Part I
A Study of
Speech Retarded Children

1 Early development, types and prevalence

Introduction

There have been few systematic follow-up studies of children with speech retardation (Beckey, 1942; Morley, 1965; Fiedler *et al.*, 1971; Sheridan, 1973; Butler *et al.*, 1973). Even fewer have focused on a specific speech milestone and subsequently attempted to ascertain its usefulness as a 'screen' criterion or to study subsequent speech and other defects shown by the children who are retarded on this milestone. This was the main theme of the current follow-up which investigated children about seven years old who had shown speech retardation at the age of three. It has been pointed out (Butler *et al.*, 1973) that seven years is a convenient age for assessment of speech and language defects because by then most of the developmental mispronunciations have disappeared spontaneously, and those that remain are either intrinsically serious or have serious psychosocial implications.

Method

General

A survey (by Newcastle Child Development Study) of the entire population of children born in Newcastle upon Tyne provided an opportunity of studying longitudinally a full sample of speech retarded children. As part of the study, health visitors had recorded the developmental milestones of these children. We defined speech retardation as failure to use 'three or more words strung together to make some sort of sense' (Neligan and Prudham, 1969) by the age of 36 months. In this way a cohort of speech retarded children was identified. Admittedly

this criterion is crude and arbitrary but it has the merit of being an objective, simple and standard way of recording developmental milestones. It is in fact a crude screening technique which has to be followed by intensive assessment and diagnosis. Using a total population sample avoids the selection bias which besets clinic and hospital studies. It also enabled us to study prevalence taking two factors into account:

(a) Although the size of the population (3300 children) was not large enough to produce reliable prevalence figures for relatively rare disorders, nevertheless, it provides a rough guide to the frequency of such disorders.
(b) Using a symptom as an ascertainment criterion does not ensure complete coverage of the disorders which it identifies. This is because all children with such disorders do not necessarily have a speech delay of the severity defined by our criteria so that the prevalence figures reported provide only a conservative estimate.

Speech retarded sample

We decided to restrict the study to those children whose parents were still living in Newcastle when the child was aged three. Of the 133 thus identified from the Newcastle Maternity Study records we found that by the age of five years:

(a) 21 cases had left the area (15·7%);
(b) eight cases (6·1%) had to be excluded from detailed analysis because of inadequate or limited data because of hospitalization, being in care, twins (due to delayed computerization of twin records) or because parents refused to co-operate;
(c) two children had died (1·5%);
(d) there were 102 remaining children (76·5%).

Matched controls

The 102 remaining children were matched with normal controls on three criteria—age, sex and postal district. The matching was an administrative exercise utilizing population records. The subsequent assessments were undertaken 'blind'. However, in subsequent analyses involving comparison of subgroups with controls the individual matching was not maintained—the total control group was used for every comparison.

Testing

A series of psychological, educational and behavioural tests were administered (T.F.) which will be described later. In addition, speech and audiometric assessments (E.S.) and clinical assessments (I.K.) were undertaken. The mean age of the speech retarded group was seven years six months at the first psychological assessment and eight years four months at the second.

Diagnosis and classification (Table I)

The initial screen was essentially to identify children who were speech retarded. The subsequent diagnostic assessment at seven years identified those speech, language and other defects of which speech retardation is a symptom. This showed clearly that the cases could be divided into two broad groups.

The *first group* consisted of those whose functioning, intellectually, psychologically or physically, was so abnormal that they were termed *pathological deviants*. Such cases fall into three relatively well-defined clinical groups and this is the basis of our operational classification.

(a) *Marked intellectual impairment* This was defined as an IQ at or below the 1st percentile on the WISC or where the child was untestable. In practical terms this means an IQ of 65 or below. This is possibly too rigorous a criterion as other authors have used a criterion of 2 standard deviations below the mean, i.e. an IQ of 70 or below (Yule and Rutter, 1970a).

(b) *Specific clinical syndromes* This included children with severe communicating disorders of childhood, such as elective mutism (Salfield, 1950; Brown *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 includes children with cerebral palsy.

