CHAPTER 5

A Follow Up of Speech Retarded Children

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Introduction

There have been few follow-up studies of children with delay of speech development which have attempted to assess outcome in a systematic and comprehensive manner. A unique opportunity to carry out this type of longitudinal follow-up occurred as a result of the Newcastle Child Development Study (Neligan *et al.*, 1974). In order to achieve this objective a series of preliminary simple steps were essential.

First, a definition of speech retardation: we decided to use the simple definition employed by the health visitors in the above study – *the failure to use three or more words strung together to make some sort of sense by the age of thirty-six months*. Admittedly this criterion is crude and arbitrary but it has the merit of being an objective, simple and standard way of recording a developmental milestone; it is, in fact, a crude screening technique which has to be followed by intensive assessment and diagnosis. Second, to select groups of children from a total population and to examine them at specified ages and by appropriate methods in order to identify the significant differences in their development. If such findings were to have more general validity it was essential for us to define the relationship between our study population and the total population from which it was drawn so that relevant

*Abstracted from *Speech Retarded and Deaf Children: Their Psychological Development* (1979) (T. Fundudis, I. Kolvin and R.F. Garside, Eds.)

comparisons could be made and conclusions drawn by workers located elsewhere. Using a total population sample avoids the selection bias which besets clinic and hospital studies.

The Newcastle Survey of Child Development enrolled survivors of the first month of life born in Newcastle during the years 1960–1962 and our baseline was their 1962 cohort. This study gave us information, which has been collected by midwives, health visitors, doctors and teachers, about the children's first five years of life. This covered perinatal, obstetric and social data, and also information about their health and development. Descriptions of the population and other aspects of the study are provided in two previous monographs (Neligan *et al.*, 1974; Neligan *et al.*, 1976). Using a total population enabled us to study prevalence taking two factors into account:

(a) Although the size of the population (3,300 children) was not large enough to produce reliable prevalence figures for relatively rare disorders, nevertheless, it provides a rough guide in an area where there is often little information.

(b) Using a symptom as an ascertainment criterion does not ensure complete coverage of the disorders which it is meant to identify. 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.

The aim of the study was to obtain a comprehensive picture of the intellectual, behavioural and physical functioning of children at school age with an earlier history of speech retardation. 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 implications.

The progress of the children with speech delay was compared with that of a matched control group. The latter consisted of children who did not suffer from speech delay and who were matched individually with our index cases on three criteria – sex, age and family neighbourhood. Of the 3,300 children born in Newcastle upon Tyne in 1962, 133

were identified as speech retarded. This constitutes 4 per cent of the population. Of these, 102 were studied more intensively when they were 7–8 years old.

Attrition

Thirty-one of the original 133 cases were not available for full testing at school age. Such losses constitute a potential source of bias so it is important to know how far the fully tested group are representative of the total cases identified as being retarded in speech at the age of three years. The distribution of the occupational social class of the families of these 31 children proved to be slightly higher 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.

The initial screen was essentially to identify children who were speech retarded. The subsequent diagnostic assessment at 7 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.

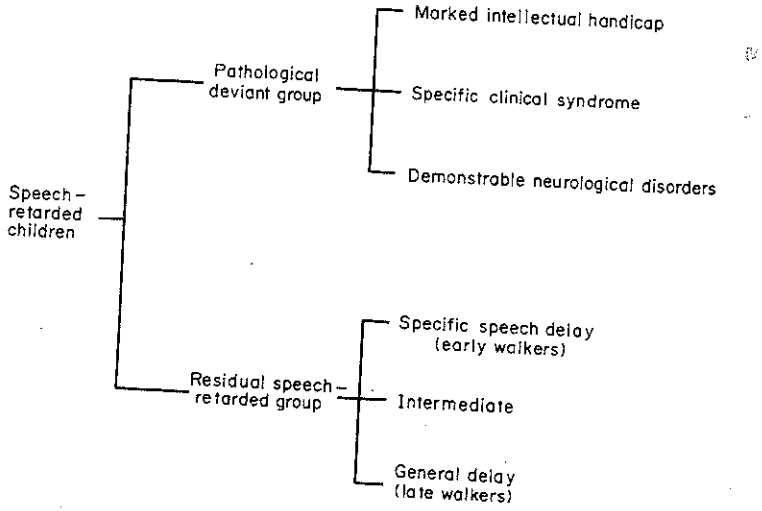
The *first group* consisted of those whose functioning, intellectually, psychologically or physically, was so abnormal that we described them as pathological deviants. Such cases fall into three relatively well-defined clinical groups:

(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 meant 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. IQ of 70 or below (Yule and Rutter, 1970).

(b) *Specific Clinical Syndromes:* This included children with severe communication 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 spastic disorders. These three categories were not intended to be mutually exclusive. For the purposes of this brief presentation 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 group* consisted of children who, after clinical examination at the age of seven years, showed no evidence of serious handicap. We have labelled them the residual speech retarded group. More sophisticated psychological assessments were necessary to delineate their characteristics.

