Chapter 14
Childhood Autism

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Strange and bizarre behaviour in young children holds a particular fascination both for professionals and for the lay public. The notion that clusters of such types of behaviour represented psychosis, perhaps different types of psychosis, or perhaps even a unitary psychosis related to adult schizophrenia, slowly began to dawn in the second half of the twentieth century. In the period leading up to the 1950s eponymous labelling of such clusters was rife, but the only label which has stood the test of time is Kanner's (1943) clear and brilliant descriptive account of the behavioural abnormalities of 'infantile autism'. This was the first major leap forward. In due course it was followed by a series of other 'leaps' representing advances in neurology, classification, aetiology and therapy, all of which were underpinned by more modern scientific methods. However, the second major leap forward was when classification and the characteristics of these psychoses were established on a more solid basis, and this has been a central feature of work in this field over the last quarter of a century.

CLASSIFICATION AND DIAGNOSIS

Diagnostic criteria

Kanner (1943) saw the condition as being characterized by three primary features – (a) a profound failure to develop social relationships and an aloneness which he termed 'autism', (b) an obsessional desire for maintaining environmental sameness, represented by a dislike of change (in domestic and personal routines, customary routes and topography) and (c) an onset within the first two years of life – together with secondary features in the sphere of communication, evidence of some circumscribed cognitive potential and abnormalities of motor behaviour. Kolvin's (1971) criteria were broadly similar – in terms of age of onset, a self-isolating pattern of social behaviour, catastrophic reactions to environmental changes and/or stereotyped motor movements. Most authors include as a diagnostic criterion an onset before the age of three years. The exception is Wing (1982b) who continues to argue against age of onset as an essential criterion; however, this may be felt to widen the concept of infantile autism unduly.

As Eisenberg (1957) has pointed out, the specification of criteria for diagnosis is crucial and essential. The hope of a number of workers has been to develop a set of diagnostic criteria based on statistical frequency analysis rather than on clinical impression (see Kolvin et al., 1971c). Rutter's (1974) solution, consisting of identifying discriminants between infantile autism and other child psychiatric disorders, controlled for IQ, sex and age, is essentially similar. He found that there were only three symptoms which were universally present and specific in terms of being significantly more frequent in the autistic group: (a) a profound and general failure to develop social relationships, (b) language retardation and (c) ritualistic or compulsive behaviour. However, as each of these symptoms can occur in the absence of the others, none can be regarded as pathognomonic.

Other workers have developed diagnostic checklists (Rimland, 1968; DeMyer et al., 1971) or screening instruments (Creak, 1964) in an attempt to make diagnosis more objective (Rimland, 1968). Using relatively structured and standardized diagnostic systems and even different systems, collaborators can apparently achieve good agreement on diagnosis (DeMyer, 1971). Although such agreement is considerably reduced when the instruments are used by non-collaborators, nevertheless, such diagnostic systems or instruments are helpful in distinguishing autistic children from other diagnostic groups (DeMyer et al., 1971); however, they are of little use in attempting to distinguish different subgroups of autism.

Nosological issues

The traditional approach to psychiatric classification, used by Kraepelin (1913), was to undertake detailed clinical examination of demonstrable features and, where possible, to study the onset, course and natural history. With the scarcity of aetiological knowledge (Slater and Roth, 1969), the early workers based their classifications on symptomatological or phenomenological criteria, seeking evidence of clustering of features that were thought to have prognostic or predictive utility. In the Anglo-American child psychiatric literature, Kanner proved the master of this descriptive classificatory technique, but he was not alone in this exercise. A wide diversity of psychotic disorders of childhood were conceptualized and were eponymously labelled: for instance, Heller (1954) described his 'dementia infantilis', a condition in which the main feature was a period of regression in behaviour and deterioration in other aspects of functioning; Despert (1951) described a condition in which the behavioural abnormalities were present at birth; another concept which has a continuing fascination is Mahler's (1952) notion of 'symbiotic psychosis', which is chiefly characterized by affectionless clinging. The relationship of these disorders was not evident, particularly as different centres used different terms for the same condition or the same term for different conditions. The most widely advanced hypothesis was that there was a single pathological process. An extension of this hypothesis was that all the varieties of childhood psychoses constituted sub-types of adult schizophrenia.

The advances which heralded order and simplicity in this complex field all relate to classification. The careful and now classic reviews by Eisenberg (1957, 1968) in the United States and by Rutter (1967, 1968) in the United Kingdom pointed to the importance of more precise diagnosis based on demonstrable behavioural symptomatology and logical classification rather than the grouping of disparate conditions under one broad heading. A crucial contribution was the distinction by Anthony (1958a, b; 1962) of the childhood psychoses by the age of onset. This conceptualization highlighted the surprising harmonies and overlaps between named syndromes within age bands, in contrast to the dysharmonies across such bands. However, the nature of the psychoses within these age bands still remained to be defined. Anthony postulated three groups:

1 Those in whom the abnormalities had an early onset, that is up to the age of three years, and a slow chronic course. Included are Kanner's syndrome, Bender's 'pseudodefective' type and Despert's 'no onset' type.
2 Those with onset between three and five years of age and in which there was an acute course followed by regression, including Heller's disease, de Sanctis and Weygandt's dementias, Bender's pseudoneurotic type, Despert's 'acute onset' type, and Mahler's symbiotic psychosis.
3 Finally, those with a late onset and a fluctuating, sub-acute course, including Bender's pseudopsychopathic type.

