

THREE ASPECTS OF CHILDHOOD AUTISM:
MOTHER-CHILD INTERACTIONS, AUTONOMIC
RESPONSIVITY, AND COGNITIVE FUNCTIONING

by

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ABSTRACTTHREE ASPECTS OF CHILDHOOD AUTISM:MOTHER-CHILD INTERACTIONS, AUTONOMIC RESPONSIVITY,
AND COGNITIVE FUNCTIONING.

A survey of the current literature published in the field of childhood autism suggested that despite a rapidly expanding body of research, we have little definitive understanding of the aetiology of the condition or a highly efficacious treatment model. However, there is a growing consensus that the autistic child suffers from a primary organic impairment, resulting in particular deficiencies in cognition and language, and consequently he displays pronounced learning difficulties.

The literature survey also indicated that there is a lack of empirical data on the manner in which parents interact with their autistic child, including the contingencies they deliver upon his characteristic responses, and the influence of the child upon the adult's behaviour. Analysis of mother-child interactions were conducted therefore, using diads with normal and autistic children and their mothers. Differences were found between the behaviour of mothers of normal children and mothers of autistic children on a number of verbal and non-verbal measures relating to their mode of interaction with these children. Such data may have potential utility for the design of generalizable behavioural treatment programmes for autistic children, with parents as the primary change agents.

The second study involved an analysis of psychophysiological data collected from a group of non-verbal autistic children who typically display infrequent, abnormal or unpredictable overt responses to important environmental events including traditional reinforcers, novelty and social stimulation. Autonomic data appeared suitable for determining the effects of such environmental stimuli, which also may have implications for treatment design and for an understanding of the aetiology of childhood autism.

The third area of investigation consisted of three studies designed to investigate further the apparent deficits and abnormalities in cognitive functioning that have been reported recently in experimental research literature. These studies involved binary sequence learning, concept attainment and probability-learning tasks, and contrasted the performance of autistic children with normal and subnormal control subjects. It was concluded from these data that there is evidence of abnormalities in functioning on cognitive tasks and that they may be particularly related to the nature of strategies adopted in such tasks and the failure to utilise task-relevant information feedback in an appropriate manner.

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CHAPTER ONE:

"DIAGNOSIS AND AETIOLOGY OF CHILDHOOD AUTISM:
A REVIEW OF THE LITERATURE"

Diagnostic considerations

Numerous aetiological hypotheses have been formulated to account for the behaviour of the autistic child. Before discussing them however, it is necessary to consider the question of definition and diagnosis of childhood autism, since this has been a source of considerable controversy. It is essential that we are aware of the nature of the population under consideration when we attempt to evaluate the results of experimental studies and case histories published in the Psychological literature. Unfortunately, there are several published reports that do not allow us to ascertain the exact nature of subjects used, but it is clear that diagnostic controversy has been a contributory factor in many of the apparently contradictory research findings and in the diversity of the aetiological hypotheses themselves.

Leo Kanner (1943), in a paper which soon became a classic, first outlined a syndrome which he labelled "Early Infantile Autism". On the basis of many years of clinical experience he suggested that there existed a group of rare, but unique, children who could not be fitted into any existing conventional diagnostic category. He listed five characteristic symptoms which he claimed would differentiate these children from those suffering from other childhood disorders:

i. An inability to relate to others from the beginning of life. This, he emphasised, was not a process of withdrawal from formerly existing relationships, as seen in the schizophrenic child, but rather what he called an extreme "autistic aloneness" which was present from birth. He reported that parents often told him of their child failing to adopt any anticipatory gestures upon being picked up, a lack of sustained eye contact, and a general indifference towards people.

ii. Failure to use language as a means of communication.

Rote memory was apparently unimpaired in these children, and echolalia and pronoun reversal were frequently observed, although language as a communication tool, as normal children use language, was not present.

iii. An anxiously obsessive desire for sameness.

This was seen in extreme reactions to apparently trivial environmental change.

iv. Fascination with objects rather than people.

This symptom was manifested in behaviours like bizarre, prolonged manipulation of favoured objects, and included repetitive flicking, spinning, rocking, or preoccupation with textures.

v. "Good" cognitive potential.

Kanner reported that in almost all the cases he studied he found examples of extraordinary isolated abilities, often in memory, music or motor skills. These abilities stood out in complete contrast to levels of functioning in social skills.

Kanner insisted that these children exhibit their characteristic symptoms within the first two years of life, and considered the first two symptoms as primary ones, the others resulting from the basic disturbance in the ability to relate to others (Eisenberg and Kanner, 1956). Since Kanner's original paper, a rapidly increasing volume of literature has been published on experimental and case-study work on these children, within the context of a broad spectrum of aetiological speculation. It is reasonable to suggest that the volume of research is out of all proportion to the frequency of the disorder. The

essentially unknown aetiology, and the unquestionable clinical fascination aroused by these children in those who work with them, would appear to be significant factors in this respect. However, one of the recurrent problems has been that of differential diagnosis, and it would appear that a number of studies refer to their subjects as "autistic" when they clearly do not correspond to Kanner's description of the syndrome. Diagnosis is complicated by the not negligible degree of overlap between symptoms seen in autistic children and certain cases of cerebral insult, aphasia, schizophrenia, subnormality, and a number of psychotic conditions. Rutter (1966) stressed how often there appears to be no clear difference in qualitative terms between infantile autism and a number of other childhood disorders. Many children do exist with some, but not all, of the features of childhood autism. Matters become even more confused when terms like "Childhood Schizophrenia", with its unfortunate inherent implication of a link with adult schizophrenia, are used by some to refer to children who do essentially fit Kanner's syndrome (Goldfarb 1961; Creak 1964; Wolf and Chess 1964), and by others to differentiate autistic children from an unrelated non-autistic group (Bender 1960; Treffert 1970). Goldfarb (1970) has commented on how often selection criteria for experimental subjects are only broadly outlined in research into childhood autism. Widely differing samples are often used in research. This is frequently a function of the referring source, programme requirements in hospital and research centres, and educational facility variations. Rimland (1974) puts the case most strongly with regard to treatment: "There is no doubt in my mind but that progress on prevention and remediation of the childhood psychoses has been impeded - I would even go so far as to say halted - by the virtual stalemate that exists in the area of diagnosis." (p.140).

Rimland (1964) designed a comprehensive checklist for childhood autism consisting of 80 questions to be answered by the parents. From this checklist a score on the 'E-2 scale' can be recorded, and he claimed that such scores will differentiate autistic children from a variety of other childhood psychoses. The questions yield data on prenatal history, degree of cerebral insult, early childhood and current behaviour. He reported that data collected from over two thousand children referred to him as 'psychotic' isolated only 9.7% who suffered from infantile autism when measured on the E-2 checklist (Rimland 1971). An item analysis revealed that individual items in the checklist discriminated properly between autistic and non-autistic children, and the E-2 scale also differentiated a sample of autistic children diagnosed by Kanner from a group of psychotic, non-autistic controls (Rimland 1974). Thus validity checks for the E-2 scale to date, appear favourable.

In this country, one of the most frequently used diagnostic schemes has been that resulting from a British Working Party report in 1961, chaired by Mildred Creak. This described nine criterion pointers to isolate a group of children who fitted what was called "The Schizophrenic syndrome of Childhood". The scheme lists criterion symptoms which largely correspond to those delineated by Kanner, although they encompass a slightly broader range of behaviours. The Working Party did not say how many criteria should be present before diagnosis should be made, although they stressed that gross impairment of emotional relationships, and retardation with islets of intelligence, were key pointers. The other criteria consisted of unawareness of personal identity, abnormal preoccupation with and attachment to objects, strong resistance to change in the environment, abnormal responses to perceptual stimuli (a mixture of hypo- and hypersensitivity), absence or retardation of speech, with echolalia and pronoun reversal common, acute and

excessive anxiety, and distorted motility patterns. Hermelin and O'Connor (1970) suggested that it would not be unreasonable to claim that children displaying four or more of these pointers may be considered as autistic. This system has the advantage of allowing a limited degree of variability in symptomatology whilst still isolating subjects from most other childhood disorders.

A well-designed Australian study (Clancy, Dugdale and Rendle-Short 1969) concluded that it is better to think of autistic children having a characteristic pattern or cluster of symptoms rather than an exclusive set of them. They broke down the nine pointers into fifty-four individual behavioural items and gave them in questionnaire form to the parents of diagnosed autists, and control groups consisting of parents of normals, subnormals, deaf and cerebral palsy children. In an attempt to ascertain the discriminative power of each of the behavioural items, a statistical cluster analysis was carried out on the data. This revealed that fourteen items appeared to characterise autistic children, and the authors concluded that a comparatively 'pure' group of autistic children may be selected if a minimum of seven items is present in each child.

Aetiological biases undoubtedly colour diagnostic considerations. Menolascino (1965) remarked that it would appear that the theoretical inclination of a given investigator, rather than the behaviour of the child, may determine the primary diagnosis. Whilst this assertion is a rather strong generalisation, it is undoubtedly a contaminating factor in some of the clinical and experimental literature. For example Kanner specifically excluded subjects with signs of cerebral insult, but most others do not. Now the question of an association between neurological impairment and childhood autism is equivocal, and this will be fully discussed. But for diagnostic purposes, no clear grounds can be cited for supposing that cases of autism with signs of neurological impairment

are fundamentally any different in behavioural terms from cases without such signs. The solution to this problem would appear to be found in the adoption of a system in which such factors as intellectual functioning, suspected neurological impairment and other associated factors are classified along separate axes to the behavioural diagnosis. Such a system has been advocated (Rutter et al., 1969; Rutter 1971, 1972). As a general approach to the classification of mental disorders in childhood, it was proposed that diagnosis of the syndrome would be restricted to axis 1, the intellectual level would be assessed and recorded on axis 2, and any possible aetiological or associative factors (such as neurological impairment indices) on axis 3. Thus each child would be diagnosed without reference to an intelligence or neurological assessment, the latter measures being used for treatment, educational and research purposes, but remaining essentially independent of diagnostic considerations. A recent evaluation of the usefulness of this system produced some encouraging results (Rutter, Shaffer and Shepherd, 1973). Their reliability study, and a clinical study, suggested that a multi-axial classification of mental disorders had distinct advantages over the International Classification of Diseases (ICD) system, particularly where specific childhood syndromes are concerned (e.g. childhood autism, hyperkinesis). Not least of these advantages is that because the former system is descriptive rather than theoretical, it can be used in a more consistent fashion by clinicians of differing theoretical orientation.

Rutter (1971) suggests that diagnosis should be restricted to children displaying three particular groups of abnormality:

- i. A profound and general failure to form social relationships.
- ii. Language retardation with impaired comprehension and echolalia.
- iii. Ritualistic and compulsive phenomena.

Other symptoms found in these children, but not unique to them, include stereotypy, short attention span, self-destructive behaviour, and delayed bowel control (Rutter and Bartak, 1971; Rutter, 1974). With the exception of 'good' cognitive potential, this system strongly resembles that of Kanner's original delineation. With the adoption of a multi-axial classification scheme, these two schemes should select very similar populations.

DeMyer et al., (1971) compared several diagnostic systems for childhood schizophrenia and infantile autism, including those of Rimland (1964), Polan and Spencer (1959), Lotter (1966), and Creak et al., (1961), and found high correlations between them. Indeed, there have been many other diagnostic schemes, well summarised by Hingten and Bryson (1972), and a large proportion of them bear a strong resemblance to Kanner's original proposals and to those of the British Working Party. Where they do differ they tend to do so by being coloured with aetiological considerations, often concerning neurological signs and intelligence scores, typically adding points to the two schemes mentioned rather than directly contradicting them. Thus whilst not necessarily disagreeing with Eisenberg's recommendation that treatment considerations should be based on clinical assessment of the possible underlying aetiology (Eisenberg 1972), international adoption of a multi-axial classificatory scheme promises considerable consensus with regard to the children concerned in the differential diagnosis of childhood autism. That is not to say that differential diagnosis is a simple matter. Indeed, Ornitz (1973) summarises the difficulties thus: "Of the many types of unusual behaviour seen in young children, the behaviour of the autistic child has been the most difficult to understand. The greater variability of the abnormal behaviour, the changes concomitant with the maturation of the child, the wide differences in degree of severity from case to case, the confusing

and inconsistent terminology which has been used to describe such children, and the lack of any physical signs have made diagnosis in the individual case a difficult and often unreliable procedure".^(p.21)/ However, the similarity between many of the numerous diagnostic schemes that have been outlined, supported by data from correlation studies like those of DeMyer et al., (1972), suggest that despite possible differences between the experimental samples isolated by the various diagnostic criteria and the likely heterogeneity of subjects within such samples, significant overlap between many of them does appear to exist. Comparison of experimental findings between studies is therefore legitimate provided we remain aware of the possibility of diagnostic variance affecting these comparisons, and we note carefully descriptions of symptoms displayed by subjects.

Having stated that a significant overlap in symptomatology exists between Childhood Autism and certain other childhood disorders, it is necessary to differentiate Childhood Autism from them. Ornitz (1973) succinctly dismisses any potential confusion between childhood autism and children suffering from major sensory defects, developmental aphasia, and environmentally deprived children. He listed several symptoms that are common in autistic children but do not appear in the other childhood disorders noted. Two differentiating characteristics that are not found in any of these non-autistic groups include the unusual hypo- and hypersensitivities, and the unwillingness to communicate found in autistic children. The extensive and profound linguistic defects of autistic children also appear to differentiate them even from those children with specific developmental receptive language disorders (Bartak, Rutter and Cox, 1975). Autistic children were found to differ from this latter dysphasic group in terms of hypersensitivity to sound, echolalia and pronoun reversal, and metaphorical language.

It appears that where language does develop in autistic children, it is not just a question of delay, but one of marked deviance in addition to being a more severe and extensive deficiency in language than in the receptive language disordered child.

The validity of the syndrome of childhood autism has been questioned most frequently however with regard to schizophrenia and mental retardation. There are those who consider autism to be simply a variety of the subnormality population, and those who see it as a precursor to adult schizophrenia (e.g. O'Gorman, 1967; Bettelheim, 1967; Bender, 1968). One can hardly consider the term subnormality as encompassing an homogeneous population, but there are many features which suggest that we cannot consider the problems of the autistic child to be solely related to subnormal functioning (Rutter, 1968, 1974; Ornitz, 1973; Clancy, Dugdale and Rendle-Short, 1969). The question would seem to be not so much whether autistic children intellectually function at the subnormal level, for clearly the vast majority of them do, but rather whether they have deficiencies and disabilities over and above those of simple intellectual retardation. Nevertheless, the question of the measured intelligence of autistic children, and the distribution of their scores, has proved to be a controversial issue until comparatively recently. Kanner (1943), Bettelheim (1967) and Rimland (1964) all suggested that autistic children are basically of normal intelligence. Such an assertion was largely based upon the reported unusual and outstanding isolated abilities and skills of these children, typically in the form of case-history data (Kanner, 1943; Cain, 1969). Empirical studies however almost invariably reveal a distribution of scores that skews heavily towards the lower end of the curve for the general population. DeMyer et al., (1974) reported that 97% of a large sample of autistic children scored in the subnormal range on the Cattell-Binet and

DeMyer profile test, and a six-year follow-up revealed strong longitudinal consistency (a correlation of over + 0.7 with initial scores). Similar, although rather smaller, figures have been reported in several studies (Gittelman and Birch, 1967; Lotter, 1966; Kolvin, 1971; Rutter, 1974; Gubbay et al., 1970). It is of course possible that low I.Q. scores are a function of 'masking' as a result of withdrawal, disturbed behaviour or negativism. Alpern (1967) rejects this however by predicting that the performance of autistic children on intelligence tests would vary considerably with fluctuations in their clinical state if this possibility were so. He reported very stable test performances over a short period of time, and others have reported high correlations in longitudinal studies (Lockyer and Rutter, 1969; DeMyer et al., 1974). Nevertheless, it is clear that autistic children do not make good testees. Their cooperation is frequently poor, attention span is limited and motivation is frequently questionable.

There are an increasing number of experimental studies demonstrating the operation of an active process of non-compliance, or negativism, which could potentially invalidate many standard test scores (Cowan, Hoddinott and Wright, 1965; Ney, 1967; Wallace, 1975). One must also question the validity of the apparent longitudinal consistency in I.Q. scores by asking how much this simply reflects the inappropriateness of many tests for these children. Mittler (1968) takes a most controversial line by suggesting that autistic children gain the label 'untestable' due to poor cooperation, attention span etc, because they only complete a very limited number of items on tests like the WISC and Stanford-Binet, and psychologists do not wish to commit themselves on such partial test scores. He suggests that the term 'untestable' tells us more about the tester than the child. This implies a complete disregard for the professionalism of psychometricians and the highly

limited validity of partial test scores. A more pertinent implication of the term 'untestable' would seem to be the appropriateness of the test selected, for the administration of intelligence tests with high verbal factor loading simply tells us what we already know - autistic children have severe language defects and may not even understand many of the test items presented.

Despite the above criticisms, the intelligence test scores of autistic children do appear to demonstrate how these children differ from those included in the general category of subnormality. Several autistic children do score well within the 'normal' range on standard intelligence tests. Rutter and Lockyer reported that between $\frac{1}{4}$ and $\frac{1}{3}$ of their subjects fell within this category, and findings of smaller but significant numbers of similar subjects have been published elsewhere (Kolvin et al., 1971; Rutter, 1968; Lotter, 1966). Similarly, unlike subnormals, wide differences consistently appear between several subtests within full-scale intelligence scores. Verbal item scores tend to be markedly below those of performance items on the WISC (Rutter, 1966; Wassing, 1965; MacCulloch and Sambrooks, 1973), with a characteristic profile emerging in which particularly low scores on 'vocabulary' and 'comprehension' are matched by superior scores on 'block design' and 'object assembly' items. Autistic children and subnormals also differ on their profiles on the Illinois Test of Psycholinguistic Abilities (ITPA), where the former have defects in almost all aspects of language functioning except auditory-vocal sequencing and especially depressed scores in encoding functions and in cross-model coding (Tubbs, 1966). The encoding deficiency appears on both verbal and non-verbal levels, and may be regarded as indicative of an expressive or communicative defect. Autistic and subnormal children also appear to differ in their

patterns of play (Tilton and Ottinger, 1964; DeMyer et al., 1967), with the autistic children displaying more oral and repetitive play and less combinational use of toys. Autistic children tend to display little desire for communication, and also unusual mixtures of hypo- and hyper-sensitivities, neither of which appear consistently in children classified as subnormal (Ornitz, 1973).

The particular deficiencies in communication and interpersonal relationships in autistic children have led some to the view that childhood autism may be a precursor to adult schizophrenia, and therefore a childhood variety of the same syndrome (O'Gorman, 1967; Bettelheim, 1967). Ornitz and Ritvo (1968a) remark that:

"... in our clinical experience with children we have observed clear transitions from early infantile autism to the thought disorder characteristic of adult schizophrenia" (p.78)

Rutter (1968; 1974) strongly disagrees, and summarises several findings which would appear to conclusively separate the two conditions. The incidence of autism is significantly greater in males than females, figures ranging from 2:1 to over 4:1 (Rutter, 1967; Kanner, 1954; Kolvin et al., 1971), whereas the sex ratio in schizophrenia is approximately unitary (Rosenthal, 1970). The high social class background found in autistic children (Treffert, 1970; Rimland, 1964) is not matched in the parents of schizophrenics (Hollingshead and Redlich, 1962), and neither is the incidence of subnormal intelligence (Pollack, 1960). A history of mental illness in the family is frequently found among cases of schizophrenia (Shields, 1967; Rosenthal, 1970), whereas this is a relatively uncommon finding in infantile autism (Gittelman and Birch, 1967; Lotter, 1967). Rutter finally points out that fluctuations in pathological status, hallucinations and delusions are all common in schizophrenia but very rare in autistic children, even in adolescence. Whilst there are those who still maintain that childhood autism is a precursor to adult

schizophrenia, one must consider the possibility that such research is conducted on rather different populations to those whose findings clearly contradict this assertion. In particular, the diagnostic label of 'Childhood Schizophrenia' appears to have led to the most confusion, as Rutter (1972) suggests: "Childhood Schizophrenia has tended to be used as a generic term to include an astonishingly heterogeneous mixture of disorders with little in common other than their severity, chronicity, and occurrence in childhood." (p.315)

Whilst the situation may not be as chaotic as this universally, it is clear that the diagnosis of childhood autism has proved to be a source of considerable controversy, confusion and conflict. It is essential that a unification of diagnostic categories and criteria be established, and one can only hope that the adoption of the multi-axial scheme as outlined by Rutter is internationally adopted. This may not solve the problem altogether, but will lead to a marked improvement in the capacity to communicate research findings on comparable populations.

EPIDEMIOLOGY

Prevalence

The prevalence of childhood autism has not been extensively studied, but those figures that have been published are closely in agreement and are very low. Lotter (1966) and Rutter (1967) recorded an incidence of 4/10,000 of the population. Lotter's study was particularly comprehensive, involving a screening of the entire

population of 8-10 year old children in a whole county. Treffert (1970), commenting on the lack of a sizeable prevalence study outside the U.K., carried out a five-year survey on all the Mental Health Clinics and hospitals in Wisconsin. He found 280 cases, which yielded a rate of 3.1/10,000.

Sex Ratio

Many epidemiological studies have demonstrated an excess of male over female cases of childhood autism. Typical ratios range from 2.6: 1 (Lotter, 1966) to 4.8:1 (Spence et al., 1973). Figures of 2.7:1 (Meyers and Goldfarb, 1961), 4:1 (Kanner, 1954), 3.5:1 (Creak and Pampiglione, 1969) and 4.3:1 (Rutter, 1967) all fall within this range. These findings of an excess of male cases is in line with figures for other childhood disorders, and especially those of suspected organic aetiology (Graham and Rutter, 1968; Lowe, 1966). These ratios are therefore not unique to childhood autism, but reflect the apparent greater susceptibility of the male to a variety of biological defects, including pre- and peri-natal injuries, and also the display of a wider range of behaviour than the female along a number of dimensions (Hutt, 1972). Anthony (1970), in a review of many studies, stated that: "... it is extremely difficult to find a pathological condition in which the incidence among girls is greater than among boys, and some authors have spoken of the handicap of being male". The sex ratio found in the incidence of childhood autism cannot however be used as conclusive evidence for

a biological aetiology, since we know that potentially powerful sex roles can operate from very early years in the child's life (Mischel, 1966; Bandura and Walters, 1963). Nevertheless it is a finding that must be considered with respect to similar ratios found in cases of known organic childhood disorders. Of particular interest is a report by Meyers and Goldfarb (1961) that on sub-dividing autistic children into those displaying clear signs of cerebral insult and those who do not, the male-female ratio of the former group proved to be significantly higher than that of the latter.

Birth Order

There have been a number of reports suggesting that childhood autism is more common among first-borns (Kanner, 1954; Rimland, 1964). However, other studies find no consistent significant relationship with birth order (Lotter, 1967; Wing, 1967). Since there is apparently no exclusive correlation with any particular birth rank, it would not appear to be of crucial aetiological significance. Whilst Wing suggests that if a first-born is autistic there may be a tendency to have another in the hope that it will be 'normal', it could equally be asserted that parents may be discouraged from further children and risking another 'abnormal' child. This may be particularly so in the case of autistic children since their behaviour demands excessive attention and is frequently highly unrewarding to parents. Neither explanation appears specific to childhood autism.

Maternal age at birth also appears to be uncorrelated with childhood autism (Schain and Yannet, 1960; Lotter, 1967)

Parental Social Class

Many investigators have reported that autistic children come from families of high socio-economic status, high intelligence and considerable educational attainment (Kanner, 1949; Rimland, 1964, 1974; Williams and Harper, 1973; Lotter, 1966). Although there are those who disagree with this assertion (e.g. Lowe, 1966; Bettelheim, 1967), whilst some studies suggest that the range of intelligence scores and social class backgrounds extends to lower levels despite high mean scores (Florsheim and Peterfreund, 1974), the overwhelming majority of studies agree with Kanner's original proposal that the parents of autistic children are of exceptional intelligence and from the 'upper' social classes. There is considerable disagreement on the significance of this finding however. Some regard it as indicative of a pathological parental factor, others suggest that it has implications concerning the genetic basis of autism (Kanner, 1949; Rimland, 1964; Ounsted, 1970), whilst still others see it as having no special clinical relevance at present (Ornitz, 1973).

It is clear however that we must consider the possibility that the high correlation between parental social class, intelligence, and childhood autism may be an artefact reflecting the varying efficiency of social and educational services, or particular types of parent being more able to use health services. Although Rimland (1974) strongly refutes the operation of such factors on the grounds of the diagnostic criteria used in non-confirmatory studies, there are more powerful arguments. Foremost amongst these are Lotter's findings in agreement with Kanner's assertion, and based on a screening survey of the whole 8-10 year old population of Middlesex (Lotter, 1966; 1967).

Prognosis

Follow-up studies on autistic children have generally reported a poor prognostic outlook (DeMyer et al., 1973; Lotter, 1974a; Kanner, 1971; Gittelman and Birch, 1967). Some symptoms, typically ritualistic behaviour and resistance to change, often diminish with increasing chronological age. However, social adjustment remains poor in the vast majority of cases, and continued institutionalisation appears to be common. Lotter (1974a) followed up 32 children found in his original survey and found that compared with controls with other severe behaviour disorders but of similar I.Q., the autistic children had a much lower frequency of employment. This seemed to be only partially related to poor motivation and concentration, a failure to appreciate social context being an equally important factor in unemployability.

Kanner, Rodrigues and Ashenden (1972) studied the case histories of 96 patients and found that only eleven had made sufficient progress in social adaptation to gain full-time employment. None of these had been institutionalised but had developed some speech by the age of five. However these were apparently not universal prognostic signs, for these features also characterised other cases where no such improvement had occurred.

Outcome has been related to the degree of language impairment, I.Q., and the original severity of the behavioural symptoms (Eisenberg, 1968; Gittelman and Birch, 1967; Lotter, 1974b). Rutter noted that if marked improvement will occur then it is usually evident by the age of six (Rutter, 1966). For those who function at the severely subnormal level the outlook is very poor regardless of other factors. For those who score above 50 on standard intelligence scales it appears that a 'fair' improvement is the most that can be expected. Those autistic children with near-normal non-verbal intelligence scores have the best

prognostic signs, especially if their language impairments are less than severe (Rutter, 1974).

Hington and Bryson (1972), in an extensive literature review, concluded that whilst the therapeutic outlook has certainly improved recently, prognosis for 'normal' functioning is most remote. They suggested that: "Until each child's specific limitations of learning are objectively determined, all methods of proven effectiveness must be used to enable him to reach his highest level of performance". One can hardly disagree with this statement although in itself it reflects the highly unsatisfactory prognostic situation. It is to be hoped that the results of longitudinal comparative educational studies and also follow-ups to home-based approaches to treatment (Howlin et al., 1973a), will yield data on important intervention factors that have implications for more promising prognoses.

AETIOLOGY

An Introductory Comment

There are those who believe that an understanding of the underlying aetiology of the condition is not necessary for a treatment programme for childhood autism, and we may therefore adopt an heteropathic approach in which cause and treatment are considered separately (e.g. Kozloff, 1973). This approach implies that the aetiology of the condition is largely an academic affair. However,

there are many who disagree with this view and consider that research into aetiological factors is of paramount importance for treatment. MacCulloch and Williams (1971) summarise the latter position thus: "Whilst these practical therapeutic approaches have in themselves importance, they are likely to remain less than maximally effective in the continuing absence of any established cause for the syndrome". Despite a commitment to objective aetiological research, inherent in a belief that treatment will depend upon such research data, there has been surprisingly little consensus achieved with regard to aetiological hypotheses.

It is true to say that there is a growing consensus amongst researchers that childhood autism is probably primarily the result of an organic defect, although the specific nature of this defect is still a matter for continued research and debate. Over the last thirty years however, the diversity of aetiological hypotheses has been both spectacular and confusing, and is well illustrated in any of the recent comprehensive literature reviews (e.g., Rutter, 1968, 1974; Hington and Bryson, 1972; Ornitz, 1973). Whilst a number of theories have stressed the interaction between biological and environmental factors, many have taken extreme 'psychogenic' or 'organic' positions. The authors of most literature reviews have therefore tended to structure them as parallelling a 'nature-nurture continuum', and it will be necessary to review the literature published on childhood autism in a similar manner since this is the way research has approached the question of aetiology.

Psychoanalytic approaches

The most frequently cited proponent of a psychodynamic approach to aetiology has been Bettelheim (1967). He suggested that from the earliest days, the child attempts to modify his environment by activity like crying and wriggling. He gradually learns that his different activities lead to differing maternal reactions, and his self-concept begins to develop together with communication. Developmental 'stages' appear at characteristic intervals. If a child is unusually sensitive, intelligent, or 'early' in his responses to the environment, a discrepancy will exist between his efforts to act upon the environment and his state of physical maturity. If, in addition to this, the environment is insensitive to this lag, inner frustration may result. Bettelheim (1967) proposed that this is the case in infantile autism. Because his 'world' is insensitive in this way, the autistic child feels that he has no control over his environment. Motivation and predictions about environmental events become severely impaired, and he attempts to protect himself by not acting through a process of increasing withdrawal, which results in a weakening of the self-concept and subsequent disintegration through lack of use. This description strongly resembles Laing's account of schizophrenia (Laing, 1960), and indeed Bettelheim regards childhood autism as a precursor to adult schizophrenia.