A further clinical classification was undertaken. This was dependent on whether these children suffered from speech retardation alone or

whether they suffered from retardation of both speech and walking. In short, we identified three subgroups: 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. In a later section we provide details of this classification.

The next problem was to reconcile the crude initial 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* – 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 therefore 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 insult at the age of two to three years there is likely to be both impairment of language and thereafter slowing of speech development.

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.

Ingram (1972) also 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 deviant as well. He finds it useful to regard this category as a spectrum of disorders 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 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 languages. 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.

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. 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 identified clinically or statistically.

Milestones and the Specificity of Speech Retardation

As previously stated, our criterion for speech retardation was a delay in using three-word sentences by thirty-six months. This corresponds to the third percentile based on Neligan and Prudham's (1969) norms for developmental milestones. We defined walking retardation as onset of walking occurring below the tenth 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 yet 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 onset of walking occurring above the 75 percentile (according to Neligan and Prudham's norms) i.e. at or below 12 months. The above method identifies three groups:

(a) Specific developmental 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 8 per 1,000 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 8 per 1,000 children.

Some Background Factors – Residual Speech Retarded Group

(i) Sex ratio

While the population sex ratio in Newcastle approaches unity (Neligan, Scott and Kolvin — personal communication) that of our speech retarded group was 1.7 boys to 1 girl. This is roughly consistent with the ratio reported in most studies of developmental disorders

where the ratio of boys to girls is in the order 2:1 to 3:1.

When the residual speech retarded group and the pathological deviants are analysed separately, the ratio is 2:1 for the former, and 1:1 for the latter. This leads to the preliminary conclusion that the sex ratio of the former group resembles that described in developmental disorders. Rutter *et al.* (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 subsequently are handicapped. In addition there is a greater vulnerability of boys to environmental stress (Neligan *et al.*, 1976.)

(ii) *Perinatal factors*

The mean gestational ages of the pathological deviant group and also the residual speech retarded group were significantly lower than that of 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 birth weight than did the controls.

(iii) *Birth Order*

The mean family size reveals a trend for the residual speech retarded to come from larger families (chi-squared = $p < .05$). In addition it was found that fewer of the speech retarded group were first born.

(iv) *Other milestones*

We also found that the residual speech retarded group achieved bladder control and unsupported walking later than the controls, but this was statistically significant for walking only. This means that our residual speech retarded group is, on the average, less retarded on walking than on speech. Possible explanations for this are provided elsewhere (Fundudis *et al.*, 1979).

(v) *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 in the number of right, mixed or left-eyed children between the group. The only significant differences concerned confusion in differentiating between the left and right side of the body; the speech retarded children were significantly worse in this respect.

Faulty cerebral dominance has been incriminated as the basis of developmental language disorders (Orton, 1937; 1934). However, in a recent survey of the literature (Rutter *et al.*, 1970), 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 not unexpected, as most of the previous studies have been mainly clinic or hospital populations.

Prevalence of Speech Retardation at the Age of Three

At the age of three years we found 4 per cent (133) of 3,300 children had retardation of speech as reported by health visitors. This is a lower percentage than that described in the 1,000 Family Study (Spence *et al.*, 1954; Morley, 1965) where a 6 per cent retardation is described at the same age using broadly similar criteria. It is of importance to note that in the 1,000 Family Study about 1 per cent were still using incomplete sentences just before starting school, but 4 per cent remained unintelligible at time of entry into school.

Prevalence of Disorders at Follow-up

Of the 102 speech retarded children studied at the age of seven 18 fell into the pathological deviant category. For simplicity the cases in the sub-categories are presented as mutually exclusive in the first column in Table 1. The second column contains overlap cases and hence the sum of frequencies is greater than 18 (see Table 1).

TABLE 1
Incidence

Pathological deviant group			
(categorised according to predominant disorder)			n = 18
		Predominant	Total
A.	Marked intellectual handicap alone	7	13
B.	Cerebral palsy	5	5
C	Specific syndromes:		
	Autism	2	2
	Elective mutism	2	2
	Severe dysphasia	1	1
	Cleft palate dysarthria alone	1	2
Residual speech-retarded group			n = 84
(includes two deaf children with no other demonstrable handicaps)			
		Total	n = 102

Pathological Deviant Group

(i) *Infantile Autism:* We found two cases with characteristic autism.

(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. Elective mutism would therefore appear to be as rare a condition as infantile autism.

(iii) *Dysphasia:* Problems of definition confound frequency and prevalence studies of childhood dysphasia. Some use both verbal behaviour and presumed aetiology as diagnostic 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 part retrospective nature of the research diagnostic exercise. In fact, we uncovered only one case which could be included in the clear-cut severe dysphasic category. Our figures therefore support the suggestion that the condition is as rare or even rarer than infantile autism.