The systematic empirical validation of the latter classification was undertaken by the Oxford and Newcastle Group (Kolvin, 1971; Kolvin et al., 1971a–e). They divided their psychotic children into three groups according to age of onset (under three, three to five and over five years). They focused on the under-three group (infantile psychotic) and the over-five group (late-onset psychotic) and used modern statistical techniques to provide validation in terms of differences of clinical features and aetiological factors between the two main groups studied.

The second group, those with an onset between three and five years old, has been described as 'disintegrative psychosis' (Rutter, 1972) and is very rare (Makita, 1966; Kolvin et al., 1971e). In this group initial development is normal but is followed by a serious deterioration of speech, language, cognition and behaviour. These children have mostly been found to be suffering from clear-cut organic disorders of varying aetiology and clinical picture (Anthony, 1958a, b, 1962; Rutter, 1968; Kolvin, 1971; Kolvin et al., 1971c).

The Newcastle/Oxford research has elaborated the distinction between infantile and later-onset psychoses, with the latter group showing more features representative of adult schizophrenia. For instance, hallucinations and delusions occur frequently in late-onset psychosis but never in infantile psychosis, even when the patients get older (Rutter, 1968; Kolvin et al., 1971c). On the other hand, gaze avoidance, finger flicking (stereotypies), resistance to change and serious retardation of speech and language are characteristic of infantile psychosis. Second, late-onset psychotics as a group tend to have only moderate intellectual impairment, but the majority of infantile psychotic children have very poor intellectual development (Kolvin et al., 1971b). Third, there is a significantly high rate of

schizophrenia in the parents of the late-onset psychotic group, compared with a low rate in the parents of the infantile psychotic group (Kallman and Roth, 1956; Rutter and Lockyer, 1967; Kolvin et al., 1971c). Fourth, there is an excess of parental personality oddities in the late-onset psychotic group (Kolvin et al., 1971a). Finally, there is evidence of an excess of cerebral dysfunction in the infantile psychotic group (Kolvin et al., 1971e). These findings (replicated by Green et al., 1984) provide definitive support for the hypothesis that infantile psychosis bears no relation to late-onset psychosis.

Rutter (1972) has reviewed the evidence distinguishing infantile psychosis from adult-type schizophrenia. A steady course is more typical in autism, while marked remissions and relapses frequently occur in schizophrenia; mental retardation is a common feature of autism but less so with schizophrenia; better visuo-spatial skills, poorer language skills and poorer intelligence are characteristic of autism but not schizophrenia; there is a marked male preponderance in autism but equality of sexes in adult schizophrenia; there is a high frequency of perinatal factors and organic factors in autism (Kolvin, 1971) but not in schizophrenia; there is a high genetic loading in the parents of schizophrenic adults but very low loading in the parents of autists (Kolvin, 1971; Rutter, 1972) and, finally, there is the bipolar distribution of onset of childhood psychosis (Kolvin, 1971) which Rutter (1972) interprets as a discontinuity between autism and schizophrenia. Rutter sees no reason for making any distinction between late-onset psychosis and adult-type schizophrenia and argues for a single disease concept to cover both of these groups. While schizophrenia may begin in later childhood (Kolvin et al., 1971e; Rutter, 1972), this is rare, and, further, it is unusual for schizophrenic symptoms to become overt before the age of seven or eight.

Hypotheses have been advanced, tested and verified that infantile autism (infantile psychosis) differs significantly from primary mental handicap (Rutter, 1966, 1967; Rutter and Lockyer, 1967; DeMyer et al., 1971–1974) and from late-onset psychosis (Kolvin, 1971; Kolvin et al., 1971a–e) and adult-type schizophrenia. There remains the question of the relationship (and the difference) between infantile autism and specific receptive developmental language disorders (dysphasia). While children in the latter group were thought to differ from autistic children, a number of overlapping features, particularly social withdrawal and repetitive activities, have been described which have led to the hypothesis that autism is an extreme variant of such language disorders. This latter hypothesis has been tested by the Maudsley group (Bartak et al., 1975, 1977; Cox et al., 1975) who compared autistic children of normal (non-verbal) intelligence with dysphasic children. Autism proved to be associated with a language deficit which was more extensive in that it spanned several different language modalities such as impairment of inner language, an impaired understanding of written language, and also involved a severe comprehension defect. Furthermore, the language impairment of the autistic group involved deviance in terms of echolalia, pronominal reversal, metaphorical language and inappropriate remarks in addition to linguistic delay. Finally, the autists used the speech they possessed poorly for social communications, whereas the dysphasic children had more impaired articulatory skills, but were better able to communicate, were far less behaviourally disturbed and much more socially mature, particularly as time passed. There is thus evidence that the language defect in autism is qualitatively different and quantitatively more severe than that in dysphasia.

Differential diagnosis

It is helpful to attempt a distinction between those disorders which, while different from infantile autism, may be confused with it and those disorders which may coexist with it. Unfortunately these two categories are not mutually exclusive. First, there are those non-autistic conditions which may have minor 'secondary' autistic features which are not sufficient to suggest an autistic syndrome. These conditions include profound deafness, developmental language disorders, psychosocial deprivation, elective mutism, schizoid personality disorder, organic brain disorders and severe mental handicap. For instance, children with profound mental handicap may have two-way social interactions which are appropriate for their developmental age or such social interactions may be moderately impaired (Wing and Gould, 1979). Second, there are the common coexisting disorders; these include hearing and other sensory defects, mental retardation, organic brain damage and language disorders.

Wing and Gould (1979) have demonstrated a relationship between severity of social interactional impairments and severity of handicap in the areas of language and cognitive ability and also severity of

organic impairment. Such findings give rise to the important question of whether there are degrees of autism. This latter notion is of crucial relevance to Asperger's syndrome (1944) which presents clinically as a schizoid-type personality disorder of childhood. Some authorities (Wing, 1981) regard it as a mild variant of autism, while others agree with Asperger that it is a personality trait (Kolvin and Goodyer, 1981).