In contrast to others who also stress the importance of mother-child relationships in aetiology, Bettelheim maintained that therapy based on attempts to modify the mother's behaviour, and the study of her personality, results in an addition to the very type of pathological process seen as crucial in aetiology. Thus the child who suffers most

from failing to acquire an autonomous personality is again not regarded as an autonomous entity in therapy and research.

Bettelheim reports high success rates using 'parentectomy' in therapy. Complete mother-child separation is maintained for several months, and in some cases parents are told that it may be for an indefinite period. Such a policy is of course highly emotive, and has inevitably received vitriolic criticism (Rimland, 1964; Schopler, 1971, 1974). Bettelheim advocates great tolerance of aberrant behaviour during therapy, the orientation being towards enabling the child to learn that he can control his environment, whilst being free from the emotional demands of the mother. Empirical studies however have invariably found that excessive tolerance of non-desired behaviour is ineffective in terms of behavioural change in any long term sense. Indeed many studies find that such tolerance actively maintains some aspects of the child's inappropriate behaviour (Kozloff, 1973; Moore and Bailey, 1973), and that the autistic child benefits most from a structured environment and from the use of parents as cotherapists (Stevens-Long and Lovaas, 1974).

Bettelheim's hypothesis also becomes weakened by recent findings which clearly suggest that much of the autistic child's behaviour is a function of genuine cognitive/perceptual deficiencies rather than any volitional withdrawal process. Thus Bettelheim believes that pronoun reversal, a frequently cited characteristic of autistic children, is a symptom of a psychic defence mechanism. He asserts that autistic children who have spontaneous echolalic speech and who do not use "I" to refer to themselves, will readily use "You", but never repeat "I". Bartak and Rutter (1974) empirically tested this assertion, and produced data which completely refuted it. It appears that pronoun reversal is a misnomer, rather it is one example of echolalia. Autistic children appear to avoid the use of "I" because it typically occurs at the

beginning of sentences and echolalia normally occurs with the last part of an utterance. When placed at the end of an utterance, autistic children readily repeated "I", contradicting the view that the child is rejecting his own existence when displaying apparent pronoun reversal.

Mahler (1949, 1952, 1960) also proposed that a number of intra-psycho processes operate in the aetiology of childhood autism, and suggested that the normal child passes through three stages:

- i. An autistic 'normal' phase from 0-3 months when the child is aware only of inner stimuli.
- ii. A symbiotic phase from 3-18 months during which he is aware of the external supply of his needs, but has no clear self-concept.
- iii. A separation-induction phase in which a self-concept develops, interpersonal relations first appear with the mother, then with members of the family and then peers.

Mahler proposed that the autistic child functions at the first stage, suggesting that while some had never developed out of it, others had regressed due to a breakdown in the mother-child relationship. The contributory factors leading to the aberrant mother-child relationship included a surprisingly heterogeneous list, amongst them being excessive proprioceptive stimulation, unpredictable mothering behaviour, and many possible traumatic experiences.

This hypothesis appears to be less than specific with regard to identifying aetiological factors, lacks any empirical support, and is clearly incorrect in the description of the first phase of normal child development. Abundant research now demonstrates that in the first few weeks of life the normal child is aware of far more than 'inner' stimuli (e.g., Bower, 1971, 1974; Kagan, 1972).

Garcia and Sarvis (1964), whilst stressing that a multi-factor aetiology is the most plausible view in the light of qualitative and quantitative differences amongst autistic children, also adopted a psychoanalytic approach. They saw autism as a reaction to an overwhelming inner or outer 'assault' at a vulnerable age (6-18 months), when the child is developing a self-concept. The autistic child construes any stress as being due to the mother's persecution, and thus himself reacts with a rejecting attitude. These analysts see this basic paranoid reaction as the core of childhood autism, for if the mother meets this rejection with counter-rejection or withdrawal, then the child becomes 'frozen' in this state of paranoid rejection. Similarly, Fraknoi and Rutter (1971) hypothesized that the autistic child's mother does not offer adequate protection from overwhelming stimuli and gratifications, nor gives the child a sense of security. Autism was seen as a defensive mode of behaviour which the child adopts in order to cope with stress and frustration. Such strong assertions concerning 'rejecting' or 'under-protective' mothering requires substantial evidence that appears to be lacking (DeMyer *et al.*, 1972; Cox *et al.*, 1975).

Many other psychoanalytic approaches have been published (Anthony, 1958; Rank, 1949; Boatman and Szurek, 1956), although noticeably fewer in the past decade. They all share the same emphasis on deviant mother-child relationships, but lay the aetiological stress upon maternal pathology. It is however rather doubtful whether all the children studied, and in particular those of Rank and Szurek collected under the term 'atypical child', do in fact conform to an acceptable definition of childhood autism. This criticism has been levelled at many of the psychoanalysts, who are frequently less than rigorous in their selection of cases and use of diagnostic criteria. The non-empirical constructs in which psychoanalytic work is set, and the accompanying

lack of experimental support, makes acceptance of their aetiological formulations rather difficult. Psychoanalytic hypotheses involve highly subjective interpretations of behaviour, and it might be thought that validity of the hypotheses therefore depends upon therapeutic results. This however, would be an oversimplification, for 'parentectomy', 'warm', or 'nurturant' surroundings, and psychotherapy may all be simply providing a beneficial environment without in any way supporting a specific aetiological position. Treatments based on analytic principles are in any case not significantly better than those based on other approaches (e.g., Ney, Palversky and Markeley, 1971).

The psychoanalysts' belief in maternal behavioural pathology has led to much debate, and this will be discussed later in relation to Kanner's writings on the subject of childhood autism. However, it should be mentioned at this juncture that little support has been found for psychoanalytical formulations. On the positive side, one can say that their approach has facilitated longitudinal study and assessment through analysis of detailed published case histories.

Other Psychodynamic Formulations

Clancy and McBride (1969) investigated autism as a developmental process within a complex family system. They stressed that primary socialisation must be viewed in terms of complex family dynamics and feedback mechanisms. During its first few months, the infant is in contact with other family members who have their own affiliative bonds, and the child's non-verbal communicative behaviour reinforces interactions with them, thus promoting bonds with each. The family members thus have to modify their pre-existing relationships to incorporate the new individual. Clancy and McBride point out that changing one relationship potentially changes all others, and thus for example the mother's relationship with her child will change her relationship with her husband and with other children.

With the autistic child, the mother often reports abnormalities in the child's behaviour in the first year of life, with typical symptoms including absence of smiling, lazy sucking, and lack of response to a human voice. Whilst these are present in some normal children, only autistic children appear to have a whole range of these characteristics which function to shape an inadequate mother-child relationship. The process is seen as interactive, with the child's abnormal behaviour eliciting inappropriate responses in the mother which in turn induce further aberrant behaviour in the child. The authors suggest that the mother's responses are characteristic, and different from those of mothers of other behaviourally difficult children. The behaviour of the autistic child becomes so aversive to the mother that contact between them becomes rapidly reduced to a minimum, thereby inhibiting the whole process of bond formation. As he grows older, the autistic child improves the skill of 'cut-off' behaviours (Chance 1962), and the family learns not to intrude on the child's isolation as it stimulates tantrums. The

obsession with sameness is seen as providing powerful negative feedback for any change in the existing set of conditions. The absence of a reciprocal mother-child bond eliminates the normal socialisation process and the subsequent acquisition of meaningful speech.

Clancy and McBride set out a treatment programme aimed at involving the whole family and not just isolated features of the child's behaviour. Intrusion upon the child's behaviour is advocated to establish a primary socialisation process, and this is in complete contrast to Bettelheim's 'permissive' programme, which is seen by these authors as the most detrimental of policies. If the child's 'cut-off' behaviour is tolerated, then he shapes the behaviour of the family and also achieves the isolation which is the very core of the problem in the autistic child. With the initial use of play media and gradual shaping, Clancy and McBride claim that many subjects become more amenable to physical and then social contact as the interactions continue, and bonds and stranger responses later appear (Clancy and McBride, 1969).

The stress upon changes in the family, and in particular the relations between members, is an important point in relation to treatment processes. It is not clear however whether it is of direct aetiological significance. No evidence is offered to support the view that parental behaviour, seen in the light of the retrospective nature of clinical diagnosis, was fundamental in the pathological process of a failure in socialisation in the autistic child.

Zaslow and Breger (1969), like Kanner, stressed the importance of the lack of a basic capacity to relate in autistic children. Human avoidance was seen as matching attempts at mastery of the non-human environment by a variety of self-initiated actions. Interpersonal schemas fail to develop due to deficiencies in the mother-child relationship and 'negative motor resistance' appears. Rage reactions or excessive pass-

ivity keeps the child independent of humans, and these reactions later develop into negative perceptual and cognitive styles. Negativism displayed towards specific figures in the environment demonstrates the child's capacity to relate to others only on his own terms. Autism is seen as the result of an interaction between a particular type of child and a particular mode of parental handling. The child's characteristics are those which lead to difficulty in holding, cuddling and orientating, and often they appear to be aggressive or hyperactive.

Zaslow and Breger report that in their clinical experience they have found the parents to be lacking in the perseverance required to cope with a difficult child. They also interpret the symptom "insistence upon sameness" in the environment as indicative of a greater reliance on place, rather than person, security. Similarly, attachment to objects is seen as an attempt to replace people with them in order to 'master' a threatening world. These interpretations bear a resemblance to some of Hudson's observations on the personality attributes of 'convergent' and 'divergent' thinking styles (Hudson, 1966). Hudson suggested that 'convergers' typically concentrated on the impersonal features of life, being most reluctant to express emotions although this rigid inhibition masks normal experiential aspects of emotion. He proposed that such a style of expressive cognition is learnt from parents who also avoided expression of strong feelings but reinforced their child for developing impersonal skills, leading to the child's perception that security lies in an impersonal field. Whilst certain characteristics of autistic behaviour may conceivably be interpreted as an extreme, pathological 'convergent' thinking style, it is clear that the normal intellectual features of convergent thinking are entirely absent.

Zaslow and Breger's hypothesis is a coherent argument, but rests almost solely for its evidence upon a particular treatment process.

This is called "rage reduction therapy", and the technique consists of reproducing the mothering act of picking up and physically comforting, and also restraining, the child when it cries during a stressful situation. Negativism is confronted and 'worked through' in an attempt to establish a positive affective relationship resembling that between a normal mother and infant. Whilst the authors claim that this technique is highly successful, it does not in itself constitute sufficient grounds for endorsing the aetiological process proposed. Zaslow and Breger's model shares much in common with that of Clancy and McBride, attributing aetiological significance to aberrant mother-child bonding. Neither are able to offer empirical support for this assertion, or make specific the nature of the deviant behaviour on the part of the mother that they feel to be crucial in the development of childhood autism.

Leo Kanner however, was most explicit in his description of the role and nature of parental involvement in the aetiology of this condition. After outlining the syndrome he labelled "infantile autism" he proposed that the fundamental disorder lay in the child's inability to relate himself to situations, and in particular people, from birth onwards (Kanner, 1943; 1949; 1965). This he envisaged as an inborn "autistic disturbance of affective contact". Despite the apparent similarity to the withdrawal seen in schizophrenia, Kanner maintained that the inability to relate to others appears from birth and is not in any way a withdrawal from formerly existing relationships. Kanner (1943; 1949) emphasised that autistic children have highly intelligent parents, few of them really warmhearted, and they are often professionals being pre-occupied with scientific, artistic or literary subjects with strictly limited interest in people. The mechanisation of their attitude to human relationships struck Kanner very forcefully in his clinical interviews with them, leading him to use the term 'emotional refrigeration'.

He illustrated his claim that the parents fitted a stereotype with very striking and dramatic case history reports (Kanner, 1943; 1949).

Kanner could find no consistent history of mental illness or marital problems in the families, and this differentiated such parents from those of schizophrenics or emotionally disturbed children. He also claimed that despite speech delay and peculiarities, autistic children do have extraordinary isolated abilities.

Kanner clearly believed in an interaction between a predisposing, biological factor, and environmental variables in the aetiology of the condition, suggesting that genetic and biochemical work may shed light on crucial factors (Kanner, 1971). Whilst he proposed that parental characteristics play an important part in the development of the syndrome, he stressed that this "... is not sufficient in itself to result in its appearance". (Eisenberg and Kanner, 1956). This point is often ignored in many critiques, where Kanner is regarded as the ultimate environmental protagonist.

Nevertheless, the question of parental abnormalities or behaviour pathology has been a central issue in aetiological controversy since the recognition of childhood autism. Eisenberg (1957) in a study of 100 cases of infantile autism, reported that 85 of the fathers showed clear stereotyped features. They tended to be obsessive, detached, humourless, and of very high I.Q. and socio-economic status. Perfectionists, but not creative, they regarded marriage as socially convenient and children as a socially expected norm. The detached behavioural symptoms of their autistic child, were seen at first as an asset by the fathers, since for them work took precedence over family life. Of the 15 fathers who did not apparently fit this stereotype, Eisenberg claimed that 11 had wives with "obvious maternal psycho-

pathology". It is worth noting that of 131 siblings in the sample studied, only three were considered to be autistic and another five emotionally disturbed. This leaves the question to be asked of why parents of autistic children were able to bring up other children as normal if their extreme behaviour is fundamental to the aetiology of their autistic child. It seems difficult to accept that this is solely because parents adopt differing attitudes and styles of rearing to their children. It may be argued that parents change their behaviour once they have had one autistic child, and this explains why so few siblings are also autistic. However, this assumes that most autistic children are first-born, and the available data suggests that this is not the case (Wing, 1967; Lotter, 1967).

Anecdotal evidence and impressions from parental interviews are certainly insufficient grounds for accepting the role of parents in the aetiology of childhood autism. A number of psychometric analyses have been published, but few provide any substantial support for a psychogenic model. Ogden et al., (1968) found 15 significant differences out of 17 features studied, between parents of autistic and parents of normal control children on the Rorschach test. Bene (1958) using the same test, reported results that suggested mothers of 'primary' autists to be less frequently able to establish social and emotional behaviour than mothers of normal children. Parish (1969) used the Edwards Personal Preference Schedule (EPPS), and the Parental Attitude Research Instrument (PARI) to look for a common mode of attitude towards child rearing in parents of autistic children. He found the greatest 'need' for both parents was to have 'deference' and 'abasement', and although they appeared to agree

on child-rearing practises, they were characterised by being obsessive, irritable, and self-abasing. Goldfarb, in a series of studies (Meyers and Goldfarb, 1961; Goldfarb, Goldfarb and Scholl, 1966; Goldfarb, Levy and Meyers, 1972), reported a characteristic mode of responding in some parents of childhood schizophrenics (diagnosed on the Creak criteria) which he labelled "parental perplexity". Such behaviour included marked uncertainty, lack of empathy and spontaneity, inconsistent parental control, and general bewilderment. These findings were based on a variety of rating measures.

It is certainly not clear that this type of retrospective testing isolates any factors of primary aetiological significance, and it is clear that other studies yield negative results on parental personality tests (e.g., Pitfield and Oppenheim, 1964). DeMyer et al., (1972b) used rated interview and questionnaire data with parents of normal, autistic, and brain-damaged children. Parents of the autistic and normal children were found to be the most similar in infant care practises, and parents of subnormals appeared as the most 'cold' and least stimulating. Cox et al., (1975) attempted to test for psychogenic factors with a design aimed to maximise the opportunities for any such factors to "show" by using autistic children of 'normal' non-verbal intelligence and with no signs of organic impairment. The control group used were "dysphasics", who present language problems for their parents not unlike that of autistic children. Using the Leyton Obsessional Inventory and structured interviews with the parents, comparative data suggested that parents of normals and dysphasics did not differ in terms of parental

'warmth', emotional demonstrativeness, or sociability. Where signs of abnormal parental behaviour do occur, they are sometimes found to be an artefact of the testing situation. Thus Schopler and Loftin (1969a) found evidence of parents of autistic children displaying conceptual thought impairment. Subsequent analysis (Schopler and Loftin, 1969b) suggested that the difference was entirely due to test anxiety resulting from the focus of assessment being directed at their behaviour towards their psychotic child.

Thus despite a number of interesting studies on the parents of autistic children, it is clear that few quantifiable dimensions of parental pathology have been produced to support the subjective clinical impressions of parental behaviour abnormalities which have been suggested in several aetiological theories. A variety of tests aimed at measuring aspects of parental personality have yielded rather mixed results. It is not clear, even when found, whether differences between parents of autists and parents of controls, are necessarily aetiologically significant. Neither can one dismiss the possibility of the existence of significant parental factors from the negative findings, since one cannot assume that the relevant features have been studied. It is also clear that the methods adopted to test parental personality factors are far from satisfactory. Problems arise with the use of projective tests of dubious validity (e.g. the Rorschach), the nature of the test situation (which does not necessarily conceal the purpose of the test from the parent), and the retrospective nature of so much of the data collected, (typically on child-rearing practices). Interview and rating measures also present methodological difficulties, and it is notable that DeMyer et al., (1972b) were cautious of their results in view of "... our need to rely on memories of 96 pairs of observers who were probably biased and unsystematic." It is unlikely that we are

being sufficiently scientific if we make definitive statements on the role of parents in the aetiology of childhood autism solely on the basis of global personality tests, attitude questionnaires, or interview data, in the light of their limitations noted above. Such methods do not in any case separate possible aetiological factors from parental reactions to severely deviant infants.

Aetiological bias, emotional involvement, and fear of attribution of blame, have led parents (Kysar, 1968; Park, 1967), and research workers (Rimland, 1964; Schopler, 1972, 1974) to react violently to any 'psychogenic' viewpoint. Schopler takes the position to the extreme of attempting to explain any psychogenic line in terms of scapegoating (Allport, 1966). Schopler claimed that the supporters of environmental determinants of childhood autism feel thwarted due to lack of aetiological knowledge, suffer from guilt evasion due to poor therapeutic results, feel fear and anxiety at the difficult interpersonal relationship and non-communicative behaviour of the autistic, and need self-enhancement as their roles are undermined by such poor therapeutic results. He suggested that the parents are thus made scapegoats for the condition of their children, and thoroughly supported Rimland (1964) in his reference to "pernicious psychodynamic theories". Such emotive statements are understandable when coming from parents, but do no credit to the professionalism of research scientists, going beyond the realm of fair criticism.

What Schopler does not appear to realise is that the conditions and the motives he attributes to psychogenic theorists could be equally applicable to those who advocate biological impairment. Thus although he may be quite correct in emphasising the pressures felt by clinicians and research staff from their lack of understanding of the aetiology of the condition and poor treatment results, such pressures could also force

attribution of childhood autism to neurological or other 'organic' impairment upon equally fragmented evidence. A biological explanation would indeed avoid the kind of alienation and indignation clearly felt by parents when faced with a 'psychogenic' hypothesis. Schopler also does not make it clear why he feels that 'scapegoating' has been used with the study of autistic children, and not with other childhood conditions of dubious origin (e.g., hyperkinesis), where similar pressures must exist. He may have been more accurate if he had suggested that there is a pressure to polarise in either 'direction' when hypothesising about the aetiology of childhood autism.

Behavioural Approaches

Behaviour modification techniques have been used to teach a variety of new behaviours to autistic children, and to reduce the emission of many inappropriate behaviours. Indeed, many psychologists regard behaviour modification as by far the most promising treatment strategy for these children (Ney, 1967; Howlin et al., 1973a; Lovaas, 1971). There have been numerous publications that have demonstrated that specific aspects of the autistic child's behaviour can be conditioned. Thus self-destructive behaviours (Lovaas and Simmons, 1969), temper tantrums (Wolf, Risley and Mees, 1964) and social withdrawal (Ney, Palvesky and Markely, 1971) have all been eliminated with behaviour modification techniques

and social cooperation (Hingten, Sanders and DeMyer, 1965), imitative speech (Lovaas et al., 1966), and eye contact (McConnell, 1967) are some examples of the many behaviours that have been established with these techniques. Long-term maintenance of such desired behavioural change has been found to depend to a large extent upon teaching parents and teachers the application of the principles of behaviour modification (Stevens-Long and Lovaas, 1974; Kozloff, 1973).

Some of the earlier behaviour modification studies were seen as having implications for an understanding of the aetiology of childhood autism. Thus Ferster (1961) saw autistic behaviour like any other behavioural pattern, as best understood within the framework of social learning theory. Autistic children were not envisaged as qualitatively different from normals, rather distinguished by a relative frequency of occurrence of behaviours in their repertoire. The autistic child thus spends significantly less time influencing the social environment in contrast to the physical one. Ferster then made the controversial assertion that a lack of parental control was fundamental in the aetiology of the condition. He proposed that the autistic child has failed to acquire any secondary, or social, reinforcement control, because the parents had failed to deliver sufficient positive reinforcement in the child's earliest years. Other humans therefore were not 'meaningful' to the autistic child because they had no social reinforcement value - they were not perceived as rewarding. Ferster then attempted to teach autistic children by establishing

conditioned and then generalised social reinforcers to build up a behavioural repertoire in which other humans would have reinforcement properties and thus 'meaning'.

The validity of this hypothesis appears to rest upon two points. First, that behaviour modification techniques enable the development of at least some social behaviour, and that conditioned social reinforcers can be acquired by autistic children. This argument is not strong, for behaviour modification techniques have been used successfully with a number of behaviour problems where quite clearly the primary aetiology is of an organic nature (e.g., token economy systems with severely sub-normals). The more recent behaviour modification programmes regard treatment as a separate entity rather than dependant upon aetiology (Kozloff, 1973).

Ferster's other source of evidence would appear to be that autistic children lack in social reinforcement control. There is evidence that this is not so, for several studies find that many of the deviant behaviours of autistic children are in fact under the control of social reinforcement, typically adult attention (Lovaas, 1971; Moore and Bailey, 1973). In addition it is possible that a lack of social reinforcement control could arise from a number of sources other than a deficiency in a social learning environment, including defects in attention or arousal.

In more recent years, behavioural work in the field of childhood autism has been directed towards treatment almost to the exclusion of aetiological investigation, and is therefore not directly relevant to

a discussion of the origins of the condition. Thus whilst Lovaas (1971) hints at early 'faulty learning', with the autistic child failing to learn symbolic rewards in early infancy and consequently failing to develop 'social' behaviour, such a deficiency could be the (primary) result of an organic deficiency. The treatment approach is essentially the same however, whatever the initial aetiology is considered to be.

Research on the effectiveness of behaviour modification techniques has yielded some promising results, potentially of great importance for the treatment and education of autistic children. With regard to aetiology however, little convincing evidence exists to support the view that these children suffer from deficiencies of learning opportunities in their early life. Nevertheless, recent investigations into the question of why there appear to be wide individual differences in responding to conditioning programmes and also unpredictable difficulties in the learning of some discriminations in contrast to others of apparently similar complexity, have led to some very interesting findings on the characteristic difficulties of these children (Lovaas, et al., 1971 ; Koegel and Schreibman, 1974). These will be discussed in relation to perceptual deficiency hypotheses.

Other Learning Theory Approaches

Leff (1968) saw the success of behaviour modification techniques as a result of the autistic child's inability to learn under ordinary circumstances. Whether due to organic or environmental factors, the early failure to solve very simple problems would severely affect subsequent learning. Such interference would thus increase in an exponential fashion with successive failures producing progressively greater decrements in the learning of new skills. The helplessness at adapting to and organising the environment, Leff believed to result in withdrawal. Thus behaviour modification is successful because it orders the environment sufficiently so that even the most severely impaired child can successfully manipulate it.

A similar analysis was offered by Churchill (1971), who although himself suggesting a primary neurobiological impairment, saw the success-failure ratio as one very important variable, and suggested that it offers a partial reconciliation between 'organic' and 'environmental' viewpoints. Churchill tested the observation that much 'pathological' behaviour disappears and reappears as a function of the success in the task in hand. Eleven autistic subjects were trained to 'read' hand signs denoting 3 colours and 3 objects. During 5 experimental sessions coloured objects were signalled, and the subject was required to select it from an array. Sessions were arranged so that one session resulted in each subject failing on 90% of the trials, the other in successful responses on 90% of the trials. Avoidance, self-stimulation and frustration all increased significantly under the 'failure' condition, and they also increased as a function of time spent in that condition. Churchill suggested that highly concerned parents, in not fully appreciating or taking account of their child's organic limitations, unwittingly increase failure by persistently demanding successful

performance on tasks beyond the child's genuine capabilities. Supportive evidence for the notion of a low success-failure ratio resulting in autistic behaviour, had been documented earlier by Hington et al., (1967). They reported that psychotic subjects working at tasks of low complexity, were more cooperative, attentive, and consistent in performance over several days, in contrast to their typical behaviour.

These are certainly interesting findings, but we must ask whether they are specific to childhood autism, or to the retarded functioning that accompanies the vast majority of these children. It may well be that many other children who function at the subnormal level, also display greater attention and less inappropriate behaviour when working at tasks of low complexity. We cannot assume that the parents of autistic children do indeed fail to take into account their child's level of functioning or at least overestimate it. Also, there is certainly evidence that this is not the case. Schopler and Reichler (1971) carried out an investigation of parents' perceptions of their psychotic child. During the initial diagnostic interview, parents were asked to estimate the level of functioning (in age) of their child in the areas of overall development, language development, motor and mental development, and social age. Psychometric assessment of the children, using the Vineland Social Maturity Scale, the WISC, the Merrill-Palmer, the Bayley, and the Leiter International test, produced scores which correlated highly with the parent's predictions in the areas of functioning listed.

Thus we should be careful in designing treatment and educational programmes such that they do not overestimate the skills of the individual child, nor proceed too rapidly. We cannot, on the basis of the evidence offered, assume that parents play a significant role in the development of childhood autism.

AN ETHOLOGICAL APPROACH

It is reflected in the diverse nature of the literature on infantile autism that those working in other fields have been attracted to the study of these children. It is thus not surprising that a recent publication from workers in the field of animal ethology has appeared. Tinbergen and Tinbergen (1972) proposed that: "... apart from aversion of the face, all other components of the social encounters of these autistic children are those shown by normal non-autistic children." They suggested that in situations such as a forced encounter with a stranger, the normal child can respond either at one extreme by approach and uninhibited smile, or at the other extreme by gaze aversion, head-turning, and stereotypy. Thus otherwise normal, but relatively timid children, can in situations involving unfamiliar elements show all the symptoms of diagnosed autistic children. The Tinbergens believe therefore, in a gradation such that the difference between autistic and non-autistic children is one of degree, the former being extremely timid and at one end of a social behaviour continuum.

Being essentially animal ethologists, they draw an analogy with bonding or socialisation that occurs in animals like the herring gull where approach-avoidance conflicts appear, and 'autistic behaviour' may be envisaged as analogous to displacement activity during such conflicts. The authors suggest that in each encounter with an adult, every infant is initially in a state of motivational conflict between 'social' and 'timidity' tendencies. Normal children quickly swing to social responsiveness due to a waning in their fear. Early environmental factors, producing an over timid child, make the autistic child take longer to resolve this conflict, and in extreme cases prevent the solution of the conflict at all. Anxiety becomes progressive, and resultant lowering of thresholds leads to fear responses to a greatly widened range of stimuli, including ultimately the normal bonding

signals like respondent smiling. The conflict keeps the autistic child in a state of high arousal, with the resultant stereotypies and regression of social developments like speech. Thus the central problem in aetiology is conceptualised as a series of frustrated attempts at socialisation, combined with constant and intense fear. Tinbergen and Tinbergen stress that treatment approaches and interactions, should effectively change the motivational state to facilitate learning, imitation and play.

Richer and Nicoll (1971) designed a playroom in which there were retreat boxes where the child could isolate himself, soft seats in an area where sitting and interaction was possible, an activity area for individual or group play, and several partitions to reduce overstimulation from others. All formal training was administered outside this room, and within it no adult intervention was used. Approaches and eye contact were only made if the child clearly signalled a desire for them. Initial results, using a time sampling method, suggested that stereotypy declined during the periods studied and interactions significantly increased.

Whilst the results of the effectiveness of the environment are contaminated with extra-playroom therapy, it seems plausible to suggest that the prototype 'playroom' may be successful as a useful precondition to effective therapy. The results also support the interesting suggestion by the Tinbergens that amidst all the diagnostic confusions, it may prove more fruitful to classify these children from a treatment rather than aetiological approach. However, treatment results will need to improve considerably for this position to be generally accepted. With regard to aetiology, several experiments do not support ethological interpretations. Thus Churchill and Bryson (1972) reported that gaze-aversion and physical avoidance of adults in a zoned playroom, did not

differentiate autistic children from controls. Similarly Hermelin and O'Connor (1967) found shorter visual inspection times among autistic children, for a variety of stimuli, but not significantly shorter times for facial stimuli compared with 'non-social' stimuli. Tinbergen's assertion that all symptoms of autistic children are seen in normal children to a certain degree does not appear to be correct. Almost all literature reviews on the subject conclude that the majority of autistic children function at the subnormal level (e.g., Rutter, 1968, 1974; Ornitz, 1973). Many of the perceptual deficiencies found in autistic children are also certainly not present in normal children (Hermelin and O'Connor, 1970; Koegel and Schreibman, 1974).