These categories were not intended to be mutually exclusive: some children showed features typical of all three categories, and were then classified 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 group* consisted of children who, after clinical examination at the age of seven, did not fall into the pathological category and hence have been described as the *Residual Speech Retarded Group*. More sophisticated

psychological assessments were necessary to categorize the various characteristics of this group (Chapter 3).

The next problem was to reconcile this crude operational classification with a clinical classification of speech and language disorders. We decided to model our classification on the work of Ingram (1959 a,b, 1969, 1972). His is essentially a functional clinical classification and we have modified it both by abbreviation and simplification to suit our research as follows:

(a) *Dysarthria/cleft palate*—disorder of speech sound production with demonstrable dysfunction or structural abnormalities of tongue, lips, teeth or palate.
(b) *Secondary speech disorders*—disorder of speech sound production associated with other diseases or environmental factors:

(i) mental defect
(ii) hearing defect
(iii) true dysphasia (acquired)
(iv) adverse environmental factors
(v) psychiatric disorders

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

(c) *Specific developmental speech disorders*—the developmental speech disorder syndrome:

(i) mild (dyslalia)
(ii) moderate (developmental expressive dysphasia)
(iii) severe (developmental receptive dysphasia, word deafness)
(iv) very severe (auditory imperception, central deafness)

Ingram (1972) sees the developmental speech disorder syndrome as a descriptive label given to children with retardation of speech development, who are otherwise apparently normal in respect of their health, intelligence and home backgrounds. The features described by Ingram are: (i) apparently healthy children; (ii) average or superior intelligence; (iii) normal home backgrounds; (iv) high male–female ratio; (v) positive family history of slow speech development; (vi) an excess of ambidexterity and left-handedness in the first degree relatives; (vii) relatives who often have had difficulties in the early stages of learning to read and write (McCready, 1962; Brain, 1965).

In the past it has been assumed that, if a child has a unitary milestone

delay and is in other ways not obviously abnormal, he will grow out of it. Such delays have been described by Rutter and Yule (1970) as representing 'extreme variations in normal development' which are 'related to the continuing growth and maturation of the brain'. Ingram, as described above, has stated what he considered to be the salient features of the developmental speech disorder syndrome. We consider that the following are some of the salient features of developmental delays (partly derived from Rutter): (i) they are more likely to occur in an isolated functional area rather than affect a wide range of milestones; (ii) they apparently are less likely to occur in younger children and children of lower mental age; (iii) they often occur where there is no evidence of structural abnormality of the brain; (iv) they tend to clear spontaneously as the child grows older; (v) boys are much more frequently affected than girls; and (vi) they may be influenced by environmental factors (Tizard, 1964).

Ingram (1972) points out that the label 'developmental speech disorder syndrome' is really a misnomer as the category comprises a heterogeneous group of articulatory and language disorders and in certain cases the speech development is not only retarded but is deviant as well. He finds it useful to regard this category as a spectrum of disorder which varies from the mild to the very severe. The mildest are the dyslalias which are defined as 'retardation of acquisition of word sounds but with normal language', i.e. the articulatory development of affected children is retarded. They are described by their parents as understanding the words but being unable to say them. Ingram also

Table I *Classification and distribution by sex*

Category speech retarded	*Total*	*Male*	*Female*
	102	65	37
1 Pathologically Deviant	18	9	9
(A) Marked intellectual handicap			
(i) Subnormality mainly	7	4	3
(ii) Subnormality plus other conditions	13	7	6
(B) Cerebral palsy	5	3	2
(C) Specific syndrome			
(a) Autistic	2		2
(b) Electively mute	2	1	1
(c) Dysphasia	1	1	
(d) Cleft palate/dysarthria			
(i) Cleft palate/dysarthria alone	1		1
(ii) Cleft palate plus other conditions	2	1	1
2 Residual Speech Retarded	84	56	28

reports that the child substitutes or omits the later acquired consonants and consonant clusters inconsistently 'though his vocabulary and grammatical structures in spoken language may be within normal limits'. Children with moderate developmental speech disorders have normal comprehension but more severe retardation of word sound acquisition and retardation of development of spoken language. The severely affected children have greater degrees of retardation of word sound acquisition, impaired development of spoken language and impaired comprehension of speech. Synonyms for these three degrees of severity are 'dyslalias', developmental expressive dysphasia and developmental receptive dysphasia, respectively.