(iv) *Dysarthria:* While Morley (1965) reports one case of cleft palate per 1,000 births, we found two per 3,300 children.

(v) *Deafness, Hearing Impairment:* Deafness is one of the major causes of delay in speech and language development (Morley, 1965). It has been estimated (Pless and Graham, 1970) that about 2 per 1,000 children have deafness severe enough to merit the use of hearing aids, but those with severe hearing loss are even less than this. We found 2 profoundly deaf children and a further 2 with less severe forms of deafness but with associated 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 only profound deafness and language retardation showed little in the way of other signs of handicap, we decided for the purposes of analysis not to include them in the pathological deviant group.

(vi) *Cerebral Palsy:* Our 5 cases of cerebral palsy gives rise to a rate which closely approximates that reported by Neligan *et al.* (1974) in the major survey of which our study forms a part.

(vii) *Intellectual Handicap:* We found 7 cases of marked intellectual handicap alone although some 13 children had both severe intellectual handicap and other associated handicaps. The rate, therefore, is about 5.1 in 1,000. 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 1,000.

The Residual Speech Retarded Group

(i) *Prevalence*

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 (1972)

specific developmental speech disorder syndrome. This gives rise to a prevalence rate of 2 to 3 per cent of children of school age.

(ii) *Cognitive, Language and Educational Development*

We have made no attempt in our study to use the newer linguistic concepts or to explore factors such as the rules of grammar and language competence (Chomsky, 1969). Like Mittler (1970), we believe that these measures are still clinically in their infancy and need highly specialised skills for their application. Instead, we have used traditional clinical psychological measures such as the Illinois Test of Psycholinguistic Abilities (Kirk *et al.*, 1968, revised edition), together with a wide range of other standard measures for assessing language development.

The residual speech retarded group scored significantly poorer than the *controls* on all the *cognitive* (intelligence, perceptual, conceptual and visualmotor 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 wider intellectual impairment. On the other hand, it may well be that the residual speech retarded group is not really a homogeneous group and therefore the above analyses may have masked patterns of abilities specific to particular subgroups. We therefore divided the residual speech retarded group according to the classification previously described. In summary, our groups consist of early walkers who we have labelled the specific speech delayed group, late walkers who we have labelled the general delayed groups and an intermediate group whose walking milestones fell between the two extremes.

An analysis of the cognitive and language development of each of these three groups at the age of seven years reveals that the functioning of all of them is depressed in relation to the controls (Table 2). First, the early walkers (i.e. the group with a specific delay in speech) have significantly poorer verbal IQ, language ability and educational achievements compared to the controls. In contrast, however, their

non-verbal IQ 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). The latter group not only perform poorly on all the above measures but also score significantly poorer on performance IQ than do the controls.

TABLE 2
Cognitive functioning at follow up

Tests	Controls = C	Residual speech-retarded: Early walkers = E	Inter walkers = I	Late walkers = L	C vs. E	C vs. I	C vs. L
Verbal							
IQ – WISC	93	85	84	81	1%	1%	1%
Non-verbal							
Performance IQ – WISC	101	99	98	87	N.S.	N.S.	1%
Language							
ITPA-Quotient	91	84	82	76	1%	1%	1%
Achievements							
Reading Quotient Schonell	94	82	81	76	1%	1%	1%

A similar pattern was found on other cognitive measures which we have used and details are available elsewhere (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.

Discussion

Awareness of the long-term sequelae of speech retardation is of paramount importance. There is evidence (Morley, 1965) 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. Further, Butler *et al.* (1973) point out that by the age of 7 years most developmental mispronunciations have disappeared spontaneously. Such apparent 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 (Ingram, 1963; Mason, 1967), in their follow-up of speech retarded children in primary schools, report that the majority have reading difficulties. In addition, Rutter (1972) points out that to read and "to understand the meaning of what he reads . . . a child must have language skills". He accordingly 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.

Summary

Our results emphasise the predictive value of a simple speech screen at the age of 3 years. About 1 in 5 of the 4 per cent of all children aged 3 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. What must be emphasised, however, is that what happens at 3 does not reflect the total position at 5, 6 or 7 years, so that a comprehensive screening programme should include periodic re-examination over the first 5 to 7 years of life. If the extreme group (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 will function normally in most respects. Our follow-up study at school age of previously speech retarded children reveals widespread impairments and in this way expands on the findings of other workers (Ingram and Reid, 1956; Mason, 1967) who have shown that a high percentage of such children later develop educational disabilities. In this chapter we describe some of the residual cognitive, language and educational deficits of these children. A comprehensive account of cognitive, speech, language, educational, physical, social and behavioural outcome is provided elsewhere (Fundudis *et al.*, 1979).