Thus the Tinbergens' hypothesis may be criticised at a number of levels, but has been attacked most vehemently by Schopler (1974) who interprets the ethological approach thus: "Childhood autism is whatever you believe it to be; the methodology is simple; assume that autism has its roots in normal development; redefine normal but shy children as autistic; have some pleasant interactions with such children and get them to act less shy. You now have a new approach to autism. If you need any validation for any of this, don't look to confusing research, simply assert personal opinion". (p.195)

Such histrionic critiques do not serve psychology well.

Environmental hypotheses: a comment

The aetiological hypotheses that have been outlined to date, have all stressed the role of psychosocial influences, including deviant mother-child relationships, parental behaviour abnormalities, and inappropriate learning environments. Although they vary in their emphasis and the precise nature of the deficiencies, and indeed the degree to which particular environmental variables are implicated, they nevertheless share a common emphasis in a belief that parents have a role to play in the aetiology of the condition. They have thus been grouped as 'psychogenic' hypotheses.

Criticisms of these hypotheses have already been noted, and as Rutter (1968) commented, since infantile autism becomes evident in very early infancy, it would require severe parental pathology to produce such an immediate effect upon the child. However, if one considers a multi-factor aetiology then the model becomes more plausible. With any severely handicapped child there will almost inevitably be a series of responses on the part of the parent to that child. Drillien (1964) concluded from a study of low birth weight infants that the quality of maternal handling and the early environment is more significant in personality development and behaviour disorder than the actual low birth-weight and pregnancy complications themselves. Similarly, Barratt (1971), in a review of pre-natal development and a variety of factors that influence it adversely, proposed that the 'match' between the needs of the child (arising from his individual pre-natal environment) and the actual environment in which he is brought up, is the most crucial determinant of subsequent development. Williams (1968) takes the point to the extreme of suggesting: "Thus one can come to the generalisation that no physical handicap leads in itself to behaviour disorder and conversely, that behaviour disorder reflects a failure of

the environment, either in the form of errors committed or of essential needs omitted in the child's early life".^(p.739) Such factors would be unlikely to be isolated by the research techniques used to date by the majority of 'psychogenic' advocates.

With regard to childhood autism in particular, Williams suggests that the emotional backlash arising from Kanner's writings (and presumably others who attempt to identify parental factors), has gone too far in denying the importance of environmental and emotional factors. A model in which environmental factors are seen as contributory, and significant, cannot be summarily dismissed. Similar types of mechanism have been suggested, with sound empirical evidence, for other childhood behaviour disorders. Thus, Prechtl (1963) studied mother-child interactions in babies included in the Minimal Brain Damage Syndrome. He proposed that normal newborns display a characteristic pattern of behaviour, essential for establishing normal relationships and development. Such behaviour includes rooting, sucking, grasping, and regular changes in state between sleep, wakefulness, crying, and smiling (Papousek, 1969). Prechtl emphasised that for the mother it seems very important that her child does display these patterns.

The Minimal Brain Damage Syndrome (MBDS) is often a sequel to pre- and peri-natal complications, yet changes in many of the infant's behaviour patterns do not arouse immediate concern for the paediatrician. Prechtl studied one thousand newborns during their first nine days, all of them having a history of pre- and peri-natal complications. One of the clearest groups that emerged from this sample was the hyperactives, who exhibited so much sudden change of state and such low thresholds, particularly to the Moro Response, that the mothers of these children were continually afraid of frightening

them. A three month follow-up study revealed that, compared with controls, mothers of these children did not show an harmonious attitude towards their child but displayed typically over-anxiety or rejection. Perhaps of more significance was the finding that 70% of the hyperactives developed the Choreiform syndrome in later life, with severe learning difficulties in school. Thus although neurologically abnormal babies do not make good partners in initial mother-child interactions, it is clear that the type of parental response to such behaviour, and thus the 'match' between the needs of the child and his environment, is crucial for their future development. This situation may conceivably be paralleled in childhood autism, for autistic children make extremely poor and unrewarding interaction partners. It is unfortunate that autistic children are typically not diagnosed as such until at least two or three, since studies on their early development are thus necessarily retrospective.

A Genetic Hypothesis

Ounsted (1970) proposed an aetiological hypothesis in which infantile autism was considered to be primarily genetically based. He suggested that a number of additive genes predispose affected children to develop a pattern of behaviour like that of extreme introversion. Since that part of the genome leading to Kanner's syndrome results in biological impairment it acquires, by selection, modifying genes which delay the deployment and enhance the variety of expression, of the main gene. The modifying genes may be those generating a pattern of development which results in a high level of intelligence in the parents. Physiological over-arousal (Hutt et al., 1964), and gaze aversion to social stimuli (Hutt and Ounsted 1966), suggest

apparent aloofness and indifference, and yet this is seen as masking extreme sensitivity to the environment. Thus Ounsted, like Hutt and the Tinbergens, stress the great social sensitivity of autistic children despite their introverted and 'detached' behaviour. However, Ounsted sees a genetic basis for this. He agrees with Kanner that parents display 'hypopaedophilia', in contrast to the 'hyperpaedophilia' shown by parents of hyperkinetic children, and explains these differences as being the result of differing social signals emitted by these children. The hyperkinetic child appears helpless, and rapid changes in his apparent emotions arouse and fixate attention in his parents. In contrast, the autistic child gives very negative social signals by not vocalizing, and by failing to establish eye contact or smiling.

Ounsted made eleven predictions from his hypothesis, and admitted that if any of them are not upheld then the validity of the theory must be seriously questioned. Two of these hypotheses however, are that siblings and parents have extroversion-introversion scores shifted to the introverted pole, and that the I.Q. scores of the parents are markedly high. These could equally be due to environmental factors and therefore do not necessarily support a genetic hypothesis. Many psychologists assert that many personality characteristics, and much that contributes to intelligence levels, is learnt by the child from his environment. The parents thus play a crucial role in the non-genetic transfer of such behaviour to their child (Bandura and Walters, 1963; Kamin, 1974; Mussen, Conger and Kagan, 1969). In addition, several studies report no differences in extroversion-introversion scores between parents of autistic children and those of normal controls.

The few genetic studies that have been conducted on childhood autism, yield little support for a genetically based model. Both Book, Nichtern and Gruenberg (1963) and Judd and Mandell (1968) failed to find any chromosomal abnormalities in autistic children. Rutter and Bartak (1971) point out that twin studies are rarely carried out on adequate sample sizes, and this is particularly so with rare disorders like childhood autism. Ornitz (1973), surveying the published literature, found only 15 "particularly satisfactory" cases. Of these, 10 monozygotic pairs were concordant in eight cases, and of five dizygotic twins one pair was concordant. He concluded that whilst this provides some evidence for a genetic model, the sample size was far from adequate, and there are sufficient doubts with regard to the diagnoses of both zygosity and the nature of the disorder in some studies. There are of course serious problems with the interpretations of twin studies where there are no control pairs reared apart. When both sets are reared together, it seems clear that monozygotic twins share a more similar environment. Lotter (1966) noted that the incidence of childhood autism is no higher in the parents and relatives than in the general population, and pooling data from three of the largest studies, Rutter (1968) found the concordance rate for siblings to be only around 2%, which is extremely low for an hereditary disorder.

Thus the evidence for a genetically based aetiological model for childhood autism rests on very little empirical data. In the light of the rare nature of the disorder, genetic hypotheses are most difficult to test, but present research suggests that it is a possible, (but unlikely), aetiological basis for childhood autism.

Biochemical Hypotheses

A number of recent studies have attempted to find a biochemical substrate which may be used both as a characteristic index of autism (and therefore as a diagnostic criterion), and also as a basis for an aetiological model. Amongst these have been reports of serotonin abnormalities in blood samples taken from autistic children. Mean serotonin levels were found to be elevated in psychotic children in studies published by Ritvo et al., (1970), Schain and Freedman (1961), and Coleman (1973), although the pattern was not consistent with autistic children. With the latter group there have been reports that it is not the free blood serotonin level itself which is characteristically deviant, but rather the platelet level, seen in an increasing efflux of serotonin from them when compared with controls (Boullin, Coleman and O'Brien, 1970; Boullin et al., 1971). In an attempt to reduce the level of serotonin in blood platelets, Ritvo et al., (1971) administered doses of L-Dopa to four autistic children over a period of 6 months. Serotonin levels dropped, but there were no behavioural changes to alter the clinical picture of the disorder.

Whilst a biochemical investigation with autistic children is quite legitimate, studies carried out to date do suffer from a number of difficulties. Not least amongst these are the very small samples used (often less than six subjects are used), and the heterogeneous nature of the children used in some studies, including those in control groups. Biochemical analyses are of course carried out on children who have been disturbed for some time, and as Kozloff (1973) notes, it may be suggested that any biochemical abnormalities found are relevant to the child's present functioning but have nothing to do with the fundamental aetiology. Indeed, it may be that any current biochemical abnormalities are themselves consequences (rather than causes) of the

autistic child's behaviour.

A biochemical hypothesis of somewhat dubious validity has been outlined by Rimland (1973, 1974). It is based upon the concept of "orthomolecular psychiatry", a term introduced by Pauling (1968) based on the belief that mental illness is caused by an imbalance in the optimal levels of vitamins present in the body. The implications are that treatment depends upon the administration of large doses of vitamins in which the patient is deficient. Rimland (1974, 1973) reports studies in which he claimed distinct improvement in children treated over several months, and that vitamin C and three of the B vitamins were the most beneficial. The samples studied included a number of different psychotic children, but the autistic children gained most from vitamin B-6 which plays an important role in the production and metabolism of serotonin. The need for this vitamin in large quantities is assumed to arise from a genetic, rather than a dietary, deficiency.

Evidence for the concept of orthomolecular psychiatry appears to rest upon the finding that a number of rare genetic disorders have been found which are characterised by the patient's need for large doses of certain vitamins. None of these disorders are related to autism, and consistent successful treatment results would appear to be a minimum requirement for even a tentative acceptance of the validity of the concept. It should be noted that one recent thorough study concluded that megavitamin therapy does not meet the criterion of scientific validity (Lipton et al., 1973)

Neurophysiological Hypotheses

Hutt et al., (1964, 1965) put forward a neurophysiological hypothesis to account for the behaviour seen in cases of infantile autism. They proposed that the non-specific activity of the

Reticular Formation is sustained at a chronically high level, inducing a very high state of arousal in the autistic child. Hutt et al., (1964) reported EEG analyses which revealed a high percentage of subjects having either:

- a) resting EEGs with very low voltage and flat desynchronised patterns whether the eyes are open or not, or:
- b) alpha dominated records with the eyes closed and flat with the eyes open.

They suggested that the presence of high frequency or desynchronised patterns may be considered evidence for an abnormally high electrocortical activity level and corresponding arousal. Stereotypy and gaze aversion were seen as mechanisms used to keep the sensory input and thus arousal, to a minimum. From this position it would be predicted that if stereotypy is a function of arousal, then an increase in sensory input, by increasing environmental complexity, would result in an increase in stereotypy. This was indeed reported by Hutt and Hutt (1968) who varied the number of people and objects in a room. The EEGs of two subjects during free-field activities were directly monitored (Hutt et al., 1965) and it was found that when stereotypy was least, (in an unfurnished room), the records showed high voltage synchronised activity. As stereotypy increased so the EEGs became increasingly desynchronised, supporting the hypothesis that stereotyped behaviours may be a protection against overstimulation.

Using concepts derived from ethology, Hutt and Ounsted (1966) proposed that since gaze aversion signifies a readiness for interaction, with more specific signals qualifying its nature, failure to fixate has a very important inhibitory effect upon social interaction. The lack of fixation in the autistic child, has led to what they believe to be a misleading description of these children as aloof and

withdrawn. Hutt and Ounsted attempted to assess the significance of gaze aversion with a systematic analysis of the incidence and morphology of actual social encounters among autistic and normal children. In the free-field situation autistic children played alone and had no verbal, eye, or physical contact with peers. In the absence of eye contact, the peers' curiosity waned and so the autistic child gradually became ignored because he eliminated any chance of social contact. Yet autistic children sought adult contact, and the total time spent in social contact did not differentiate the two groups. Rather, the nature of the encounters differed in that autistic children were found to stay for much longer periods in close proximity to an adult, being cuddled and sitting on the adult's knee. Hutt and Vaizey (1966) studied group density and its effect on behaviour. The main effect of increasing such density was to force autistic children to the perimeter of the room and to increase contact with adults but not peers. Nevertheless, this was accompanied by lack of eye contact.

In an experimental room where model faces were placed on stands spaced evenly throughout the floorspace, Hutt and Ounsted (1966) found that normal subjects spent longest looking at expressive faces, whilst least at the blank faces. Autistic children however, displayed their longest fixation times at the non-facial stimuli (door, windows etc.), and their shortest at the faces. They also used locomotor strategies actively to avoid exploring the faces. Video-tape analysis showed that despite the paucity of fixations, fractional glances were used to yield sufficient information to locate and monitor them. In the diadic situation with a normal peer, whenever the peer was not fixating the autistic child, the latter again used fractional glances to monitor position. The authors commented that

these glances were too selective and accurate to permit an explanation in terms of perceptual deficits. The implications of gaze aversion are extensive, and much affectational behaviour is contingent upon making and maintaining some degree of eye contact. They pointed out that it may result in attenuated responsiveness and reduced stimulation from the adult. It is not surprising therefore that where operant conditioning has established tolerance of eye contact 'social' behaviour tends to improve also (Currie and Brannigan, 1970). In child development, eye contact appears to be a necessary precursor to smiling (Ambrose, 1961). Even if the autistic child does smile frequently in infancy, which some retrospective parental reports suggest is rare, it is infrequently accompanied by eye contact, and it is this which signals direction. Ambivalent maternal attitudes may develop due to positive feelings aroused by the child's outstretched arms but also negative feelings from the lack of eye to eye contact. The result may be a 'double-bind' situation for the mother, resulting in an aberrant mother-child relationship.

There are several findings however, which contradict this model. Metz (1967) measured stimulation level preferences in autistic children by recording the sound level each subject selected by operating a lever controlling a tape recorder. Results supported the clinical observations of unusual responsiveness to sound levels, but in contrast to Hutt's predictions, autistic subjects not only preferred but also acted to maintain, significantly higher levels of stimulation than normals. Churchill and Bryson (1972) studied the looking and approach behaviour of autistic children and normals as a function of adult attention using a zoned playroom in which adult behaviour was systematically varied. Visual fixation times and

physical avoidance did not separate the groups with either preoccupied or attentive adults.

Stereotypy is envisaged as a 'defence' behaviour to prevent overstimulation on the over-arousal hypothesis, yet several studies report that such behaviour continues even in periods of highly reduced sensory stimulation (Sorosky et al., 1968), and does not always increase with increasing environmental complexity (Ornitz et al., 1970; Hermelin and O'Connor, 1970).

EEG data in relation to an over-arousal hypothesis also provides contradictory evidence to that forwarded by Hutt et al., (1965). Thus Hermelin and O'Connor (1968) found that the occipital alpha measures taken did not differentiate autistic and normal children during conditions of high or low visual stimulation. Autistic children however, did show higher levels of this measure of arousal during continuous auditory stimulation. Creak and Pampiglione (1969) analysed the EEG records of 35 autistic children, and only very occasionally found that there was a record of low amplitude throughout, of the type suggested as supportive of an over-arousal hypothesis. Bernal and Miller (1971) compared autonomic measures of reactivity and habituation (the cardiac peak-to-valley and the G.S.R.) between autistic and normal subjects. The GSR data suggested that normal children responded more to the first three of a series of either tones or flashes, but the groups did not differ in terms of the cardiac measure or either measure of habituation. An over-arousal model would predict that autistic children have slower habituation rates. Bernal and Miller also found no evidence of any electrodermal or cardiac indices and tonic state change during stereotypy or self-stimulatory behaviour. Again, this is discordant with an over-arousal model, for it assumes that such

behaviours occur during periods of high arousal in the autistic child, and is a mechanism used by them to reduce stimulation (and thus to lower the arousal level).

Rimland (1964) placed an entirely different interpretation upon the behaviour shown by autistic children in suggesting that their symptoms indicated a pathological under-aroused nervous system. A malfunction in the ARAS was implicated as the origin of the disorder. The hypothesis proposed that inadequate arousal from the sensory input results in an inability to relate new stimuli to remembered experience. It is not entirely clear how the mechanism operates, although one assumes that a minimum level of arousal may be necessary for the information processing required in the coordination of perception, retrieval, and memory systems essential for recognition functions. In infancy this process leads to a failure to associate biological reward with social reinforcement and subsequent asocial behaviour. Thus a biological defect accounts for the failure to acquire the secondary reinforcement control which the early behaviourists attributed to environmental deficiencies. Rimland envisaged the excellent rote memory of autistic children, their extreme literalness and the delayed echolalia in their speech, and the bizarre mixture of hypo- and hypersensitivity, as evidence for a closed-loop phenomenon. Thus he suggested that stimuli enter the nervous system of the autistic child, they are stored in an unmodified fashion, and output and input become almost indistinguishable.

Many of the studies that fail to support the Hutt's model of an over-arousal defect, also contradict the notion of an under-aroused neurophysiology (e.g., Creak and Pampiglione, 1969; Bernal and Miller, 1971). But the fact that many of the same behavioural symptoms of the

autistic child are cited as evidence for totally opposing views of a neurophysiological disorder, may be seen by some as a reflection of the speculative and highly inferential nature of both hypotheses.

Aspects of both the neurophysiological hypotheses just described are incorporated in a third hypothesis; Ornitz and Ritvo (1968) described a single pathologic process they believed to be common to early infantile autism, certain cases of childhood schizophrenia, the 'atypical child', symbiotic psychosis, and children with general abnormal sensitivities. They suggested that these descriptive categories are varieties of a unitary disorder, and classified the multitude of symptoms observed into a number of sub-clusters, including disturbances of perception, motor behaviour, relating, language, and developmental rate and sequence. Ornitz and Ritvo attempted to relate the symptom clusters of infantile autism to a pathologic mechanism which results in an inability to maintain constancy of perception. Thus identical percepts from the environment are not experienced by the autistic child as the same each time they appear. The defect is envisaged as reflecting a basic failure in the CNS, so that environmental stimuli are either not adequately modulated or are unevenly amplified. This failure results in random over- or under-loading of the CNS without relation to the sensory input. The 'under-loading' is seen in the attention to apparently inconsequential stimuli, and scrutiny of tactile or visual detail. 'Overloading' is often reflected in sudden panic reactions to almost minimal stimuli, and in hand flapping and whirling and other stereotyped behaviours.

This underlying pathologic process common to the syndrome involves, according to the authors, the presence of states of hyper-excitation and inhibition which interfere with the normal capacity to

maintain perceptual constancy. These states are seen as reflecting a dissociation of physiological equilibrium between facilitatory and inhibitory systems which regulate sensory inputs. It is of course crucial for Ornitz and Ritvo to explain how multivariate expressions occur from the same basic underlying mechanism. This they attempt to do by suggesting that if the dissociated inhibitory and excitatory states are not too severe, then percepts are available for imitation, and the concepts of self and non-self can be distinguished. In this case, only disturbances of perception and motor behaviour, are reflected in symptomatology, and such cases constitute the "atypical child" or children with "unusual sensitivities". If communicative speech develops, the disturbances in relatedness are not too severe, but a thought disorder develops and diagnosis of childhood schizophrenia is made. If however, a more severe dissociation occurs, then disturbances in relatedness, language and perception are also present and such children become labelled autistic. If, in the early arrested development of the autistic child, a failure to distinguish self from non-self occurs, then such a child is labelled as falling in the category of "symbiotic psychosis". Ornitz and Ritvo do not explain how the severity of dissociation determines the variety of syndromes proposed. Their suggested continuum is merely descriptive. Thus for example it is assumed that because the autistic child has disturbances of perception, language and relatedness then his hypothesised dissociation must be greater than that of the childhood schizophrenic.

It has been shown that the brain is not a simple passive recipient of incoming stimuli, rather it exerts an active influence on inputs at many levels (French, 1960; Livingstone, 1959). It has also been shown that the converse applies, and so a true homeostatic

regulation of input to the brain almost certainly exists. Pompeiana and Morrison (1966), have shown that REM sleep is composed of a state of tonic inhibition punctuated by episodes of phasic excitation and inhibition, and it is by extrapolating from this type of finding and research, that Ornitz and Ritvo proposed a neurophysiological basis for their theory (Ornitz and Ritvo, 1968b). Their hypothesis is that the behavioural states of apparent excitation and inhibition seen in infantile autism, result from a pathologic uncoupling and disruptive breaking into waking life of the phasic excitatory and inhibitory influences of central vestibular system origin, normally found as the neurophysiological substratum of REM sleep. They cited a series of studies to support their theory: Ornitz et al., (1968) reported that the normal phasic inhibition of the Auditory Evoked Response (AER) during REM sleep, is markedly overridden in autistic children. Ornitz et al., (1969) found a significantly greater amount of 10-15 cps activity, along with a reduction in number of eye movements during REM sleep in autistic children compared with controls. The latter finding was postulated as due to a deficient or inhibited vestibular function. The reduction in association between the synchronous slow waves and eye movement bursts, further suggested impairment of an organising influence underlying integration of cortical and oculomotor activity. The authors suggested that this may be a function of maturational factors in view of the 10-15 cps component in the REM sleep EEG, and the tendency to approach more normal patterns with increasing age. Timsit-Bertheir (1973) published data on some EEG work with autistic children which supported a perceptual inconstancy model. He found that the primary component of the average evoked potential (AEP) is identical in autistic and normal children, but that they differ in the form of the secondary component of this response.

Autistic children showed a much stronger secondary component, seen especially in REM sleep, which suggests a disruption of integrative cortical functions which may be linked to an imbalance in the excitatory and inhibitory systems regulating the sensory input.

There are findings however, which do not agree with this aetiological hypothesis. Ornitz, Ritvo and Walter (1965) had in fact earlier reported no EEG, eye movement, heart rate, or muscle tone abnormalities in autistic and schizophrenic children during seven nights of continuous recording. Ornitz et al., (1974) predicted that the recovery time of the Average Evoked Response (AER) during sleep would differentiate autistic and normal children. No differences were observed however, in the recovery ratio or in latency and amplitude measures. Findings of consistent cortical evoked responses (CER) to 'familiar' tone-word stimuli (Small, 1971), and consistent performance on psychometric tests over time (Alpern, 1967) do not support a perceptual inconstancy model. Ornitz and Ritvo (1968a, 1968b) themselves cite much of their evidence from work done on adult schizophrenics, and justify this by stating that they have demonstrated a relationship between this condition and childhood autism: "As we have shown a clinical relationship between early infantile autism and later schizophrenia, our findings of a failure in phasic inhibition during REM sleep in young autistic children may have more general applicability to schizophrenic reactions in both children and adults". (p.78) As has been noted already in relation to differential diagnosis, childhood autism and adult schizophrenia are separate conditions (Rimland, 1964; Rutter, 1968, 1974). Thus some of the studies cited, and much of the clinical analogy drawn in the course of their argument, is of dubious relevance to childhood autism.

More recently, Ornitz became concerned at the independent treatment of motility disturbances in relation to the uncoupling of excitatory and inhibitory systems. He suggested that we must consider the interaction between the modulation of sensory input and the bizarre motor output frequently observed in autistic children (Ornitz, 1974).

Several experimental learning studies have suggested that feedback from self-generated motor responses is needed for efficient learning in autistic children (Hermelin and O'Connor, 1970). Thus these children appear to learn via position cues and manipulation responses rather than via normal perceptual processes. Ornitz suggested that the bizarre, repetitive motor output of the autistic child is actually a compensatory mechanism to help the autistic child make sense out of sensation via kinaesthetic feedback. Neurophysiological studies by Ornitz et al., (1969) on the oculomotor response to vestibulosensory stimuli in the presence of visuosensory stimuli, have demonstrated that there is an inhibition of the postrotatory nystagmus in autistic children. Similarly, vestibular mediated phasic eye movements during REM sleep (induced by rocking a bed), are smaller than normal controls in autistic children. The vestibular system is thus implicated in the disorder. Neurophysiological work on the vestibular system reveals that these nuclei directly modulate motor output at the time of sensory input, and also sensory input at the time of motor output (Ornitz, 1974). Now whilst the cortical centres may inhibit vestibular function alone, the findings of a depressed oculomotor response to vestibular stimuli suggest a dysfunction of cerebellar or brain-stem influence. Thus autism is seen in this revised model, as a result of a dysfunction of subcortical mechanisms, involving connections of the vestibular system, cerebellum, and brain-stem producing the characteristic sensorimotor behaviour of the autistic child.

MacCulloch and Williams (1971) agree with this model, and take the argument further by specifying the anatomical locus of a proposed lesion that may account for the behaviour seen in autistic children. They suggested that any single circumscribed cite for a

lesion which could produce such a severe multiple handicap, must be one where the pathways are adjacent and in the dorsal lower brain-stem. They propose a site where posterior column tracts, vestibular, and auditory afferent pathways mutually decussate. This area contains the reticular formation, cardiorespiratory centres, and cranial nerve nuclei, and the exact lesion is proposed to be in the nucleus of the Tractus Solitarius, as a result of peri-natal anoxia (MacCulloch and Sambrooks, 1973). It is suggested that this lesion results in underdampening of much of the ascending reticular formation activity, impairing perceptual and arousal functions and leading to states of perceptual inconstancy. The authors predict that the undampened arousal system will lead to rapid cardiac acceleration and deceleration, seen in high heart rate variance. They report a study in which they telemetered the heart rate of autistic, subnormal, and normal children taking maximum and minimum values of the recorded heart rate as an index of heart rate variance. Results were in accordance with their predictions in that no differences were found in mean heart rates, but the variance values for autistic children were higher than that of the controls. Unfortunately the study was poorly designed. Chronological age was not a controlled factor, and it is known that this affects heart rate measures. More important, subjects were allowed free movement in the experimental setting during telemetry and thus activity levels amongst the subject groups was not controlled. Indeed, the authors admit that a wide variation in exploratory behaviour occurred between subjects. Thus individual differences amongst subjects could account for the results found. MacCulloch and Williams conclude that dysfunction in heart rate modulation occurs in autistic children, and that the increase in heart rate variability suggests partial failure of negative feedback between cardio-acceleratory and deceleratory components of the brain-stem cardio-

regulatory centre. Clearly, better designed studies need to be conducted before we can accept such firm conclusions.

The neurophysiological hypotheses that have been outlined, have all developed from studying the overt behaviour of the autistic child, constructing an hypothesis concerning an underlying neurophysiological defect, and then looking for experimental supportive evidence in terms of physiological measures. But this approach can be criticised on two levels. First, to speak of a general level of arousal, and also of 'excitatory' and 'inhibitory' states is almost certainly an oversimplification. It may be quite possible to categorise overt behaviour into apparent periods of hyper- and hypo-activity, and hypersensitivity or hyperresponsiveness; but one cannot then assume that there will be underlying neurophysiological parallels. Indeed, it is quite possible that the behaviour will in any event determine, or at least influence, the physiology. Secondly, and perhaps more important, even if we did accept that the autistic child is over-aroused, under-aroused, or in a continued state of fluctuation between the two, our search for physiological measures to support our assertions must be modified by our knowledge, or rather lack of it, concerning what it is that we are measuring. Indeed, Johnson (1970) concluded after reviewing an extensive series of studies, that the state of consciousness of the subject must first be known before the physiological significance and possible behavioural meaning of measures like the EEG and various autonomic responses, can possibly be inferred. In other words, it may not be a valid approach to propose a physiological 'state' hypothesis to explain behaviour, and then support that hypothesis with EEG and other physiological measures, as the models described have done. Rather, as Johnson concludes: "Instead of using autonomic and EEG measures to define state, the reverse appears more appropriate. We

must first determine the state before we can interpret our physiological measures". (p.515). Johnson found that the same physiological responses and visceral changes may occur in more than one state of consciousness, and questioned whether we can thus infer that they have the same meaning, and the same physiological significance in each state. He pointed out that there is considerable support for the view that the EEG is in particular a poor correlate of behaviour. Thus the alpha rhythm attenuates during high states of arousal, but paradoxically also decreases as the subject becomes drowsy. It also is present during REM sleep (Johnson et al., 1969), and whilst alpha blocking occurs in response to a stimulus during waking, and alpha enhancement during stage 1 sleep, during REM sleep there was apparently no blocking or enhancement to a much more intense stimulus. Thus REM sleep alpha appears to have different response characteristics which are not predictable from other states with similar alpha activity. Johnson and his colleagues also report the inability of even large scale computer analyses to differentiate consistently between stage 1 and REM sleep. The EEGs of awake subjects with low voltage activity also could not be differentiated by computer analysis from stage 1 or REM sleep (Lubin et al., 1969). Other physiological measures fair little better. Thus Johnson and Lubin (1966), and Koumas et al., (1968), induced large GSRs during the deepest (stage 4) period of sleep, yet waking electrodermal activity is commonly used as a measure of arousal. The corollary to the finding that similar visceral responses occur in differing states, appears to apply also. One example of this is that evoked responses to the same stimuli differ in differing states, seen in the finding that the heart-rate response to stimuli is larger during sleep than waking (Hord, Lubin and Johnson, 1966). As Johnson (1970) pointed out, even with multivariate recording techniques and multiple

physiological measures, the frequent dissociation among physiological variables makes it difficult to conceptualise a simple physiological or psychological model that would allow us to generalise as to meaning and the probable response pattern of individual variables as the subject goes from one state to another, or even when he remains within the same state. Thus a simple arousal continuum on which a subject's position can be clearly defined by his responses, is not a viable proposition since autonomic measures and the EEG fail to indicate the state of consciousness.