From Table I it will be seen that included in our pathological deviant category are Ingram's dysarthrias and secondary speech disorders. Our 'Residual Speech Retarded' Group comprised the remainder of the children and one could argue that these fall into the developmental speech disorder syndrome provided we widen Ingram's inclusive criteria to cover as well the dull range of intelligence and without stipulating a normal home background. It remains to be seen whether characteristic features of the syndrome can be indentified clinically or statistically.

Some background factors

Social class and speech retardation (Table II)

The breadwinner's occupation was coded in accordance with the Registrar General's Classification of Occupations (Great Britain, 1951). The speech retarded group's family social class tended to be lower than that of a random sample of Newcastle children (Neligan *et al.*, 1976). This is broadly in accord with the findings of other speech studies (Beckey, 1942; Morley, 1965; Butler *et al.*, 1973). However, as the control group in our study had been matched for postal district, we had expected that the controls and the speech retarded group would have had a similar distribution of social class; this was confirmed though the speech retarded group still showed a slight downward trend. The explanation which seems most plausible is that, even within urban areas or neighbourhoods, there is a relationship between child handicap and social class factors.

Sex differences

Rutter and Tizard (1970) report that there is a tendency for biological and perinatal hazards to occur more frequently among young males

than females, with the inevitable consequence that a higher number of boys than girls are subsequently handicapped. This may be the basis for the greater vulnerability of boys to handicap, but is certainly not the whole answer (Ounsted and Taylor, 1972).

In most studies of developmental disorders, male:female ratios in the order of 2:1 to 3:1 are usually described. While the population sex ratio in Newcastle (1·04:1) approaches unity (Neligan *et al.*, 1976) that of our speech retarded group was 1·7:1.

When the Residual Speech Retarded Group and the pathological deviants are analysed separately, the ratio is 2:1 for the former, and 1:1

Table II *Social class distribution*

Social class	Controls (C)	Total speech retarded (T)	Residual speech retarded (R)	Random sample (RS)[a] $n = 208$
I + II	8 (7·9%)	5 (4·9%)	5 (5·7%)	9%
III	63 (61·8%)	53 (51·9%)	45 (53·5%)	61·0%
IV + V plus	31 (30·4%)	44 (43%)	34 (40·2%)	29·5%
Total	102 (100% approx.)	102 (100% approx.)	84 (100%)	(100%)
Statistic Chi-squared		C *vs* T = 3·81 2 d.f: NS	C *vs* R = 2·1 2 d.f: NS	T *vs* RS = 6·1 2 d.f. $p < 0·05$ R *vs* RS = 3·4 2 d.f. NS

NS = Not Significant
[a] From Neligan *et al.* (1976)

for the latter. This leads to the preliminary conclusion that the sex ratio of the former group resembles that described in developmental disorders.

We wondered whether there would be any change in the sex ratio in the Speech Retarded Group if we took into account whether they were early or late walkers and we found that the ratios were 1·7:1 and 2·1:1 respectively. In other words, the ratio does not change substantially when we separately analyse early and late walking speech retarded children.

In brief, as already described, separate analysis of the pathological deviant group and of the Residual Speech Retarded Group gave male:female ratios of 1:1 and 2:1 respectively, suggesting that the high male:female ratios reported in the literature (Butler *et al.*, 1973) are

probably more related to developmental delays than to disorders with an organic basis.

Milestones and the specificity of speech retardation

The other signs of developmental retardation are of considerable import. We were anxious to find out whether the milestone delay in children in the Residual Speech Retarded Group was specific to speech or general in nature.

There are a number of ways of tackling the above question but we confined ourselves to attempting to identify a group of children with speech delay which was not associated with a delay in beginning to walk.

