CHAPTER 10

Speech and Language Disorders of Childhood

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Introduction

A distinction needs to be made between speech and language and for the purposes of this chapter relatively simple definitions, modified from Sheridan (1972), will suffice. *Speech* is the ability to produce spoken sounds or more systematised vocalisations to express verbal symbols or words. *Language* is a cognitive skill in which there is a symbolisation of ideas or concepts for the purposes of communication in a social context, the communication having both expressive and receptive components. This distinction allows us to understand how the inability to speak, such as that which occurs in various forms of mutism, is not incompatible with the presence of language. As a corollary to this, it is well known that 'parrotting'—the imitation of speech without understanding—is not necessarily accompanied by underlying language facility. However, this distinction only sketches in the polar extremes, and such clear-cut cases are rare. Further, it ignores the effects on speech of certain language disorders, and the effects on both speech and language of certain organic disorders. Finally, for the purposes of clinical assessment, it is necessary to have a reasonable knowledge of the norms of speech and language development.

A common question in general practice and developmental paediatrics concerns the long-term consequences of delays in the 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 'grow out of it'. 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. However, two applied clinical questions remain. First, which children with a delay in starting to speak will eventually catch up, and which will have persistent speech or language problems or other handicaps? Second, what is the differential diagnosis in the case of those who have persistent problems?

In a brief chapter such as this there is no space to discuss theoretical aspects of the development of speech and language, particularly the new psycholinguistic concepts of the rules of grammar and language competence (Chomsky 1957, 1965; Menyuk and Looney 1972), which are summarized in the relevant published reports. We will concentrate on those types of disorders which present in clinical settings, such as deafness, specific developmental speech/language disorders and elective mutism. Hence, in this chapter, apart from classification, we will focus on those disorders which are of particular significance to developmental psychiatry but will not include those resulting from mental handicap or brain damage.

Classification of speech and language disorders

One of the the more useful clinical classifications is that of Ingram (1959*a*, *b*; 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 cleft palate. In these conditions there is either demonstrable dysfunction or structural abnormalities of tongue, lips, teeth or palate. (They will not be described further.)

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

Ingram (1972) points out that acquired dysphasia implies the loss of acquired language functions, so a birth-injured child cannot be described as having lost language functions, but more accurately as showing a retardation of speech and language development. If a child suffers serious brain damage at the age of two to three years, there is likely to be both impairment of language and, thereafter, slowing of speech development.

Developmental speech and language disorder. This is a descriptive label given to children with retardation of speech/language development, who are otherwise apparently normal.

Specific developmental speech/language disorder syndrome. This is an important condition and merits a more detailed description. In the past it has been assumed that if a child has an isolated delay of a major milestone, such as speech, and is in other ways not obviously abnormal, he will 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 extreme variations in normal development (Rutter and 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 of the syndrome 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 male:female ratio. Others, too, assert that the course of the condition may be influenced by environmental factors (Tizard 1964).

Ingram (1972) considers 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 clinically as a spectrum of disorder which ranges from the mild to the very severe, as follows.

The mildest forms are the *dyslalias*, which are simply delays in the development of articulation, giving rise to 'retardation of acquisition and word sounds but with normal language' (Ingram 1972). While the vocabulary and grammatical structures in spoken language may lie within normal limits, the child inconsistently substitutes or omits the

later-acquired consonant or consonant clusters. The parents usually report that the children's understanding of words is greater than their ability to say them. Most authorities consider that, commonly, these are 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*. Children with these disorders have normal comprehension but more severe retardation of word sound acquisition and retardation of development of spoken language.

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 development, and often deviant spoken language and impaired comprehension of speech. 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 *et al.* 1971).

Synonyms for these three degrees of severity are 'dyslalias', 'developmental expressive dysphasias' and 'developmental receptive dysphasias', respectively. Some earlier research (Morley *et al.* 1950, Miller *et al.* 1960) suggested that the dyslasias show natural improvement with age 'which suggests a functional disturbance in which the development of language outstrips the accurate use of articulation'. However, this important project studied neither outcome on a wide range of cognitive skills nor behavioural functioning.