BEHAVIOUR OF INFANTILE AUTISTIC CHILDREN

Social relationships

The poor relational ability which these children display is most disconcerting and distressing for the parents and relatives. In essence this consists of a maintenance of social distance and aloofness (autism). Autistic children generally avoid contact with people and mix and play very poorly with other children. They show poor ability to initiate or to respond to social overtures: three forms of impaired social interaction have been described – namely aloof, passive and odd (Wing and Gould, 1979) – with aloofness being more characteristic at lower levels of IQ. Autistic children gaze equally little at faces and at objects (O'Connor and Hermelin, 1967; Wing, 1982b). Some think that the tendency of these children to avoid eye contact (gaze avoidance) is part and parcel of the above relational difficulty.

Disorders of communication

Most autistic children have difficulties in the use of verbal and non-verbal methods of communication (Rutter, 1978c). Some remain mute and most have serious delays or abnormalities of speech and symbolic language. The more common abnormalities consist of a tendency to echo or repeat words or phrases, use of meaningless words or phrases, reversal of pronouns and inappropriate use of prepositions and conjunctions, and general immaturity of speech. When it develops, their speech is frequently stereotyped, monotonous and pedantic. In addition, there are difficulties of comprehension and in the use of gestures and imitation. All the above suggest that the underlying disorder is similar to developmental receptive dysphasia. Such children cannot cope with the nuances of communication or the subtleties of humour and often have little appropriate variation in facial expression, which parallels their poor response to the nuances of humour.

Imagination and purpose in play and behaviour

Especially in the pre-school years, autistic children tend to wander around aimlessly, devoid of the usual constructive and exploratory ventures seen in toddlers, who commonly display considerable curiosity about their environment. In autistic children play tends to be neither creative, imaginative nor constructive; nor do they display pretend play, but rather seem to prefer mechanical or repetitive pursuits. However, some circumscribed symbolic imaginative interests have been described in a small minority of autistic children (Wing et al., 1977).

Ritualistic and compulsive behaviour

Autistic children often insist obsessionally on a particular routine in their daily lives, and their resistance to any change – either of the routine or of objects or people in their environment – creates major management problems. Some children develop a deep attachment to an unusual object, such as a piece of string or a cup, from which they cannot easily be parted. These behaviours have been interpreted as an attempt by the child to create some order and constancy (Kanner, 1943) in a confusing and chaotic world. Kanner considered them to represent a desire to maintain 'sameness' in the environment.

Catastrophic reactions

Autistic children tend to cling to these rituals or compulsions and may react with considerable, 'catastrophic' distress to interruptions of these or other changes in their routine. Nevertheless, such reactions do not always seem to have obvious determinants. Some children are apparently suddenly abnormally distressed by an everyday object, such as a table or a jug, but for other catastrophic reactions there are obvious explanations. The stimuli to which the children react so disastrously appear to be specific to each child; for instance, one may react to reorganization of furniture within the home, another to an unfamiliar route on a journey, and another to a change in a dressing routine.

Motor abnormalities

These are particularly evident when the child is distressed or excited. The most characteristic consists of finger stereotypies and hand-flapping. Facial

grimaces, jumping, toe walking, pirouetting and a number of other repetitive activities are seen. Some autistic children are over-active while others may be underactive.

Sensory disorders

One of the conundrums of autism is that these children tend to show a greater response to proximal sensory stimuli than to distal ones: whereas they are likely to ignore aural or visual stimuli they seem to explore the external world by touching, tasting and smelling objects. There are notable exceptions to such general tendencies, in that they are often transiently alert to new, strange noises and appear fascinated by rotatory or pendular movements. Nevertheless, the ignoring of sound stimuli and the failure to respond to startle stimuli often suggests a degree of deafness and it is not always easy to exclude this; often information from parents about the child's response to ordinary domestic auditory stimuli may provide helpful clues. Some autistic children display a dangerous combination of a lack of response to painful stimuli plus fearlessness.

Mood and aggressive behaviour

Rages and tantrums and self-directed aggression are relatively frequent; although most episodes do not appear to have obvious precipitants, many authors speculate that these behaviours may be a reaction, either to frustration following poor ability to communicate or to boredom. Self-destructive behaviour, such as biting of hands, face-slapping and head-banging, generates considerable anxiety in parents.

INTELLECTUAL DEVELOPMENT

The impression of good intellectual potential which was gained by earlier authors was probably based on the attractive physical appearance of autistic children and the special ability of a minority of autists on non-verbal, non-symbolic tasks. A few autistic children show exceptional abilities in circumscribed areas, such as with jigsaw puzzles, or have remarkable memories for numbers or tunes.

Subsequent research has shown that autism is associated with major intellectual deficits, with about half having IQs below 50 and another quarter between 50 and 70 (Rutter and Lockyer, 1967; Kolvin et al., 1971b). Nevertheless, Rutter and Lockyer point out that autistic children with low IQs have symptoms similar to those with higher IQs and so 'mental subnormality as a concept is insufficient to account for the autism.' Fluctuations (Rutter, 1970) in IQ over time (DeMyer et al., 1974) in autistic children have proved to be similar to those found in normal children. However, there are marked fluctuations on sub-test scores reflecting different types of skills: thus a simple averaging of sub-tests may mask any individual strengths and weaknesses.