As previously stated, our criterion for speech retardation was failure in using three-word sentences by 36 months. This corresponds to the 97th percentile based on Neligan and Prudham's (1969) norms for developmental milestones (based on the total population of births in Newcastle upon Tyne over a three-year period). We defined walking retardation as walking later than the 90th percentile (using Neligan and Prudham's norms)* which in practice meant grouping of children according to whether they were walking before or after 16 months. A child was therefore considered to have a general delay in milestone achievement if he was retarded in speech and in addition had not walked unsupported by 16 months. Next, we identified a group of children who were retarded in speech but walked early, i.e. had a specific speech delay. For this purpose early walking was defined as walking at or below the 25th percentile (according to Neligan and Prudham's norms), i.e. at or before 12 months. The above method identified three groups:

(a) Specific speech delay, i.e. a group of 25 children who walked early but were speech retarded. Using the above criteria the minimal rate is about eight per 1000 children.
(b) An intermediate group of 34 (who were speech retarded but walked by 16 months).
(c) General milestone delay, i.e. a group of 23 children who walked late and also were speech retarded. Using the above criteria the minimal rate is again about eight per 1000 children.

* Neligan and Prudham's percentile norms are presented in reverse of the more usual form; the 97th percentile would, therefore, presumably have read as the 3rd percentile, and so on, in the more customary form.

Two children who proved to be deaf were excluded from this classification (see p. 16 and Chapter 3).

Other milestones

From Table IIIa it will be seen that the Residual Speech Retarded Group achieved bladder control and unsupported walking later than the controls, but this was statistically significant for walking only. However, it will be noted that these statistics do not take into consideration the

Table IIIa *Milestones—three-year-old data*

	Controls	Residual speech retarded	Pathological deviant	Control *vs* RSR group
Bladder dry by day n =	94	72	7	
m (in months)	23·8	25·8		NS
s.d.	5·9	5·8		
Not yet dry n =	8	12	11	
Walking unsupported n =	102	84	10	[a]
m (in months)	13·4	14·5		
s.d.	2·1	3·7		
Not walking n =	0	0	8	

[a] $p<0{\cdot}01$.

higher percentage of the Residual Speech Retarded (14%) who were not yet dry by day at three years of age compared to the controls (8%). While the mean age of walking of the controls is close to Neligan and Prudham's (1969) 50th percentile, that of the speech retarded group is closer to their 75th percentile level. This means that our Residual Speech Retarded Group is, on average, less retarded in walking than in speech. Some may interpret these findings as reflecting uneven maturation while others may interpret them as an expected regression to the mean. Further, it will be noted that the pathological deviants' other milestone development (bladder control) is seriously retarded.

Laterality

Faulty cerebral dominance has been implicated as the basis of developmental language disorders (Orton, 1937, 1934). However, in a recent

survey of the literature (Rutter and Yule, 1970a), it was found that reports of excess of left and/or mixed laterality in speech retarded children tend to be highly contradictory with 'as many reports of negative findings as of positive findings'. This is, of course, to be expected in that most of the previous studies have been clinic or hospital ones. The same is true of studies of reading retarded children where high rates of left or mixed-handedness have not been found even when the study was epidemiological rather than hospital-based (Belmont and Birch, 1965; Douglas *et al.*, 1967; Rutter and Yule, 1970a).

The current research provided further opportunities for studying such questions. As shown in Table IIIb, there are no significant differences in

Table IIIb *Dominance and laterality at age of eight*

	Controls	Residual speech retarded
Hand preference (Harris) Right dominance	68%	62%
Hand–foot dominance discrepancy	52%	51%
Hand–eye dominance discrepancy	40%	49%
Left–right differentiation difficulty	19% (19 out of 99)	37%[b]

[b] $p < 0{\cdot}01$.

terms of right, mixed or left-handedness between the controls and the Residual Speech Retarded Group; nor were there any significant differences in the number of right, mixed or left-eyed children between the groups. The only significant differences concerned the ability to differentiate between the right and left sides of the body; the speech retarded children were significantly worse in this respect ($p < 0{\cdot}01$). This important finding is re-examined in relation to intellectual developments in Chapter 3. These somewhat surprising findings provide unequivocal evidence that an unselected sample of children with delayed speech development show no more left hand or eye preference or inconsistencies of preference (mixed handedness or eyedness) than the normal population. It remains to be seen whether different types of classification may lead to the emergence of associations which have been masked by the lack of conceptualization. This question is examined in subsequent chapters.