Prevalence

Most studies of prevalence of speech and language disorders are based on relatively small populations, so it is not possible for them to provide accurate estimates of the prevalence of the rare conditions. In the Newcastle-upon-Tyne 'thousand family study', Morley (1965) reported that 5.7 per cent of 944 children were not using two- to three-word phrases at three years. However, in the Newcastle child development study, Fundudis *et al.* (1979) report that 4 per cent of 3300 three-year-old children failed to use three or more words strung together to make some sort of sense. In London, Stevenson and Richman (1976) studied expressive language delay at the same age, but their criterion for children who could not be assessed with standardized tests was the inability to speak in three-word phrases. They report a rate of 3.1 per cent in 705 children. The different rates for retardation of speech and language reported in these population studies are likely to reflect the definitions used. The following therefore constitutes 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 seen 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, and over half of these children will have language or articulation defects, or both (Rutter and Mittler 1972).

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

hearing aids (Reed 1970, Pless and Graham 1970), and the rate of profound deafness is 0.8/1000 (Fundudis *et al.* 1979).

Infantile autism. A rate of 2-4/10,000 children has been described by Lotter (1966) in his Middlesex survey.

Elective mutism. Some workers consider that this is a relatively common condition, but others consider that the frequency depends on the age under scrutiny and whether the condition is broadly or narrowly defined. In their population study, Fundudis *et al.* (1979) report a prevalence rate of 0.8/1000 at the age of seven to eight years, and therefore conclude that it is as rare a syndrome as infantile autism.

Severe dysphasia. This grave and persisting disorder has proved to be rare and has been estimated to occur in 0.7/1000 children in Scotland (Ingram 1963) and in 0.4/1000 in Newcastle (Fundudis *et al.* 1979).

Cleft palate dysarthria. The rate is approximately 1/1000 births (Morley 1965, Fundudis *et al.* 1979).

Developmental speech/language disorder syndrome. The Newcastle child development study indicates that this syndrome occurs in 33/1000 children of school age (Fundudis *et al.* 1979). It seems likely that the cases studied in Newcastle have much in common with those described by Stevenson and Richman (1976) as having expressive language delay: it is not surprising, therefore, that they report a similar rate of 31/1000.

SOME CLINICAL CONDITIONS

In this section we provide an up-to-date account of four conditions which are of particular clinical interest: speech retardation, infantile autism, deafness and mutism.

1. *Speech retardation: the Newcastle child development study*

The Newcastle Survey of Child Development enrolled survivors of the first month of life during the years 1960 to 1962. It was decided to study the 1962 cohort, with special reference to speech retardation, and to examine the children at specified ages and by appropriate methods in order to identify the significant differences in their development. Information about the children's first five years of life included perinatal, obstetric and social data and also details of health and development (Neligan *et al.* 1974, 1976).

The initial screen was essentially to identify children with speech delay, and the simple definition of speech retardation employed in the study was the failure to use three or more words strung together to make some sort of sense by the age of 36 months. Subsequent diagnostic assessment at seven years identified those speech, language and other defects of which speech retardation is a symptom. This showed that the cases could be divided into two broad groups (see Fig. 1).

The first main group consisted of those whose intellectual, psychological or physical functioning was so abnormal that they were described as *pathological deviants.* Such cases fall into three relatively well-defined clinical subgroups:

(a) *Marked intellectual impairment.* This was defined as an IQ at or below the first percentile on the WISC, or if the child was untestable. In practical terms this meant an IQ

of 65 or below. This is possibly too rigorous a criterion, as other authors have used a criterion of 2SD below the mean, *i.e.* IQ of 70 or below (Yule and Rutter 1970).
(b) *Specific clinical syndromes.* This group included children with severe communication disorders of childhood, such as elective mutism (Salfield 1950, Browne *et al.* 1963), infantile autism (Creak 1961, Rutter 1968, Kolvin *et al.* 1971) and cleft palate/dysarthrias or severe language disorders.
(c) *Demonstrable neurological disorders.* This included children with spastic disorders. These three categories were not intended to be mutually exclusive. For the purposes of this chapter we have classified disorders according to the most predominant feature. Finally, we decided that deafness alone should not constitute sufficient grounds for labelling the child pathologically deviant.

The second main group, which we have designated the *residual speech-retarded* group, consisted of children who showed no evidence of serious handicap on clinical examination at the age of seven years. More sophisticated psychological assessments were necessary to delineate their characteristics.

A further clinical classification was undertaken, depending on whether these children suffered from speech retardation alone or from retardation of both speech and walking. In short, we identified three subgroups of speech-retarded children: those who *walked early* comprised the *specific speech-delayed* group; those who *walked late* comprised the *general-delayed* group; and those whose walking milestones were *average* comprised the *intermediate* group.