The evidence for the neurophysiological hypotheses described, rests on two sources. The first is upon the behaviour of the autistic child, particularly stereotypy and apparent variability in responsiveness. But it is speculative to link this with any particular neurophysiological defect, and hence the second source of evidence is drawn from EEG and autonomic measurement. In view of this critique of such studies, it requires much further work to substantiate these aetiological theories, which at present remain little beyond the stage of tentative, and largely unsubstantiated, speculation.

Autism as a Cerebral Damage Syndrome

A substantial quantity of literature has been documented on pre- and perinatal events in relation to later behaviour, and in particular significant associations between complications during these periods and certain neuropsychiatric and psychological disorders (Pollack and Woerner, 1966; Pasamanick and Knobloch, 1966; Barratt, 1971). A number of studies have reported a significant percentage of cases of autism with pre- and perinatal difficulties (Rutt and Offord, 1971; Kolvin, et al., 1971; Taft and Goldfarb, 1964; Gittelman and

Birch, 1967). However, it must be stressed that much data comes from retrospective reports, and also differs in that some studies include cases with clear signs of cerebral insult (e.g., Gittelman and Birch, 1967) whilst others do not (e.g., Vorster, 1960). Ornitz (1973) comments that when reviewing studies the lack of uniformity of diagnostic criteria, and the frequent lack of similar controls, makes firm inferences rather difficult. Rutter (1974) concludes that "It appears that there might be a slight excess of perinatal complications but this excess, if confirmed, is not great enough for them to be regarded as a common cause of autism". However, difficulties with the collection of data, the selection of criteria for determining pre- and perinatal difficulties, and problems of interpretation of the current literature on the subject, should not force us to be too hasty in our dismissal of the possibility of such complications playing a role in an interactive aetiological model of childhood autism.

Whilst we may remain uncertain as to the question of prenatal complications and childhood autism, if they do play a role in the development of the condition it is possible that they may do so via consequent cerebral damage. Indeed, several studies report significant numbers of autistic children with evidence of cerebral damage. Thus Kolvin et al., (1971) found 25% of their subjects displaying "strong evidence" of cerebral dysfunction (Profound EEG abnormalities, history of epileptic fits). Gubbay, Lobascher and Kingerlee (1970) reported 56% of their sample displaying "unequivocal brain disease", and Gittelman and Birch (1967) cite figures of up to 80% with central nervous system (CNS) pathology. 29 of the 35 subjects studied in Creak and Pampiglione's (1969) study showed abnormal EEG records, and several other studies publish significant, but smaller figures (White, DeMyer and DeMyer, 1964; Schain and Yannet, 1960;

Ritvo et al., 1970).

However, it is clear that we cannot assume that autistic children are necessarily all suffering from cerebral insult of some kind. Kanner claimed that none of his cases displayed any such evidence, and certainly most studies find significant numbers of cases without such signs. Thus for example Kolvin et al., (1971) found almost half his sample presenting measures of cerebral functioning indistinguishable from normal controls, and similar findings have been frequently published (Bryson, 1970; Goldfarb, 1961). There are problems with the measures taken, these frequently consisting of EEG and "soft" neurological signs, and we must be cautious in our inferences on the basis of these measures for they cannot be used as definitive indices of neurological impairment. Certainly the EEG and behavioural measures that are frequently cited as evidence of cerebral dysfunction in autistic children, are not specific to these children (Rutter, 1968). Equally, the vast majority of children with a diagnosis of cerebral insult display none of the characteristics of childhood autism (Rutter, Graham and Yule, 1970). The relationship between neurological dysfunction and behavioural symptoms is unclear. Many children who do have such organic defects, as verified anatomically or with neurological examinations, do not exhibit the behaviour that we presume to be characteristic of 'brain damage' (Chess, 1972). A diagnosis of brain dysfunction in the absence of specific neurological signs, and based solely on behavioural findings, appears unjustified. Thus impairment, albeit of a severe nature, in areas of cognitive, linguistic, and perceptual functioning, is not necessarily indicative of cerebral damage. We know that extreme environmental conditions can lead to marked deficiencies in these areas

(Rutter and Mittler, 1972).

Despite the problems outlined above, it is clear that a number of autistic children do display signs of neurological impairment, and this number tends to increase as one looks at older children in follow-up studies (Lotter, 1974b), and has been found to correlate with subnormal functioning (Gittelman and Birch, 1967). These signs are not definitive, rather suggestive of such dysfunction. Given that significant numbers of autistic children display these signs at no stage in their development, do we therefore require a subclassification within childhood autism? The answer would appear to be that unless there are clear behavioural differences between autistic children with signs of cerebral dysfunction and those without, it would be better to regard cerebral dysfunction as an associative factor recorded on a multi-axial classificatory scheme.

A similar position may be adopted with regard to pre- and perinatal difficulties, where such factors appear to be associated with many autistic children but are not a prerequisite for the condition. It is known that certain pain-relieving drugs can have long-term effects upon the behaviour of the neonate on measures such as habituation, cuddliness, and orientation (Aleksandrowicz and Alekandrowicz, 1974). Such effects naturally produce adverse influences upon mother-child interactions. However, it is more likely that such pre- and perinatal events will produce their effects via varying degrees of cerebral damage.

The value of information on pre- and perinatal events is weakened by its retrospective nature, and in many cases by the absence of specific data. Also, there are many problems with neurological dysfunction data. Our techniques for measuring cerebral dysfunction are still less than sophisticated, and it is possible that later

developments will enable us to attribute autism solely to a variety of brain damage. At present this appears unlikely, and an aetiological hypothesis based entirely upon a presumption of neurological impairment, quite possibly as a sequel to pre- and perinatal events, is unjustified. It may prove to be a necessary factor, but is most unlikely to be a sufficient one to explain the specificity of the syndrome.

Autism as a Severe Disorder of Language

Rutter (1968) proposed that childhood autism may be the result of a central disorder of language, including both comprehension and production, whilst 'social' symptoms were seen as secondary to the fundamental linguistic defect. He noted that there are several examples of children who became less socially withdrawn with increasing age, and yet in whom there were no apparent improvements in language functioning (Rutter, 1966, 1967). Thus he concluded that whilst there may be a severe social handicap accompanying autism, it is not likely to be causal, at least as far as the primary symptoms of impairment of intellect and language ability are concerned.

It is universally accepted that autistic children do display severe language deficits. As has already been noted, these children display markedly inferior scores on verbal subtests of intelligence tests like the Weschler Intelligence Scale for Children (WISC) in contrast to performance (Wassing, 1965; Rutter, 1966). Illinois Test of Psycholinguistic Abilities (ITPA) test profiles also demonstrate severe defects in almost all aspects of language functioning, especially in encoding functions and cross-modal coding (Tubbs, 1966). Deficiencies in the transfer of information from one modality to another has also been demonstrated experimentally (Hermelin and

O'Connor, 1970; Bryson, 1972). Echolalia and pronoun reversal are now seen by most as a reflection of comprehension defects (Bartak and Rutter, 1974). Many experimental studies of language functioning report severe deficiencies. Typical of these is the finding of Hermelin and O'Connor (1968) that autistic children re-group semantically a list of verbal items upon recall significantly less than controls. When subjects were asked to recall nonsense phrases and correct sentences, autistic children recalled in terms of recency rather than semantics. Such behaviour demonstrates deficiencies in verbal coding, semantic patterning, and the use of 'meaning'.

Churchill (1972) suggested that childhood autism and childhood schizophrenia are both extreme examples of "Developmental Aphasia", and that it is possible to separate aphasic and autistic children only on the basis of quantitative terms. He claimed that the similarities in sex ratios, incidence of apparent brain damage, phenomenology in terms of case histories and symptoms like echolalia and pronoun reversal, and response to therapy ('social' symptoms being more responsive), were sufficient grounds for implicating the qualitative similarity of the two conditions. Churchill taught groups of aphasic and autistic children a 9-word 'language' in the form of labels for objects and colours. Generalisation, modality, and perceptual-motor association tasks demonstrated that all subjects made errors in processing information or making association "... which would seem logically pre-requisites for language competence", (Churchill, 1972)^(p.90). Each subject yielded idiosyncratic error patterns that were stable over time. The limit on the ability to generalise verbally or generate spontaneity appeared to be common to both groups.

This however, is insufficient evidence for a model that places the two conditions on the same continuum. It merely emphasises that

both groups suffer from certain common marked linguistic deficiencies, whilst we know that the autistic child has other symptoms. The odd mannerisms, insistence on sameness, stereotypy and 'asocial' behaviour of the autistic child is not found in aphasics. Neither is the bizarre mixture of hypo- and hypersensitivities, frequently found in autistic children, or delayed echolalia (Ornitz, 1973). Rutter (1971), in contrast to Churchill, believes that the groups do differ in terms of gesture and 'inner' language, pronoun reversal and echolalia, and 'social' symptoms.

The extreme severity, and the specificity of the language deficiencies found in autistic children, have been clearly outlined by Bartak, Rutter and Cox (1975). Looking for factors which appear necessary for childhood autism rather than simply being associated with it (e.g., retardation), they compared autistic children of normal non-verbal intelligence with a group of children with severe defects in receptive language. This latter group display some 'autistic symptoms' and share with them a marked deficiency in the understanding of language. Behavioural assessments were made in the comparative study using the WISC, the Merrill-Palmer, the Reynell Developmental Language scale, the Peabody Picture Vocabulary Test, a behavioural checklist, and the Vineland Social Maturity scale. Autistic children were less mature on the Vineland scores and showed more behavioural 'disturbance' in terms of stereotypy, gaze aversion, temper tantrums, and non-cooperation. On the cognitive assessment there were also clear differences between the two types of child. Verbal-performance distinctions were greater with autistic children, and both Reynell and Peabody scores showed that the latter group suffered from greater comprehension defects. They used less non-verbal expression than the receptive language disordered children, were more hypersensitive,

and produced more metaphorical language, echolalia and pronoun reversal. Thus where language does develop in autistic children, it appears that it is not just delayed, but also markedly deviant as well as being more severe than that seen in receptive language disordered children. The authors in fact noted that in behavioural terms, there was even less overlap between the two groups.

Thus it appears that autistic children have grave deficiencies in almost all areas of language functioning which differentiate them from other children with delayed speech or even "dysphasia". Perhaps the most handicapping aspect is the gross impairment in comprehension, which certainly explains behaviours like echolalia and pronoun reversal, and quite possibly their lack of use of gesture. Rutter (1968) believes that other behaviours such as compulsions, stereotypes, and obsessions may be seen as maladaptive attempts to cope with the defects of language, although it does not seem altogether clear why these particular behaviours should develop. However, this hypothesis fails to predict two sets of symptoms frequently found in autistic children. Some symptoms appear in very early infancy, typically failure to adopt anticipatory postures, smiles, and hyper- and hypo-sensitivities. In some cases, one or more of these symptoms appear in the first few weeks of life, if one accepts the accuracy of at least a significant number of retrospective reports. It is known that almost from birth, infants can make increasingly fine auditory discriminations (Friedlander, 1970). Whilst this may be seen as the precursor to language comprehension, it seems unlikely that a failure to learn these earliest discriminations can be considered the basis of the first symptoms of childhood autism. Congenitally deaf children, who presumably are also unable to make these early discriminations, do not develop childhood autism.

A second set of symptoms which appear in autistic children may be included in a category labelled 'negativism'. Several studies have demonstrated experimentally the existence of these behaviours in the repertoires of a significant number of autistic children (e.g., Ney, 1967; Wallace, 1975). An aetiological hypothesis based on a model of childhood autism as a central disorder of language, would not predict, and indeed does not account for, an active process of non-compliance. Such a model may predict failure at certain experimental tasks, inappropriate or random responses, or a failure to respond at all to linguistic commands or language based tasks. It is possible that these consequently develop into active resistance. However, this model does not directly predict an active resistance to compliance and motivationally based fluctuations in linguistic competence. But Ney, Palversky and Markely (1971), in a comparison of the effectiveness of operant conditioning and play therapy treatment regimes, published results which suggested that at least some language defects in autistic children may be only apparent ones resulting from anti-communicative behaviour. Similarly, Morrison, Miller and Mejia (1971) found that in response to adult verbal requests, the subject's performance on a series of discrimination tasks rapidly changed from random to better than chance when one of the objects to be discriminated was replaced by a preferred object. This suggests that failure to discriminate was not caused by an inability to understand the request, but rather by motivational variables concerning compliance and resistance to requests. Cowan, Hoddinott and Wright (1965) reported evidence of negativism, with suitable contingent reward changing performance on a number of discrimination tasks. Negativism does not therefore appear to be simply another maladaptive response mode adopted by autistic children when confronted with tasks, particularly verbal ones, beyond their genuine ability. Wallace (1975) provided further data to support this

assertion by showing that negativism did not increase with increasing complexity of verbal requests requiring verbal responses.

It may be concluded therefore that language deficiencies in the autistic child are fundamental to the difficulties that the environment presents to him, but if one considers them in relation to a single factor aetiological theory, it does not appear to be sufficient to account for all his characteristic behaviours.

Autism as a Multiple Handicap

Wing (1966, 1969) suggested that autistic children suffer from multiple dysfunctions involving language, perceptual, motor, and 'behaviour' abnormalities. Any child suffering from a combination of aphasia, visual-perceptual problems, and an inability to understand and use gesture, could be correctly diagnosed as autistic. Wing (1971) compared the behaviour of a group of autistic children in their first seven years with children suffering from receptive aphasia, executive aphasia, and partial blind/deaf children using parental questionnaires. The linguistic problems of autistic children closely resembled the first two syndromes, and visual-perceptual problems and difficulties with non-verbal communication, were very similar to the latter group. Wing (1969) suggested that the number and severity of the different defects in social behaviour are related to the number of sensory, perceptual or executive handicaps found in combination in each child. However, there are symptoms characteristic of autistic children which do not appear in the other handicapped children that Wing describes. Ornitz (1973) points out that in neither aphasia or partially blind/deaf children are there the bizarre hyper- and hyposensitivities that are found in autistic children. Perhaps the symptom that appears most unique to autistic children is their apparent failure to use any mode

of communication and their asocial nature. Certainly the partially blind/deaf show active interests in their environment and learn other channels of communication than via their impaired function. Executive aphasics use non-verbal gesture and communication, and even the receptive aphasic children use such communication far more than autistic children (Bartak, Rutter and Cox, 1975). All three groups tend to be more socially mature and responsive than autistic children.

Perhaps the strength of Wing's argument lies in the difficulties in upholding a unitary impairment hypothesis. Thus Wing and Wing (1971) point out that a central language disorder cannot explain the difficulty in coping with skilled movement, postural problems, and immature physical development. Puzzling anomalies, seen in apparent outstanding abilities (typically in mathematics, music or memory), yet chronic linguistic impairment, do apparently exist. Similarly, the memory for exact location of objects in a room or verbatim recollection of T.V. commercials, seem odd in contrast to the perceptual difficulties extensively reported (e.g., Hermelin and O'Connor, 1970; Bryson, 1970). It is true that in syndromes like "aphasia" several 'autistic elements' appear. Also, many of the impairments found in autistic children can often vary apparently independently of others in severity. Thus child A may be very poor at auditory perceptual problems but be very skilful at fine motor control, yet child B displays the converse.

But having said this, it is difficult to appraise the Wings' theory as anything other than descriptive, rather than an explanatory hypothesis. They have apparently grouped symptoms together, assumed that no single mechanism or impairment is common, and thus concluded that the autistic child is multiply handicapped. He is considered the end result of adding together the impairments of aphasics and the part-

ially blind/deaf. No prediction or provision is made for the possibility of interaction among the impairments. Such an additive model is not concordant with our increasing awareness of the interactive nature of behaviour problems in handicapped children (Chess, 1969; Williams, 1968). It would seem quite possible for a multiplicative interaction to yield characteristics that are quite different from the sum of the individual components themselves.

Whilst the Wings' formulation may be seen to account for variation in the clinical picture amongst autistic children since each dysfunction could occur in varying degrees of severity (Wing, 1971), it may be simply describing diagnostic variance or the overlap in symptomatology with certain other childhood disorders. It is difficult to envisage this hypothesis as increasing our understanding of the underlying aetiology of the condition even if it were acceptable. The contradictory evidence noted does not make it so.

Perceptual Deficiencies and Childhood Autism

There now exists a considerable body of experimental evidence that autistic children suffer from marked, and characteristic, deficiencies in areas of perceptual functioning. The exact origin of these malfunctions is unknown, although it is generally considered to be of a biological origin and neurologically based.

Difficulties in cross-modal coding have been observed in many independent studies with autistic children and a variety of controls, and these have clear implications for the deviant behaviours seen in these children. This deficiency was first noted in psychometric studies (Tubbs, 1966; Whittam, Simon and Mittler, 1965). More recently, cross-modal coding deficits have been reported in the

failure of autistic children to imitate body movements successfully (Churchill, 1969; Hingten and Churchill, 1971). Such behaviour requires short-term visual memory and transfer to the motor system. DeMyer et al., (1971, 1972a) found that autistic children can spontaneously use objects (demonstrating an intact motor system), can imitate motor-object behaviours (where there is help from a visual reference point), but have grave difficulty imitating pure body movements (where there are no memory or immediate visual cues). Thus they conclude that there is a deficiency in motor-visual association. Similar performance is found in patients with ideomotor apraxia (Mittler, 1970). Hermelin and O'Connor (1970) reported that autistic children are helped less by the addition of visual cues in a motor performance task than either normal or retarded controls. Bryson (1972) carried out a series of matching tasks under simultaneous and delayed presentation conditions. For both conditions autistic children showed serious auditory-visual association defects.

There have been several studies which have yielded data suggesting deficiencies in the visual memory of autistic children (Schopler, 1966; Frith and Hermelin, 1969; Bryson, 1972).

DeMyer et al., (1972) remarked that together with cross-modal coding deficits, this may explain the asocial nature of the autistic child and many of his 'social' symptoms. Thus non-verbal communication in early infancy, such as the smile, wave, or anticipatory gesture, is not learnt because of the failure to remember visually his own or others' motor actions together with the inability to transfer information from the visual to the motor system. Failures in acquiring secondary reinforcers and in imitation are added to deficiencies in auditory processing. This may explain why the autistic child does not develop 'social' behaviour, and can frequently only appear to

understand those things that have regular repetitive patterns, such as television commercials and locationally permanent objects.

Hermelin and O'Connor (1970) summarise several experiments which make a significant contribution to our understanding of the perceptual deficits in autistic children. Sensory dominance tests suggested that visual stimuli are dominant over auditory stimuli for both normals and autistic children in choice tasks, but in contrast to controls it appears that touch dominates sound for autistic subjects. Frith and Hermelin (1969) report that autistic children make less efficient use of visual cues and more of motor feedback compared with controls, and this is concordant with reports that autistic children prefer the use of proximal rather than distal receptors (Goldfarb, 1964; Schopler, 1965). Hermelin and O'Connor note that vision may aid performance on many motor tasks by some 'planning' process or anticipatory set, which autistic children fail to use. They conducted a picture series completion task with varying degrees of motor and visual cues for autistic children, and found that 'higher perceptual ability' subjects did use predominantly visual strategies but 'lower perceptual ability' subject did not (perceptual ability was scored from tasks adapted from the Frostig test). They concluded that with increasing development, the performance and strategies of autistic children become more like that of normals, but more retarded autistic children are apparently unable to integrate cues from different modalities.

Differential performance between autistic children was also reported by Hermelin and O'Connor in discrimination learning experiments, when subjects were split into those displaying some spoken (although deviant) language and those who were effectively mute (non-verbal group). The latter subgroup performed at chance level through-

out discrimination learning on brightness, size, shape and direction dimensions, but 'verbal' autists found shape and direction more difficult than size and brightness (Hermelin and O'Connor, 1965). Data on visual inspection times for pairs of stimuli varying in identity, size, brightness, colour, complexity and 'meaningfulness' however, suggested that despite short inspection times, the relative distribution of fixation for each dimension was the same for autistic and control subjects (O'Connor and Hermelin, 1967a). This may suggest a difference in discriminatory capacity in contrast to discriminatory learning seen in the previous study. Nevertheless, further experiments on identity, direction, and position discriminations, also yielded differences between verbal and non-verbal autists. These differences may be important in allowing us to study impairments possibly not directly related to verbal deficits, and a dysfunction fundamental to autism rather than associated with it.

Hermelin and O'Connor suggest that language defects may be simply a reflection of a more general coding or perceptual impairment, rather than the basic cause of the inability to structure the environment. Thus they tested non-verbal operations which parallell the type of functions that are found to be impaired in analysis of linguistic defects (e.g., Tubbs, 1966). These included seriation, cross-modal coding, immediate memory, and cue-matching tests (O'Connor and Hermelin, 1965). Non-verbal autistic children responded at chance level throughout all tests, but verbal autists only responded this way on the seriation and cross-modal coding tasks and were comparable to normal subjects on the memory and cue-matching tests. As noted earlier, several studies report similar cross-modal deficiencies in autistic children on a number of association tasks (Bryson, 1972; DeMyer et al., 1971, 1972a). Hermelin and O'Connor then tested

autistic subjects on a unidirectional seriation task not requiring the concept of reversibility, using a series of picture cards and varying the relationship between them from random to a meaningful sequence (O'Connor and Hermelin, 1967b). Autists made more errors than normal controls, but nevertheless they could order this series thereby using meaningful order as an aid to recall, and appeared to be able to process visually presented semantic sequences. They then carried out experiments to see whether such ordering was in any way comparable with the ordering of words in the construction of sentences. Using selected monosyllabic words from a vocabulary list for subnormals, sixteen 4-word sentences were constructed varying in frequency of occurrence together and in meaning. Results suggested that for subnormal controls, words matched for frequency of presentation were easier to recall than when in random order. For autistic subjects this difference was much less pronounced, and the authors concluded that perhaps failure to recall words was due to the lack of ability to predict the items on the basis of previous knowledge of the nature in which they are organised. A further experiment in which words were arranged in meaningful and in random order, revealed that recency was a very important factor for autistic children compared with controls. Hermelin and O'Connor then investigated whether not only grammatical structure, but also semantic appreciation, is impaired in autists. Using word strings exceeding the immediate memory span, it was found that recoding of semantically related items into their related 'clusters' upon recall, appeared significantly more often for controls. This suggests that autistic children lack the ability to associate words semantically, their recall depending on acoustic and phonetic aspects of speech. Varying stress and meaningfulness, subsequent work suggested that higher memory span autists did use grammar, but only to

a very limited extent. Stressed words were recalled better than unstressed words, but the nature of the words appeared irrelevant.

This supports the notion of an 'echo-box' memory system.

A final aspect of perceptual deficits in autistic children arose from operant programmes in which wide individual differences were noted in the acquisition of several discriminations. It was also noted that autistic children on operant programmes frequently showed rapid improvement in some areas and slow, minimal gains in others for no apparent reason. In addition to the mixture of hyper- and hypo-sensitivities frequently reported, this inconsistency and variability was related to an attentional hypothesis (Lovaas et al., 1971).

Lovaas found that when confronted with a complex stimulus, autistic children selectively responded to only one element in the complex cue. Thus in discrimination learning tasks, only one dimension is attended to. They speculated that such a process of 'stimulus overselectivity' may retard the acquisition of many behaviours, particularly language and interpersonal and intellectual behaviours. Further evidence for an inability to utilise simultaneous stimuli comes from a study in which autistic subjects were taught to respond to a simple 2-component cue. When later they were presented with the cues individually, they still only responded to one component (Lovaas and Schreibman, 1971).

Lovaas believes that autistic children do not show a preference for any one modality, or an impairment in any one modality. Rather the problem is one of an overselection of modalities (Stevens-Long and Lovaas, 1974). He notes that such an overselectivity would seriously impede the learning of contiguous or near contiguous stimuli, which is fundamental in learning. Such a defect has of course implications for the way autistic children should be taught. Koegal and Schreibman (1974) present data that suggests we must provide intra-

stimulus prompts (e.g., emphasis of the critical component of the discrimination) rather than the more typical extra-stimulus prompts (additional cues), in the light of overselectivity, for language and educational learning programmes.

Kovattana and Kraemer (1974) also found evidence for a process of stimulus overselectivity in autistic children, using colour, size and shape discriminations. They also noted that it applied only to non-verbal autistic children, for verbal autists used all three cues. The latter subgroup performed better than subnormals control subjects, and thus it appears that both mental age and language development are important variables in predicting behaviour on these tasks.

To summarise, a large body of evidence now suggests that autistic children have marked perceptual deficits. Cross-modal coding deficiencies have been reported in many studies. Hermelin and O'Connor have documented an extensive and impressive series of experiments. They conclude that very short inspection times, the use of motor cues in preference to visual ones, and the inability to organise reversible sequences, indicates a defect in the capacity to classify and interpret visual material. This pattern is matched in the auditory channel, with defects in semantic and syntactic aspects of language. These perceptual defects result in an inability to appreciate meaningfulness and order in the sensory input as a whole. Lovaas and his colleagues report a process of stimulus overselectivity in at least the visual channel, which clearly has a profound inhibiting effect upon the normal process of learning.

The differential performance of verbal and non-verbal autistic children would appear to imply one of three things: There may be heterogeneous experimental samples under study, such that only the 'non-verbal' autists constitute acceptable cases of childhood

autism. This appears most unlikely, in view of the diagnostic criteria used by Hermelin and O'Connor. It is also possible that in verbal autists we are observing what is fundamental to childhood autism itself, whereas with non-verbal autistic children, associative additional linguistic deficits add to the perceptual deficiencies measured. Finally, one may view the verbal/non-verbal difference in terms of a gradation of autistic behaviour, thus degrees of childhood autism exist. The latter assumption makes research into the minimum necessary dysfunctions for childhood autism a difficult task.

Autism as a Cognitive Disorder

Frith (1968, 1970a, 1970b) extended the approach of Hermelin and O'Connor by studying general cognitive functioning using pattern perception measures. As Miller (1967) pointed out, virtually all cognitive phenomena involve pattern perception, since the reduction of the sensory input by categorising is the only way of dealing with huge inputs. Frith carried out a series of immediate recall tasks using binary sequences as the stimulus input. Such sequences are extremely useful because they can be generalised, are abstract, yet can be organised into definite patterns and can be presented in many modalities. Using sequences of sufficient length as to exceed the immediate memory span thereby enforcing 'coding' for correct recall, normal subjects reflected the dominant structure of the presented series (input) upon recalling sequences of verbal and visual stimuli. These subjects exaggerated the dominant features of the patterns in recall (output), and learning errors could be predicted on the basis of the dominant feature of the pattern. Autistic children however, did not feature extract in this manner, but imposed their own simple stereotyped

patterns on the recall measures (e.g., strict alternation or perseveration). Errors could not be predicted on the basis of the input sequence, but rather upon the autistic child's own internal 'rules' regardless of the structure of the input pattern.

In an experiment on the spontaneous production of sequences, subjects were first required to predict each successive colour in a random sequence of red and black cards, and secondly to set out a row of counters using two colours. Older normal children very accurately 'probability matched' in the first task, while younger subjects did not. Autistic children imposed their own simple rules in the sequences they produced. In the second task, whilst normal children imposed a non-random structure in their responses, the autistic subjects produced much shorter, stereotyped patterns. Thus under conditions of unstructured input, there was a strong tendency for pattern imposition in both groups, although for the autistic child the rigidity of imposition and simplicity of the pattern was particularly marked.

It appears therefore as if the autistic child treats structured, patterned input as if it were random. He appears to be lacking in the capacity for feature extraction from ordered input, with structured sequences being treated in the same way as random ones, with the imposition of simple patterns which although themselves non-random, are nevertheless stimulus independent. The consistent results with a variety of materials and sequences suggests that this model of cognitive behaviour in autistic children appears to be independent of modality and material. The apparent inability to differentiate between structured and random input reflects, perhaps, the dominance of internal factors. If, as Frith suggests, one can envisage cognitive activity, as involving the matching of stimulus input to an internal schema or plan (paralleling the Piagetian

notion of "assimilation" and "accommodation"), one could suggest that autistic children are characterised by an imbalance in favour of "assimilation".

Hermelin and O'Connor (1967) reported that autistic children do not treat meaningful and syntactically well-formed sentences as different from random word strings. Frith's work has shown that this is apparently not restricted to language, but also to pattern detection in visual and auditory modalities. It may therefore apply to cognition as a whole. The inability to feature extract and use redundancy, thereby treating the input as random, appears to explain the linguistic behaviour. Echolalia and stereotyped repetition may be seen as a ^{often} reflection of an internal speech rule and this is/ the only linguistic output of the autistic child. Frith correctly points out that the appreciation of rules by extracting important features, would seem to be crucial to social interaction and play, skills which are clearly impaired in these children.

Frith (1972) further examined self-generated rules using materials that enabled the possibility of more complex pattern production. In the first task subjects used rubber stamps and inkpads to produce spontaneous patterns of either two or four colours, and in a second task subjects were encouraged to produce a series of tones on a xylophone. Frith reported that "low" mental age autistic subjects displayed significantly greater restriction and rule adherence than other subjects. All autistic children, regardless of mental age however, revealed less 'originality' in their spontaneous sequences than controls. A modality comparison suggested that autistic children produced more complex tone than colour sequences, and more complex tone sequences than even normal controls. They were also characterised by less restriction and greater originality with tone sequences compared

with visual patterns. Commenting on the finding that rule adherence did not increase with increasing mental age in autistic children, Frith suggested that for non-autistic children it appears that in the development of rule-governed behaviour a transition point occurs where rules are no longer used loosely but become more strict. Frith concluded that this point could be at the end of the critical period for early language development as this depends upon the flexibility of rule application. The lack of flexibility of autistic children may thus inhibit the learning of new rules.