Acknowledgements

Details of acknowledgements are provided elsewhere. We would, however, like to acknowledge our main co-authors — Mrs. G.S. George, Dr. H.I.J. van der Spuy, Mrs. J.S.H. Nolan and Mrs. E. Scanlon. In addition we are indebted to Mrs. M. Blackburn for administrative and secretarial help.

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The Family and Social Origins of Antisocial Behaviour

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Introduction

The 1940s saw two landmarks in the attempts to unravel the psychosocial origins of antisocial behaviour and delinquency in childhood: the first consisted of clinical research and linked separation from mothers with juvenile delinquency (Bowlby, 1946); the second was based on multivariate analyses and this gave rise to a suggested association between different types of antisocial behaviour with different kinds of family patterns (Hewitt and Jenkins, 1949). Both, while preliminary, were seminal exercises which generated a multitude of questions and provoked widespread research. Subsequent advances were dependent on more precise definitions, diagnosis and the development of classifications which had a sounder scientific basis.

Description and Definition

Mischievous behaviour in childhood is not uncommon and is of greater concern to parents and teachers than to the children themselves. Nevertheless, only a small proportion of such behaviour is sufficient to impair relationships with peers or adults or is seen as contravening social norms. Such disorders of conduct include behaviour which is non-delinquent such as bullying, lying, disruption in classroom, etc. and which may also include truancy and stealing. Inevitably, such behaviour will overlap with what is viewed officially as delinquent behaviour. Delinquency is a common and major problem, especially amongst disaffected youth of the inner neighbourhoods of industrialized cities. It is one variety of conduct disorder in which the children enact potentially illegal behaviours, with some of the acts being known to legal agencies, while others are known only through self-reports (Farrington, 1973).

In this chapter we confine ourselves to school-age delinquent and non-delinquent conduct disorders. As to diagnosis, there is general agreement about the range of symptoms which are subsumed under the category of conduct disorder such as lying, stealing, truancy, disobedience, destructiveness, aggression, poor relationships and wandering. But there has been a lack of agreement about the

level or threshold of severity and duration of such symptoms (Offord *et al.*, 1986). The question arises as to when the constellation of symptoms should be considered as a disorder. The answer is, if the behaviour handicaps the child himself or his social or family environment. Furthermore, such behaviours may reveal themselves in either the *home* (1) or the *school* (2), or *both* (3) with the former (i.e. 1 or 2) being labelled *situational* and the latter (i.e. 3) *pervasive*.

Classification

One of the main subclassifications of antisocial behaviour disorders derives from multivariate approaches; factor analysis of child behaviour data invariably has demonstrated emotional and conduct disturbance patterns (Peterson, 1961; Wolff, 1971; Kolvin *et al.*, 1975). Such patterns emerge at all ages but it is only the classic endeavour by Hewitt and Jenkins (1949) that gave rise to a distinction between the so-called unsocialized aggressive behaviour and socialized group delinquency. This distinction has formed the basis of the scheme employed in the DSM-III, which defines a conduct disorder as a persistent pattern of conduct in which the basic rights of others and also major age-appropriate societal norms or rules are violated. Criteria for diagnosis include disturbance of at least six months' duration. In the DSM-III, a number of broad categories of conduct disorder are described. The first is the *under-socialized aggressive type*. This includes manifestations of such behaviours as physical violence against persons or property and theft outside the home. In addition, there is failure to establish a normal degree of affection, empathy or bond with others, as evidenced by no more than one of five indicators of social attachment (for instance, one or more lasting peer group friendships). The duration of such conduct disorder is defined as at least six months. The behaviour of youths over 18 can be labelled as conduct disorder provided that the behaviour does not meet the criteria for antisocial personality disorder.

The *second* category is conduct disorder of the *under-socialized non-aggressive type*. The diagnostic criteria consist, first, of repetitive and persistent patterns of non-aggressive conduct: these include chronic violation of a variety of social rules, repeated running away from home overnight and consistent serious lying and stealing not involving confrontation with the victim. Secondly, there is a failure to establish a normal degree of affection, empathy or bond with others. Thirdly, again there is a duration of at least six months.

A *third* category consists of conduct disorder, of the *socialized aggressive* type. In some ways, this overlaps with the previous category, but the main features are a repetitive and persistent pattern of aggressive conduct. On this occasion, there is evidence of social attachment to others as indicated by at least two of five patterns relating to social attachments.

The *fourth* category consists of conduct disorders that are *socialized non-aggressive*. This again overlaps with the others and has three main diagnostic criteria: first, a persistent pattern of non-aggressive conduct; secondly, evidence of social attachment to others; third, duration of at least six months; the youth must be

below the age of 18 if the conduct disorder is to fulfil the criteria for antisocial personality disorder.

There is a *further* category of atypical conduct disorder that is a rag-bag of antisocial behaviours which cannot be included under the specified subtypes of conduct disorder.