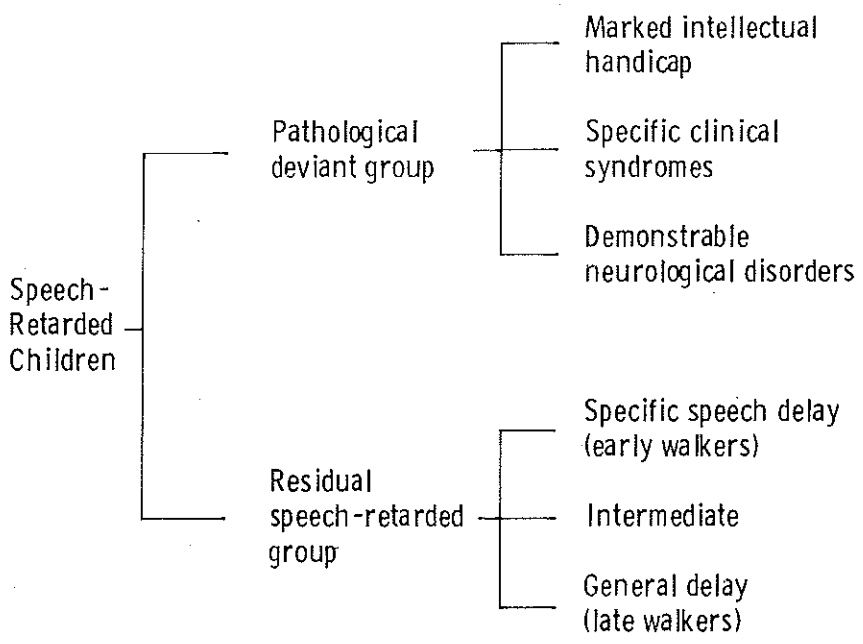

Fig. 1. Classification.

Some background factors in speech-delayed children

Perinatal factors. The mean gestational ages of both the pathological deviant group and the residual speech-retarded group were significantly lower than the mean for the controls. This finding is similar to that described by Butler *et al.* (1973) in children with speech defects. However, in contrast to the Butler study, we found that our total speech-retarded group had a significantly lower birthweight than that of the controls.

Birth order. The mean family size reveals a trend for the residual speech-retarded group to come from larger families (χ^2 = $p<0.05$). In addition, fewer of the speech-retarded group were first-born.

Other milestones. We also found that the residual speech-retarded group achieved bladder control and walked later than the controls, but this only proved statistically significant in the case of walking. Thus our residual speech-retarded group is, on average, less retarded on walking than on speech. Possible explanations for this are provided elsewhere (Fundudis *et al.* 1979).

Laterality. There were no significant differences in terms of right-, mixed- or left-handedness between the controls and the speech-retarded group, nor were there any significant differences between the groups in the number of right-, mixed- or left-eyed children. The only significant difference was confusion in differentiating between the left and right sides of the body; the speech-retarded children were significantly worse at this. Faulty cerebral dominance has been incriminated as the basis of developmental language disorders (Orton 1934, 1937). However, in a recent survey of the literature it was found that reports of an excess of left and/or mixed laterality in speech-retarded children tended to be highly contradictory, with 'as many reports of negative findings as of positive findings' (Rutter *et al.* 1970). This is not unexpected, as most of the previous studies have been mainly of clinic or hospital populations.

Developmental delays. A family history of developmental delays occurred more frequently in the speech-retarded group, which confirms an often-reported association.

Associated problems in speech-retarded children

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

Follow-up studies

This brings us to outcome. 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 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 residual cohort of children with

serious speech retardation is likely to overlap extensively in speech and language development with the Great Ormond Street cohort of children with language delay (Stevenson and Richman 1976), and both cohorts mainly consist of children with moderate to severe developmental speech/language disorders. The Newcastle research can therefore be used to answer some of the questions previously posed about outcome.

The residual speech-retarded group scored significantly less than the controls on all the cognitive (intelligence, perceptual, conceptual and visuo-motor abilities) and language tests. This applied equally to the global test scores and to the subtest scores. Full details of statistical and other findings are reported in our main publication (Fundudis *et al.* 1979). The initial interpretation of these results is that the poorer functioning of the residual speech-retarded group reflects wide intellectual impairment. On the other hand, it may well be that this is not really a homogeneous group, therefore the above analyses may have masked patterns of abilities specific to particular subgroups. We therefore divided the group according to the classification previously described—specific speech-delayed, general-delayed and intermediate.

An analysis of the cognitive and language development of each of these three groups at the age of seven years reveals that they all have depressed functioning compared with the controls. First, the early walkers (*i.e.* the group with a specific delay in speech) have a specific impairment of verbal ability, language ability and educational achievements compared with the controls; in contrast, their non-verbal ability compares very favourably with that of the control group. This pattern is similar for the intermediate group but not so for the late walkers (*i.e.* the general-delayed group). Children in the latter group not only perform poorly on all the above measures but also score significantly poorer on performance IQ than do the controls.