Perinatal factors

The mean gestational ages of the pathological deviant group and also the Residual Speech Retarded Group were significantly shorter than

that of the controls. This finding is similar to that described by Butler *et al.* (1973) in children with speech defects. However, unlike the Butler study, we did find significant differences in birth weight between our total speech retarded group and the controls. The only other perinatal index which revealed differences was that of the second stage of labour which proved significantly longer in our total speech retarded group than the controls.

Birth order

Fewer of the speech retarded children were first-born. While this achieves statistical significance (Table IV) the absolute figures are not as

Table IV *Sibling order*

Position	Controls		Residual Speech Retarded Group	
	n	%	*n*	%
Eldest/only	35	35	15	19
2nd or 3rd	41	41	48	57
4th or 5th	20	20	14	17
6th or more	6	6	7	9

Significance–Chi-squared = 8·0, 3 d.f, $p < 0{\cdot}05$.

impressive as in the National Child Development Study (Butler *et al.*, 1973). On the other hand, the mean family size reveals a trend for the Residual Speech Retarded to come from larger families (mean of Residual Speech Retarded Group = 4·35; of controls = 3·37).

Prevalence findings

We have already noted that 31 of the original 133 cases were not available for full testing at school age. Such losses constitute a potential source of bias and hence it is important to know how far the fully tested group can be regarded as representative of the total cases identified as being retarded in speech at the age of three years. Fortunately, information gathered in the perinatal period and in the first five years of life was

contained in the records of the Newcastle Child Development Study (Neligan *et al.*, 1974). We confined ourselves to checking basic characteristics where fuller information was likely to be available. The characteristics chosen were occupational social class of breadwinner and the rate of serious handicap in the children. The distribution of the occupational social class of the families of these 31 children proved to be slightly better and the rate of serious handicap no greater than in the group available for assessment (with the exception of the two children who had died). We therefore concluded that those children who were not seen were unlikely to differ significantly from those assessed at school age.

When calculating prevalence rates it is necessary to make appropriate adjustment for such losses. Our formula for this consists of multiplying the rate of disorder by a factor of 1·30.

Prevalence of speech retardation at the age of three

At the age of three years some 4% (133) of 3300 children had retardation of speech reported by health visitors. This is a lower percentage than that described in the 1000 family study (Spence *et al.*, 1954; Morley, 1965) where a 6% retardation is described at the same age using broadly similar criteria in a random sample of 114 children. However, it is likely that these slight differences can be accounted for both in terms of the definitions of incomplete sentences (simpler in our study) and the fact that the data in the 1000 family study was derived from an examination by speech therapists. It is of importance to note that in the 1000 family study about 1% were still using incomplete sentences just before starting school.

Disorders at follow-up: prevalence and comment

Of the 102 speech retarded children studied at the age of seven, 18 (17·6%) fell into the pathological deviant category. The subcategories in Table I are not intended to be mutually exclusive and hence the sum of the frequencies in these is greater than the total number of cases.

(a) *Pathological deviants*

(i) *Infantile autism* A rate of two to four per 10,000 children has been described by Lotter (1966) in his Middlesex survey. Our rate of 0·8 per 1000 is broadly consistent with Lotter's rate. This is of considerable

importance as there has been some speculation about whether the rate reported by Lotter is only applicable to the more affluent southern areas of England. The above figures suggest this rate is true for both the north and the south and for both affluent and industrial areas of the country.

(ii) *Elective mutism* We recorded only two 'nuclear' electively mute children (Tramer, 1934) with an inordinate and selective shyness of strangers severe enough to persist into the seventh year of life. Browne (1963) feels that elective mutism is more common than is generally believed. However, the frequency is dependent on whether it is broadly or narrowly conceived and defined. Indeed, norms for shyness relative to age would enable a clearer distinction to be made between unusual shyness which occurs frequently in the pre-school period (Kolvin and Nolan, 1978) and the pathological shyness which occurs in elective mutism. We have used the latter, more narrow definition and therefore consider 0·8 per 1000 as a minimum prevalence rate particularly as there remains the theoretical possibility that electively mute children could have slipped through our screen at the three-year-old stage. Appropriate enquiries were made to ascertain if any cases had been referred to colleagues, both for the purpose of this study and a specific study of elective mutism (Kolvin and Fundudis, 1979) but no further cases were uncovered. Elective mutism would therefore appear to be as rare a syndrome as infantile autism.