EPIDEMIOLOGICAL FACTORS

Prevalence

The work of Lotter (1967) has emphasized the rarity of the condition (that is, 4 per 10 000) when early childhood psychosis is tightly defined. When more widely defined, the rate jumps to 21 per 10 000 (Wing et al., 1976). However, such rates need to be viewed with caution as the nature of this wider group of psychotic children is not yet clear. In addition, such epidemiological rates, even when narrowly defined, are far greater than the so-called administrative rates (numbers in the community who are known to the local services) and hence should be used with caution in planning services, to avoid over-provision.

Sex ratio

There is a high male : female ratio ranging from 3 : 1 or 4 : 1 in hospital series (Creak and Ini, 1960; Rutter and Lockyer, 1967; Kolvin et al., 1971d) to 2.9 : 1 in epidemiological research (Lotter, 1967).

ORIGINS

Social and psychological factors

Previously, it has been commonly assumed that the fundamental determinants of psychiatric disturbance in childhood are parental personality, attitudes and emotional disturbance (Kolvin et al., 1971a). Whereas, with few exceptions (Ritvo et al., 1971), clinical studies have reported that the parents of autistic children came predominantly from the upper social strata and were of above-average intelligence (Creak and Ini, 1960; Rutter et al., 1967; Kolvin et al., 1971c), population surveys have either reported a weaker association (Lotter, 1967) or

have not confirmed such findings (Wing, 1980). Nevertheless, such findings have been used to support the notion of an origin for childhood autism in environmental and family factors, one of the earliest environmental explanations being based on descriptions of social formality and lack of warmth of mothers (Kanner, 1943, 1949) and of fathers (Eisenberg, 1957). Such notions soon led to the concept of 'refrigerator parents', who were said to have obsessive and cold personalities, despite these features being relatively common characteristics of the public manners of middle- to upper-class parents.

Unfortunately, the basic premise was not validated before explanations were considered to have been proved: a plethora of similar theories have been advanced, mainly stemming from clinical practice, most of which are totally unacceptable on the grounds of being based on heterogeneous or unduly small samples; nor were objective and reliable measures used to accumulate empirical data upon which a theory could be based (Kolvin et al., 1971a). Finally, not only were some of the theories that were developed incapable of generating testable hypotheses, but also some theorists did not perform the two axiomatic methodological steps of demonstrating a correlation between child and parent variables and of carrying out experimental or observational studies in an attempt to validate a cause–effect relationship; neither did most theorists consider the possibility that the syndrome might have a multifactorial basis.

Modern research has not confirmed the parental personality stereotype, irrespective of the method of assessment. DeMyer et al. (1973) reported that child-care practices of parents of autistic children were similar to those of the parents of matched, normal children. Cox et al., (1975) used objective clinical interviews plus parental self-rating inventories and reported that although mothers of autistic children showed less warmth to their autistic child, this could be attributable to the difficulty of showing warmth to an unresponsive child.

Not only is there no evidence that autism is secondary to abnormal parental personalities or unusual child-rearing practices, but also there is no evidence of a link between extremely depriving environmental circumstances (Rutter, 1972) or faulty conditioning (Ferster, 1961) and infantile autism. It would be difficult to imagine how any of these extreme environmental experiences could give rise to the complex but specific patterns of impairments in such diverse areas as cognitive, perceptual, motor and autonomic functioning (Wing, 1976). Further, even where organic factors have been ruled out, with children of normal intelligence being studied, there is good evidence of a cognitive defect and so 'it seems entirely improbable that the aetiology is entirely psychogenic' (Bartak et al., 1975). The sum total of these studies and reviews provides strong refutation of a psychogenic hypothesis. In addition, there is suggestive evidence (Bell, 1968, 1971; Cox et al., 1975) that some of the parental social reactions may be secondary to the autism and that social isolation, when present in mothers of autists, may follow the onset of the disorder (Kolvin et al., 1971a).

Biological factors

There is a great deal of evidence to suggest that autism may be the final common behavioural expression of a wide variety of organic-cum-developmental influences. First, presumptive evidence of cerebral injury has been obtained from studies of perinatal complications, with particularly high rates being reported in clinical studies (Gittelman and Birch, 1967; Lobascher et al., 1970; Kolvin et al., 1971e), but the evidence from population studies is less impressive (Lotter, 1967; Treffert, 1970). Second, in about half of the cases in the two major hospital series there is evidence of cerebral dysfunction and up to one autistic patient in three in the course of time develops epileptic fits (Rutter et al., 1967; Kolvin et al., 1971e; DeMyer et al., 1973). EEG studies confirm this evidence, with high rates of clear-cut abnormalities (spike or spike and wave activity) (White et al., 1964; Gubbay et al., 1970; Kolvin et al., 1971e; Small, 1975). Other workers have shown an excess of abnormalities of evoked potentials during waking and sleep (Ornitz, 1970; Small, 1971). Epileptic fits are usually associated with lower levels of IQ (Rutter, 1970). Third, certain organic conditions appear to have a close relationship with infantile autism: for instance, it occurs a hundred times more frequently in children whose mothers have had rubella in pregnancy than in the general population (Chess, 1971); it is also associated with previous episodes of infantile spasms (Kolvin et al., 1971e; Taft and Cohen, 1971) and with congenital or profound blindness (Freedman, 1971) and has been described in association with a variety of other specific cerebral organic conditions (Darby, 1976). One group of workers (Hauser et al., 1975) report enlargement of

the temporal horn of the left lateral ventricles, with atrophy of the adjacent area of the left medial temporal lobe. But there is doubt about the homogeneity of the sample and there is a suggestion that the cases were highly selected, with heavy loadings of neurological disorder and, indeed, other questions have been raised about methodology (Lancet, 1976). There are also suggestions of involvement of the dominant hemisphere (Hermelin, 1966) and of the non-dominant hemisphere, which is concerned with both comprehension and expression of emotion (Ross and Mesulam, 1979). However, it is unlikely that the lesion would be confined to one or other of the hemispheres.