A similar pattern was found on the other cognitive and behavioural measures we used, details of which are available in Fundudis *et al.* (1979). In brief, the early walkers, with one exception, do as well as the controls on non-verbal tests, but again do consistently worse on verbal and language tests. The late walkers do significantly worse than the controls, even on non-verbal tests. In addition, combined delay of speech and walking is likely to predict poor cognitive and language development and poor educational achievement.

Previous reports have shown that childen with speech defects are prone to psychological difficulties (Solomon 1961, Rutter *et al.* 1970, Sheridan 1973). However, there are few studies of the effects of earlier speech and language difficulties on behaviour. The Newcastle research provides such data: the previously speech-retarded children had an excess of classroom disturbance and also of introversion and social withdrawal. Combined delay of speech and walking is particularly likely to predict behavioural problems (Fundudis *et al.* 1979). The Newcastle workers also describe a statistically significant but not high correlation between poor language development and deviant behaviour. Because the primary problem studied was an early speech delay, they conclude that the disorder of speech and language preceded the behavioural deviance and was therefore more likely to be primary, but the mechanisms by which the behavioural deviance occurs are not clear. Possible mediating mechanisms include communication problems, social relationship problems, academic failure or associated brain damage (Rutter 1972).

Awareness of the long-term sequelae of speech retardation is of paramount importance. There is evidence that the frequent use of incomplete sentences at the age of 3 years 9 months rapidly improves, so that one year later very few children have this disability (Morley 1965). Further, Butler *et al.* (1973) point out that by the age of seven years most developmental mispronunciations have disappeared spontaneously. Such apparently spontaneous improvement does not necessarily mean that henceforth all will be well, as there are reports of worrying long-term consequences. For example, the Edinburgh research group, in their follow-up of speech-retarded children in primary schools, report that the majority have reading difficulties (Ingram 1963, Mason 1967). In addition, Rutter (1972) points out that to read and 'to understand the meaning of what he reads . . . a child must have language skills'. Accordingly, he argues that speech-delayed children are likely to have reading delays as well, because both reflect language impairment. Our findings suggest that speech delay is a better predictor of impaired verbal intelligence than of performance intelligence, and that a combined delay in speech and walking is a good predictor of poor cognitive, language and educational development. We have other evidence to demonstrate that, at the age of seven, the three subgroups of speech-retarded children have residual linguistic difficulties. The extent of the difficulties is closely tied to their previous prowess or delay in walking.

Less is known about longer-term consequences. The literature suggests that most children with speech and language problems and who are not physically or mentally handicapped will eventually speak normally and prove to have reasonable emotional adjustment, but persistent educational difficulties. However, such an optimistic prognosis may be influenced by the selectivity of the samples upon which such conclusions are based (Ingram 1963, Garvey and Gordan 1973, Renfrew and Geary 1973).

Our results emphasize the predictive value of a simple speech screen at the age of three years. About one in five of the 4 per cent of all children aged three years who were speech-retarded were later found to have serious language, intellectual or physical handicaps. This underlines the value of an early screening exercise (Butler *et al.* 1973) in identifying children with handicaps who may need more intensive assessment or help with appropriate placement. Nevertheless, the situation at the age of three years does not reflect the total position at five, six or seven years, so that a comprehensive screening programme should include periodic re-examination over the first five to seven years of life. If the extreme (pathological deviant) group is set aside, it was commonly assumed that the children in the residual group would soon overcome their speech disability and thereafter would function normally in most respects. Our follow-up study at school age of previously speech-retarded children revealed widespread cognitive, language and educational disabilities, some of which we describe in this chapter. A comprehensive account of cognitive, speech, language, educational, physical, social and behavioural outcome is provided in Fundudis *et al.* (1979).

Finally, the other findings show that the subgroups of the residual speech-retarded group, who were identified on the basis of their speech and walking milestones, differed from the controls in important ways. The pattern of cognitive performance of the specific speech-delayed group was broadly similar to that of children who were

clinically considered to be *dysphasic* (Olson 1961, Weiner 1972). Fundudis and colleagues argue that this constitutes circumstantial evidence that the specific speech-delayed group had previously suffered from a type of developmental dysphasia. Some of the evidence adduced by Fundudis and colleagues in support of this hypothesis include good non-verbal IQ in contrast to relatively poor grammatical abilities, lack of vocabulary comprehension, poor expression of ideas and reading attainments, poor word-sound discrimination and immaturity of speech articulation. In addition, hearing was also within normal limits. These workers also assert that the specific speech-delayed group appears to represent the less severe end of the spectrum of the developmental speech/language disorder syndrome described by Ingram, in which there appears to be a reasonably good prognosis. However, children in the generally delayed group showed widespread retardation of verbal and non-verbal ability, language and educational attainments.