Unfortunately, the validation of the above classification has proved difficult, so it is not surprising that the DSM-III-R suggests modifications—for instance conduct disorder is subdivided into three types: (1) a group type, in which most of the above behaviours occur in the company of peers; (2) a solitary, aggressive type, in which there is predominantly aggressive behaviour that is initiated by the child or adolescent alone; and (3) an undifferentiated type, for those children or adolescents with a mixture of clinical features that are not easily classified into any previous two subcategories. However, in addition, conduct disorders are assessed on a scale running from mild through moderate to severe, according to the number of problems in excess of those needed to make the diagnosis (i.e. over three) and the degree of general handicap or actual physical injury that is inflicted on the environment. It is not surprising that these amendments have been made, as Hewitt and Jenkins had based their research on a clinical population that was heavily weighted for delinquency. However, the details of these distinctions have not been widely replicated (Field, 1967).

The ICD-9 offers a variation of the above classifications (Rutter *et al.*, 1976). It defines conduct disorders as those mainly involving aggressive and destructive behaviours, and disorders involving delinquency. It is used at any age for abnormal behaviour that is not part of any other psychiatric condition. To be included, the behaviour needs to be frequent and severe, and it must be abnormal in its context. Such disorders differ from personality disorder in the absence of deeply ingrained maladaptive patterns of behaviour present from adolescence or earlier. The ICD-9 describes four subcategories: unsocialized disturbance of conduct; socialized disturbance of conduct; compulsive conduct disorder; and finally mixed disturbance of conduct and emotions.

The relationship of attention-deficit disorders and hyperkinetic disorders to conduct disorders merits clarification: there is, undoubtedly, a degree of overlap with conduct disorders but longitudinal research (Schacher *et al.*, 1981) suggests differences in outcome that would support such a distinction.

However, there is as yet no good evidence that non-delinquent and delinquent conduct disorders can be differentiated adequately in terms of behavioural features, family and social background and outcome (Moore *et al.*, 1979). Nor, indeed, is there as yet good evidence to support the division of conduct disorders into those with and without aggression. Nevertheless, the strength of the work of Hewitt and Jenkins was that behavioural patterns were found to be associated with family patterns similarly derived from multivariate analysis: unsocialized behaviour correlated with broken homes and family rejection; socialized delinquency correlated with parental neglect and social disadvantage, rather than rejection. But so far this work has not been replicated or validated adequately.

Origins

The origins of delinquency have been of central interest to psychologists and sociologists alike. Sociologists have been interested in the delinquent subculture and the fact that delinquents tend to be concentrated in the poorer areas of the inner city—areas with their own 'subculture'. One of the main advocates of the sociological theories which abounded in the mid-1950s stated that it was not sufficient to understand the delinquent subculture in negative terms as due to disorganization or even culture conflict, but rather that the delinquent feels that he has an inferior status in the wider community and when he meets others who are similarly afflicted, he acquires a new sense of self-esteem which allows the rejection of the values of a wider community (Cohen, 1956). One of the influential views advanced was one which suggested that delinquent acts reflected frustration generated by inequality in material goods. Other theories suggested that living in disadvantage gave rise to a belief that different social norms were permissible within a delinquent subculture (Cloward and Ohlin, 1960).

In summary, such theories suggest that the origins of delinquency reside mainly in endeavours to compensate for the influences of society that deny them equality in the social and material sense. Joining groups allows individuals to identify with group values and thus satisfy their needs by illegitimate means (West, 1985). West points out that such theories have become highly politicized.

On the other hand, one of the psychological explanatory theories advances the view that it is important to distinguish between the individual who has a psychological motivation or basis for becoming delinquent and the so-called 'sociologic delinquent' (Johnson, 1959). However, there has been a reaction against this position by those who consider that such theories lend support to the tendency to label those who are social rebels as psychologically ill or deviant.

Earlier research concentrated on single-factor theories, seeking factors which seemed to be of predominant importance; these included maternal separation, family criminality and other family patterns. However, the specificity of identified patterns has not been validated and further there is a consensus that this approach was far too simplistic. More often than not it has been found that there are multiple factors operating simultaneously; and it has proved difficult to pull apart those variables that usually act in concert (Rutter and Wolkind, 1985). Thus in the last decade the trend has been to appreciate that few factors operate in isolation and to view delinquency as being multifactorially determined and to attempt to highlight the relative importance of these family and parental factors. It is these themes that this chapter addresses. They include personality and criminality; the possibility of a genetic basis; social deprivation and inequality; family disruption; inadequate or weak family relationships; questionable quality of care; questionable child-rearing practices; and, finally, the question of models of conduct.

Family criminality

There is a striking link between a child's delinquency and parental criminality or social deviance (Farrington and West, 1981, Offord, 1982); for instance, 40% of

the sons of recidivist fathers also proved to be recidivist (West, 1985). These are important studies but need to be supplemented by twin and adoption studies when trying to explore the possibility of genetic influence (Shields, 1977; Hutchings and Mednick, 1977; Bohman, 1978): such studies report only a small genetic influence regarding juvenile delinquency. However, it would seem that parental criminality is not the decisive factor, as the association applies as well to persistent parental personality and social difficulties, often represented by drinking problems and a poor work record. In these circumstances, it is not surprising that more recent reviews suggest that both heredity and environment influence delinquency and that genetic factors act by making the individual more vulnerable to adverse environmental influences.