(iii) *Dysphasia* Similar problems of definition confound frequency and prevalence studies of childhoold dysphasias. Some use both verbal behaviour and presumed aetiology as criteria whereas others use verbal behaviour alone (Ingram and Reid, 1956; Morley, 1965; Lenneberg, 1967; Eisenson, 1968). Even though one can theoretically make the distinction between an acquired dysphasia (Ingram, 1972) and the moderate or severe form of the specific developmental speech disorder syndrome (Ingram, 1972), which some would label as developmental expressive or developmental receptive dysphasia respectively, in practice we found the distinction difficult because of the partly retrospective nature of the diagnostic exercise. In fact, we uncovered only one case which could be included in the clear-cut severe dysphasic category. This represents a rate of 0·4 per 1000 children. The prevalence of serious and persisting language disorder has been estimated as approximately 0·7 per 1000 in Scotland (Ingram, 1963) at the age of five, and 0·8 per 1000 on the Isle of Wight (Rutter *et al.*, 1970a). MacKeith and Rutter (1972) point out that there are no reliable figures for persistent disorders involving a defect in language comprehension, and estimate that the rate is very much less than that for infantile autism. Our figures support the suggestion that the condition is as rare or even rarer than infantile autism.

(iv) *Cleft palate/Dysarthria* Morley (1965) reports one case of cleft palate per 1000 births and we found two which represents a rate of 0·8 per 1000.

(v) *Deafness, hearing impairment* Deafness is one of the major causes of delay in language development (Morley, 1965). It has been estimated (Pless and Graham, 1970) that about two per 1000 children have deafness severe enough to merit the use of hearing aids. But the rate in the case of profound deafness is even lower than this. The latter estimate is supported by our own two cases with profound hearing loss—a rate of 0·8 per 1000. Additionally another two, though not profoundly deaf, were in special educational settings for children with multiple handicaps. This leads to a rate similar to those reported by Barton *et al.*, (1962), Reed (1970) and Neligan *et al.* (1974) in the major Newcastle survey of which this study forms a part. (As the two cases presenting with profound deafness and language retardation showed only minimal signs of other handicaps we decided for the purposes of this study not to include them in the pathological deviant group in our subsequent analyses.)

We defined hearing impairment as hearing loss of 20 decibels or more on at least four out of the seven frequencies tested in each ear. Any loss of less than 20 decibels was considered to be within the normal range, provided that there was no high frequency loss. Details of audiometric assessments are presented in Chapter 5. Hearing loss of 20 decibels or more occurred in 15% of the 80 children in the Residual Speech Retarded Group, which is not significantly higher than the controls where it occurred at a 9% rate. This is, however, double the rate reported by Anderson (1967) but we do not consider the findings of these two studies comparable because of the different definitions. Such rates are predictably lower in a survey than in clinic populations—for instance, Morley (1965) reported that over one-third of 280 children referred to her with delayed speech development had 'insufficient hearing'.

(vi) *Cerebral palsy* The cerebral palsy rate (Mackeith and Rutter, 1972) is two to three per 1000 (Ingram, 1955; Rutter *et al.*, 1970). If cases with severe intellectual handicaps are excluded the rate falls to one per 1000 who have, in addition, language retardation. Our rate for a combination of cerebral palsy and speech and possible language retardation is about 2·0 per 1000. This closely approximates the rate reported by Neligan *et al.* (1974) in the major survey of which our study forms a part.