2. *Infantile autism*

This is an intriguing condition which is well described in the literature; therefore we will confine ourselves to an account of the associated speech and language problems. Often such children not only have delayed speech, but also delayed and deviant language (Rutter 1965).

Even their babble may be impaired (Ricks 1975). Subsequently only about a half these children develop or acquire useful speech and, even when they do the onset is often delayed. Those who remain mute tend not to use gesture as a form of communication (as do children who are deaf or who have developmental speech/language disorders). Some may display defects in articulation, but more often there are defects in pronunciation and their speech is immature. Where speech is present it may be staccato or jerky, rather flat and monotonous and rarely accompanied by appropriate facial expression; other characteristic abnormalities include echolalia, a reversal of pronouns, and a paucity of spontaneous speech or use of speech for the purposes of communication. Furthermore, most autistic children show a surprising lack of interest in speech and their understanding of spoken language is poor. Nor do they appear able to cope with symbolic gesture and mime, but rather only with a primitive form of non-verbal communication in which they indicate their needs by pulling an adult by the arm towards the desired object, without looking at the adult. Older autistic children, even those with normal intelligence, often have residual speech and language disorders, combined with a curious use of metaphors and a rather mechanical expression. The development of 'inner language' is often impaired and this is reflected in their paucity of imaginative play; furthermore, few reach the stage of imitative play (Ricks and Wing 1976).

These empirical findings highlight the fact that not only do autistic children tend to be retarded in language, but their pattern of linguistic abilities is deviant as well. This suggests that one of the important determinants of autism is a specific defect involving language, covering both comprehension and production of language and language modalities. This hypothesis is supported by the finding that the abnormality of response of autistic children to sensory stimuli tends to resemble that seen in developmental receptive dysphasia, but in autism the language deficit is more severe

(Bartak *et al.* 1975). Some workers have suggested that such defects could make a contribution to the impairment of social relationships (Churchill 1972). Further, it has been postulated that deficiencies in language modalities make the natural environment of the autistic child so complex that it inhibits him from making predictions from it or achieving mastery of it (Churchill 1971, DeMeyer *et al.* 1972). This is a complex problem and it is still not clear whether the primary disorder is one of language or whether the language disorder arises from a more widespread cognitive deficit (Rutter 1974), or from widespread cerebral dysfunction (Damasio and Maurer 1978).

Finally, the development of speech and language make an important contribution to prognosis. Mutism, in particular, has a poor prognosis for infantile autism; a more favourable prognosis is associated with the development of useful speech by the age of five. Further, the more developed the demonstrable language at initial assessment and the greater the evidence of 'inner language', as reflected by the creativeness of play, the better the prognosis (DeMeyer *et al.* 1973). However, even in the more intelligent older child with good speech development, one can often identify residual difficulties with rhythm and repetitiveness of speech and of language (Rutter *et al.* 1967).

3. *Deafness*

Because deafness is rare and because the deaf infant sporadically babbles, the defect may remain unsuspected and undiagnosed in early infancy. Thereafter, parents may be alerted because of a poor expressional response to sudden meaningful noises, or the condition may be detected from about nine months of age using developmental screening tests. Further, moderate deafness is a potentially hidden or masked handicap: any unusual behaviour might be attributed to intellectual handicap or emotional disturbance (Freeman 1975).

Likely causes of profound deafness can be identified by careful investigation of the history: at least one-third of cases are considered to have a genetic basis, and a substantial number have significant organic causes. Further, depending on the source of the sample, between one-third and two-thirds have associated handicaps, which are particularly common where deafness has a perinatal rather than a genetic basis (Freeman 1975). These include such conditions as serious postnatal middle-ear infections and neonatal hyperbilirubinaemia. Recent work has revealed that hearing-impaired children suffered more than the usual number of minor childhood fevers, and also major infections during the first five years of life: there is a step-wise increase in the incidence of such conditions from the normal-hearing to the part-hearing to the profoundly deaf child (Fundudis *et al.* 1979).