It is evident that we have little idea what the specific brain dysfunction comprises; however, the diversity of EEG and seizure patterns, which range from focal epilepsy to the widespread disorganization of hypsarrhythmia (Rutter et al., 1967; Kolvin et al., 1971e; Taft and Cohen, 1971), appears to argue against a single underlying homogeneous pathological mechanism. Indeed, on the basis of knowledge available at that time, in 1974 Rutter found it impossible to localize the lesion and favoured the hypothesis of a non-specific syndrome of biological impairment. Nevertheless, a number of unifying theories have been advanced. For instance, Damasio and Maurer (1978) attempted to incriminate dysfunction of a single and yet widely based system consisting of the bilateral neural structures of the mesolimbic cortex. They postulated that autism might be the result of macroscopic or microscopic changes in the above-mentioned target areas or in structures influencing them. Thus, the broadly homogeneous functional aberration of autism might be the result of very different types of structural disorganization. Unfortunately, this theory is so far-ranging that it is incapable of generating more specific testable hypotheses. An allied theory is that multiple neurological deficits do not necessarily imply multiple aetiologies but are dependent on the abnormality impinging on a specific relevant brain centre or centres, such as association areas (Crawley, 1971) or language areas (Wing, 1971). This is a plausible explanation, which could account for the multiple handicaps or impairments of language, perception, motor ability and behaviour described by Wing and Wing (1971): 'a number of different brain functions could be affected by, for example, a single genetic or biochemical abnormality, or anatomical proximity could make different brain centres vulnerable to the same lesion'. Hence, these workers assert that any condition which produces abnormality or delayed maturation of relevant brain areas could theoretically lead to the impairments of infantile autism.

Other workers have incriminated subcortical dysfunction which directly or indirectly affects the reticular activating system (Des Lauriers and Carlson, 1969) involving either under- or over-arousal (Hutt et al., 1964, 1965) or an imbalance between these systems. However, the empirical evidence advanced in support of these sometimes conflicting theories tends to be rather questionable.

Cognitive factors

As implied above, the pattern of cognitive functioning in autism suggests a specific defect involving language not only in terms of comprehension but also in terms of conceptual skills in thinking (Rutter, 1968, 1974; Rutter et al., 1971). Not only are the earliest signs of language (babbling) deficient (Ricks, 1972), but subsequently there is evidence of impairments in comprehension and in production of language and language modalities, such as gesture (Tubbs, 1966). In addition, there are 'pragmatical deficiencies', which relate to inappropriate use of speech and language in a social context (Frith, 1982). Further, the experimental evidence available implicates an impaired ability to conceptualize and symbolize and to process meaningful and temporally patterned stimuli (Hermelin and O'Connor, 1970; Frith, 1971). Hermelin and Frith (1971) assert that the deficit is central and appears to consist of an inability to encode information in a meaningful way; this results in an autistic child having difficulty in 'making sense of what he sees and hears' (Frith, 1982). Despite their poorer ability in processing meaningful information, autistic children tend to have good rote memory.

It has been suggested that such cognitive defects could impair social interactions (Churchill, 1972), but the problem with this theory is the existence of autistic children who are not cognitively impaired and yet are socially impaired (Frith, 1982). In addition, it is not clear whether the disorder is primarily one of language or whether the language disorder arises from a more widespread cognitive deficit (Rutter, 1974) or more widespread cerebral dysfunction as described above (Damasio and Maurer, 1978).

A related and plausible theory is one that implicates non-specific cerebral dysfunction and suggests that, whatever the pathophysiology, the mechanism involves a learning disorder (DeMyer et al., 1972a) which hinders the acquisition of language (Rutter, 1968; Hermelin, 1971; Wing, 1971). The theory suggests that the learning disorder also hinders the acquisition of certain visuomotor acts such as the imitation of body movements. These disabilities in turn impair the child's ability to establish social relationships with others. Indeed, Churchill (1972) circumscribes the theory by implicating specific anomalies of areas subserving perceptual, sensorimotor and language functions. He suggests that autistic children share, with non-psychotic brain-damaged children, various perceptual–motor deficits, but the essence of the psychotic condition is a central language deficit allied to, but more severe than, that found in children with a dysphasic disorder. In support of this theory is the finding that the abnormality of response by autistic children to sensory stimuli resembles that seen in developmental receptive dysphasia and congenital partial blindness/deafness, particularly when associated with maternal rubella.

Other workers have pointed to the association between impaired interpersonal relationships and impaired imaginative play in autistic children (Rutter, 1978b; Wing and Gould, 1979). Hermelin (1978) suggests, on the basis of her experimental work, that this reflects a deficit in symbolic thought. On the basis of the above-mentioned facts and a presumed social deficit existing in autistic children at a Piagetian 'pre-symbolic level' (Piaget, 1951), Frith (1982) speculates 'that this deficit alone may prevent the development of imaginative play'. She goes on to suggest that normal children are likely to play with toy objects as if they were social objects and this in turn gives rise to pretend play, while autistic children rather treat people as objects. Such a theory takes us back forty years to Kanner's original assertion that a profound social impairment is primary and explanatory of the more widespread disturbances.