(vii) *Intellectual handicap* The single most common cause of slow speech development in paediatric clinics is mental handicap (Ingram, 1972). Ingram points out that this is to be expected as it has been shown in a number of studies (Illingworth, 1966) that while motor milestone in a significant proportion of intellectually handicapped children may be

within normal limits 'milestones of speech, adaptive and social behaviour development are invariably achieved late. But even so the degree of retardation of speech development is not always proportional to the severity of mental defect in later life.' Finally, Ingram (1972) points out that intellectually handicapped children tend to have a high degree of environmental deprivation and a conspicuous proportion of hearing defects compared to children of average intelligence.

The rate of handicap is dependent on the definition employed. Rutter *et al.* (1970a) defined it as an IQ of less than 70, and reported that over 2½% have intellectual handicap and that over half of these show severe language impairment or articulation defects or both. Our criterion was more rigorous, i.e. an IQ of 65 or below (1st percentile or below) using the WISC performance norms. We decided to use these norms because it was considered invalid to use a verbal criterion in a group of speech retarded children. When we included cases of autism and cerebral palsy who have severe intellectual handicap, we found that some 13 children had both severe intellectual handicap and speech retardation. The rate, therefore, is about 5·1 in 1000. If we employ the criterion of a performance IQ of 70 or less then the numbers marginally increase to 14 and the rate becomes 5·5 per 1000. An analysis of our data (see Chapter 3) reveals that if we had used the full scale IQ as a criterion the picture would have dramatically changed. The use of the performance IQ as a criterion was based on clinical experience where at times it can be demonstrated that some children with poor speech and language abilities can function better on non-verbal tests (Rutter, 1972). Research on autistic children (Rutter and Lockyer, 1967) has provided evidence of the predictive importance of performance tests. However, Rutter (1972) points out that such findings need to be validated on other language disordered populations.

(b) *The Residual Speech Retarded Group and its relationship to the developmental speech disorder syndrome*

It will be remembered that these comprise the remainder of the children who did not fall into the category of being 'pathologically deviant'. If we make no qualification about intelligence or home background (provided we exclude children with severe intellectual handicap) these children could be considered as falling into Ingram's specific developmental speech disorder syndrome. The prevalence rate is then in the vicinity of 3% of children of school age.

Questions still remain about the severity of the disorder: how many children are mildly affected, having retardation of acquisition of word sounds, but with normal language (dyslalias), and how many children are severely affected, with retarded spoken language development

(dysphasias). Such difficulties are underlined by Mittler (1970), who points out that there is considerable disagreement about the difference between 'the mere late development of language and some kind of specific developmental disorders of the dysphasic type'. Further, language comprehension normally precedes language production (Fraser *et al.*, 1963; Lovell and Dixon, 1967) which leads mothers of even normally developing children to comment that their children can understand a great deal before they can speak. By the same token, children with developmental disorders may have a normal understanding of language before they can talk (Lenneberg, 1962). From clinical experience this is particularly true of the 'dyslalias' and developmental expressive dysphasias where, in the latter, the discrepancies can be particularly marked. Obviously such discrepancies, though helpful in diagnosis, can hardly be considered specific to the dysphasias.

Clearly, diagnosis undertaken after the age of seven cannot easily identify which children were dyslalic and which dysphasic, as the disorders mostly correct themselves (Morley, 1965). However, more sophisticated psychological testing theoretically could reveal a cluster of cases with a pattern similar to that obtained with dysphasic children (Olson, 1961). This will be the subject of a subsequent chapter.

Fluency and articulatory ability at the age of seven

Disorders of word sound production can occur in the dysarthrias, with defective sensory input, and in the developmental speech disorder syndrome. We studied articulation using the Edinburgh articulation test, which led to quantitative assessments of the child's articulatory skills. (In consultation with Mr Pellowe, of the Linguistic Department of the University of Newcastle upon Tyne, we modified the test slightly to accommodate local dialectical variation.) This test (Anthony, 1971) provides norms only up to the age of $5\frac{3}{4}$ years. Obviously such norms are not applicable to our children. On the other hand, it appeared reasonable to use this test as a means of obtaining standardized measures of articulation for comparison of age-matched groups of children. In Table Va it will be seen that the Residual Speech Retarded Group have a significantly lower mean score of correct items than the controls. The incorrect items were further analysed using a modification of Anthony's (1971) qualitative assessment system. She describes five categories of errors and we have varied this using the following three categories only: (a) almost mature; (b) immature (including very immature); and (c) atypical substitutions. The atypical errors were rather infrequent in both groups, but the most striking example of it occurred in the one pathological deviant case who was