While reiterating that autistic children produced more complex sequences with tones, and generally approached 'normal' patterning in this modality, Frith merely commented that: "This brings to mind the clinical impression that autistic children are often musical". (p.1

If this finding is in fact valid, it would present problems for a theory of a general cognitive dysfunction for it is hypothesised as a non-modality specific defect. However, it should be noted that her experiment in the visual modality used a control method to prevent visual matching by requiring different colour dots on different pages of a book, and the colour of the inkpads was hidden from sight. Yet in the auditory modality the "sustaining power" or decay properties of the xylophone notes, may have allowed auditory comparison between notes, and thus invalidated Frith's control for immediate memory effects.

But despite these methodological considerations, it is interesting to find that Frith reported autistic children approaching 'normal' patterning with spontaneous production of tone sequences. This appears particularly relevant to studies conducted by others reporting differential performances on a number of tasks. Studies finding quite startling changes in performance from apparent negativism or random responding to successful discrimination learning have already been discussed (e.g., Cowan, Hoddinott and Wright, 1965; Morrison, Miller and Mejia, 1971). Serafica (1971), while testing Bettelheim's

assertion that autistic children function at the fourth stage of the Piagetian Sensorimotor scale, found that subjects performed at a significantly 'higher' level when using preferred stimulus materials. These studies lead one to question how real some of the suggested cognitive defects are. If some of them are only of an apparent nature, then motivational variables may be important. It may prove useful to repeat some of Frith's experiments using preferred, familiar stimulus materials, perhaps those of known high affective value for each child.

Not all behaviours displayed by autistic children fit the cognitive defect hypothesis outlined. Frith concluded that the autistic child treats the world as if it were random, imposing simple patterns upon it. Yet the studies demonstrating an active process of negativism, avoidance of eye contact as an active process, the bizarre attachments, and the effects of group density (Hutt and Vaizey, 1966), all suggest anything but a random approach to the 'social' world.

Whilst we may question the degree or generality of 'cognitive pathology', or as Rutter (1974) succinctly puts it, the: "boundaries of the cognitive defect", it is clear that autistic children do have pronounced deficiencies in perceptual, linguistic, and coding functions. Wassing (1975) concluded from a psychometric analysis of intellectual functioning in autistic children, that if the tasks are at a purely perceptual level, the child can achieve quite well. But he fails at "thinking proper", perhaps a result of a disability in dealing with imaginary situations and things. He fails to objectify what is not present in his immediate, concrete environment. Tilton and Ottinger (1964) analysed toy play behaviour of autistic and control children, and concluded that the former may have unique deficiencies in perceiving relationships between objects. Hermelin (1972) summarised

studies aimed at observing how autistic, blind, and deaf children code events in terms of spatial location and temporal sequences. Using designs in which subjects could choose in their method of coding, she concluded that autistic children resemble normals in coding in visual terms to locate a point in space, but resembled deafts in the coding of visually presented verbal items. They seemed less able to locate events according to their temporal positions. Thus autistic children do not appear to process visual material inadequately, rather they have a disinclination to use words as coding units. It is not a question of an abnormality in the structure of sensory channels, rather it is a case of the availability of codes for organisation, which may be relatively independent of the modality in which the stimuli are presented.

Rutter (1974) notes that many cognitive processes are indeed related to language. There is some evidence of a relationship between language and sequencing skills (Rutter and Yule, 1975), and speech delay has been linked to sequencing and auditory deficiencies as well as specific impairments of speech. The particular deficiencies of the autistic child are certainly broader and deeper than those seen in receptive language disordered children (Bartak, Rutter and Cox, 1975), but whether these extra deficiencies reflect impairment in aspects of cognition beyond those involved in language, Rutter believes to an unanswered question to date.

To a large extent this latter problem is of academic, and possibly dubious, importance. It rests upon the largely obsolete question of the relationship between language and thought. As Hermelin and O'Connor (1970) point out, perhaps thinking and cognition depend upon language, and logic is simply a convention. Therefore a lack of linguistic skills results in a lack of a system into which experience can be coded. On the other hand it is possible that a cognitive or

coding defect precludes the development and acquisition of language, much of which at least we know involves learning. Thus the language impairment may be seen as symptomatic of cognitive pathology consisting of impairment in the processing of temporally structured input. The separation of language and thought is more likely to be an artificial one for the two are dialectically related (Vygotsky, 1962). It may be more fruitful to conclude that there is now sufficient evidence to believe that autistic children suffer marked impairments in areas which we traditionally call perceptual, language, and cognitive functioning.

The Question of a Multi-Variate Aetiology

Single factor aetiological hypotheses to date have failed to fully identify the relevant details of the origins of childhood autism. It is not surprising therefore, that it has been suggested that the condition may be the result of a number of factors and that childhood autism may be the end-product of a number of possible aetiologies (Chess, 1969; Schopler and Reichler, 1971). Weiland (1971) concluded in his analysis of treatment approaches that: "The very heterogeneity of manifestations and responses of this syndrome, make it very likely that we are dealing with a set of symptoms which, like fever, can be the result of a variety of causes, and therefore requires a selectivity of treatment".

The emotional nature of much of the aetiological controversy has undoubtedly led to prejudice and polemics, which favour single factor hypotheses. Eisenberg (1971) pleaded in his introduction to a quite recent symposium: "Let us hope that we may all avoid the self-defeating nature-nurture opposition that dooms the proponents of either camp to ignore the findings of the other, and to do a disservice to

their own research by failing to control the other variable". (p.4). Rutter (1974) notes that much aetiological research is conducted on the basis of nothing more than an assumption that childhood autism has a single cause. We have long ago recognised that there are several distinct conditions included in the category of 'mental retardation'. Goldfarb (1965) commented that we cannot assume a single aetiology simply because we have derived a unitary classification for these children.

However, it does not necessarily enlighten our knowledge of the condition by simply grouping together a host of single factors found to correlate with it, and conclude that each child suffers from one of a number of possible combinations of these factors. One must consider the interaction between factors, and the probable multiplicative nature of such an interaction. Chess (1969) stressed the need to study the simultaneous and interactive operation of environmental and organic components in the aetiology of the syndrome. She emphasised that the effect of an organic factor is determined solely by interaction with the environment, and can be demonstrated only by its social expression. Chess illustrated the complexity and multiplicative nature of aetiological variables by suggesting that if a child with a defective capacity to learn socially flexible patterns of behaviour meets environmental demands he cannot fulfil, these demands will lead to bewilderment and further deviant behaviour. This in turn adds to the non-adaptive, repetitive, withdrawn behaviour already in his repertoire. At school, many more social demands are made, and he may become upset at changes in his routine. Expectations become more complex, and his distorted responses may invoke rebuke, with resultant anxiety. Thus Chess sees the crucial role of the environment epitomised in the case of two subjects with the same type and

degree of brain damage, with the differential reactions of other people to identical deviant behaviour, both at home and at school, largely determining development and outcome.

One example of a multi-variate aetiological hypothesis is that of Goldfarb (1961, 1964). He suggested that multiple and combined aetiological factors exist in childhood autism, these being very general categories of impairment, of intrinsic organic origin and of extrinsic environmental origin. Each child may be characterised by a varying combination of these two types of factor. Goldfarb proposed that one could envisage all autistic children upon a continuum such that at one end are placed somatically intact children with pathological environments, and at the other end are somatically impaired children in a normal environment. In between, and this Goldfarb believes applies to the great majority, are cases characterised by varying combinations of the two types of factor. It should perhaps be noted at this point that although Goldfarb himself referred to his subjects as "childhood schizophrenics", he used the diagnostic criteria of the B.W.P. (Creak et al., 1961) which corresponds closely to Kanner's concept of autism.

Goldfarb sees childhood schizophrenia as an end manifestation of a sequence of interactions. Cerebral dysfunction is viewed as restricting the child's adaptive competence, also stimulating certain types of response in the parents, and these in turn reinforce particular behaviour in the child (Taft and Goldfarb, 1964). Meyers and Goldfarb (1961) attempted to test what they claimed to be a repeated clinical observation of a particular set of responses in parents of childhood schizophrenics, collected under the term "parental perplexity". This parental behaviour is characterised by passivity, marked uncertainty, lack of spontaneity, absence of empathy with the child, bewilderment at his bizarre behaviour, and absence of forthright

parental control. Meyers and Goldfarb suggested that this behaviour is often disguised by a compensatory dependance upon 'mechanical intellectualisation', and the use of an inflexible set of parental rules. These are features shared with the parental stereotype stressed by Kanner (1943, 1949) and Eisenberg (1957). The "parental perplexity" is seen as presenting the child with inconsistent and confusing environmental experiences, an absence of necessary reinforcement for organising and integrating the sensory world at a very early age, and a resultant defect in the self-concept (Meyers and Goldfarb, 1961). Goldfarb sees the formlessness of the child's world resulting, alternatively, from his own intrinsic inability to structure his environment. Those childhood schizophrenics with brain damage and associated defects in perceptual, cognitive, and output functioning, will also display impairments in ego and self-identity development, and at the extreme end of Goldfarb's continuum will do so on the basis of cerebral dysfunction alone. Some parents will show deviant behaviour but others will behave totally within accepted norms. Such variability, whilst apparent when reviewing a number of studies in the literature, has not been fully accounted for in most other aetiological theories.

Meyers and Goldfarb (1961) carried out three studies to investigate the 'parental perplexity hypothesis'. Using a participant observer technique, patterns of family interaction were rated on a number of scales measuring various aspects of 'parental perplexity'. A child neurologist, naive in respect of the nature of the study, divided subjects into those displaying signs of clear neurological defects ("organic" group), and those not displaying these signs ("non-organic" group). The "organic" group revealed no significant differences on the rated scales compared with controls, but the "non-organic" group did, both these findings being predicted from the hypothesis.

To eliminate any contaminating effects from case history, diagnostic, and psychopathological knowledge, subjects very recently admitted to the Ittelson Research Centre were observed in a second study and the same findings emerged. One obvious criticism of this study is that parents may be simply reacting to a difficult child. Goldfarb (1959) reported that it is the "organic" group who display the most deviant behaviour, using three naive observers to rate deviancy, and yet they had the mothers who displayed the least 'perplexity'. However, knowledge of "organic" impairment may well alter the responses of parents to the child in contrast to parents without such information.

The limits of clinical judgement and rating scales were recognised by Goldfarb, and later work was directed at systematic study of more precise aspects of parental behaviour. Goldfarb, Goldfarb and Scholl (1966) proposed that parental perplexity would be reflected in the speech of mothers of childhood schizophrenics and would be inferior to that of normals. Thus childhood schizophrenics would have poorer speech models to emulate than normals. A speech pathologist evaluated the speech of childhood schizophrenics and their mothers in a structured situation. Measures rated included volume, pitch, voice 'quality', rate, phrasing, fluency, stress, intonation, and communication of meaning and mood. Significant differences were frequently reported between experimental groups and controls. The authors claim that the poor communication of thought and feeling in mothers of childhood schizophrenics indicates the lack of the most important instrument for enhancing normal attachment responses in their children, for educating and for guiding their growth, and for assisting them to achieve order and clarity. This is quite apart from being a poor speech model for the learning of the acoustic and grammatical aspects of language. It is true that the childhood schizophrenic does have disorders of perception, integration and

executive functions due to his own intrinsic impairment (Goldfarb, 1961). But the mother's modelling failures are seen as an addition to the child's initial defects through the provision of an inadequate^{linguistic} and affective environment.

Goldfarb, Levy and Meyers (1966) studied the clarity and use of the mother's speech to her child, and reported several deviant aspects. These included: (1) Failure to stimulate the child's interest in active communication, being typically rather passive and withdrawn; (2) Failure to maintain a continuous flow of communication; (3) Failure to reinforce normal and acceptable speech, seen in the lack of correcting bizarre expressions, relevance and logic in the child's utterances; (4) Active confounding of the child by the use of evasive, incongruent and illogical expression; (5) Failure to grasp or active disregard of the child's cues, either intellectual or affective; (6) A failure to cope with the communication deficiencies of the child arising from the severity of his deviations and deficiencies.

Goldfarb, Levy and Meyers (1972), comparing the performance of the two sub-groups, found significantly lower clarity scores for mothers of 'non-organic' subjects, in line with their hypothesis of the continuum of an interaction between organic and environmental factors. These mothers showed particularly unclear communication and accompanying regulation and orientation of their children. The child responds to this with apparent puzzlement and disinterest. As the authors remark: "Most important of all, in the face of environmental ambiguity, they are not actively stimulated to develop increasingly differentiated language and purposeful responses". Goldfarb also reports parental language deficiencies in referential communication tasks in support of his hypothesis of aberrant patterns of communication in the families (Goldfarb, Yudkowitz and Goldfarb, 1973). Referential communication

tasks involve the construction and communication of a verbal description of a target object, in order that a recipient may be able to discriminate this target from an array. The task thus involves a degree of verbal competence, but more important, role-playing skills. It has been frequently used in this context in developmental studies (e.g., Krauss and Glucksberg, 1969).

Goldfarb and his colleagues have based their work around an attractive hypothesis which does account for many apparent discrepancies between studies revealing parental pathology and no consistent neurological impairment, and on the other hand those reporting such impairment and no consistent parental pathology. Thus whilst appearing not to differ in overt behaviour, subject samples used in these studies may possibly have been skewed in opposing directions along the hypothesised aetiological continuum. Not only does Goldfarb stress the interaction of determinants, but also he provides for individual differences within samples of childhood schizophrenics and for the apparent heterogeneity of subjects within samples. Whilst conceding that an 'organic' basis may underlie some cases, the impairment varying in degree, Goldfarb has studied in some depth the nature of the role of parents in aetiology. Thus not only may the negative studies on the contribution of parents have been a function of the sample studied, but also the techniques of measurement may well have been inappropriate. Studying behaviour in parental interviews and on personality questionnaires are comparatively imprecise, unreliable methods of assessment. More complex, less manifest parental behaviours than 'rejection' or 'overprotection' may be important (Goldfarb, 1961). The study of family interactions is a more comprehensive and relevant technique for analysing more subtle aspects of their behaviour. Indeed, there is an onus upon those who believe there is a parental contribution to the aetiology to explain how it

functions in the development of the syndrome. Many children with a known degree of cerebral damage present behaviour problems from birth and it must be explained how reactional behaviour from these parents is different from that of the parents of autistic children. However, although Goldfarb has studied an important aspect, that of mother-child verbal interactions, his data was collected by observer ratings with the accompanying contamination from the particularly subjective nature of such ratings.

Howlin et al., (1975b) commented that Goldfarb's techniques are not altogether appropriate for studying fundamental variables. Thus the use of surprise visits are not conducive to the measurement of typical behaviours, the periods studied are frequently very short, only verbal deficits are analysed, and categories of speech scored are somewhat 'loose' in definition.

Perhaps the most important merit of Goldfarb's work is his emphasis upon mother-child interactions, for whatever organic limitations there may be acting on the child, it is via this interaction that the child will learn, or fail to learn, so much of the necessary information for self-identity and organisation of his world. If this relationship is pathological or aberrant in any way, then treatment programmes must involve the mother as well as the child. It is clear that many parents reward several inappropriate verbal and non-verbal demands, frequently with contingent attention or compliance with the child's demands (e.g., Kozloff, 1973; Lovaas, 1971). This may be due either to a desire to maintain some form of communication however basic it may be, or to pressures to pacify a highly 'demanding' child. Ferritor (1970) emphasised that whatever the initial origin, once started, the 'autistic pattern' may be maintained by pathogenic

mother-child exchanges. He taught parents to reward and attend only to attempts at appropriate verbalisations. A three-month follow-up study suggested that all subjects displayed increased verbalisations, and parental training had increased reciprocity in structured situations, and increased the frequency and complexity of such interactions.

It must be noted that whatever deficiencies have developed in parent-child communications, Goldfarb's work does not necessarily tell us about a primary 'psychogenic' aetiological factor. His testing was inevitably conducted long after the initial diagnosis of the child. Any deviations in the parents' speech is more likely to be a function of a reaction to the child's behaviour and the knowledge that he is autistic, with the accompanying expectations that this diagnosis carries. This criticism is not of course intended to imply an irrelevance to such research. Indeed, any deficiencies in parent-child communication, however they arise, is clearly of utmost importance to treatment considerations. Most psychologists working in this field would not deny the importance of mother-child interactions or their relevance to the maintenance of some aspects of autistic behaviour. However, an appraisal of Goldfarb's data may lead one to conclude that he has identified some aspects of parental behaviour that may be important for a number (although by no means all), of autistic children. These parental behaviours are unlikely to be a primary aetiological determinant of the condition, but have implications for treatment and the current level of functioning of a significant number of these children.

Recent research has demonstrated the existence of severe perceptual, cognitive, and linguistic defects in autistic children. This does not relegate the importance of the environment in determining

the child's future development to a secondary role. It is the 'match' between the child's needs arising from his deficiencies, and his immediate environment, that we must strive to understand and optimize.

CHAPTER TWO:

"EXPERIMENTAL STUDIES: THE RATIONALE"

From a study of the literature on childhood autism to date, there appear to be at least four general conclusions to be drawn. First, it is clear that our knowledge of the aetiology of the condition is deficient in many respects. There is an abundance of aetiological hypotheses, a number of which are now largely discredited or substantially lacking in supportive evidence (e.g., psychodynamic and genetic models). Some are constructed in such a way as to be untestable or certainly not amenable to experimental analysis (e.g., most psychodynamic formulations). Some hypotheses clearly require further refinement, specificity, and research. Foremost amongst these may be considered those approaches stressing perceptual, cognitive, and language deficiencies, and certain of the neurophysiological and biochemical dysfunctioning hypotheses.

Diagnostic confusion, arising from both the use of differing criteria and from imprecision in behavioural descriptions, have added to the difficulties in appraising aetiological research. Caution must be adopted in the acceptance of any aetiological formulation or research finding where satisfactory subject population descriptions are not detailed. A multi-axial classificatory scheme would appear to be the most appropriate format to adopt, with a clear denotation of the behaviours displayed and the criteria used in ascribing the diagnostic label of childhood autism to the children under consideration.

It must be concluded however, that a plethora of unsatisfactory aetiological hypotheses exist, and we are still a considerable way from a full understanding of the origins of childhood autism.

The second conclusion is that much of the well-designed research has led to a growing consensus that childhood autism is a syndrome with a primary biological origin. The precise nature of the impairment is unknown, but there is much evidence that these children

display impairments in perception, language, and cognition. Thus it seems that we can delineate deficiencies in functioning in autistic children, which we may presume to have an 'organic' basis, without being able to specify their exact origin.

Our knowledge, or rather lack of it, concerning the precise aetiology of childhood autism, means that at present our treatment procedures and educational methods remain, to a considerable extent, independent of aetiological considerations. It is quite possible that they will remain so even when our understanding of the aetiology is complete. The most promising current approaches involve a variety of behaviour modification techniques with programmes designed for the individual requirements of each child. The third conclusion to be drawn from the literature to date however, is that despite the apparent independence of treatment (including education) and aetiology, we must incorporate our knowledge of specific perceptual, language, and cognitive deficits into these treatment programmes. Our techniques will be significantly less than maximally effective if we fail to do so. Thus data on the limitations of the power of secondary reinforcers, on stimulus overselectivity, cross-modal information processing difficulties, severe comprehension and expressive defects in language, and the use of internal stereotyped 'rules' are merely examples of research that has forceful implications for the way we design treatment and educational programmes. The use of 'intra-stimulus' prompts and the initial use of primary rewards in any discrimination learning setting, are also only examples of the way we may use our research findings in this manner. The vast majority of studies that have yielded data on specific deficiencies in the autistic child are of recent origin. There are several areas of functioning that also need exploring. Further research in these areas of deficit is needed to confirm the

proposed areas of dysfunction, to test the degree and generality of the deficit, and to look for possible further characteristic abnormalities in functioning.

A final conclusion may be that despite the extensive volume of research published since the original delineation of the syndrome, there remain many unanswered questions. The autistic child presents intriguing, but urgent, problems. It is essential that further research attempts to answer some of these questions and to add substantially to our knowledge of his characteristic deficiencies and his milieu, in order that we may provide an optimal environment tailored to his individual needs. At present the prognostic situation is very poor.

This thesis was therefore directed towards such research, and consists of three major areas of enquiry. The first concerns an analysis of certain features of the autistic child's environment. Where 'psychogenic' protagonists report evidence of environmental variables that they consider to be relevant to the aetiology of childhood autism, their arguments are frequently refuted on methodological grounds and also because it is clear that they have not separated a primary aetiological factor from a parental reaction to a severely behaviourally disturbed child. These criticisms are undoubtedly valid, but do not necessarily imply that such features are unimportant. Clearly the reactions of parents to the autistic child's deviant behaviours are of prime concern to those involved in treatment and education. We know little of the way in which parents attempt to cope with the autistic child's demands arising from his intrinsic deficiencies. In addition to individual variations amongst these children, there are many features common to them. There is a need therefore to study common difficulties and common strategies used by

parents in an attempt to overcome these problems. We are deficient in specific data on what may be common to the type of linguistic environment parents provide for these children, the type of reinforcement contingencies they deliver, their method of interaction with the autistic child, responses that may maintain some aspects of the deviant behaviour, and many other features of parental behaviour. The first study to be reported attempts to yield information on some of these points via a comparative analysis of parent-child interactions with normal and autistic children.

A second area of research concerns the measurement of autonomic responses of autistic children to certain environmental stimuli. Many autistic children are non-verbal and all display severe expressive and communication defects. Similarly, in many situations no overt responses to environmental stimuli are observed, and such apparent unresponsiveness has led to clinical descriptions of the autistic child as aloof and severely withdrawn and also to aetiological hypotheses concerning arousal and attentional defects. Despite these qualities, little research on autonomic responses of the autistic child has been conducted, although it may be potentially most fruitful, perhaps especially with completely non-verbal subjects. The second study was concerned with such autonomic measurement, involving responses to selected, experimentally controlled stimuli.

The third aspect of childhood autism under study involves analysis of the learning difficulties characteristic of the autistic child. A process of stimulus overselectivity would indeed provide learning difficulties, but it seems that there are others. It has been proposed that these children suffer from marked defects in cognitive functioning which may differentiate them from other handicapped children. The importance of such defects for treatment and education

has been outlined. Further research in this area is warranted. Current research has chiefly used the errors found in the learning of binary sequences as an index of such cognitive dysfunction. It is clear that these defects must be evident in activity not directly related to language competence, and therefore tasks requiring conceptual functioning may be a suitable medium to investigate proposed deficits. In addition to error analysis on such tasks, an investigation of the strategies used by subjects, including their use of task-relevant information feedback, may yield further data on the depth and generality of cognitive defects.

The experimental studies to be reported concerned themselves, therefore, with these three aspects of childhood autism.

CHAPTER THREE:

"MOTHER-CHILD INTERACTIONS WITH NORMAL AND AUTISTIC CHILDREN"

Introduction

For a number of childhood behaviour disorders it is clear that whatever factors may be aetiologically relevant, many aspects of the child's deviant behaviours are influenced and indeed in some cases maintained, by parental behaviour (Werry & Wollersheim, 1967; Wahler, 1969). It is also apparent that the same may apply to certain symptoms displayed by autistic children (Moore and Bailey, 1973; Kozloff, 1973; Lovaas, 1971). There has been a rapid growth in recent years therefore in home-based behaviour modification programmes involving parents as co-therapists (O'Dell, 1974). However, before any appraisal of relevant features in the child's environment, and before any modification of either the child's or parents' behaviour, it is necessary to obtain appropriate normative data. It is proposed that the relevant variables in the child's environment, and particularly the degree and nature of parental control, cannot be satisfactorily assessed with any degree of reliability and accuracy from simply interviewing parents, administering personality tests, or designing parental questionnaires. The studies that use such techniques to investigate parental characteristics are in any case aetiologically orientated (e.g., Ogden et al., 1968; Parish, 1969; Eisenberg, 1957). These methods also assume that parents are aware of their behaviour in relation to their child and the effect of the child's behaviour upon themselves. Neither assumption is warranted.

A more appropriate method would be to study parent-child interactions, which constitute the major channel through which both parent and child exert behaviour control and which is one of the principle learning situations for the child. Due to practical restrictions on the present study, it was necessary to restrict the analysis of interactions to those between mother and child, although

this was by no means intended to display a lack of regard for father-child interactions.

Kozloff (1973) points out that parenthood is perhaps the one major occupation and skill not taught formally in any way, rather being learnt by trial and error. Thus when severe disturbances in the child do occur (as in childhood autism), parents are ill equipped to deal with them and only then do they receive any professional advice. The child's disturbance is frequently effectively excused, thus changing important role expectations. If there is a further deterioration in behaviour, then the child's influence in the home often increases due to inadvertent reinforcement of inappropriate behaviour, consisting usually of demands, disruptive behaviour, non-compliance and negativism in the case of childhood autism. Kozloff maintains that at this point, no matter how the disturbance initially arose, the parents need training in teaching the child socialisation. This is a crucial point, for whatever factors are aetiologically significant, the autistic child's behaviour disorder has inevitably disrupted the home environment and rendered normal interactions between parent and child non-optimal, if not directly maladaptive, for both parent and child. Thus Kozloff believes in a heteropathic approach, where cause and treatment can be entirely separated. This is in direct contrast to the homeopathic treatment implied by the medical model of behaviour disorder.

It is clear that with our present state of knowledge and from current research findings, we cannot yet unequivocally separate the cause-effect problem. Parents may be reacting to the child's initial deviant behaviour, and this is as plausible as suggestions that their behaviour is directly aetiologically relevant. In addition, initial causal factors may not be the ones that maintain

'autistic' behaviour. Williams (1968) asserts that: "We have long ago abandoned the idea that good mothering is something innate in all women but we are only at the threshold of understanding the multitude of mechanisms through which mother and child stimulate each other to desirable responses". It is clear that the parental personality characteristics reported are not necessarily causal, and we must take special care not to use any parental 'type' or behaviour pattern descriptions in a perjorative fashion. Parents are as likely to be as much under the control of the child's behaviour as the child is under the control of the parent. Indeed, Kozloff proposes that we must look upon the autistic child and his environment within the framework of social exchange theory.

A review of the current literature suggests that there is a strong probability that childhood autism has an initial aetiology of an organic nature. But it is also clear that the autistic child's environment will be crucial in determining at least some of his intellectual and social development, and particularly the severity and persistence of many associated symptoms. Rutter (1974), after proposing a primary organic aetiology, maintained that: "Of course this does not mean that family and other environmental factors do not influence the behaviour of autistic children. Obviously they do, just as they influence that of any children - normal or handicapped. There are special considerations in this connection as the cognitive handicaps of the autistic child impair his responses to the world about him. If there is to be optimal development, normal experiences are not enough, because they may not impinge upon him". (p.155)

It therefore seems most important that we investigate the nature of the environment that is provided for the autistic child. Taft (1964) clearly agrees with such an assertion: "Continuing ex-

plorations into the interactions of the child and his environment are essential if we are to understand the pathophysiology of infantile autism. Behavioural scientists are well aware of the unsettled questions regarding classification, the sampling problems and the almost infinite permutations and combinations that are inherent when experiential factors are compared with genetic and constitutional factors". (p.105)

The present study therefore attempted to analyse features of mother-child interactions without in any way claiming to be an aetiological study. Psychogenic aetiological theories and studies investigating possible relevant parental characteristics have involved much research on parental attitude and personality measures. It was felt that not only are such characteristics too global and imprecise, but they oversimplify the situation and are also too divorced from the environment directly relevant to the child, i.e., the interaction situation between child and parent.

While behaviour therapists and modifiers have outlined many of the important features in the autistic child's environment which do appear to maintain some of his deviant behaviour (e.g., Lovaas, 1971; Kozloff, 1973), their treatment-based approaches have inevitably resulted in research largely based on single case studies or very small samples. While the efficacy of a behaviour modification programme can indeed be assessed in such designs, the question of the generality of findings and the relation of the data to mother-normal child interactions cannot be assessed from such studies. These are both questions which not only the research psychologists are interested in, for experiments with autistic and control groups will yield data potentially useful in the design of generalised treatment programmes and also normative data on the mother-child interaction. One recent study (Wahl et al., 1974) on mother-child interactions with non-

deviant children suggested that such interactions are themselves non-optimal in terms of the efficiency of parental management within an operant framework analysis. A further problem with behaviour therapy studies of most varieties is their rejection of the usefulness of traditional diagnostic categories, since their treatment approach to most disorders is essentially similar, leading to references to their subjects as "autistic-like" (e.g., Craighead, O'Leary and Allen, 1973). This naturally casts a suspicion on the homogeneity of their subjects where criteria or detailed behavioural notes are not presented. Other limitations of such studies have been outlined (e.g., Howlin et al., 1973b).

Language deficits are undoubtedly part of the fundamental problems in childhood autism, and investigating the current linguistic environment for the autistic child would seem to be of some importance. Marshall, Hergrenes and Goldstein (1973) propose that: "In assessing speech and language development, the environment of the child, including his relationship with those playing a vital role in shaping that environment, should be considered....The adequacy of a child's skills may be conditioned by the speech patterns of the environment. The child's earliest experiences are in relationship to his parents and more specifically, his mother. The mother provides the most consistent speech pattern in the child's environment. A systematic analysis of verbal interactions has the potential for providing a base from which rehabilitative procedures might be developed". (p.415).

