000 children of school age (Fundudis *et al.*, 1979). It is likely that the population studied is similar to that described by Stevenson and Richman (1976) as showing expressive language delay: it is not surprising, therefore, that the latter report a similar rate of 31 in 1000.

Types of disorder

Specific developmental speech/language disorder syndrome

In the past it was assumed that if a child had an isolated delay in speech development and was not obviously abnormal in other ways he would usually grow out of it. This is the basis of the specific speech/language disorder syndrome. In this sense the syndrome may be considered to represent an extreme variation in normal development (Rutter & Yule, 1970). The salient features have been described by Ingram (1972): it affects apparently physically healthy children with normal hearing and average intelligence, coming from normal home backgrounds. Other commonly reported features include a family history of slow speech development; an excess of close relatives who have ambidexterity or left-handedness or who previously had difficulties in the early stages of learning to read and write (McCready, 1962; Brain, 1965); and a high ratio of males to females. Others assert that the course of the condition may be influenced by environmental factors (Tizard, 1964).

Ingram's view is that the syndrome comprises a heterogeneous group of articulatory and language disorders in which speech development may not only be delayed but deviant as well. Nevertheless, he finds it useful to regard it as a spectrum of clinical disorder which range from mild to very severe.

The mildest are the dyslalias, which are simply delays in development of articulation giving rise to 'retardation of acquisition of word sounds but with normal language' (Ingram, 1972). Most authorities consider that these are commonly developmental mispronunciations which usually have little predictive importance for later cognitive or linguistic development.

The moderate developmental speech/language disorders are the developmental expressive dysphasias. The affected children have greater degrees of retardation of word sound acquisition, impaired development of and often deviant spoken language, and impaired comprehension of speech.

The severe developmental speech/language disorders are serious disorders of language comprehension. The affected children have greater degrees of retardation of word sound acquisition, impaired developmental and often deviant spoken language, and impaired comprehension. One report claims that

this form is not only equally common in boys and girls but that partial high-tone hearing impairment may accompany it (Rutter, 1972).

More recent research has shown that, as a group, speech-retarded children have shorter gestational ages and lower birth weights than controls and are less frequently first-born (Fundudis *et al.*, 1979). Early reports of left-handedness or mixed laterality in developmental speech/language disorders (Orton, 1934, 1937) have not been consistently confirmed (Rutter & Yule, 1970) and this is particularly true in the case of population studies (Rutter & Yule, 1970; Fundudis *et al.*, 1979).

Modern epidemiological research reveals that the rate of problem behaviour in 3-year-old children with language delay is four times that in a random sample of children (Stevenson & Richman, 1978). Furthermore, 50 per cent of the language delay group were also retarded in their non-verbal abilities (Stevenson & Richman, 1976, 1978). However, the predictive value of such early assessments is not great and the crucial question that remains is, how many children with language delay at three years will catch up in their cognitive and language ability and lose their behavioural disturbance?

One in five of the Newcastle cohort who were previously speech retarded were found at school age to have serious language, intellectual, or physical handicaps and to have been labelled a 'pathological deviant' group. If this extreme group is set aside we are left with a residual group of speech-retarded children. There are firm grounds for believing that this group of children with serious speech retardation is very similar in speech and language development to the children with language delay described by Stevenson and Richman (1976) and both of these groups consist mainly of children with moderate or severe developmental speech/language disorders. The Newcastle research can, therefore, be used to answer some of the questions about outcome posed in this section. A high percentage of the residual speech-retarded children group still had significant cognitive and educational impairment, poor language development, including poor expressive language skills, and more restricted type of language expression (as defined by Bernstein, 1962). In addition, the prognosis in terms of behaviour is likewise poor. An important finding was that speech delay is a better predictor of impaired verbal intelligence than of performance intelligence. Furthermore, combined delay of speech and walking significantly predicts poor cognitive and language development, less educational achievement, behavioural problems, and also introversion and social withdrawal (Fundudis *et al.*, 1979).

Acquired dysphasia

This consists of a loss of acquired language functions (Ingram, 1972). Hence, a birth-injured child cannot be described as having lost language functions, but more accurately as showing retardation of development of speech and language. In a child aged two to three years there is likely to be an impediment of language and thereafter slowing of speech.

Psychological mutism

This has two forms: traumatic and elective mutism.

Traumatic mutism has an acute onset following a psychological or physical shock or injury. Some consider it to be a hysterical phenomenon and the literature suggests that it is common, but a clinical survey does not corroborate this (Kolvin & Fundudis, 1981).