Maturational factors

There remain those autistic children with higher IQs with little or no evidence of neurological dysfunction and fewer demonstrable cognitive deficits, whose disorder cannot easily be accounted for by the above theories. Rutter and colleagues (1971) have, in the past, speculated that in these cases autism may be due to a kind of maturational delay which is associated with a physiological developmental language disorder. However, despite its attraction, the support for this hypothesis is reduced by the fact that very few autistic children ever recover completely (Rutter et al., 1971).

Biochemical and genetic factors

Biochemical studies do not suggest an inborn error of metabolism (Guthrie and Wyatt, 1975). Very occasionally, defects have been identified, such as hyperserotonaemia, but there has often been insufficient rigor in the definition of the groups studied and in standardization of the assay procedures, and the findings have seldom been replicated. While the possibility remains that infantile psychosis is aetiologically heterogeneous, such studies have not as yet identified a sub-group which have other features which cluster together in a meaningful way.

Has autism a genetic basis? First, are there genetic links with schizophrenia? When a tight definition of psychosis is applied, in comparison with the general population there is no evidence of excess of schizophrenia in parents of children with early-onset psychosis (Kanner, 1954; Creak and Ini, 1960; Rutter and Lockyer, 1967; Kolvin et al., 1971d); this is in contrast to late-onset psychosis, which appears to be genetically linked to adult schizophrenia (Kallman and Roth, 1956; Kolvin et al., 1971d).

What about other types of genetic causation of autism? Hanson and Gotteman (1982) have reviewed the evidence for a genetic basis. First, they point out that inheritance of autism is not of a simple Mendelian pattern. In addition, no chromosomal abnormalities had been identified (Böök et al., 1963; Wolraich et al., 1970). However, an excess of fragile X marker aberrations has been reported in the male autistic population (Gillberg and Wahlström, 1985), and this appears to be specific to autistic psychoses. Second, if rare mutations were the cause of autism then it is to be expected that neither parents nor siblings would be affected; in fact, while there are no reports of parents of autistic children being autistic, there is a slight excess (i.e. under 0.5%) amongst siblings in the British series (Creak and Ini, 1960; Rutter, 1967; Rutter and Lockyer, 1967; Kolvin et al., 1971d). In addition, none of the other findings fit theoretical expectations for a rare mutation: there is not an

equal sex ratio and the identical-twin concordance rate is also low (Folstein and Rutter, 1977); hence a simple dominant mutation theory is unsupportable.

Third, a theory of polygenic inheritance is not supportable: there are no distinctive qualities in the parents, for instance of personality, which would suggest they have a milder version of their child's condition. The sibling rate, though very low, could support a notion of familial clustering, as could the twin concordance, but the latter is equally consistent with an explanation in terms of perinatal brain damage (Hanson and Gotteman, 1982). Finally, as in their twin study, discordance was usually associated with brain damage; Folstein and Rutter (1977) suggest that infantile autism may be determined by a combination of genetic predisposition and brain damage. The one plausible genetic explanation relates to fragile X chromosome anomalies. Jörgenson et al. (1984) suggest these make the child vulnerable to deviant speech/language development; interaction with different pre- or neonatal brain insults may give rise to one sub-group of autism. However, there is no identifiable genetic basis for most cases of childhood autism.

Mention must be drawn to the personality disorder of childhood originally described by Asperger (1944), comprising gross lack of skills in social diplomacy, associated with a degree of naivety, giving rise to an impairment of social relationships. He considered that this personality variant must be transmitted genetically. Van Krevelen (1971) has postulated that such a personality could be turned into an autistic psychosis by earlier brain damage. However, there remain doubts about the relationship of this disorder to infantile autism (Wolff and Barlow, 1979; Kolvin and Goodyer, 1981).

OUTCOME AND PROGNOSIS

Outcome

The clinical features of infantile autism tend to vary with the age of the child and his stage of development. For instance, in early infancy a characteristic feature is a failure to cuddle; babbling may be absent and there may also be a failure to engage in pre-verbal mother–baby social interactions. In the toddler a prominent feature consists of profound social withdrawal and avoidance of gaze, together with language abnormalities and repetitive activities. This is the peak period for the more florid symptoms of the disorder. Subsequently, many children continue to have difficulties in interpersonal relationships, particularly outside the home, and this may be because the adults not in regular contact with the child are unaccustomed to interpreting his communications or making allowance for his behaviour.

Older autistic children show an inability to appreciate the nuances in social relationships and have a lack of appreciation of other people's feelings. Often in adolescence there is a real desire for close friendships, but the lack of social skills and appropriate empathy makes these difficult. While most of the other symptoms disappear eventually, it is not uncommon for obsessional and ritualistic symptoms not only to persist, but to expand. When the child attends infant school the prominent features are inactivity, inertia and educational difficulties.

In spite of the modern methods of treatment, the outcome in adolescence and adulthood is mostly poor in terms of intellectual development, overall adjustment and, additionally, work potential in adulthood. Irrespective of whether the sample is drawn from a clinic (Rutter et al., 1967; Kanner, 1971; DeMyer et al., 1973) or epidemiologically based (Lotter, 1974b) the picture of the outcome is roughly the same. While two out of three autistic children remain severely handicapped, one out of four does fair-to-moderately, with some continuing social and relational problems; however, only just over one child in ten (Kanner, 1971) develops adequately in terms of intellect and social adjustment and is able to survive in an unsheltered work situation (Rutter et al., 1967; DeMyer et al., 1973). Even those who improve may show continuing difficulties of relationships and oddities of personality.