diagnosed as being clearly dysphasic. As shown in Table Va the mean number of immature errors was significantly greater for the speech retarded group. From Table Vb, which provides a distribution analysis of these immature errors, it can be seen that some 70% of the speech retarded group (as compared to 35% of the controls) had six or more immature errors. These findings again emphasize the importance of following up the total sample of children to ascertain their later speech development.

Table Va *Assessment of speech at seven years using Edinburgh articulation test*

		Controls (n = 100)	Residual speech retarded (n = 80)	Statistical significance
Correct score	m	59·5	52·5	p <0·01
	s.d.	8·6	12·4	
Immature errors of articulation	m	4·9	9·9	p <0·01
	s.d.	6·2	7·6	

Table Vb *Distribution immature errors of articulation*

Range	Controls	Residual speech retarded
0–5	65 (65%)	24 (30%)
6–14	26 (26%)	40 (50%)
15+	9 (9%)	16 (20%)

Sig. Chi-squared = 21·9, 2 d.f., p <0·01

Using the Andrews and Harris scale (1964) we found one mild stammer in each group. Andrews and Harris' work suggested that about 3–4% of children of school age stammered for a period, but in only about 1% of children was this more persistent. Our findings appear to be in accord with the latter figure as our assessment of stammer was based on a conversation with a child at one point in time. There is a suggestion in the literature (Andrews and Harris, 1964; Ingram, 1972) that stammering is more common in children who have been delayed in language development. As our sample was rather small it is difficult to be absolutely sure, but it does not provide evidence that stammering is more common in children who have previously been speech retarded.

Concluding comments

Our results emphasize the predictive value of a simple speech screen at the age of three years. About one in five of the 4% of all children aged three years who were speech retarded were later found to have serious language, intellectual or physical handicaps. It is also important to note that almost half of these handicapped children were found to be functioning rather poorly in what appeared to be inappropriate educational settings. This underlines the value of an early screening exercise (Butler *et al.*, 1973) in identifying children with handicaps who need help, for both accurate diagnosis and appropriate placement. Such a screening procedure could be applied quite easily by a health visitor or a general practitioner who could then refer the identified children to the appropriate paediatric clinic. Findings from the national study (Butler *et al.*, 1973) support the view that it would be preferable to institute screening procedures before the age of seven.

Some might argue that screening at the age of three years would pick out too many 'false positives', comprising subsequently spontaneously remitting cases of developmental speech delay. However, as yet, there is inadequate information about how this group of children function intellectually, educationally, behaviourally and socially at older ages. This is the subject of later chapters in the book. What must be emphasized is that what happens at three does not reflect the total position at five, six or seven years, so that a comprehensive screening programme should include periodic re-screening over the first five to seven years of life.

We have put forward arguments to support the theory that the Residual Speech Retarded Group can be considered as falling into the developmental speech disorder category. However, while the distribution of certain factors, such as the sex ratio, support this view, othejs, such as laterality, do not. Further, it is evident that even if most cases can be, by a *tour de force*, included in a so-called developmental speech disorder category, this category is clearly heterogeneous (Ingram, 1972). For instance, our research has demonstrated that there are at least two subgroups—one with general delay of milestones and one with a specific delay of speech. In a subsequent chapter we examine the profile of features found in the above subgroups and those reported in the literature which contribute to the syndrome of developmental dysphasia.

Finally, we have to consider the influence of adverse environmental factors (McCarthy, 1954; Bernstein, 1961, 1962). We have not, in fact, tried to separate a group of children suffering from sociocultural retardation, mainly because such a procedure implies that once a group of children has been separated environmental influences subsequently do not adversely affect the remaining children. We believe instead that

there is a continuum of adverse environmental factors; in a subsequent chapter we will attempt to quantify these factors and to compare their frequency in our speech retarded group with our control group.