Elective mutism is the term coined by Tramer (1934) to describe a fascinating condition where talking is confined to a familiar situation and to a small group of intimates. This, too, is a rare condition and is distinguished from the excessive shyness that occurs relatively frequently in reception classes in schools (Wright, 1968; Brown & Lloyd, 1975), by its severity and its persistence. Earlier accounts were usually anecdotal but recent studies of more representative series of cases indicate that such accounts are often misleading: more girls than boys are affected, which is unusual for childhood disorders (Wright, 1968; Kolvin & Fundudis, 1981); furthermore, elective mute children are born significantly early in the sibship. There is evidence of slow or uneven development compared with controls, including delay in the beginning of speech, excess of developmental mispronunciations, EEG immaturity, and associated problems of speech, bowel and bladder function (Kolvin & Fundudis, 1981), poorer non-verbal intelligence, and a high rate of associated behavioural problems.

With regard to aetiology, some workers suggest that mutism may be secondary to some biologically based symptoms – for instance, some children avoid speaking because they are teased when they mispronounce words (Rutter, 1977). On the other hand, the literature is also replete with single or small-

group studies postulating psycho-dynamic bases such as faulty mother–child relationships (Parker *et al.*, 1960), family neurosis (Browne *et al.*, 1963) or psychological trauma in infancy (Salfield, 1950). In the recent Newcastle study, personality problems of parents, particularly those concerning social relationships, proved common. In addition, more than double the number of parents of elective mute children attended psychiatric clinics than did those of the control children.

Finally, follow-up reveals that elective mutism is a more intractable disorder than most of the other non-organic, non-psychotic psychiatric disorders of childhood, with less than 50 per cent subsequently improving (Kolvin & Fundudis, 1981).

Deafness

Both because of its rarity and because the deaf infant sporadically babbles, deafness may remain unsuspected in early infancy. Subsequently, parents may be alerted because of a poor expressive response to sudden meaningful noises, or the condition may be detected using developmental screening tests from about 9 months of age. Likely causes of profound deafness can be identified by careful history and include such conditions as serious post-natal middle-ear infections and neonatal hyperbilirubinaemia. In contrast, epidemiological studies usually do not reveal an excess of social and family pathology in families of deaf children (Fundudis *et al.*, 1979).

In a short review it is possible only to touch on some of the complex theoretical issues concerning speech, language, and intellectual development of deaf children (Moores, 1972; Meadow, 1975). In profoundly deaf children the development/acquisition of speech and language is different from that of children with normal hearing, but may also differ for different deaf groups according to the modes of communication used by those taking care of them. The difficulties of language acquisition which deaf children encounter cover not only more superficial language skills, such as spoken language, but also include inner language abilities (Meadow, 1975). On the other hand, there is evidence that the rules of language learned by deaf children in their earlier years are similar to those of hearing children (Fundudis *et al.*, 1979).

The literature provides much evidence that childhood deafness hampers the development of language and verbal abilities, but not necessarily that of non-verbal abilities (Wiley, 1971; Fundudis *et al.*, 1979). However, this comparatively poorer performance on cognitive tasks appears to improve with age and appropriate stimulation. In addition, there is evidence that the academic achievements, particularly progress in reading, of deaf children are poorer than those of hearing children (US Office of Demographic Studies, 1973; Conrad, 1977). Some consider that these cognitive and educational impairments are more an indictment of the educational system than an inevitable consequence of deafness (Vernon, 1976). Surprisingly, deaf children of deaf parents often show better linguistic development than deaf children of hearing parents – which seems to favour an early introduction of non-verbal methods of communication.

Research findings usually report that hearing children are better adjusted socially and behaviourally than deaf children: in the case of profoundly deaf children, antisocial disturbance often reveals itself in school (Fundudis *et al.*, 1979). Furthermore, deaf children raised in families where other members are deaf often prove better adjusted than those raised in families where other members are not deaf (Myklebust, 1964).

Organic brain damage may cause deafness and contribute to the poorer intellectual and educational performance of such children. However, there is no way of estimating the extent of this contribution.

Social influences

While some workers emphasize the biological determinants of speech and language, others stress the importance of psychosocial influences. Chomsky (1969) postulates the presence of an innate language acquisition device which determines the deep-seated properties of organization and structure which are found in all human languages. Lenneberg (1967), too, argues that the emergence of speech is most easily accounted for by maturational changes which are only moderately affected by abnormal factors in the child's environment. He and others (Critchley, 1967) have pointed out that children of deaf parents were found to babble appropriately and to develop speech adequately, despite being brought up in a grossly abnormal linguistic environment.