Goldfarb and his colleagues have carried out much research on mother-child interactions within an experimental, non-treatment framework (Meyers and Goldfarb, 1961; Goldfarb, Goldfarb & Scholl, 1966; Goldfarb, Levy & Meyers, 1972; Goldfarb, Yudkovitz and Goldfarb, 1973). This research has been reviewed in Chapter 1. Goldfarb reported

several deviant aspects in the clarity and use of speech by the mother when interacting with her autistic child. As Goldfarb himself pointed out, the apparent poor communication of thought and feeling indicates a lack of the most important instrument for enhancing normal attachment responses in the child, for educating and guiding their growth, and for assisting them to achieve order and clarity in their environment. This is quite apart from mothers being poor speech models for the learning of the acoustic and grammatical aspects of language. These modelling failures are clearly most interesting and whilst Goldfarb related them to his aetiological hypothesis of a continuum of an interaction between biological and environmental factors, one does not need to subscribe to his position and aetiological inferences to appreciate the significance of these findings. In a recent interesting study (Goldfarb, Yudkowitz and Goldfarb, 1973), Goldfarb reported aberrant patterns of communication between the mother and autistic child when engaged in referential communication tasks (c.f. Kraus and Glucksberg, 1969) which is a situation clearly related to the home learning environment of the child. Referential communication tasks involve one member of a diad constructing a verbal description of a target object in order that a recipient may be able to select that target from an array. Howlin et al., (1973b) underline the importance of defects in the linguistic environment for a child by noting that: "While Psychologists may disagree over the mechanisms underlying the acquisition of language in the young child, there is common agreement on one major point: a child's information about the specific language he is to learn must come from the language which he hears spoken around him". (p.317)

Goldfarb's studies do display what appear to be four limitations however:

- 1) They concentrate on the verbal aspects of mother-child interactions and especially the mother's speech. Many important features of interactions are non-verbal, and we know that there are important non-verbal aspects of communication (Argyle, 1969; Stern, 1969). Indeed, since autistic children are reported to have a strong tendency to gaze avert (Hutt et al., 1964; Tinbergen, 1972), it would, for example, be interesting to study the effects that this may have upon mother-child interactions.
- 2) Goldfarb's studies not only concentrate on mother's speech but also most of the measures reported were based on observer ratings procedures, (e.g., "clarity" and "spontaneity" of speech, or "meeting/not meeting" child's demands). Whilst rating measures are not uncommon in psychological research, and the use of naive observers and reliability checks increases the strength of the data, it would seem that the use of less inferential, more 'concrete' empirical measures where possible, would be desirable.
- 3) The interaction periods studied are frequently very short, and often are of an atypical nature (e.g., 'surprise' parental visits).
- 4) Perhaps the most important weakness in Goldfarb's studies lies in the treatment of the mother and her behaviour as an individual unit in the interaction situation. As Kozloff (1973) advocated, we need to observe the reciprocal relationship between the behaviour of the parent and the child. We must ask questions of the kind: "If the mother elicits behaviour x, what is the child's response to this?" or "What maternal behaviour tends to precede child behaviour y?". The mother-child interaction is a process of social exchange, where each partner modifies the behaviour of the other.

Howlin et al., (1975) report an interesting pilot study on the analysis of speech to autistic children by their mothers. Several

measures were taken of speech over longer time periods than Goldfarb used. The data suggested that a high percentage of questions are presented to autistic children even when they are non-verbal, although the children themselves ask very few questions. Verbal reinforcement was quite frequent in mothers of verbal autistic children, but much lower in mothers of non-verbal children. Similarly, corrections, directed mimicry, and prompting were much more frequently observed in the speech of mothers of non-verbal autistic children. Finally, the amount of imitative speech produced by mothers was very low, being less frequent than that often reported in studies where mothers speak to normal children in the very early stages of productive language development. The authors of this study propose that there are a number of possible hypotheses which this data may support. Amongst these are the possibility that low frequency speech in autistic children is associated with a lack of reinforcement of spontaneous utterances, and that inaccuracies in the child's speech, where present, may be a function of a poor parental speech model or a lack of feedback via corrections and prompts. It is possible that mothers fail to stimulate and encourage speech: the study reported a high level of non-interactive speech consisting of commands and statements rather than reinforcement and modelling behaviours. The authors also note that their data may reveal effects that are not specific to autistic children, but rather to many children with language delays.

This study has produced some intriguing results, but it must be born in mind that it suffers from the difficulties of most pilots in the use of a small sample and insufficient controls. However, it fully warrants the proposed full-scale study to follow it, and the speech analysis scheme outlined promises to lead to the identification of deficiencies in the speech of mothers to their autistic children.

Goldfarb's studies were based on comparisons between experimental groups of autistic children with their own mothers and control group children and their own mothers. The control groups themselves consisted in some studies of normal children but in others children with "non-psychotic behavioural pathology". It would seem that there are further questions to be asked. We may wish to know how much any differences in the behaviour of mothers of autists and mothers of controls are dependant upon the immediate deviant behaviour of the autistic child in the interaction situation. Thus if mothers of autistic children are atypical in their interaction behaviour, it is necessary to establish whether this is a function of the immediate deviant behaviour of the autistic child or whether this is a long-standing 'mode' or 'style' of interacting with children in general, including non-autistic children.

In the present study therefore, mothers of autistic children were also studied while interacting with normal children, and control mothers whilst interacting with autistic children. Also, the question arises as to the effect of familiarity upon the interaction. Goldfarb's studies employed mothers of autists interacting with their own autistic child and control mothers with their own normal child. We may now wish to ask whether any characteristics of being a mother of an autistic child are restricted to interactions with their own child, rather than any autistic child. Thus in addition to interacting with their own child and with a normal child, mothers of autistic children also interacted with an unfamiliar autistic child. [Familiarity in this context refers to the familiarity of the mother with her own, individual child]. Thus it was intended to look at the effects of interacting with an autistic child, but one that was not familiar to the mother.

If consistent differences appear between mothers of autistic and control children, with both familiar and unfamiliar children, then inferences about more stable interactional characteristics of the mothers can be made, rather than about specific responses to the immediate behaviour of any individual interaction partner.

Thus to summarise, there were six separate 'types' of interaction included in the design of the current study: Each mother of an autistic child interacted with (i) her own autistic child; (ii) with a normal child (unfamiliar to her); and (iii) with another autistic child (with whom she was unfamiliar). Each mother of a normal child interacted with (iv) her own normal child, (v) with an autistic child (unfamiliar to her), and (vi) with another normal child (with whom she was unfamiliar).

The present study had five major aims:

- 1) To collect empirical data on the nature of mother-child interactions using a large number of both verbal and non-verbal measures, collecting data from both the mother and child. The data to be collected was intended to be as free as possible from any subjective rating procedures.
- 2) To assess some of the relationships between the child's and the mother's behaviour.
- 3) To compare the data collected from interactions involving autistic children and their mothers with data from control mothers and their children.
- 4) To assess the relative effects on mothers of interactions with (a) autistic children (b) normal children (c) familiar children [i.e., familiar to the mother] (d) unfamiliar children.
- 5) To investigate whether any interaction characteristics of being a mother of an autistic child, are restricted to interactions with her

own child (viz effect of familiarity), interactions with autistic children in general, both familiar and unfamiliar to the mother (i.e., effects of familiarity with "autism" and its behavioural correlates), or are not restricted (i.e., appear in interactions with both normal and autistic children, familiar and unfamiliar).

METHOD

a) Subjects

The autistic children selected for experimental study had all received a psychiatric diagnosis of childhood autism. However, this only served as an initial screening for suitable subjects. The parents of a group of these children were contacted and each were then paid a home visit. A questionnaire was verbally administered to at least one parent, and typically this was the mother. The questionnaire was based on the British Working Party "9 points" (Creak et al., 1961) but with a number of additional questions. These included questions on prenatal history, family size, diagnostic history, earliest symptoms, education etc. A copy of this questionnaire is included in the Appendix (Table A).

On the basis of the questionnaire data, autistic subjects were selected for experimental study if they displayed at least four of the British Working Party (BWP) "pointers" (Creak et al., 1961), and it was considered essential that they displayed in these, three particular groups of symptoms. These were first a gross and sustained impairment of emotional relationships with other people (from BWP pointer number 1). Second, severe speech and language difficulties, typically including echolalia, fragmented speech, or no speech at all, and profound comprehension defects, (from pointer number 7). Finally, either ritualistic, compulsive or obsessional behaviour (from pointers

3 or 4 or 8). Thus the selection criteria for subject inclusion in the present study corresponded closely to the description of childhood autism as outlined by Rutter (1971), but was based upon questionnaire data derived from the Creak et al., (1961) 9 pointers for autistic behaviour disorders.

Questionnaire data on the twelve subjects selected for the present study and its pilot, revealed that the average age of subjects used in the study was 7.6 years (range 5.2 - 10.2), with the mean age at which a psychiatric diagnosis of childhood autism was made being 3.6 years. 75% of the mothers reported pre- or paranatal complications of a wide variety (e.g., Toxaemia, breech presentation, anoxia, sedative drugs). A symptom found in all cases selected, but not included in the selection criteria, was that of marked negativism.

b) Pilot Study

A pilot study was conducted using two autistic children and their mothers, together with two mothers of normal children of the same chronological age as the autistic children. Each child interacted separately with his own mother, the mother of another autistic child, and the mother of a normal child. Videotaped recordings were made of these interactions, and the procedures used and measures taken were essentially the same as those outlined in the description of the present study. The pilot study enabled the rehearsal of appropriate videotape recording techniques, the refinement of unambiguous instructions to the mothers, and the development of measures relevant to the analysis of the mother-child interactions.

c) Procedure

A total of ten autistic children and their mothers, together with ten normal children and their mothers took part in this study.

The ten normal control children and their mothers were selected from a list of volunteers collected via local schools, W.I's and Mothers' Unions. The selected control children were matched for chronological age, sex, and parental social class background with the experimental subjects. The social class background of the two groups of children were scored on the basis of ratings in the Registrar General's (1970) Classification of Occupations, and a Fischer Exact Probability test revealed that the two distributions did not significantly differ ($p = 0.37$).

Both experimental and control children were within the 5-10 years (C.A.) age group. For autistic children the mean chronological age was 7.2yrs (range 5.2 - 9.5), and for normals 7.5yrs (range 5.5 - 10.0). The normal children were matched with the autistic children on chronological age (rather than mental age for example), for three major reasons:

- 1) It was felt that of particular interest in this study would be differences in terms of developmental behaviour norms, rather than pure intellectual or cognitive differences.
- 2) Normal children matched with autistic children on mental age would clearly have ~~Lower~~ chronological ages, and would therefore require differing toys and puzzles as an interaction medium which may have affected some of the interaction measures to be taken.
- 3) Since it was intended to measure any differences in the behaviour of mothers towards the two types of child, it was important to control for discriminatory behaviour on the part of the mother as a function of the chronological age of her interaction partner. There is a

growing body of evidence to suggest that this discrimination does occur and is of significance in language acquisition (Roberts & Fraser, 1972; Snow, 1972; Broen, 1971). Also, matching on C.A. ensured that all mothers brought with them current experience of interacting with children of the same age to the experimental setting.

Social class matching was necessary to control for linguistic competence and social class correlates of mother-child verbal interactions. These variables have been demonstrated to be of importance in social interactions with children (Hess and Shipman, 1968; Heider, 1971). Since there is a high consensus on the finding that a large percentage of autistic children come from middle-class backgrounds (Kanner, 1949; Rutter and Lockyer, 1967; Kolvin, et al., 1971), it was important that the mothers of control children were not selected at random, but rather were matched with the mothers of autistic children on this dimension.

Ten experimental sessions were held over a period of twelve weeks. The sessions were arranged in pairs and each pair required two autistic children and their mothers, and two control children and their mothers. In session A one autistic child and a matched normal child were present, together with their respective mothers, plus the mothers of the other autistic and normal children who had been assigned to the experimental session pair. In session B the same four mothers took part together with the autistic child and matched normal child who did not take part in session A. Thus each session required four mothers and two children, sessions A and B differing only with regard to the children present. The study involved a total of five such pairs of experimental sessions, the sessions within any one pair being separated by approximately one week.

At the commencement of each experimental session, mothers and children were assembled in a waiting room and were told that they would be taking part in a comparative study of toy play and simple task completion behaviour with autistic and normal children. Mothers were told that the interactions would be videotaped, and that they would be able to see the videotaped recording themselves afterwards if they so wished, but they would otherwise be confidential. One child and one mother were then selected and taken into the child laboratory. The laboratory contained a central table with two seats arranged on fixed adjacent positions at the table, to control for seating (and thus ease of facing) position. Two remote-controlled cameras were mounted in corners of the laboratory, and were made as inconspicuous as possible. A microphone was hung from a central point in the ceiling, well above the normal visual field of the seated subjects. The laboratory was otherwise clear of any distracting material, and was connected to a recording and monitoring room by a closed door. The mother and child were seated at the table and the following instructions were given to the mother:

"I would like you to take this (your) child into this room and sit down with him/her at the table. When you think that he/she is reasonably settled, I would like you to talk to him/her and perhaps tell him/her a story about anything you like. It doesn't really matter what you say in the story or conversation, it is just so that we can look at him/her when an adult is talking to him/her, and to get him/her used to the surroundings. After a few minutes, I shall pop in and ask you to play with him/her. I would like you to try and get him/her to do something very simple with you. I shall bring in a selection of toys

and puzzles, and again it doesn't matter which one you or the child chooses to play with. I would just like you to try and get him/her to do something very simple with the toys or puzzles. You might for example play with the moving duck, or show him/her how the puppet works or help him/her to do this simple formboard puzzle. Try and select only one toy or puzzle at a time, since he/she may become distracted by all of them at once."

Mothers were not given highly specific instruction about the required activity from the child, other than that it should be as constructive and cooperative as possible, in an attempt to observe a more 'natural' mode of interaction in the play/learning situation. The initial part of the interaction, when the mother was talking or telling a story to the child, was not in fact analysed. It was intended as a period for initial familiarisation with the child, with the setting, and with the awareness of being observed via cameras. All mothers were verbally praised for their behaviour when E brought in the toys and puzzles before the second stage of the interaction. The initial period of interaction lasted for ten minutes, the toy play section being of 10 minutes' duration and videotape recorded throughout.

A total of six such diadic interactions were recorded at each session, yielding sixty interactions in the whole study. Each interaction was labelled in terms of the child (normal or autistic), mother (mother of normal or mother of autistic), and familiarity (familiar with child or mother not familiar with child). 'Familiarity' was defined in terms of familiarity of the mother with the child, 'familiar' interactions involving only the child with its own, parental mother. All other interactions were labelled 'unfamiliar'.

This labelling thus yielded six "types" of interaction (see table 1A):

CHILD	"TYPE" OF MOTHER	FAMILIARITY
autistic (A.C.)	mother of autistic child (M.A.) (own mother)	familiar (F)
autistic (A.C.)	mother of other autistic child (M.O.A.)	unfamiliar (U.F.)
autistic (A.C.)	mother of normal child (M.N.)	unfamiliar (U.F.)
normal (N.C.)	mother of normal child (M.N.) (own mother)	familiar (F)
normal (N.C.)	mother of other normal child (M.O.N.)	unfamiliar (U.F.)
normal (N.C.)	mother of autistic child (M.A.)	unfamiliar (U.F.)

TABLE 1A: SIX "TYPES" OF INTERACTION

The mother-child diads were balanced so that at any one session there were three interactions involving mothers of autistic children and three with mothers of normals, three involving autistic children and three with normal children. Thus there was one interaction of each of the "types" listed in table 1. The order of the "types" of interaction within sessions was rotated between sessions to control for any order effects such as time spent in waiting room, familiarity of child with toys (from previous interactions involving the child), familiarity with the laboratory etc. Autistic and normal children however were always alternated from one interaction to the next in an attempt to reduce fatigue and any distress, particularly with the autistic children, when separated from their own mother.

The videotaped recordings of the mother-child interactions were analysed in detail over a period of three months. Video recordings were found essential in the analysis of high-speed behaviour (e.g., fractional glances), and accurate and reliable assessment of many of the measures taken. Slow speed playback of some behaviours provided much of the accuracy and detail required. Complete typed transcripts of the mother-child dialogue were also prepared.

d) Variables measured

There were seven general categories of analysis, yielding a total of 101 measures for analysis from each interaction, 70 of which were completely independent of each other (others consisting of absolute and percentage scores of the same variable for example). The categories were:

1) MOTHER-CHILD FACIAL POSITIONS

Each interaction was analysed in terms of the frequency of occurrence of four facial positions, these being mutual facing (AF/CF), mother facing-child not facing (AF/CNF), mother not facing-child facing (ANF/CF) and neither facing (ANF/CNF). Despite the use of close-up lenses on recording cameras, 'facing' could not be defined in terms of direct eye contact with absolute certainty. It was felt that in any case a more useful, and certainly more accurate, operational definition of "facing" would be when an interaction member was clearly directing his/her gaze at the partner's face or upper trunk, or in the case of the mother, also when she was directly watching the child's hands when engaged in activity with the interaction medium (the toys and puzzles). Thus 'facing' did not necessarily require actual looking at the partner's

eyes, although in the majority of instances this probably did occur.

The present definition of facing was intended to be a functional one. Thus for example, a definition of facing requiring the judged observations of actual eye contact would imply that a mother clearly attending to and observing her child's activity with one of the puzzles or toys, was not in fact facing the child. Equally, although the lack of actual mutual eye contact between autistic children and others is well documented (Hutt et al., 1964) and gaze aversion is sometimes considered symptomatic of such children (Kolvin et al., 1971; Hutt and Ounsted, 1966), such a child who turns towards an adult and perhaps even approaches the adult, yet only looks at the upper trunk, is clearly 'facing' the adult although not actually establishing mutual eye contact. Thus the definition of 'facing' in this study was not based solely on actual eye to eye contact, but rather on the basis of directed gaze within strictly defined limits. In practice, little difficulty was experienced in inferring the presence of any one of the four defined facial positions at any given time during the interactions. Indeed, a reliability check on the scoring of the facial positions (number of occurrences of each position), taken on a random sample of interactions, yielded strong evidence of a high degree of consensus between observers. A Wilcoxon Matched pairs signed-ranks test on the sets of scores of "number of occurrences of each facial position" for six randomly selected interactions for the two observers (naive post-graduates), revealed that there was no significant difference between the two observers' scores ($T = 102$; $T = 81$ or less for sig.). A Spearman's Rank correlation on the same data yielded a correlation coefficient of 0.93 between the scores for two observers over the four facial positions.

The frequency and duration of each facial position was scored throughout each interactions, and the percentage of time spent in each position as a function of total interaction time was also calculated. For each position, the number and percentage of child terminations of that position, and the number and percentage of adult terminations were recorded. Also, at each occurrence of a facial position, the average duration before the child terminated that position, or the average duration before the adult terminated it, was scored. By summing the total time spent in the mutual facing and adult facing-child not facing (AF/CNF) positions, the total percentage of time spent facing the child by the adult could be calculated for each interaction. Similarly, summing the mutual facing (AF/CF) and adult not facing-child facing (ANF/CF) positions, yielded a figure for the total percentage of time the child had spent facing the adult.

As each facial position was superseded by another, the transition was recorded, and thus at the end of each interaction the transitional probabilities of moving from each specific facial position to each of the other three were calculated. A 4 x 4 transitional probability matrix was then drawn up for each interaction.

2) Stereotyped behaviours

The stereotypy displayed by the autistic children during the course of the interactions was recorded and scored. Whilst the specific nature of stereotypy was highly idiosyncratic, its occurrence was clearly observed and could be defined as any apparently irrelevant, often bizarre, behaviour that was performed repetitively at high frequency. The expression of stereotypy often took the form of hand flapping or shaking, repetitive stroking of surfaces, head rocking or finger "poking". The total number of separate occurrences of stereo-

typy (i.e., with a minimum of 5 seconds between exhibited occurrences of stereotypy), and the total duration of time spent exhibiting stereotypy, were scored for each interaction. It was hypothesised that stereotyped behaviours may be in some instances related to antecedent maternal behaviour, and that this stimulus control may come through maternal 'demands' in the form of questions, verbal commands, or non-verbal demands (e.g., when the mother offers a puzzle or toy to the child, or when she physically approaches the child). Thus the number and percentage of stereotyped behaviours that were preceded (in the immediately preceding 5 second or less) by such "demands" upon the child, were recorded and scored.

3) Total Body Movements (TBM)

It was suggested that not only may stereotypy be under the control of various maternal demands, but that this may also be so for total body movements by the child away from the adult. A total body movement (TBM) for both child and adult was defined as a whole body movement away from or towards an interaction partner, which involved a turning from the waist upwards of at least 90° (i.e., not just a simple head turn), and more frequently it involved actually walking towards (and facing) or away from (and facing away), an interaction partner. The number of such TBM by the child towards the mother and the number away from her, were recorded. The number and percentage of these movements that were preceded (within at most 5 secs.), by maternal "demands", were also recorded. The number of TBM by the adult away from, and towards, the child were scored.

4) Cooperation and Negativism

Aspects of cooperation and negativism were analysed and scored. Cooperation was defined as an active non-verbal response from the child which involved compliance with a verbal command, or question, or non-verbal demand, from the mother. This frequently took the form for example, of placing pieces in a puzzle, handing something to the mother, or turning to face or moving towards the mother, all of these in response to requests to do so from the mother.

Negativism was defined as an active non-verbal response on the part of the child, to resist compliance with a verbal command or request, and occasionally simple outbursts of aggression or temper tantrums. Negativism often took the form of throwing toys around the room, pushing the mother or a toy away, hitting the mother, and running or completely turning away from her.

Mother's responses to both cooperation and negativism were scored in terms of whether the mother rewarded, punished or ignored the relevant behaviours. Reward was scored in terms of verbal praise or approval, and punishment in terms of verbal or physical reprimand. Other responses to these behaviours were not scored. From these figures, the number and percentage of reinforcement 'errors' displayed by the mother in any interaction, (defined as cooperative behaviours unrewarded or negativism that was rewarded), and the number and percentage of punishment 'errors' (defined as negativism unpunished or cooperative behaviours punished), were calculated for each interaction.*

(* See also section on verbal cooperation in category 7).

5) Toy Play + puzzle completion

In each interaction the total number of seconds the child spent in constructive toy play or puzzle completion (i.e., not exhibiting stereotypy with any of the pieces of toy or puzzle), was recorded. Also noted were the number of different toys that each child played with in each interaction. A score was only recorded when the child was actively playing with the toy/puzzle for at least ten seconds.

6) Mother's speech to child

From the typed dialogue scripts, the total number of words, utterances, and mean utterance length were calculated for each interaction. Similarly, the number and percentage of verbal statements, questions, and verbal commands were recorded. The Type-Token Ratio (TTR) was calculated from the first 200 words spoken in each recorded interaction, and the number of separate utterances conveying verbal reward, and also verbal punishment were scored. Utterances were delineated on the basis of both grammatical and phonetic cues.

The number of 'guiding' statements were then summed for each interaction. A 'guiding' statement was defined as an explanatory statement from the mother, designed to aid the child in the solution to a puzzle or to explain the use or properties of any particular toy. 'Guiding' statements did not include purely directive verbal commands like "Put this piece there" or "This goes here". Rather, they included statements of the kind: "That round one doesn't fit, but you could try a different shape"; or "If you turn it upside down it will make a lovely noise". As a general rule, 'guiding' statements were those seen as explanatory, informational, facilitatory statements

enabling the child to learn, in contrast to simple directives or commands. (The recognition of "guiding" statements was aided by the concurrent use of the visual information in the videotape, and also the typed transcripts.) Metaphorical questions, recognised largely from the intonation in the verbal recordings rather than the typed transcripts, were scored as verbal statements since they were functionally non-interrogative.

7) Child's Speech

The child's speech to the mother in each interaction was similarly analysed in terms of total number of words, utterances and mean utterance length, and also number and percentage of verbal commands, questions and statements. In addition, the number and percentage of verbal replies to mother's questions were also scored. In category 4 (cooperation-negativism) only non-verbal cooperation was considered. However, it would appear plausible to suggest that a verbal reply to a question is also indicative of a willingness to cooperate in the interaction, although of course we must remember that for the non-verbal autists only the non-verbal channel is available to them to cooperate. Thus separate analyses on non-verbal cooperation (section 4), and verbal cooperation, were carried out in addition to an overall cooperation score consisting of the combined scores for these two aspects of cooperative behaviour.

RESULTS

The six 'types' of interaction listed in table 1A were analysed in terms of three major comparisons:

- i. Autistic Vs normal child interactions
- ii. Familiar Vs unfamiliar interactions
- iii. Interactions with mothers of autistic children Vs interactions with mothers of normal children

This enabled the separation of the effects of type of child, type of mother, and familiarity of child and mother with each other. This was achieved through the use of an analysis of variance on the 101 measures taken, consisting of a 2 (child) x 3 (mother) ANOVA carried out on programme RGANOVA (R.Gillett) after Winer (ch.5). Multiple comparisons were conducted using the Scheffe contrast method (Scheffe, 1959). This test is comparatively conservative, and leads to few type 1 errors (Ferguson, 1966: p.297).

The major significant results were as follows: *

AUTISTIC Vs NORMAL CHILD DIFFERENCES: SIGNIFICANT RESULTS

a) Facial Positions

(1) Diads involving autistic and normal children differed significantly in the absolute length of time spent in the mutual facing position, this being longer for diads with autistic children; ($F=4.49$; $df=1,18$; sig. 5% level). However, there were no differences in the total number of separate occurrences of this position ($F=0.09$; $df=1,18$).

(2) The number of adult terminations of the mutual facing position tended to be greater with autistic children, ($F=3.14$; $F=4.41$ for significance), but the average duration before this position was terminated by the adult was longer also with autistic children ($F=4.46$; $df=1,18$; sig. 5% level).

(3) The total duration and percentage of time spent in the position adult facing/child not facing (AF/CNF) was longer with autistic children. ($F=9.35$; $df=1,18$; sig. 1% level; and $F=7.63$; $df=1,18$;

* RELEVANT MEANS, WHERE NOT CITED IN THE TEXT, MAY BE FOUND IN APPENDIX D. A SIMPLER, MORE READILY COMPREHENSIBLE ANALYSIS OF THE FOLLOWING DATA CAN BE FOUND IN APPENDIX E. READERS MAY FIND THIS USEFUL WHEN CONSIDERING THE DETAILED ANALYSIS OF RESULTS WHICH FOLLOWS.

sig. $2\frac{1}{2}\%$ level respectively).

- (4) The average duration before the adult terminated the position AF/CNF was longer with autistic children. ($F=8.94$; $df=1,18$; sig. 1% level).
- (5) The total time spent in the neither facing position (ANF/CNF) was greater for normal children. ($F=10.51$; $df=1,18$; sig. 1% level).
- (6) The average time before the adult terminated the neither facing position tended to be longer with normal children. ($F=4.08$; $F=4.41$ for significance).
- (7) The total percentage of time spent facing the child by the adult (for all positions) was greater in diads involving autistic children. ($F=9.51$; $df=1,18$; sig. 1% level).
- (8) With the probability of transition from the position adult not facing/child facing (ANF/CF) to mutual facing, there was a strong tendency for an interaction such that with the autistic children the mothers of normal children gave the highest probability but with normal children the mothers of autistic children gave the higher probability. ($F=3.04$; $F=3.26$ for significance. See Figure I/i).
- (9) With the probability of transition from ANF/CF to ANF/CNF (i.e., child termination of this position to neither facing), this probability was greater with autistic children when with mothers of autistic children, but with normal children it was greater with mothers of normal children. ($F=3.52$; $df=2,36$; sig. 5% level). Own mothers resembled mothers of normal children by having a greater probability with normal children than autistic children, although this difference was smaller. This interaction is illustrated in Figure I/ii.