Prognosis

The most important prognostic factor is the *testable level of intelligence* (Rutter et al., 1967). Outcome can be envisaged as being closely tied to the degree of associated handicap. For instance, DeMyer and her colleagues have demonstrated that children tend to remain in the same intellectual academic/work category in which they were initially assigned, rather than improving with time. However, there is an important variation: while the mean IQ of the population of autists remains stable, autists who are rated as having the best potential at initial assessment show considerable gains in verbal IQ with the passage of time (DeMyer et al., 1973).

The following constitutes a useful guide. Those in the lowest bands of intelligence (with IQs below 50), constituting about 40% of the autistic population, will comprise the majority of those who remain severely handicapped; the next band (with IQs ranging from 50 to 70), constituting about 30% of the autistic population, contains a high proportion of those who do moderately well; the highest band (with IQs above 70), constituting again about 30% of the autistic population, comprises the majority of those who develop adequately. In the course of time some movement does occur between bands, but upward movement, while unusual, tends to be confined to adjacent bands. There is therefore little support for the belief that autistic children have latent intelligence (Wing, 1976). Careful psychometric assessment by an experienced child psychologist constitutes a fundamental basis of prognosis and also a useful guide to educational placement. Inter-related with these cognitive factors are *organic* factors; not only is IQ related to severity of cumulative evidence of brain damage (Kolvin et al., 1971e), but so too is prognosis (Gubbay et al., 1970; Small 1975).

The question of the *effects of treatment in relation to IQ* has been studied by DeMyer et al. (1974). Their work suggests that while children with initial IQs above 50 showed a greater increase in IQ than untreated autistic children in the same IQ range, those with IQs below 40 showed no differential effect. Furthermore, after treatment the verbal IQ gains achieved during treatment tended to be maintained in the autists with higher initial IQs but stagnated in the middle groups or were even lost in those in the lower IQ groups.

The next set of important prognostic factors is in the area of *speech, language and communication.* It is well known that an improved prognosis is associated with the development of meaningful speech by the age of five years, but about half do not achieve this. The more communicative the child, or the better developed the speech or language at initial assessment, and the more constructive or symbolic the play (as these reflect inner language), the better the development of conversational speech later (DeMyer et al., 1973). Mutism has a particularly poor prognosis. However, even where speech improves considerably, there are often residual difficulties with speech rhythm, repetitiveness, and with abstract concepts (Rutter et al., 1967; DeMyer et al., 1973).

A poor prognosis is also associated with the severity of psychosis (DeMyer et al., 1973) and a slow rate of losing the more florid autistic symptoms (Kolvin, 1972a). If substantial improvement is to occur it will usually show itself by the age of seven years (Rutter, 1967). The child with a good prognosis is one with a good IQ, little evidence of cerebral dysfunction, early speech and language development, mild symptomatology which he rapidly loses, and who is given appropriate behaviour modification and adequate schooling.

It has been suggested that the origins of the usually reported poor work record are three-fold: poor cognitive development, adverse temperamental features, such as inertia, inactivity and poor concentration, and poor social skills in the work setting (Lotter, 1974b). However, environmental factors, particularly home factors, must play a part and Lotter (1974b) commends the importance of a harmonious home atmosphere.

TREATMENT

The multiplicity and diversity of treatments which have hitherto been used in early childhood autism are an indication of the ineffectiveness of most of them. Further, the previous extravagant claims that were made for many of the approaches subsequently have had to be substantially modified. Such treatments have included electro-convulsive therapy, intensive psychotherapy of the parents and/or the child, high-dosage vitamin supplements, pharmacotherapy, and the Delacato (1974) programme of physical stimulation.

Early diagnosis and counselling

The need for early diagnosis in autism has been questioned. So far, there is no clear-cut evidence that early intervention affects the underlying handicaps or the child's progress. Some UK researchers have reported that younger children respond more rapidly (Hemsley et al., 1978), but some US researchers have denied this (Goldfarb, 1974). However, even if early intervention does not lead to substantial improvement, inadequate systematic stimulation and behavioural training from an early age can actually result in deterioration (Lovaas et al., 1973). Further, early diagnosis is helpful to parents who may have gone through a period of distress and exasperation at their child's behaviour, but who can now more easily understand this behaviour and will want to know more about its

causes. It also enables them to seek referral of the child to an appropriate specialist centre with a multi-disciplinary staff who have had wide experience in assessing such children's skills and handicaps. Careful and detailed medical assessment is indicated in all cases.

The child's condition must be described carefully and patiently and explanations and advice will need to be given, preferably with both parents present. The nature of the condition and the implications may be difficult for parents to understand. The clinician must be well-informed and experienced so that he or she is able to give a balanced opinion with due frankness and compassion. There are other important facets to counselling: for instance, it is helpful for the parents to know that there does not appear to be a major genetic component and that autism is not in any way related to schizophrenia; this should help to dissipate anxiety about a genetic predisposition to developing the latter condition. Further, practical advice and support can be given concerning the day-to-day handling, how to stimulate and play with the child, and guidance and direction about general management, education, holiday relief and so on.

Two important components in treatment are counselling in conjunction with the use of parents as 'co-therapists' in home-based treatment programmes (Schopler and Reichler, 1971a, b; Howlin et al., 1973) and the use of parent support associations (Wing, 1981). Finally, it must be stressed here that not only has intensive analytical psychotherapy of parents of autistic children not worked as a cure for autism, on occasion it has been counterproductive. It can generate a deep sense of guilt in parents, implying that their personality has contributed to their child's autism.