In contrast, epidemiological studies usually do not reveal excesses of social and family pathology in families of deaf children (Fundudis *et al.* 1979). However, as far as parental attitudes are concerned, the picture appears to be that parents are more supportive and less critical of their deaf children than is usual with normal children, and these parents tend to be more protective (Freeman 1975, Fundudis *et al.* 1979) and more closely supervise their children's activities (Schlesinger and Meadow 1972). Their management is also more strict, particularly in the case of the profoundly deaf (Fundudis *et al.* 1979). Further, family disorganization and social problems are no more frequent in the families of hearing-impaired children (Freeman 1975).

In a brief review it is possible to touch on only some of the complex theoretical issues concerning speech, language and intellectual development of deaf children (Moores 1972, Meadow 1975). Profoundly deaf children's mode of speech and language development or acquisition is different from that of normal-hearing children, and may also differ for different deaf groups, according to the modes of communication used by their supervisors. The difficulties of language acquisition which deaf children encounter include not only more superficial language skills such as speech and speech reading, but also 'inner language' abilities (Meadow 1975). Despite such differences, there is evidence that the rules of language learned by deaf children in their earlier years are similar to those of hearing children (Meadow 1975). An indication of the grave extent of the speech handicaps suffered by deaf children comes from a Department of Education and Science survey (Great Britain: DES 1964) which revealed that most profoundly or moderately deaf school-children of normal intelligence have unintelligible speech.

There are many published reports to show that childhood deafness hampers the development of language and verbal abilities, but not necessarily that of non-verbal abilities (*e.g.* Wiley 1971, Fundudis *et al.* 1979). More recent work suggests that the more adversely affected the child's hearing, the greater will be his disability, not only on verbal but also on non-verbal and educational tests.

The deaf are not a homogeneous group, and their division into part-hearing and profoundly deaf subgroups reflects this distinction: cognitive impairments of the part-hearing are relatively moderate compared with the profoundly deaf (Fundudis *et al.* 1979). Further, non-verbal ability has a normal distribution among the deaf who do not have other handicaps (Furth 1966, Freeman *et al.* 1975). However, the comparatively poorer performance on cognitive tasks appears to improve with age and appropriate stimulation. There is also evidence that the academic achievements of deaf children, particularly progress in reading, are poorer than those of hearing children (US Office of Demographic Studies 1973, Conrad 1977). Further, while it might be expected that deaf children would tend to be reliant on reading for the expansion of their knowledge, the evidence is that they read fewer books than their hearing counterparts (Fundudis *et al.* 1979). Such facts are consistent with the majority of deaf children being 'functionally illiterate when they leave school' (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 have better educational achievements than deaf children of hearing parents (Meadow 1968, Vernon and Koh 1970), which suggests that the early introduction of non-verbal or manual methods of communication is beneficial.

The two main educational traditions in teaching profoundly deaf children are the oral and the manual. Those advocating the oral method maintain that manual methods of communication will inhibit the development of speech. The contrary view of advocates of manual forms of communication is equally unacceptable. These dogmatic views are unsupported by research and condemn children to limited forms of communication and opportunities for learning. The oral and manual methods have now been replaced by *total communication*, which includes oral and variations of

sign language tailored to the needs of the individual child. Dogmatic standpoints are likely to have done more harm than good, resulting in the loss of countless opportunities for incidental learning (Freeman 1975).

Research findings usually report that although hearing children are better adjusted socially and behaviourally than deaf children, the majority of the latter group are not psychiatrically disturbed. However, in the case of profoundly deaf children, antisocial disturbance often reveals itself in school (Williams 1970, Fundudis *et al.* 1979). Other symptoms commonly reported, such as impulsiveness in adolescence (Altshuler *et al.* 1976) and irregularity in temperament (Fundudis *et al.* 1979), may simply reflect variations of this disorder. Other workers point to social immaturity (Myklebust 1964) and poor skills in social relationships, including susceptibility to teasing and bullying (Rodda 1970, Fundudis *et al.* 1979). While the balance of evidence suggests that the severity of the behaviour disorder is related to the degree of hearing loss (Peckham *et al.* 1972, Fundudis *et al.* 1979), certain workers attribute the behavioural problems of deaf children to frustration as a result of speech and language difficulties (Levine and Wagner 1974). It is noticeable that deaf children raised in families in which other members are deaf often prove to be better adjusted than those raised in families in which other members are not deaf (Myklebust 1964).

Organic brain-damage may cause deafness and also contribute to the poorer intellectual and educational performance of such children. However, it is far from easy to estimate the extent of this contribution.