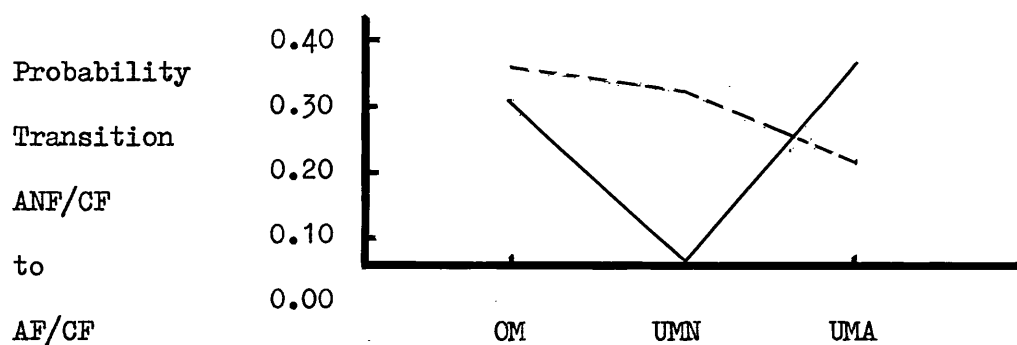
Figure I/i

Figure I/i:- PROBABILITY ANF/CF TRANSITION TO AF/CF X
MOTHER TYPE

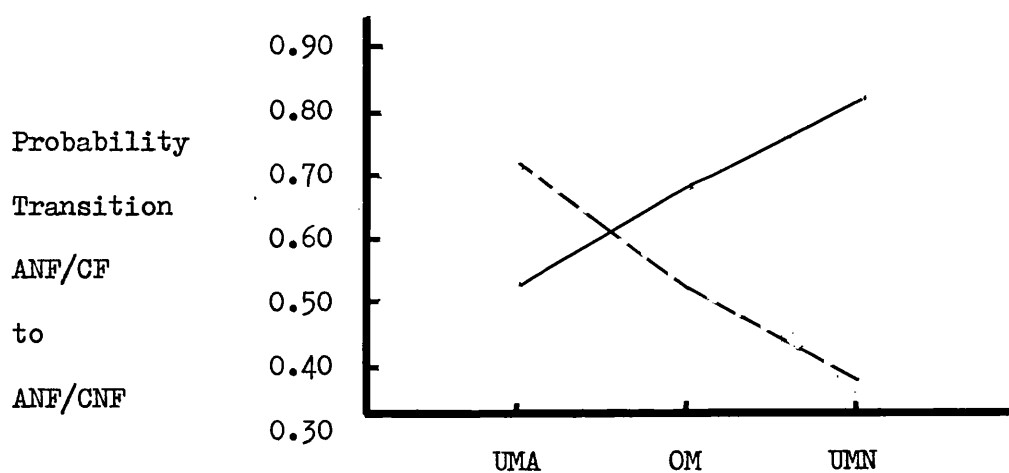
Figure I/ii

Figure I/ii:- MOTHER "TYPES"
PROBABILITY OF TRANSITION ANF/CF to ANF/CNF X
MOTHER TYPE

/ = Normal Children / = Autistic Children
 \ \

b) Stereotypy

(10) Autistic children naturally displayed more stereotyped behaviours than normal children ($F=17.88$; $df=1,18$; sig. 0.1% level), during a greater duration of the interaction time ($F=21.21$; $df=1,18$; sig. 0.1% level), with a greater number and percentage of antecedent non-verbal demands, commands, and questions (NVDQ) ($F=23.24$; $df=1,18$; sig. 0.1% level, & $F=30.06$; $df=1,18$; sig. 0.1% level respectively).

(11) The total number of separate occurrences of stereotypy in autistic children was 279, of which 133 or 47.7% were preceded by NVDQ.

c) -----Total body movements (TBM)

(12) Autistic children displayed a greater number of TBM towards the mothers than normal children ($F=12.46$; $df=1,18$; sig. 1% level), with a greater number of these movements preceded by NVDQ ($F=13.73$; $df=1,18$; sig. 1% level), and a greater percentage of such antecedents ($F=45.29$; $df=1,18$; sig. 0.1% level). 70% of all TBM towards the mothers were preceded by NVDQ.

(13) Autistic children displayed a greater number of TBM away from mothers than normal children ($F=15.91$; $df=1,18$; sig. 0.1% level), preceded by more total NVDQ ($F=14.72$; $df=1,18$; sig. 1% level) and a greater percentage of NVDQ ($F=83.15$; $df=1,18$; sig. 0.1% level).

(14) The mothers displayed a greater number of TBM away from the autistic child and also towards him, compared with normal children ($F=6.23$; $df=1,18$; sig. 2 $\frac{1}{2}$ % level, and $F=11.63$; $df=1,18$; sig. 1% level, respectively).

d) Cooperation-negativism

(15) Autistic and normal children did not differ in the number of non-verbal cooperative responses that they displayed to mothers.

($F=0.34$, $df=1,18$). The mean number of cooperative (non-verbal) responses for autistic children was 10.6 per interaction, and for normal children 9.4/interaction.

(16) A greater number of the cooperative responses of the autistic children were rewarded when compared with the cooperative responses of normal children. ($F=7.29$; $df=1,18$; sig. $2\frac{1}{2}\%$ level). 44% of the cooperative responses of the autistic children were rewarded in contrast to 23% of the normal children's cooperative responses.

(17) Autistic children displayed more negativistic behaviour than normal children. ($F=7.35$; $df=1,18$; sig. $2\frac{1}{2}\%$ level).

(18) Normal children experienced a higher percentage of reinforcement 'errors' from mothers than autistic children did ($F=9.35$; $df=1,18$; sig. 1% level). The average percentage of such errors with autistic children was 56.6% whereas for normal children it was 76.9%.

(19) Mothers displayed a greater number and percentage of punishment 'errors' with autistic children ($F=6.33$; $df=1,18$; sig. $2\frac{1}{2}\%$ level, and $F=13.86$; $df=1,18$; sig. 1% level respectively). A mean of 82% of the negativistic behaviours of the autistic children were unpunished.

(20) If verbal replies are scored as cooperative responses and added to the non-verbal cooperative response data, then normal children were found to display greater overall cooperation (verbal + non-verbal) than autistic children. ($F=5.00$; $df=1,18$; sig. 5% level).

e) Toy play

(21) Normal children spent longer in constructive toy play than autistic children ($F=67.41$; $df=1,18$; sig. 0.1% level), but there were

no significant differences in the number of different toys used per interaction. ($F=2.00$; $F=4.41$ for significance).

f) Mothers' speech to child

(22) Mothers spoke more utterances to autistic children ($F=7.52$; $df=1,18$; sig. $2\frac{1}{2}\%$ level), but the mean utterance length was longer with normal children. ($F=23.63$; $df=1,18$; sig. 0.1% level).

(23) A greater number of 'guiding' statements were directed towards normal children in contrast to autistic children. ($F=6.55$; $df=1,18$; sig. $2\frac{1}{2}\%$ level).

(24) A greater percentage of verbal statements were spoken to normal children in contrast to autistic children. ($F=21.95$; $df=1,18$; sig. 0.1% level).

(25) A greater number of questions were directed towards the autistic children ($F=4.43$; $df=1,18$; sig. 5% level), although the percentage of speech that contained questions did not differentiate autistic children and normals. ($F=0.06$; $df=1,18$).

(26) A greater number and percentage of verbal commands were spoken to autistic children. ($F=30.32$; $df=1,18$; sig. 0.1% level; and $F=22.09$; $df=1,18$; sig. 0.1% level).

(27) The TTR (type-token ratio) was higher when mothers were speaking to normal children. ($F=32.17$; $df=1,18$; sig. 0.1% level).

(28) A greater amount of verbal reward and verbal punishment was directed to the autistic children. ($F=17.32$; $df=1,18$; sig. 0.1% level; and $F=4.56$; $df=1,18$; sig. 5% level).

g) Child's speech to mothers

(29) Normal children spoke a greater number of words in total ($F=42.41$; $df=1,18$; sig. 0.1% level), utterances in total ($F=27.23$; $df=1,18$; sig. 0.1% level), and with a longer mean utterance length than

autistic children. ($F=84.58$; $df=1,18$; sig. 0.1% level).

(30) Normal children spoke a greater number and percentage of verbal statements to mothers. ($F=31.02$; $df=1,18$; sig. 0.1% level; and $F=5.67$; $df=1,18$; sig. 5% level).

(31) Normal children spoke a greater number of verbal commands than autistic children. ($F=6.14$; $df=1,18$; sig. 2 $\frac{1}{2}$ % level).

(32) Normal children made a greater number of verbal replies in their speech to mother's questions, and produced a greater percentage of replies in their speech. ($F=14.29$; $df=1,18$; sig. 1% level; and $F=18.74$; $df=1,18$; sig. 0.1% level).

EFFECTS OF "TYPE" OF MOTHER ON INTERACTIONS

(33) With the probability of transition from ANF/CF to AF/CF (i.e., adult termination to mutual facing), there was a strong tendency for an interaction between mothers and children such that with autistic children the mothers of normal children gave the higher probability but with normal children the mothers of autistic children gave the higher probability. Own mothers resembled mothers of normal children, although the differences between the two types of child were smaller. ($F=3.04$; $F=3.26$ for significance). (See Figure I/i).

(34) The probability of transition from ANF/CF to ANF/CNF (i.e., child termination to neither facing), was greater with mothers of autistic children when with autistic children, but with mothers of normal children it was greatest when they were with normal children. Own mothers resembled mothers of normal children in that there was a higher transitional probability when they interacted with normal children. ($F=3.58$; $df=2,36$; sig. 5% level). This interaction is illustrated in Figure I/ii.

Cooperation-negativism

(35) There was a significant difference between mothers in their ability to elicit cooperative responses (non-verbal) from the types of child. Mothers of normal children elicited a greater number of cooperative responses from both types of child than mothers of autistic children. ($F=5.07$; $df=2,36$; sig. $2\frac{1}{2}\%$ level). Mothers of normal children induced an average of 14.2 cooperative responses per interaction with autistic children and 10.7 with normal children. Mothers of autistic children induced a mean of 8.7 cooperative responses with autistic children and 7.8 with normal children.

(36) Mothers differed significantly in the percentage of punishment 'errors' they made with both types of child. ($F=5.03$; $df=2,36$; sig. $2\frac{1}{2}\%$ level). Own mothers made significantly more than the unfamiliar mothers of autistic children and normal children (Scheffe $F=3.71$; sig. 5%), and mothers of normal children made fewer than own mothers or mothers of autistic children. (Scheffe $F=3.82$; $df=3,36$; sig. 5%).

Mother's speech

(37) Mothers differed significantly in the mean utterance length that they spoke to both types of child. ($F=3.44$; $df=2,36$; sig. 5% level). Own mothers differed significantly from unfamiliar mothers of autistic and normal children by producing a shorter mean utterance length. ($F=3.39$; $df=2,36$; sig. 5% level).

(38) For both types of child, mothers differed significantly in the number of questions that they asked. ($F=6.50$; $df=2,36$; sig. $2\frac{1}{2}\%$ level). Multiple comparison analysis revealed that mothers of normal children asked more questions than either mothers of autistic children or own mothers. (Scheffe $F=5.92$; $df=2,36$; sig. 1% level). Also, own mothers asked significantly fewer questions than either

mothers of autistic children or mothers of normal children.

(Scheffe $F=4.62$; $df=2,36$; sig. $2\frac{1}{2}\%$ level).

(39) Mothers differed in the percentage of questions in their speech that they asked of children. ($F=6.12$; $df=2,36$; sig. 1% level).

A multiple comparison analysis revealed that both mothers of normal and mothers of autistic children asked a greater percentage of questions in their speech than own mothers. (Scheffe $F=5.43$; $df=2,36$; sig. 1% level).

(40) Mothers differed significantly in the percentage of verbal commands that they directed to the children. ($F=14.60$; $df=2,36$; sig. 1% level). Own mothers directed far more commands to both types of child, and especially to autistic children.

(41) Mothers differed significantly in the number of words in total that they spoke to both types of child. ($F=4.58$; $df=2,36$; sig. $2\frac{1}{2}\%$ level). A multiple comparison analysis revealed that mothers of normal children spoke significantly more words than own mothers or mothers of autistic children (Scheffe $F=3.48$; $df=2,36$; sig. 5% level), and own mothers spoke significantly fewer words than mothers of autistic and normal children. (Scheffe $F=3.38$; $df=2,36$; sig. 5% level).

EFFECTS OF FAMILIARITY UPON INTERACTIONS

(42) Familiar and unfamiliar diads differed in the total number of words they spoke ($F=4.58$; $df=2,36$; sig. $2\frac{1}{2}\%$ level), with familiar (own) mothers speaking significantly fewer words than unfamiliar mothers.

(Scheffe $F=3.38$; $df=2,36$; sig. 5% level).

(43) Familiar and unfamiliar mothers also differed in their mean utterance length ($F=3.44$; $df=2,36$; sig. 5% level), with familiar mothers speaking with a shorter mean utterance length than unfamiliar mothers.

(Scheffe $F=3.34$; $df=2,36$; sig. 5% level).

(44) Interactions involving familiar and unfamiliar diads differed in the absolute number, and also percentage, of questions that were directed to the children. ($F=6.50$; $df=2,36$; sig. 1% level; and $F=6.12$; $df=2,36$; sig. 1% level). Familiar mothers asked fewer questions in total (Scheffe $F=4.62$; $df=2,36$; sig. 2½% level), and a smaller percentage of questions in their speech. (Scheffe $F=5.43$; $df=2,36$; sig. 1% level).

(45) Familiarity affected the percentage of punishment 'errors' made by mothers. ($F=5.03$; $df=2,36$; sig. 2½% level). Familiar mothers made significantly more errors than unfamiliar mothers. (Scheffe $F=3.71$; $df=2,36$; sig. 5% level).

(46) Familiar mothers directed a higher percentage of verbal commands in their speech than mothers unfamiliar to the children. ($F=14.60$; $df=2,36$; sig. 1% level; Scheffe $F=11.79$; $df=2,36$; sig. 1% level).

These results may be summarised as follows:

SUMMARY OF MAJOR RESULTS

a) Child effects

(1) Autistic children spent longer in mutual facing than diads involving normal children. However, when the mother and child were mutually facing, the mother turned to face away more often when interacting with autistic children, although mothers maintained mutual facing for longer periods before facing away with autistic children.

(2) Mothers spent a longer total time facing the autistic child. In addition to the longer periods spent in mutual facing with autistic children, this finding was also due to a longer duration and percentage of time spent in the adult facing/child not facing position with autistic children. With autistic children, the average duration before

the adult terminated the position adult facing/child not facing by looking away, was longer than with normal children. Predictably therefore, normal children spent longer in the neither facing position and the average duration before the adult terminated this position, (by turning to face the child), was also longer with normal children.

(3) There was an interaction between type of child and mother such that when the child was facing the mother but the mother was not facing the child, the autistic child was more likely to turn away himself when with mothers of autistic children in contrast to mothers of normal children, but the normal child was more likely to turn away from mothers of normal children. However, there was also a tendency for an interaction between type of mother and child such that when it was the mothers who terminated this position by turning to face the child (thus establishing mutual facing), with autistic children the mothers of normal children were more likely to do so than mothers of autistic children. With normal children the mothers of autistic children were more likely to turn towards the child.

(4) Predictably, the autistic children displayed more stereotypy than normal children, with an accompanying higher percentage of antecedent maternal "demands". The mean number of stereotyped behaviours per interaction was 9.3, displayed on average during 43.6 seconds of interaction time and preceded on 47% occasions by maternal "demands".

(5) Autistic children displayed more total body movements, both towards and away from, mothers than normal children. These movements were preceded by a higher percentage of maternal "demands". The mothers themselves displayed a greater number of total body movements towards and away from autistic children.

(6) Whilst normal children did not exhibit a greater degree of non-verbal cooperation than autistic children, a greater number of the cooperative responses of the autistic children were rewarded.

Therefore normal children elicited a higher percentage of reinforcement 'errors' from mothers than autistic children. If verbal replies to questions are also considered as an aspect of cooperative behaviour, then summing the number of non-verbal and verbal cooperative responses revealed that normal children did display more cooperation than autistic children. Autistic children predictably displayed more negativism than normal children, and mothers displayed a greater degree of punishment 'errors' with autistic children.

(7) There were no significant differences between the types of children in terms of number of toys/puzzles used, but the normal children did spend longer overall times in constructive toy play.

(8) Autistic children received a greater number of utterances from mothers, but the mean utterance length was shorter for autistic children and the TTR was lower. Normal children received a greater percentage of verbal statements from mothers in contrast to autistic children, including also more 'guiding' statements. More verbal commands were directed to the autistic child, and they received more verbal reward but also more verbal punishment. More questions were directed to the autistic child than normal child, but since the former received a greater total number of utterances, the percentage of total utterances that contained questions, did not differ between autistic and normal children.

(9) Normal children spoke more words and utterances in total to the mothers than autistic children, and their speech had a longer mean utterance length. Normal children emitted more verbal statements, more commands and made a greater number of verbal replies to mother's questions than autistic children.

Familiarity effects

- (1) In unfamiliar diads, the mothers spoke with a longer mean utterance length than familiar mothers, and also spoke more words in total.
- (2) Familiar mothers (i.e., the own mothers) asked a significantly smaller number and percentage of questions to both types of child than unfamiliar mothers, but made more verbal commands.
- (3) Familiar mothers made more punishment 'errors' than unfamiliar mothers.

Effects of type of mother

- (1) There was an interaction between type of child and type of mother such that when the child was facing but the mother was not, the autistic child was more likely to turn away from the mother (thus establishing a neither facing position) in interactions involving mothers of autistic children. Conversely, the normal child was more likely to turn away from mothers of normals.
- (2) There was also an interaction between type of child and type of mother with the probability of the adult terminating the ANF/CF position, and thus turning to the child to establish mutual facing. The mothers of autistic children tended to be more likely to establish mutual facing from this position when interacting with normal children, but mothers of normals were more likely to do so with autistic children. Own mothers resembled unfamiliar mothers of normal children by displaying a greater probability of turning towards the autistic child, rather than the normal child.
- (3) Mothers of normal children elicited a greater number of non-verbal cooperative responses from both types of child than mothers of autistic children.
- (4) Mothers of normal children made fewer "punishment errors" than either mothers of autistic children or the own mothers, the latter

making the most of such errors.

(5) Mothers of normal children spoke a greater number of words in total to both types of child than the own mothers or mothers of autistic children. Own mothers spoke the least words.

(6) Own mothers produced a shorter mean utterance length than mothers of autistic children and mothers of normal children unfamiliar with the child.

(7) Mothers differed significantly in the number of questions they asked their interaction partners. Mothers of normal children (unfamiliar with the child) asked more questions of both types of child than either mothers of autistic children (unfamiliar with the child) or the own mothers. The own mothers asked the least questions. When expressed as a percentage of their total speech, own mothers also asked a smaller percentage of questions to both types of child than either mothers of autistic children or normal children unfamiliar with the child.

(8) Own mothers directed the highest percentage of verbal commands in their speech and this was especially marked in interactions with autistic children. Mothers of normal children directed the least commands to children.

DISCUSSION

Lack of eye contact, or gaze aversion, is a commonly cited characteristic of autistic children, (Creak et al., 1961; Hutt and Ounsted, 1966; Castell, 1968; Kanner, 1943). This symptom has been used to support hypotheses concerning both CNS pathology (Schopler, 1965; Rimland, 1964), and emotional withdrawal, (Bettelheim, 1967; Zaslow and Breger, 1968). However, the results of studies designed to measure the degree of gaze aversion have not produced consistent results. Thus whilst Hutt and Ounsted (1966) reported that autistic children

spend significantly less time looking at the faces of adults than normal children, Churchill and Bryson (1972) presented data which did not support this assertion. Equally, O'Connor and Hermelin (1967a) found that autistic and retarded children do not differ in visual preference for social stimuli (pictures of faces) in contrast to non-social stimuli, although overall fixation time for all stimuli was less with autistic children, who spent longer in non-directed gazing. Hutt and Ounsted claimed that autistic children, when they do fixate, use fractional glances to monitor social stimuli in their environment, this consisting of rapid glances of very brief duration which, presumably, parents in the home environment may miss altogether. Such glances can only be accurately observed and measured with the use of videotaped recordings and repeated slow-motion playback. The use of such a technique in the present study did indeed reveal a number of such glances. However, it would appear that the contradictory findings in the degree of eye contact can perhaps be explained in terms of its measurement and the environmental conditions under which eye contact is measured.

With regard to this latter question of the method and conditions of measurement of eye contact; if autistic children are over "aroused" (Hutt et al., 1964) or simply very timid (Tinbergen and Tinbergen, 1972), then the degree of eye contact may be a function of the complexity or indeed "demand" in the environment. Thus the number and behaviour of adults or peers in the environment in which eye contact is measured, may affect fixation time. This would parallel the assertion that stereotypy increases with environmental complexity (Hutt and Hutt, 1968). Perhaps of more significance is the measurement of actual eye contact and gaze aversion. Even with the use of videotaped playback at slow speeds in the present study it was not considered possible to infer direct eye-to-eye contact with any strong

degree of confidence, although the less specific direction of gaze was clearly measurable.

In the present study, the "facing" position was defined in terms of an interaction partner facing the other by directing the gaze towards the upper trunk, neck or face. This almost certainly did not always involve actual mutual eye-to-eye contact, although it was felt that it was operationally and functionally a better description of "facing" than that requiring an inference of mutual eye contact. It was felt that "facing" as defined above implied that an interaction partner was attending to the behaviour of the partner and also signalling a readiness to interact. Thus it is possible that contradictory research findings could be due, for example, to different criteria used to define what have been referred to (and often interchangeably) as "eye contact", "fixation", "looking" and "facing".

In the present study, with the definition of "facing" given above, it was found that there were no significant differences between autistic and normal children in terms of total number of occurrences of mutual facing. Indeed, there was a longer duration of interaction time in the mutual facing position with diads involving the autistic children. The mean time actually spent in mutual facing was significantly longer with autistic children (22.7 seconds/interaction), and this was almost twice as long as in diads involving normal children (12.1 secs.). There were no differences between autistic and normal children in the time spent facing the adult when the adult herself was not facing (i.e., the ANF/CF position). These results are not intended to imply that autistic children do not necessarily gaze avert in the sense of failing to establish mutual eye-to-eye contact; rather it is suggested that they do not differ in the amount of "facing" the adult, (and thus the social "signals" that go with it). These results suggest that in

fact they spend longer in mutual facing under the experimental conditions used in this study.

Hutt and Ounsted (1966) state that directed gaze signifies a readiness for interaction, and that a failure to fixate has a very important inhibitory affect upon social interaction. They point out that much affectational behaviour is contingent upon the child making and maintaining some degree of eye contact. Gaze avoidance will thus result in attenuated responsiveness from the adult and a reduction in stimulation for that adult. In early infancy, eye contact is claimed to be a necessary precursor to smiling (Ambrose, 1963) and Hutt and Ounsted point out that although autistic children can (and do) smile, when unaccompanied by eye contact the initial maternal enthusiasm may wane. Eye contact signals the direction of smile, and thus with an autistic child in early infancy, ambivalent maternal attitudes may develop due to positive feelings aroused by other cues (e.g., outstretched arms) and yet negative feelings from the lack of eye contact. It is not necessary to support Hutt and Ounsted's proposal that gaze avoidance is a mechanism to reduce arousal in order to agree with their assertions concerning the importance of eye contact. Many parents of autistic children do report perplexity and disappointment at their child's gaze avoidance and clearly it can have inhibiting effects upon interactions. Since the present study was not intended to measure eye-to-eye contact, the results cannot illuminate directly on the contradictory findings with regard to direct gaze aversion. What the present results do suggest is that if there is a significant degree of gaze aversion, then this must apply solely to eye-to-eye contact, since there was certainly no evidence of autistic children displaying less "facing". There would appear to be two implications here:

- 1) It may be possible to train parents to interpret their child's "facing" as positive feedback, as a reinforcer for interacting with their child, by teaching them that a child "facing" is indeed signalling attention and a readiness to interact, and that actual eye-to-eye contact need not be the only way such a willingness can be signalled. Thus we might try to teach mothers of autistic children to find facing, (in addition to eye-contact), as a reinforcer during interactions.
- 2) As an alternative to the above, or indeed in conjunction with it, actual eye-to-eye contact could be established through the use of a behaviour modification technique using the parents as co-therapists. Since the present study yielded results which suggest that "facing" at least is in fact a well established behaviour, it could be used as an initial approximation, or baseline, from which shaping actual eye contact could proceed. Some success has already been reported with the reduction of gaze avoidance using operant techniques, (Brooks, Morrow and Gray, 1968; McConnell, 1967).

The findings that mothers themselves tended to terminate mutual facing more frequently when interacting with autistic rather than normal children, and yet spend longer periods maintaining the mutual facing before such terminations, appears at first sight to be rather contradictory results. However, one explanation could be that mothers tolerate longer periods of mutual facing with the autistic child because there is less immediate feedback from the autistic child. This may occur because the autistic child does not readily establish actual eye-contact despite "facing", and as the current results suggest, he gives less verbal feedback in terms of both overall verbal output and replies to questions. If the autistic child is not providing the same degree of positive feedback or reinforcement for the mother in the mutual facing position, then this might explain why mothers termin-

ate mutual facing, by turning away, more frequently with the autistic child in contrast to the normal child. This in turn may result in autistic children receiving less reinforcement for "facing" mothers in an interaction situation.

Mothers also spent longer facing the autistic child overall, and despite the occurrence of longer periods of mutual facing with such children, it is clear that this finding was also due to longer periods spent in the adult facing/child not facing (AF/CNF) position. This latter position was maintained for longer periods before the adult turned away. Predictably therefore, diads involving normal children spent longer in the neither facing position, and with normal children this position was maintained longer before the adult turned to face the child. These results suggest that mothers find it necessary to watch the autistic child far more than the normal child, when neither child was facing the adult. This may have been related to the finding that autistic children spent significantly less time in toy play activity, and thus spent longer displaying the non-required behaviour (including stereotypy).

More verbal commands were directed by mothers towards the autistic child, many of them instructing him to engage in the required (toy play) activity. Not surprisingly therefore, the autistic children commanded greater visual attention from mothers, provided one assumes that less visual attention was directed towards the child once he was playing with the toys/puzzles. In the present study, no separate data had been recorded for maternal behaviour solely when the child was engaged in constructive play. However, simple observation from a replay of the videotaped recordings, suggested that mothers do not appear to spend less time facing the autistic child when he is engaged in constructive toy play, although this may be so for normal children.

An alternative explanation for these findings is that since the autistic child spoke significantly less than the normal child, and indeed five were effectively non-verbal, the visual modality was of particular importance for the mothers in receiving any feedback from the autistic child. For normal children, naturally much feedback was signalled to the mothers via speech, which does not necessarily require the mother to "face" the child. However, it must be pointed out that verbal dialogue might itself be a cue or discriminative stimulus for mutual "facing".

When the child was facing a mother who was herself not facing (ANF/CF), autistic children were more likely to turn away from a mother of an autistic child (MA) than a mother of a normal child (MN). This may have been because mothers of autistic children themselves tended to be less likely to terminate this position by turning to the child and thus establishing mutual facing, (although this trend was not statistically significant), and this may have been particularly so with unfamiliar mothers of autistic children. The position adult not facing/child facing (ANF/CF) was not maintained for very long by either partner (for autistic children diads a mean of 1.98 secs., and for normal children 1.96 secs.), and so it would seem that unless the mother fairly rapidly turned to face the child, then the child would turn away himself.

In contrast to the above result, when normal children were facing the mother but the mother was facing away, they were more likely to turn away from mothers of normal children than mothers of autistic children. This also seems likely to be related to the fact that mothers of normal children were less likely themselves to turn to face the child, and particularly the unfamiliar mothers of normal children. Thus in terms of a "sensitivity" to the child facing the adult and thus

signalling attention and readiness to interact, mothers of autistic children (and particularly those unfamiliar with the child) were least "sensitive" to the autistic child in terms of turning to face the child. Similarly, mothers of normal children (and particularly those unfamiliar with the child) were least sensitive to normal children.

This might suggest that familiarity with the "type" of child, in contrast with familiarity with the individual child (as we have used the term 'familiarity' to date), influences the mothers' "sensitivity" to the child's behaviour. Thus mothers of autistic children, being familiar with the characteristics of autism and the behaviours of autistic children, were less "sensitive" to autistic children in the interactions. Mothers of normal children on the other hand, are unfamiliar with the characteristic behaviour of autistic children and are therefore more sensitive to his facing position than that of the normal child. One might tentatively go further and say that mothers of autistic children are less "sensitive" to the autistic child "facing" because they do not find mutual facing as rewarding as mothers normally do when interacting with normal children. It would seem possible that this may be because the autistic child fails to establish mutual eye-contact very often. But when interacting with the normal child, mutual facing is rewarding for the mothers of autistic children due to the relative ease and frequency of establishing mutual eye contact. Thus she is more "sensitive" to the facing position displayed by the normal child. For the mother of a normal child when interacting with an autistic child, being unfamiliar with autism and its characteristics, results in continued attempts to establish mutual eye-contact, in addition to a probable high level of curiosity inherent in interacting with such an atypical child for the first time.

These inferences do, of course, depend on the assumption that "sensitivity" is a function of: (a) Speed of perceiving that the child

is "facing"; (b) More important, having recognised that the child is facing, turning to face the child herself.

One might add however, that a mother who ignores her child's "facing", (by not turning to face the child), would at an intuitive level appear to be effectively rejecting the child's signal of a readiness to interact.

Stereotypy did not appear more frequently or for any longer periods with any one "type" of mother. There were also no differences in the degree of stereotypy with unfamiliar mothers in contrast to mothers familiar with their interaction partner. However, 48% of the appearances of stereotypy in the autistic child, were preceded by either verbal commands, non-verbal demands, or questions, from the mother. This is a higher percentage than one might expect by chance, and it would appear therefore that stereotypy may be displayed partially, although not wholly, as a function of the degree of environmental "demand" placed upon the child.

Hutt et al., (1964) reported that stereotypy in autistic children appeared to be a function of environmental "complexity". Such behaviour increased as the number of people and objects in the immediate environment increased. Hutt et al., (1965) later reported that monitored EEG recordings suggested that stereotypy increased with EEG correlates of arousal, and thus stereotypy may be seen as a function of arousal. The Hutt's proposed that the autistic child suffers from an overactivated Reticular Formation, maintaining the autistic child in a chronically overaroused state. Thus any increase in environmental complexity and therefore environmental stimulation, would overload the child with sensory information, and so stereotypy acts as a mechanism to reduce stimulation and reduce arousal to a minimum.