Disadvantage and poverty

Conduct disorders, including delinquency, occur more frequently in the lower social strata (West and Farrington, 1977). At least a moderate link is often reported between low social status and delinquency: the Newcastle Study (Kolvin *et al.*, 1988, 1990) reveals a close relationship between offences and occupational status of the family breadwinner, rising from 5% of males from Class I to 26% from Class III and 42% from Class IV and V and lower strata. In their review of the evidence, Rutter and Giller (1983), conclude that the relationship resides mainly at the extremes of the social scale; they question, as well, the extent to which the differences can be accounted for in terms of the differential attitudes to treatment of working class juveniles by the legal system. However, the Newcastle figures relate to more serious delinquency where there is less possibility of variations in legal management and hence such associations are less likely to be explicable in terms of attitudinal and management differences by legal agencies. Furthermore, an even stronger association is found for family deprivation and delinquency, which suggests at least some direct effect of adverse family influences. Indeed, this research links antisocial behaviour more closely with environmental deprivation than with social class (Kolvin *et al.*, 1989).

The question therefore arises of whether poverty has a direct bearing on deliquency (Rutter and Giller, 1983). They note that the major increase in the standard of housing this century does not seem to have been accompanied by a corresponding reduction in crime. However, this is a superficial observation as it would appear that more people are living in poor conditions than are exhibited in the face of official statistics and that community expectations have risen with changing patterns of experience (Townsend, 1979). These factors, taken together, suggest that it is inequality in income rather than overall level of wealth, that may predispose to crime.

Deprivation

This gives rise to the question of the relationship between deprivation and offending. Such issues need to be examined in longitudinal studies. In the Newcastle Study, the proportion of males who offended varied according to the

degree of deprivation, ranging from one in six males from non-deprived families to six in ten from families in multiple deprivation. Thus, offending appears to be 'dose-related', i.e. related to the degree of severity of family deprivation in the pre-school years. The above research suggests that some forms of family deprivation appeared less harmful than others; for instance, the criminality rate was relatively lower in the case of parental illness than where there was poor quality of parenting and poor care of the home and children. Moreover, there were significant correlations between the two latter criteria of deprivation, which implies that they are not independent of each other and it is not easy to estimate the relative importance of such explanatory factors in the genesis of delinquency.

Family and parenting variables

A wide number of parenting variables correlate with delinquency. The question arises as to which of these are causal or are most of them a reflection of a third factor common to all (Rutter and Giller, 1983)? With the complexity of such family interactions, care must be taken to avoid *a priori* assumptions regarding the significance of any particular variable. A good example of this is *family size*, where West (1985) revealed that 42% of persistent delinquents came from families with more than seven children. However, as Rutter and Giller (1983) have pointed out, the fall in the fertility rate over the past two decades does not seem to have induced a corresponding fall in crime, which suggests that any assumed link between large families and delinquency is indirect. The crucial link may not be the family size itself but associated factors such as overcrowding, socioeconomic disadvantage, less intensive family interactions, poor child discipline and supervision—all of the above may be underpinned by a lack of parental skills, foresight or planning.

Other factors can be examined in a similar way (Rutter, 1985). For instance, at one time, *family disruption or broken homes* were seen as central to delinquency, but Rutter (1971) quite rightly pointed to the alternative explanation, that the underlying determinant may be pre-existing and continuing family discord and quarrelling. Rutter and Giller (1983) advance evidence that divorce and separation are strongly associated with delinquency, whereas death is only weakly associated. They also point out that discord in both broken and unbroken homes appears to be strongly associated with delinquency and conclude that discord is more important than family breakdown.

They also go on to pose the question of why divorce is still associated with the continued increased risk of delinquency and point out that divorce does not necessarily mean resolution of marital conflict (Wallerstein and Kelly, 1980). Thus, children are particularly vulnerable if they live in homes where divorce has been preceded by seriously discordant family relationships (Rutter and Giller, 1983).