4. *Mutism*

There are a number of forms of mutism, which conveniently can be divided into those with a presumed physical basis and those considered to be psychological. The first includes that type of mutism which may be associated with profound deafness, serious mental handicap, autism or akinetic mutism.

There are two types of psychological mutism, both of which are rare and have a dramatic presentation. *Traumatic mutism* is said to follow an acute psychological or physical shock. Some consider it to be an hysterical phenomenon and fictional literature suggests that it is common, but a clinical survey suggests that in fact it is an extremely rare condition (Kolvin and Fundudis 1981).

Elective mutism is the term coined by Tramer (1934) to describe a fascinating condition in which talking is restricted by the child to a small group of intimates and familiar situations, the most common of these being the child's home. This, too, is a rare condition and is distinguished from the excessive shyness which occurs relatively frequently in reception classes in schools (Wright 1968, Brown and Lloyd 1975) by its severity and its persistence. Confusion may arise with infant-school children who do not speak when first attending school (Brown and Lloyd 1975), but over 90 per cent of these improve spontaneously over the first school year. Kolvin and Fundudis (1981) consider that only the residual small percentage of children not speaking for as long as a year after starting school would be similar to those children clinically diagnosed as elective mutes. Previously, there was a tendency to emphasize the importance of motivation in diagnosis, which was accompanied by suggestions that abnormalities in speech or language precluded the diagnosis of elective mutism. However, such earlier views were

based on anecdotes: 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 and 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 onset of speech, an excess of developmental mispronunciations, EEG immaturity and associated problems of speech, bowel and bladder function (Kolvin and Fundudis 1981), poorer non-verbal intelligence, a high rate of associated behavioural problems and certain adverse temperamental traits.

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). Others suggest that there may be an important maturational component which slowly diminishes with age. On the other hand, there are many published reports of single or small-group studies in which a psychodynamic basis is postulated, such as a faulty mother-child relationship (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 (Kolvin and Fundudis 1981). In addition, more than double the number of parents of elective mute children attended psychiatric clinics than did those of the control children. With the diversity of factors which could contribute to the aetiology, it is likely that the origins are multifactorial and the condition heterogeneous.

Finally, follow-up reveals that less than 50 per cent of elective mutes subsequently improve, which suggests that it is a more intractable disorder than most of the other non-organic, non-psychotic psychiatric disorders of childhood (Kolvin and Fundudis 1981).

SOME ENVIRONMENTAL FACTORS

The part played by environmental factors in the development of speech and language has been exaggerated by the behaviourists (Skinner 1957). This extreme viewpoint has been rejected by Chomsky (1969), who postulates the presence of an innate specific mechanism—a 'language acquisition device'—which determines the structure of all human languages. Lenneberg (1966) gives support to the latter theory by arguing persuasively that the emergence of speech and language is most easily accounted for by maturational changes which are only moderately affected by abnormal factors in the child's environment. For instance, children of deaf parents were found to babble appropriately and to develop speech adequately, despite being brought up in a grossly abnormal linguistic environment. In a more recent review, Fundudis *et al.* (1979) concluded, as had Bruner *et al.* (1966), that it is unlikely that narrow, extreme theories will eventually prove to have anything more than moderate validity and that wider multifactor theories are likely to explain much more. Bearing these qualifications in mind, it will be helpful here to give some indication of what has emerged from research.

Bernstein (1962) has found, despite controlling for intelligence, that when comparing middle-class children with working-class children he is able to distinguish an

elaborated code, mainly used by the middle classes, which is characterized by a wide syntactical repertoire to express ideas and by complex grammatical sentences of good syntactical construction. The working class use a more *restricted* code. Some see this as a continuum rather than an absolute distinction. Bernstein emphasizes usage—that working-class children simply use the elaborated code less. As to mechanisms, it has been suggested that middle-class mothers are more specific and informative in talking to children and answering questions, and in turn their children are more specific in their use of language (Rackstraw and Robinson 1967, Brandis and Henderson 1970). However, such wide and clear-cut differences are not always found, which gives rise to questions about the method and context of such research.

There is also evidence that impoverishment of stimulation, particularly prolonged institutionalization during infancy and early childhood, has an adverse effect on language development and verbal intelligence (Brodbeck and Irwin 1946, Provence and Lipton 1962). More recent research suggests that the critical factor is not institutionalization, but rather the quality and intensity of communication between the child and his caretakers. For instance in institutions where the quality of care is good there are few adverse effects (Tizard *et al.* 1972) and similarly, Kibbutzim children are not affected (Kohen-Raz 1968).