Environmental stimulation (Irwin, 1960; Routh, 1969) and interchange, particularly with adults, facilitates progress in vocalization and language development in the early years (Cazden, 1966; Brown *et al.*, 1969). The converse is also true, in that it has been

shown that speech and language may be adversely affected, as may verbal intelligence, in those situations which reflect an impoverished social environment, as in those institutions where there is inadequate emphasis on the quality and intensity of stimulation (Brodbeck & Irwin, 1946; Tizard, 1970) in large families and in the case of multiple births. However, even serious impoverishment of the social environment appears to give rise to only moderate degrees of impaired vocalization (Provence & Lipton, 1962), speech delay and language retardation, with the retardation being confined mainly to language expression (rather than to comprehension (Klaus and Gray, 1968)), and retardation of educational performance.

On the basis of the above brief review, it would seem that those more moderate theories which stress interaction of innate factors and social influences are likely to have more general validity (Bruner *et al.*, 1966). We can conclude that adverse social factors must be very unusual to seriously impede the emergence of speech in a normal infant.

Assessment

In the assessment of children with speech and language problems a preliminary formulation and differential diagnosis can usually be made on the basis of relatively simple assessment. The main areas of inquiry include biographical information; observation of the child during interview; assessment of hearing, speech, and language; a study of the child's play and capacity for imitation; cognitive ability; social and behavioural functioning; and, finally, neurological assessment.

Biographical information

This may provide information not only about psychosocial influences, but also about relevant physical factors, such as cerebral insults, history of clumsiness, and so forth.

Observation

This constitutes an integral part of all other forms of assessment. Most clinicians, even while gathering biographical information, will use observation to determine whether the young utilize clues from parents, or display the clumsiness of gait typical of the child with cerebral palsy.

Hearing

This should always be assessed, with the clinician checking whether the child responds to a wide range of auditory stimuli, including responses to sudden or unusual noises and a variety of domestic noises in the absence of visual or gestural clues. Where there are any doubts, expert assessment is indicated, which may include audiometry or even EEG audiometry and other specialized techniques.

Speech and language in its various forms

Speech may be delayed, or articulation skills deviant. For instance, articulation may be defective in deafness, immature in mental retardation and developmental speech/language disorders, defective and immature in autism and variably disordered in elective mutism. The articulation defect in dysarthria is characteristic.

Language can be assessed in a number of modalities, for example, spoken, sign (manual), or written. Language comprehension can be tested by assessing the child's ability to understand simple commands both *with* and *without* the provision of visual and gestural clues. Careful observation will indicate by what means the child is trying to understand his environment. Evidence of language *production* can be obtained from the way the child communicates through gesture, mime and conversation (Rutter, 1977). 'Inner language', which reflects an understanding of a symbolic code can be indirectly assessed by observing whether there is a meaningful use of objects and by constructiveness and creativity in play.

In *profound deafness* the child does not attend to, or respond to, auditory stimuli, but will extensively use gesture, attempt conversation when older, and may be constructive in his play. In many respects the child with the most severe form of a *developmental speech/language disorder* will show a similar pattern and, indeed, there may only be limited evidence of inner language, but usually there will be evidence of normal hearing. In milder forms of development delay there is usually no suggestion of deafness but rather a lack of understanding of verbal but not non-verbal communication and usually there is reasonable evidence of inner language. The younger *autistic child* may not respond meaningfully to auditory stimuli, and, indeed, may tend not to respond to any form of verbal or non-verbal communication. There is little indirect evidence of language either in terms of gesture, mime, imitation or play. In *mental handicap* there is usually no evidence of deafness, but rather of limited language abilities and usually a delay in

development of articulation, together with other evidence of slowness of development.

Constructive play and imitation

Evidence of constructive play reflects the presence of inner language and augurs well for the growth of language in general. Gestural imitation and play constitutes a form of communication which is impaired in infantile autism and in severe language retardation.

Cognitive ability

Parental reports of the child's social abilities and play, combined with clinical observation, provide only a rough guide to the child's cognitive level. However, clinical impressions can be most deceptive and need to be validated by careful psychometric assessment of both non-verbal intellectual skills and language performance. A wide range of tests are now available, which limit the amount of spoken instruction, and measure performance rather than verbal abilities and a wide range of language skills (Mittler, 1972). Even if formal testing is not possible, a social maturity scale, which employs evidence from the mother or the person taking care of the child, may be profitably employed to provide a crude estimate of intellectual level.

Social and general behaviour

Accounts from parents complemented by direct observation may provide useful information about the basis of the speech delay – the manifold social and behavioural abnormalities of autism; the absence of startle to loud noises or alerting to domestic noises – or those reflecting social behaviour of peers, such as occur in the young deaf; and the social withdrawal of the electively mute.

Neurological assessment

If the clinician is not experienced in neurological examination of the child, important signs may be missed. Collaboration with a paediatrician or paediatric neurologist will usually ensure identification of relevant physical anomalies.