Family relationships

One key theoretical issue has been the subject of the effects of separation from parents on a child (Bowlby, 1946, 1951, 1969). Rutter (1980) suggests that it is

the context and circumstances of separation rather than the mere fact of separation that are important—it is the quality of the relationship prior to the separation as well as the quality of care subsequent to the separation. Disturbances of emotions and behaviour are more evident when separations arise because of family discord and disruption (Rutter, 1971). Thus, the persistent effects of separation stem from the prior chronic family discord rather than from the separation experience, *per se*; or it could be a consequence of the subsequent poorer quality of child care (Yarrow and Klein, 1980). In addition, admission to foster care is associated with an increased rate of psychiatric disorder when such admissions occur with children from disharmonious and disadvantaged families (Wolkind and Rutter, 1973). A single admission to hospital has not been shown to be related to later psychiatric difficulties, but when there are multiple hospital admissions (and such circumstances are more likely to be related to family disadvantage), then there is a significant increase in subsequent conduct disorder and delinquency (Quinton and Rutter, 1976; Douglas, 1975). A major question is the subject of affectionless psychopathy, originally described by Bowlby (1946, 1951). More recent evidence concerning this derives from Roy (1983), Wolkind (1974), Tizard and Tizard (1971), Tizard and Reese (1975) and Tizard and Hodges (1978), all relating to institutionalized children. The balance of evidence from the above researches implicates disturbed social behaviour of the exposed children which, although different from the controls, especially in their school settings, does not amount to the type of behaviour originally described as affectionless psychopathy. This has given rise to the conclusion that early lack of bonding experiences did not inhibit these children from making true attachments in later childhood but did appear to influence other social relationships (Rutter and Wolkind, 1985). This brings us back to the question of attachments and relationships which are crucial issues in child psychiatry and psychology.

There is good evidence (Campos *et al.*, 1983; Sroufe, 1983) that securely attached infants are more sociable with adults and show greater competence with peers, more positive affect and a higher self-esteem (Rutter and Wolkind, 1985). There remains the issue of whether the quality of attachments in infancy predicts adult relationships and adult psychological functioning. In a review of the evidence, Rutter and Wolkind (1985) point out that discordant family relationships in early life, including a lack of parental affection, are associated with a greater likelihood of emotional and personality disorders in adult life (Brown *et al.*, 1986; Quinton and Rutter, 1985; Wolkind and Kruk, 1984). Furthermore, at any point in time, an individual's social behaviour is likely to reflect both current social circumstances and previous social experiences (Rutter and Wolkind, 1985).

Moving away from attachments in early life, Rutter (1985) points out that weak family relationships are often found to be associated with delinquency. He goes on to point out that it is important to make a distinction between family discord and weak family relationships. One setting in which there is a lesser amount of discordant and quarrelsome interactions is institutional; but here the children are less likely to form close attachments with their care-takers. All the evidence suggests that conduct disorders are commoner amongst children reared from infancy in such institutional settings (Rutter *et al.*, 1983; Wolkind, 1974).

Rutter therefore concludes that 'Weak family relationships are important in their own right, quite apart from their association with discord'. It is not only that children who lack secure attachments in infancy are going to have impaired social relationships in adolescence, but also that the development of internal controls appears to depend on an affectional identification with parents. Evidence in support of such notions is moderately strong.

One of the more influential theories about the origins of delinquency was Hirschi's (1969) control theory which asserts that lack of attachment to parents, peers and school gives rise to a lack of conventional attitudes and eventually juvenile delinquency. Aspects of this theory have been examined in a number of large-scale studies in senior schools the United States utilizing self-administered, self-report delinquency measures and usually self-report measures of parent–child relationships. Three important findings emerge. First, closeness to father (rather than to mother) may be a better predictor of delinquent behaviour, especially amongst males (Johnson, 1987). Second, that parental and school attachment, conventional beliefs and involvement in youth subculture activities contribute to only low levels of variance of delinquency (Wiatrowski and Anderson, 1987). However, this poor explanatory power of the data in the last study may well be due to an insufficiency of variation of delinquent behaviour, especially at extremes when using a population of high school sophomores. For instance, when combining high school and juvenile correctional institution data Hirschi's model was only supported when delinquent companions were included as an additional explanatory factor in the causal path analysis (Thompson *et al.*, 1984). Furthermore, the latter authors contend that the findings are more consistent with a social learning than a control theory. However, irrespective of whether the findings are construed within a control or a learning theory model, the contribution of positive attachments in protecting children from delinquent behaviour is again confirmed.

Quality of parenting

Another theme concerns quality of parenting. The Newcastle research highlights the importance of poor-quality parenting, especially poor mothering (Kolvin *et al.*, 1990). However, it is not only quality of care that is likely to be important: parental supervision has a particularly strong influence as well (Farrington and West, 1981; Patterson, 1982). West and Farrington (1973, 1977) speculate that poor parental supervision and the associated freedom from restraint, together with police practice, are contributory factors in the determination of levels of delinquency, with poor family and social circumstances playing some part, however marginal, in police decisions to prosecute. Patterson (1982) suggests four ways in which the behaviour of children may be influenced by family rearing patterns: there may be, first, no clear expectations of acceptable child behaviour; second, a lack of monitoring of child behaviour; third, a lack of strategies for following through disciplinary plans in a logical way; fourth, lack of problem-solving techniques in the face of family crises. Thus, some families do not appear

to know what their children are doing and their disciplinary management techniques are not very efficient (Rutter, 1985).