The Newcastle population studies provide support for the view that social-class factors have a less severe effect than was previously believed (Morley 1965, Fundudis *et al.* 1979). There is also evidence to suggest that such effects are at least partially reversible by appropriate help (Rutter and Mittler 1972). Further, even serious impoverishment of the social environment appears to give rise only to moderate degrees of impaired vocalisation (Provence and Lipton 1962), speech delay and language retardation. The effects are confined mainly to language expression rather than to comprehension (Klaus and Gray 1968), but there is also retardation in educational performance.

There is also evidence that other environmental factors such as large family size and multiple birth are associated with relative delays in language development: Rutter and Mittler (1972) offer the explanation that this is due to the insufficiency of clarity and quality of adult-child interactions and communication in these situations.

Assessment

The assessment of speech and language disorders is complex, because of their diversity and because such disorders may involve paediatrics, neurology, psychiatry, psychology, linguistics, education and sociology. Nevertheless, there are some useful guides in the literature which indicate that a *preliminary formulation* and *differential diagnosis* can usually be made on the basis of relatively simple but systematic assessment (Rutter 1977).

Biography can provide information not only about familial factors, including familial deafness, speech delays and psychosocial influences, but also about relevant physical factors such as cerebral damage or a history of clumsiness, which may suggest cerebral palsy. In-depth interviews of the parents will also provide evidence of previous parental suspicions and anxieties which often may have been ignored and thus delayed the diagnosis (Freeman 1975).

Observation is central to all other forms of assessment. Most clinicians, even while gathering biographical information, will observe whether the child uses clues from facial expression or gesture in communication, responds to verbal or gestural instructions from parents, or displays the clumsiness of gait typical of the child with cerebral palsy. Much can be learnt about the child's speech, hearing and behaviour if he remains in the consulting room during the history-taking. It may be necessary to observe the child in his own home, *i.e.* his natural habitat.

Hearing should always be assessed, the clinician checking whether the child responds to a wide range of auditory stimuli, including alerting to sudden or unusual noises and responding to 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 specialised techniques.

Speech and language in their various forms

Speech may be delayed or defective (deviant). For instance it 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 across a number of modalities, *e.g.* spoken, sign (manual) or written. Language *comprehension* can be tested by checking the ability to understand simple commands, both with and without 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, such as a toy tea-set or telephone, and by the constructiveness and creativity of the child's play (Rutter 1977).

If there is *profound deafness* the child is unlikely to attend to, or respond to, auditory stimuli, but may use gestures extensively and attempt conversation when older; there may also be evidence of constructiveness in play. He is often particularly attentive to facial expression. In many respects the child with the most severe form of a *developmental speech/language* disorder will have a similar pattern, and indeed there may be only limited evidence of 'inner language', but usually there will be evidence of normal hearing. Again, the child is likely to be attentive to facial expression. In milder forms of developmental delay there is usually little suggestion of deafness but, instead, 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 tends not to respond to any form of verbal or non-verbal communication; and usually does not respond to his mother's facial expression. There is little indirect evidence of language, in terms of gesture, mime, imitation or play. Children with *mental handicap* usually have no evidence of deafness, but rather have limited language abilities and a delay in the development of articulation, together with other evidence of slowed development.

Imaginative and constructive play and imitation. Evidence of imaginative constructive play reflects the presence of 'inner language' and augurs well for the growth of language and development in general. Gesture, imitation and play constitute a form of communication which is impaired in infantile autism and in severe language retardation. Constructive and imaginative play may be good and reasonably advanced in deaf childen and those with developmental language delays, but it is likely to be more restricted in children with infantile autism and mental handicap.

Cognitive ability. Accounts from parents about the child's social abilities and play, combined with clinical observation, provide only a rough guide to the child's intellectual level. 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). Not infrequently, children with speech delay may be mistakenly diagnosed as mentally handicapped because they are assessed by intelligence tests which rely heavily on verbal tasks. The most useful picture of the child's cognitive assets and deficiencies may be obtained from testing for specific skills (Berger and Yule 1972). Even if formal testing is not possible, a social maturity scale which employs evidence from the mother or caretaker may be profitably used to provide a crude estimate of intellectual level.

Social and general behaviour. Descriptions by parents are usefully complemented by direct observation, which may provide hints about the basis of the speech delay. Useful signs may comprise the manifold social and behavioural abnormalities of autism, the absence of startle to loud noises or alerting to domestic noises, the anomalies of social behaviour when in the company of peers (such as occurs in the young deaf child), and social withdrawal and a tendency to avert the face in the case of the electively mute.

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