Pharmacotherapy

Pharmacotherapy cannot modify the course of severity of the disorder. Further, there is no specific drug treatment for any associated brain damage. Nevertheless, anticonvulsants are useful in the 25–33% of autistic children who suffer from seizure disorders; sedatives and tranquillizers may be useful to allay specific symptoms, such as overactivity or aggressiveness (Wing 1976). Hypnotics may be indicated if sleeplessness becomes a severe problem. Unfortunately, the response to drugs tends to be idiosyncratic and dosage has to be tailored to the individual child.

Operant approaches

The main focus of treatment has now moved to those educational and operant conditioning approaches geared to help the child overcome social, behavioural, educational and linguistic difficulties. However, the optimism engendered by the introduction of operant conditioning has been tempered by subsequent evaluation (Lovaas et al., 1973). As often occurs with new therapeutic techniques, its usefulness has been exaggerated: improvement often may be specific to particular situations, and also transient; it should be considered as one of a range of techniques which must be tailored to the individual child and his or her handicaps. There is evidence that operant techniques work best in conjunction with educational measures as part of a general training approach (Lovaas et al., 1973; Wing, 1976). These basically comprise systematic encouragement of appropriate behaviour and discouragement of inappropriate behaviour using a programme tailored to the individual child. Operant principles can be applied to increase imitative behaviours, to encourage play and to improve self-help skills (Everard, 1976). In adolescence, training in social skills may increase the ability to socialize. Most clinicians have now turned away from using punitive forms of discouragement, partly because therapists dislike them, many questioning the morality of the use of noxious stimuli; in addition, there is little evidence of their long-term effectiveness. However, physical aversive measures have been used as a last resort in those severely handicapped autistic children who are prone to serious self-injury.

Home based approaches

It is well known that autistic children may not generalize skills acquired in one setting, in particular the school, to other settings (Hermelin, 1972; Rincover and Koegel, 1975). This has encouraged involvement of parents in the child's treatment: for instance, a variant of the operant conditioning approach has been developed by Schopler and Reichler (1971a,b), who use the parents' high degree of motivation to help children with learning and the acquisition of practical and social skills. These ideas have been expanded by the Maudsley group (Rutter and Sussenwein, 1971; Howlin et al., 1973) to a home-based approach in association with multiple impact therapy adapted to the needs of the individual child. They focus on social and linguistic

development and the removal of maladaptive behaviours, using a range of operant techniques together with appropriate parental counselling. In the treatment programmes the parents are helped either to participate in treatment or to be the main vehicle for carrying this out. These workers report considerable improvement in behaviour, social responsiveness and language abilities.

Special education

Work by the Maudsley group constitutes a landmark in the evaluation of special educational treatment of autistic children (Bartak and Rutter, 1973; Rutter and Bartak, 1973). They report considerable social, behavioural and scholastic gains even with those children with the most marked behavioural disturbance (Rutter and Bartak, 1973). Furthermore, it would appear that greater educational benefits derive from specific teaching in a well-controlled and more structured classroom situation (Rutter and Bartak, 1973). However, it is to be noted that the autists' understanding of what they learn often lags behind the mechanical skills that they achieve. This has important implications for the curriculum: particular emphasis should be given to language comprehension and social skills training. For instance, language training and education should be directed towards helping the autistic child to understand language and not merely the use of words. Operant techniques have been developed for children who have acquired some spoken language. The techniques are geared to helping the child to imitate sounds, expand the use of words and phrases and to couple this with comprehension. Allied to this is the reduction of echolalic and other jargon and inappropriate speech (Sloane and MacAulay, 1968). Attention should also be given to the needs of those 50% of autistic children who do not develop meaningful speech, and to the possibility of training in the use of alternative forms of communication (Deich and Hodges 1977), for instance through imitation, gesture and drawing.

Inevitably, educational approaches need to be wide-ranging and geared to a child's level of development rather than to his chronological age, and therefore must be preceded by careful assessment (Everard, 1976; Jeffree et al., 1977). In addition, there is much evidence to suggest that the tasks with which autistic children are presented must be reduced to small steps which are within their grasp. High staff:pupil ratios are therefore especially important, in that they facilitate attention to curricular details and provide opportunities for staff–child interactions and appropriate interventions.

Residential approaches

As discussed above, improvement in school or hospital units does not necessarily generalize to the home environment unless effective reinforcement is available at home and at a parental level (Lovaas et al., 1973; Rutter and Bartak, 1973). Indeed, the emphasis is now on the importance of extensive parental involvement, which allows parents to maintain or enhance their skills, and consequently the importance of residential management is apparently questioned. (Schopler and Reichler, 1971a, b; Lovaas et al., 1973; Rutter and Bartak, 1973). On the other hand, Menolascino (1973) has argued in favour of a specific residential or milieu setting for fostering relational contacts, for planning and tailoring behaviour modification programmes and, finally, for evaluating the efficacy of education and speech training. In addition, the varying abilities of families to cope with their autistic children must be borne in mind: before the family cracks under the strain, the professional advisers should consider residential placements, usually on a short-term basis.

Mental handicap alone or with autism

There remains the important question of whether the autist who is seriously retarded merits any different treatment from the mentally handicapped child without autism. Rutter and Bartak (1973) point out that autistic children (at least in the pre-school and infant school years) are dependent on the active intervention of adults for social and educational progress; mixing with non-autistic children at this age is apparently of no significant benefit to the autist. Nevertheless, for children with an IQ of less than 40 the educational benefits are minimal, despite the best schooling available. However, because most handicapped children can benefit from attempts to educate them (Bartak and Rutter 1973; Lotter 1974b), on humanitarian grounds all children, no matter what their degree of handicap, merit educational help. The evidence therefore is in favour of the view that advocacy on behalf of the brighter autistic child is advocacy on behalf of all autistic children but with resources and facilities appropriate to their potential.

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