Models of behaviour

Here, the implication is that parents of children with antisocial behaviour often have provided a model of antisocial attitude and conduct with which the children can identify or which they can copy (Rutter, 1985; Kelvin, 1969).

Evidence from longitudinal studies

The plethora of ideas which have emerged from cross-sectional studies reflect the difficulties of separating the contribution of different types of family influences. However, longitudinal studies have proved more fruitful. For instance, changes in family circumstances may influence a delinquent outcome—improvement in family relationships (Rutter, 1971) or reduction in family deprivation (Kolvin *et al.*, 1990) is associated with a reduced rate of conduct disturbance in the case of the former and of delinquency in the case of the latter. Such fluctuations become even more impressive if it can be demonstrated that increases and decreases in family pathology are associated with changes in subsequent offending. In the Newcastle Study (Kolvin *et al.*, 1990) if families moved into deprivation, there was a 50% increase in the rate of subsequent offending by their male offspring; if they moved out of deprivation, there was a 40% decrease. This suggests that there are certain factors which serve to counter the motivation to crime, despite youths coming from high-risk backgrounds. In the Newcastle Study, resilience in the face of deprivation was characterized by a number of factors operating on the junior school years. Most prominent amongst these was a good care-giving environment and hence it is argued that good parenting protects youths against the acquisition of a criminal record, irrespective of poverty or other forms of social deprivation. Other studies suggest close personal supervision of boys by parents is similarly protective and beneficial (West and Farrington, 1973, 1977).

Statistical analyses

Modern methods of analysis address themselves to the converse of pulling apart of those variables which usually act in concert and consist of putting them together in order to ascertain any identifiable patterns on the one hand or important predictors within any set of predictors of conduct disorder or delinquency on the other. Finally there are attempts to ascertain pathways to antisocial behaviour, which may contribute to identifying relevant processes and mechanisms. Let us start with predictions. Earlier attempts to develop formulas that would give reasonably accurate predictions of delinquency have not fulfilled the anticipated promise (Glueck and Glueck, 1950, 1964) as subsequent attempts to use the indices that were developed have shown poor predictive performance (Dootjes, 1972). Furthermore, in these early studies, much of the original data was

retrospective and therefore the claims about predictive utility must be viewed with caution. Unfortunately, more modern research suggests that multiple regression techniques give, if anything, worse prediction than the simpler methods originally utilized (Farrington, 1983). It is likely that this relatively poor prediction has more to do with the insensitivity of the predictor measures than the statistical techniques employed. The limitation of prediction studies has been well summarized by Rutter and Giller (1983), who draw two conclusions: first, that many youths theoretically at high risk do not necessarily become delinquent; second, that a substantial minority not at high risk do become delinquent. Nevertheless, some researchers have demonstrated the potency of prediction analysis utilizing early life psychosocial data: for instance, the Newcastle Study (Kolvin *et al.*, 1990) report that some two-thirds of boys coming from multiply deprived homes subsequently become delinquent. In addition, in the Cambridge Study, only two behavioural measures and five background measures were found to be independently predictive of delinquency: delinquents tended to be among those boys who were rated troublesome and daring at school; boys who were at risk coming from poorest or largest families; had criminal parents or parents who supervised them poorly or who had low IQs (West and Farrington, 1973, 1977). Further, the Newcastle group (Kolvin *et al.*, 1990) report that the only measure representing family environment that made an independent significant contribution to prediction of criminality was poor child care and mothering. Factors that failed to make a significant independent contribution were marital disruption, parental illness, those representing adverse social circumstances, parental personality, mother's age at marriage, occupational status and neighbourhood influences. However, an index of deprivation derived from the summing of scores of deprivation proved to be a good predictor of subsequent criminality. So too, was family size, which probably directly reflects poor home circumstances. Furthermore, in a series of analyses, prior juvenile delinquency proved to be the most powerful predictor of later criminality.

Finally, there is some evidence of specificity in relation to two types of family experiences. First, whereas marital disruption and parental illness were predictive of offences committed during the school years only, poor mothering was a significant predictor of criminality after the age of 15 years (Kolvin *et al.*, 1990). In the United States, McCord (1979) reported that, whereas home atmosphere was reliably related to criminal behaviour, parental absence failed to distinguish the criminal from the non-criminal, which again supports the notion that mere separation, or absence of parents, on its own does not have a significant effect on antisocial behaviour.

Summary

Although some of the evidence is conflicting, a number of facts have become established and these in turn have given rise to a diversity of theories to explain the associations between offending, antisocial behaviour and family and social influences. Certain theories have social and family and others psychological bases,

yet others seek genetic explanations. However, previously fashionable single factor theories are now considered too simplistic and have given way to multifactorial explanations. Furthermore, while longitudinal approaches have helped to clarify a number of the important issues, as have the application of multivariate analyses, the correlations that are found account for an important but not a substantial proportion of the variance.

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