Whilst the present results could be interpreted as supporting this hypothesis, since, "demands" placed upon the child might be increasing environmental stimulation levels and thus arousal, another explanation is also tenable. As Kozloff (1973) points out, we must look upon the interaction as a social exchange where the behaviour of one individual yields consequences for the behaviour of the other - interactions are based on reciprocity. We may therefore ask what effects stereotypy has upon the mother. It is possible that stereotypy acts as a "cut-off" act (Chance, 1962) in the same manner that the Hutts see the function of gaze aversion. Stereotypy contingent upon maternal "demands" may result in a gradual extinction of maternal "demands", or certainly it would seem that stereotypy is unlikely to reinforce such "demands". Stereotypy could therefore be seen as an avoidance response from the autistic child rather than a mechanism to reduce physiological arousal. This is supported by the finding that a high percentage (75%) of total body movements away from the adult were preceded by maternal "demands".

We may now ask why maternal "demands" could be aversive for the autistic child. Whilst this may be connected with the broader question of how childhood autism develops, in a restricted sense the answer may be partially related to the problem of the child's apparent abilities. Churchill (1971) reported a study in which he found that much "pathological" behaviour of the autistic child disappears and reappears as a function of the success of the task in hand. Stereotypy, amongst other deviant behaviours, disappeared when the experimental situation was arranged so that the autistic child could not fail at the required tasks. Similarly, Hington et al., (1967) found that autistic children working at tasks of very low complexity, were both attentive and cooperative in contrast to their behaviour during more complex tasks. Churchill feels that the autistic child may be living in a

perpetual "failure" condition. The parents, not fully understanding or taking sufficient account of the child's limitations, may unwittingly increase failure by persistently demanding successful performances on tasks remaining beyond the child's genuine capabilities. If this were so, it is not difficult to imagine how many maternal "demands" may have become aversive to the autistic child. What remains is to explain why stereotypy is the selected avoidance response of the autistic child, although one can see how operationally at least, it effectively precludes any possibility of reciprocal interaction. Stereotypy is not uncommon in the subnormal population as a whole, where it is often considered to be a self-stimulating behaviour. Such behaviour may however be considered to serve more than one function in the autistic child.

Autistic children predictably displayed more total body movements both towards, and away from, the adult. These movements were preceded on 70% occasions, when towards the mother, by maternal "demands", and on 75% occasions when the movement was away from the mother. Thus whilst a high percentage of "demands" preceded movements away from the mother, (and these movements may appear to be avoidance responses to maternal "demands"), the movements towards the mothers were preceded by a similar percentage of "demands". However, whilst the specific nature of the "demands", (verbal commands, verbal and non-verbal demands, and questions) were not differentiated, it was observed that a larger proportion of those directed to the child preceding movements towards the mother were either commanding or "coaxing" the child to move towards the adult. Thus these body movements towards the adult were often cooperative, rather than avoidance, responses. An empirical differentiation between the nature of the "demands" would therefore have proved useful.

Autistic and normal children did not differ significantly in

the number of non-verbal cooperative responses that they exhibited. However, since a greater number of verbal commands were directed to the autistic child, there would seem to have been a greater number of potential opportunities for him to cooperate. In addition, this finding is restricted to purely non-verbal cooperative responses, although as noted earlier this is the major channel for cooperation for the autistic children.

Mothers of normal children elicited more non-verbal cooperative responses from the children than mothers of autistic children. This result applies where both types of mother were unfamiliar with the individual child. Since this finding is apparently not dependent upon familiarity with the child, one must infer that some factor associated with being a mother of an autistic child results in the child exhibiting fewer cooperative responses. This may be related to the finding that mothers of normal children asked more questions of both types of child, and indeed spoke a greater total of words overall. Thus one possible explanation is that mothers of autistic children, being familiar with the general characteristics of "autism" and the limitations in the autistic child's abilities and behaviours, make fewer "demands" upon the child and this generalises to interactions involving normal children. However, this appears unlikely for, although mothers of normal children asked more questions overall, the mothers of autistic children made more verbal commands than mothers of normals. Thus it seems unlikely that mothers of autistic children were making less overall "demands" upon the children.

The finding that both types of child actually display more non-verbal cooperative responses with mothers of normal children, suggests that mothers of autistic children may be either understanding the child's capabilities or interacting with him in such a way that the child is less cooperative and compliant than he is with mothers of normal children.

Indeed, one might have predicted that mothers of autistic children, being aware of the characteristics of autistic children, would be more skilled and adept at eliciting cooperation from the autistic children. Clearly, the present findings suggest that for neither type of child is this so.

Perhaps somewhat surprisingly, although autistic children were rewarded in total more frequently for their cooperative responses, (44% of cooperative responses of autistic children were rewarded; 23% for normal children), the "types" of mother did not differ significantly in the number of rewards they made contingent upon a cooperative response. Thus the greater cooperative behaviour of the autistic child with mothers of normal children does not appear to be due to greater use of overt reinforcement. It was noted however that mothers of normal children did display more contingent reward than mothers of autistic children in absolute number (means:- 2.7/interaction and 4.6/interaction respectively), though this trend was not statistically significant. Thus the origin of the differential display of cooperation must remain open to question. Further research on the possibility of "non-optimal" interaction behaviour in mothers of autistic children would appear to be warranted, for it is most important that the natural environment of the autistic child is optimally matched with the needs of that child arising from his particular deficiencies. The importance of parental behaviour is stressed by Moore and Bailey (1973): "It has become increasingly clear that if changes in a child's behaviour are to be maintained, the significant adults, usually parents, must be systematically trained to cope with these behaviours". (p.497)

Since the number of reinforcement "errors" made by mothers was greater for diads involving normal children, (mean % for autistic children was 56.6%, for normal children 76.9%), it would appear that

mothers feel it necessary to reward the non-verbal cooperative responses of autistic children more often than those of normal children, despite the finding that the children did not differ in the total number of such cooperative responses displayed. This may be related to the relative abilities of the children and to differences in their qualitative behaviour. Autistic children have relatively limited skills in contrast to normal children, and in addition display far more negativism. The mothers may have been rewarding cooperative behaviour more frequently with autistic children in an attempt to differentially reinforce cooperation and negativism.

If verbal replies to questions are considered as another feature of cooperation in addition to the non-verbal cooperation, the summing of these two aspects of cooperation revealed that normal children predictably did display more overall cooperative behaviour.

The finding that autistic children displayed more negativism than normal children is not surprising, and supports other research that suggests that autistic children display a significant degree of negativism (e.g., Morrison, Miller and Mejia, 1971), and also the parental questionnaire data reported in this study. However, of more interest was the finding that such negativism was infrequently punished. Analysis of mean punishment "error" data revealed that 82% of all negativistic behaviours remained unpunished. Indeed, it was observed that on many occasions such behaviours were reinforced with attention. This figure for punishment "errors" is very high, even taking into account the awareness of the mothers that the interactions were being observed and recorded. One might also predict that the negativism would cause as much embarrassment to the mothers as the use of punishment, and there would therefore be pressure to attempt to suppress such behaviours with the use of social or even physical punishment.

Mothers of normal children made fewer punishment errors than mothers of autistic children. This again suggests non-optimal contingency management from mothers of autistic children.

The finding that mothers often reinforced negativistic behaviours, through delivery of contingent attention, supports findings that many deviant behaviours of the autistic child are inadvertently reinforced by parents, (Lovaas et al., 1965; Kozloff, 1973). Moore and Bailey (1973), whilst conducting a behaviour modification programme with an autistic child, also noted that punishment "errors" from the parents were far more frequent than reinforcement "errors" during base-line assessment. The present findings support this assertion, particularly with the own mothers, and also imply that a programme teaching parents of autistic children the use of social and physical punishment in the modification of "negativistic" aspects of autistic behaviour, could prove most useful. Such schemes are of course not restricted to autistic children, and have been successfully used with children displaying inappropriate behaviour in other psychiatric disorders, (e.g., Bernal et al., 1968; Wahler, 1969).

Whilst there were no significant differences between autistic and normal children in terms of the number of toys and puzzles they used, the normal children spent longer total time in constructive toy play. (Mean time for autistic children = 51% interaction time, for normals = 90%). This is predictable on the basis of the findings that autistic children spend significantly longer periods displaying stereotypy, negativism, and movements away from the adult, in addition to using the toys in non-constructive play.

Autistic children received a greater number of utterances from mothers than normal children, although the mean utterance length was shorter with the former, as was the type-token ratio (TTR). This

indicates that the mothers were modifying the construction of their speech when interacting with autistic children in contrast to normal children. Since the children were matched on chronological age, it is clear that such a discrimination must be based on the linguistic and intellectual abilities of the children. For mothers who were unfamiliar with a particular child, such speech modifications were inevitably based largely on estimations made in a comparatively short period of familiarisation prior to the experimental video recordings, in addition to internal "models" or expectancies currently held about the comprehension abilities of autistic and normal children of the relevant chronological age. It would seem safe to conclude that mothers familiar with the child (i.e., the 'own' mothers), would construct their speech largely on the basis of their past experience of the linguistic and intellectual abilities of the individual child.

That all mothers verbally discriminated between autistic and normal children is not surprising since they display differences in comprehension cues and feedback. Much recent research has focussed on modification of the mothers' speech when interacting with different children. In the field of language acquisition, several studies have shown that the linguistic input to the child is very different to that of the adult. Snow (1972), using 2 and 10 year olds in mother-child interactions, found that on a number of verbal measures including indices of sentence complexity and length, the mothers' speech differed when addressing 2 and 10 year olds. Broen (1971) reported similar significant differences in finer discriminations, (2 Vs 5 Yr olds), and Roberts and Fraser (1972) found age affects amongst speech to $1\frac{1}{2}$, $2\frac{1}{2}$, 4 and 6 year olds. Similar examples of speech modification were found by Moerk (1974), and by Spradlin and Rosenberg (1964) using retardates and normals. Snow, in an attempt to establish what it is that stimulates the mother to modify her speech, had 'present'

versus 'absent' conditions. In the latter condition, the mother was asked to imagine the presence of a child of a particular age, and Snow found that age discriminations largely disappeared. It seemed that something beyond the actual social role or mother's 'plan' or 'model' of the appropriate level of communication was operating in the child "present" condition. The children themselves therefore appear to play an important role in eliciting speech modifications via feedback, including features like comprehension and attention cues. This is not only a vital factor in enabling the child to acquire speech, but is also an excellent example of measuring the mother's "sensitivity" to feedback from her child.

In the present study, the analysis revealed that mothers spoke more to the autistic child than the normal child in terms of total number of utterances, although the mean utterance length was shorter in speech to the autistic child and the TTR (a measure of lexical variance), was lower. Such modifications are highly functional. Thus Browning (1974) reported how the number of correct responses to verbal commands rapidly increased in autistic children as the length of the utterance was reduced. Mothers however appeared to feel it necessary to speak more to the autistic child, in the present study. This may be related to the predictable finding that autistic children spoke far less to mothers than normal children did, and naturally this left more interaction time for mothers' speech. However, this seems an unlikely explanation, for the two-way dialogue or conversation which characterised interactions with normal children would appear to require a greater degree of output from the mother. A more probable explanation is found in the differential non-verbal behaviour of the children. Thus autistic children, displaying more negativism and less time spent in independent constructive toy play, invite a greater degree of verbal supervision. Indeed, not surprisingly more verbal commands were directed to the autistic child, and they received more verbal reward and punishment.

Mother's speech contained a higher percentage of verbal statements when directed to normal children in contrast to autistic children, but the finding that more "guiding" statements were spoken to the normal children was particularly surprising. Autistic children need far more "guiding" statements than normal children in view of both the severely limited abilities and apparent preference for less constructive use of toys, (Tilton and Ottinger, 1964), seen in autistic children. This finding is particularly important in the light of Churchill's evidence (Churchill, 1971) that much of the inappropriate behaviour of autistic children may be a function of the success:failure ratio of the child on required tasks. "Guiding" statements from the mother are necessary for the autistic child to maximise his chances of success, and thus reduce deviant behaviour.

The modification of utterance length and the TTR by mothers when speaking to the two types of child, is evidence that mothers were clearly sensitive to some measure of grammatical and lexical complexity for the child. Since autistic and normal children were matched on chronological age, mothers did appear to modify aspects of their speech as a function not just of chronological age, but also of mental age and overt behaviour.

More questions were directed to autistic children than normal children, but since autistic children received a greater total of utterances, the percentage of speech that consisted of questions did not differ between the two types of child. Thus autistic children did not receive proportionally more questions than normal children.

While the present study revealed that all mothers made modifications in their speech when speaking to autistic and normal children in terms of number of utterances, utterance length and TTR, there were also differences in the modification between the types of mother depending on their familiarity with the child. In interactions

where mothers were unfamiliar with the child, mothers spoke more words in total and with a longer mean utterance length with both types of child. This might suggest that since unfamiliar mothers make their speech modifications on the basis of generalised expectancies or "models" from experience with other children, plus estimations on the basis of the few minutes prior to the recording section of the interactions and the child's current behaviour in the interaction, these mothers were comparatively inaccurate in their estimations of the child's comprehension, tending to overestimate their comprehension abilities. This explanation is based on the assumption that the own mothers, or familiar mothers, do indeed "match" their speech construction to the abilities of the child in an optimal fashion. It is also restricted to a measure of grammatical modification since the TTR did not differ between familiar and unfamiliar mothers.

It is possible that these results may well be related to other findings concerning the mother's speech to the child which suggests that speech modification is not dependant solely on familiarity but also on the "type" of mother. Mothers of normal children, unfamiliar with their interaction partners, spoke a greater number of words in total to both types of child than mothers of autistic children also unfamiliar with the child. These results would appear to be related to the type of utterances and their contents. Mothers of normal children (unfamiliar with the child) asked more questions of both types of child than mothers of autistic children (also unfamiliar with the child). In addition, both mothers of autistic children and mothers of normal children unfamiliar with the child, produced a greater percentage of questions in their speech than the own mothers. Thus it was the percentage of questions, rather than the absolute number, which appeared to be solely dependant upon familiarity.

Autistic and normal children predictably differed in several aspects of their own speech. Normal children spoke more in terms of utterances and words and gave mothers, particularly those unfamiliar with the child, more verbal information on which to estimate the required level or appropriateness of their speech to the child. Since the normal children spoke with a longer mean utterance length, it would not be surprising if mothers used this as one of the guides to the child's own comprehension, and so "matched" her own speech on this basis of this estimated level.

Normal children made more verbal statements and commands than autistic children, and made a greater percentage of replies to mothers' questions. As discussed earlier, this latter measure may be seen as one measure of the degree of cooperation displayed by the child.

In conclusion, it is suggested that the present study may indicate a number of features which should be present in a treatment programme for autistic children in which parents are used as cotherapists. It should be emphasised that these suggestions are only tentative.

These might include an emphasis on the recognition of the child facing the mother as indicative of a willingness to interact, despite a lack of eye-to-eye contact. Mutual facing should be encouraged and reinforced, and maintained for as long as the child permits, perhaps as a first stage in the shaping of sustained eye-to-eye contact. In addition, emphasis may be placed upon the need to make "demands" upon the child only within his known capabilities, breaking down required activities into smaller sub-components such that the child learns to succeed. Such programmes are used with many mentally handicapped children, but may be especially important for autistic children. Consistent contingent reward is clearly necessary for cooperative behaviour, but parents may need particular training in the use of extinction and "time-out" procedures for non-adaptive behaviours like negativism.

Finally, comprehension and intellectual ability-appropriate speech modifications, although clearly made by mothers of autistic children, may be supplemented by a greater use of speech including "guiding" statements, which in this study were far more frequently directed towards normal children.

CHAPTER FOUR:

"AUTONOMIC RESPONSIVITY IN AUTISTIC CHILDREN"

Introduction

It has been suggested that one of the fundamental problems for the autistic child is his failure to learn secondary, symbolic rewards (Ferster, 1961; Lovaas, 1971). Secondary reinforcement control is clearly most important for the normal child, for it takes over the controlling functions of the primary rewards that operate in early infancy. Many behaviourists believe that the lack of social development in the autistic child is the result of his failure to work for symbolic rewards.

Whether the failure to acquire secondary reinforcement control over the autistic child is a function of inappropriate reinforcement contingencies in early life (Ferster, 1961) or of an organic defect whose precise nature is unknown (Rimland, 1964), this failure is defined by behaviourists in operational terms. (Thus 'social' reward does not increase the emission of desired behaviours, and 'social' punishment does not suppress inappropriate behaviours). This type of analysis yields information concerning the effectiveness of secondary rewards, but does not tell us whether such rewards fail to gain control over behaviour because they have no value or meaning for the autistic child (i.e., they act as neutral stimuli), or whether they do possess values but they are not the values that they appear to hold for other children. Thus Lovaas, et al., (1965) reported that social contact (typically a positive secondary reinforcer for normal children) actually increased the frequency of inappropriate behaviours like tantrums and self-destructive behaviour, whilst isolation from interpersonal events (considered as secondary punishment) reduced such deviant behaviour.

It is necessary to distinguish between the possibility of secondary rewards being 'meaningless' or valueless to the autistic child, and holding inappropriate values for him. Certainly experimental secondary reinforcement control is possible, for behaviour modification

programmes involving the pairing of social reward with the termination of aversive stimulation have apparently been successful in the shaping of several desired behaviours like mutual eye contact and physical contact (Lovaas, Schaeffer and Simmons, 1966; Lovaas, 1971). Rather than simply observing overt behaviour, it may be possible to assess the 'meaningfulness' of traditional secondary rewards through the use of other indices. Autonomic measures appear to be particularly suitable for this purpose, monitored during conditions of both primary and secondary reward. They enable a distinction to be made between a failure to respond to social reward despite perceiving that reward, and a failure to perceive the reward itself.

Hutt et al., (1964, 1965) proposed that the autistic child suffers from states of chronic over-arousal, and report behavioural and EEG data to support this hypothesis (Hutt and Ounsted, 1966; Hutt et al., 1965; Hutt and Vaizey, 1966). This research has been reviewed in chapter 1 (pp.49-54) where it was also noted that evidence contradicting this model has been reported (e.g., Bernal and Miller, 1971; Hermelin and O'Connor, 1970; Churchill and Bryson, 1972). Inferences from EEG records are not unequivocal (Johnson, 1970), and the situation is further complicated with autistic children by the apparent high frequency of EEG abnormalities (Kolvin et al., 1971; Gittelman and Birch, 1967). An over-arousal model thus requires testing on rather less ambiguous measures, and again an autonomic measure may prove useful in this context. It has been noted that the autistic child appears to be particularly sensitive to 'social' stimulation on measures of overt behaviour like stereotypy and gaze aversion (e.g., Hutt and Vaizey, 1966). It may therefore prove interesting to measure the relative response magnitude to 'social' and non-social stimuli, using an autonomic index.

Predictions from a second physiological aetiological model are also amenable to tests on autonomic measures. If the autistic child experiences fluctuating physiological states and a process of perceptual inconstancy (Ornitz and Ritvo, 1968a, 1968b; MacCulloch and Williams, 1971), then predictions of transient periods of hypo- and hypersensitivity, and wide differences in the responsivity of any individual over time, logically follow. An inability to maintain constancy of perception would naturally lead to grave problems of recognition, and a serious failure to habituate to incoming environmental stimuli. A comparative analysis of responses to familiar and unfamiliar stimuli may therefore yield data that will enable some assessment of the utility of this aetiological model.

As discussed in chapter 1 (pp.55-59) supportive evidence to date for the hypothesis of perceptual inconstancy rests largely on the basis of physiological measures taken during the sleep periods of autistic children rather than during behaviour exhibited in the waking state (and which thus characterises the autistic child). Other evidence consists of measures of heart rate variance taken during a methodologically unsound study (MacCulloch and Williams, 1971), in which neither age or activity levels were controlled. It is proposed therefore that the selection and monitoring of an appropriate autonomic measure during experimentally controlled stimulus conditions, is an appropriate technique for testing possible autonomic abnormalities in childhood autism.

A final consideration in the design of the present study arises from the repeated clinical observation of the behavioural unresponsivity of the autistic child. Many of these children are non-verbal, and all of them display severe expressive and communication deficits. The lack of overt response to many environmental stimuli has

led to the description of the autistic child as aloof and severely withdrawn, and indeed to a number of the aetiological hypotheses concerning arousal and attentional defects. These descriptions and hypotheses are however largely inferential, and surprisingly little research has been conducted on autonomic responsivity. The present study was therefore designed to collect such empirical data on the autonomic responsivity of the autistic child.

Method

a) Subjects

A group of seven autistic children were selected for participation in the present study. The subjects had been diagnosed as autistic according to the criteria outlined in chapter 3 (pp.112-113). Thus all subjects displayed at least four of the Creak et al., (1961) 9 pointers, and these included those symptoms related to language deficiencies, obsessional and ritualistic behaviour, and social relatedness. In addition, the subjects used in the present study were selected because they were all effectively non-verbal. It was felt that autonomic data from such subjects may be especially enlightening in view of their complete failure to communicate verbally.

From the available sample of autistic children who displayed the required criteria of childhood autism, seven subjects were effectively mute and were therefore selected for participation in the present study. Technical difficulties, experienced during the recording of the telemetered heartrate, resulted in the discarding of the records of two subjects. For one, the heart rate record contained a very weak signal of insufficient strength for data analysis. This was attributed to a transmitter fault together with difficulty experienced in tuning-in the receiver to the telemetered signal. A second subject's record was spoilt by a failure in the FM magnetic tape recorder drive. Thus com-

plete records were obtained for five subjects.

b) Autonomic Measure

The chosen autonomic measure for this study was heart rate. This measure was favoured for two major reasons. First, the heart rate is comparatively speaking a technically simple autonomic measure to monitor. This is an important consideration, for there are many practical problems inherent in physiological measurement with autistic children (White, 1974). The limited cooperative behaviour, susceptibility to panic reactions, and rejection of much that is novel, make the autistic child a very difficult experimental subject. Second, the cardiac measures are free from many of the inferential problems associated with EEG and GSR records. Whilst there may be some difficulties associated with the interpretation of the psychological significance of heart rate changes, we at least may be more certain of what is actually being measured than with most other autonomic measures.

The specific cardiac index taken was the peak-to-valley (P-V), defined as the difference between the highest heart rate and lowest heart rate occurring during the specified baseline and the response periods. In this study, the peak-to-valley difference (P-V) was measured during the ten seconds immediately preceding the experimental stimulus (the baseline period), and the twenty seconds following the stimulus presentation (the response period). The mean heart rate was not selected as the experimental measure since it suffers from two clear deficiencies. The mean heart rate as a response measure does not take into account the fact that the heart rate response is typically not monophasic. There is however no general agreement on the exact shape of such a response (Johnson and Lubin, 1972). In addition to this limitation, because of individual differences in heart rate reactivity, comparison of absolute heart rate changes or means, is seldom a valid method

of assessing the effects of an experimental variable between subjects. Thus what for one subject is a high heart rate, might for another be a low one; and what for one subject is a small heart rate change, might for another be a large change. Brener (1967) suggests that it is better to look at sequential patterns of heart rate changes rather than overall means, and concludes that there is strong evidence that the most reliable measure of heart rate response is the peak-to-valley difference (P-V). Thus the peak-to-valley difference measure (P-V) was selected as a response measure in the present study.

c) Recording Technique

The method for recording heart rate from autistic children as unobtrusively as possible was considered to be radio telemetry. Where possible, an attempt was made to reduce the subject preparation time to a minimum in view of the highly limited tolerance of such activity by autistic children. The electrode placement sites were selected on the upper and lower regions of the sternum. An accurate recording of individual elements of electrical correlates of the cardiac cycle was not necessary for the present study. Therefore the above mentioned sites on the sternum were selected for ease of electrode placement and subsequent concealment under the subject's clothes. These selected electrode sites also minimized body movement EMG artefacts on the autonomic recordings, for few muscles traverse the sternum. Thus the frequency of the R-wave of the cardiac PQRS complex was the specific measure of heart rate selected.

The electrode sites for each child were acetone-prepared, and the skin was mildly abraded with electrode jelly. The electrodes were filled with Cambridge electrode jelly, and together with a transmitter, were taped to the chest using disposable adhesive discs and tape. The Devices 102F transmitter converted the cardiac electrical activity to a

signal received by a Nelson-Jones receiver-tuner. The tuner was coupled to an SE Laboratory 4-channel FM portable magnetic tape recorder. Thus the heart rate was directly recorded on one channel of a magnetic tape for subsequent analysis.

Where necessary, subjects were given several preparation rehearsal sessions to adapt to this procedure, since for some it initially proved to be an anxiety-provoking novel test procedure. Subjects did not take part in the study until they were sufficiently unconcerned with the preparation procedure, as judged from behavioural observations. After preparation, the electrodes and transmitter were covered with the subjects' clothes and thereafter they showed little awareness of them.

d) Procedure

The experimental room was partitioned into two halves. In the first of these E1 monitored the telemetered heart rate. In the second section the subject sat at a table, and E2 sat adjacent to this table. The subject was led into the experimental room and seated at a table. Whilst E2 interacted with him during a five-minute adaptation period, E1 conducted the fine tuning necessary to pick up the cardiac signal and to establish an acceptable recording of the heart rate. Recording times were kept as constant as possible to balance for possible changes over a 24 hour cycle.

The heart rate data was directly recorded on one channel of the FM tape recorder. On a second channel E1 placed a verbal commentary on the experimental procedure including the timing of stimulus presentation and any relevant behaviours (e.g., stereotypy).

The experimental design consisted of the programmed presentation of seven experimental conditions. Before each condition a ten second baseline period was recorded, for which comparative behavioural

quiescence was a prerequisite, and E2 avoided the possibility of any interaction with the subject by completely ignoring him. When an acceptable baseline period had been recorded (yielding an even physiological record), E1 signalled E2 to present the relevant stimulus and noted the stimulus presentation point verbally on the second channel of the tape. A twenty second post-stimulus period then followed during which E2 again avoided any interaction with the subject.

This procedure was repeated for all stimulus conditions and for each subject. Continuous heart rate recordings and commentary were made on the magnetic tape throughout, and at least twenty seconds were allowed to elapse between the end of a post-stimulus period and the next baseline. Heart rate data were also recorded during all periods of exhibited stereotypy, although where this occurred during an experimental stimulus presentation, that condition was aborted and subsequently repeated to prevent contamination of heart rate data with stereotyped behaviours. The seven stimulus conditions were as listed below:

- i. "Adult approach" E2 approached the subject, and in close proximity (within three feet), looked directly at him but did not verbally communicate.
- ii. "Adult verbal demand" - E2 commanded the subject to complete a simple task (e.g., "pick up this ball"). These commands had been previously established as within the subject's capabilities.
- iii. "Social reward" - E2 smiled and gave verbal praise.
- iv. "Primary reward" - E2 gave the subject a sweet.

- v. "Social punishment" - E2 verbally reprimanded the subject in this condition, and wherever possible this was delivered contingent upon some non-desired behaviour (e.g., child attempts to get up from table).
- vi. "Unfamiliar stimulus" - E2 presented the subject with a novel stimulus, consisting of a toy completely novel to him (ascertained from teacher and parent reports).
- vii. "Familiar stimulus" - E2 presented the subject with a toy that he had frequently played with.

An eighth condition ("primary punishment") was removed from the design when it became clear that it was too stressful for subjects. Each subject received each of the seven stimulus conditions a total of five times during the course of the experiment, making a total of 35 separate stimulus presentations per subject. The conditions were presented to each subject in the same sequence, although the sequence was rotated such that each subject had different starting points in this sequence. However, there was an additional constraint upon the presentation order such that instances of familiar and unfamiliar stimuli were not consecutively presented, and the same applied to conditions of primary and social reward. This was an attempt to control for possible habituation to reward and stimulus materials.

Results

The heart rate data was recorded on magnetic tape, together with the verbal commentary delineating baseline, stimulus, and response periods. This data was subsequently fed directly into a PDP8 Lab 8/E computer, and analysed with programme H.R.ANALYZER. This programme

was individually written for the current data, enabling the identification of the magnitude of each inter-beat interval (IBI) over the experimental conditions. For each condition H.R.ANALYZER gave a print-out of the IBIs for the 10 second baseline period, identification of the point of stimulus presentation, and the IBIs during the 20 second post-stimulus (response) period. The programme also calculated maximum and minimum IBI values and the mean heart rate for both baseline and response periods. A copy of this programme is shown in appendix B.

Having identified the baseline and heart rate response inter-beat interval values for each subject in each condition, it was then considered necessary to establish whether any significant dependency existed between initial baseline levels of heart rate and the response levels. This dependency variable, known as the Law of Initial Values (LIV), is clearly important and although outlined several years ago (Wilder, 1956), is quite frequently uncontrolled in psychophysiological studies. Wilder stated that the magnitude of a response to an external stimulus is related to the prestimulus level. Benjamin (1967) formulated the resultant problem for autonomic data interpretation thus: if group or individual A has a prestimulus level different from B, and the response to a stimulus is dependent on the initial level, how can one compare the response to A with the response to B independently of the differing initial levels?

Several workers have suggested that analysis of covariance (anacova), rather than analysis of variance, must be used on such data (e.g., Lacey, 1956; Benjamin, 1963). But first it is necessary to assess the degree of linear dependency between initial and response levels, before the relevant statistical analysis is chosen. Benjamin (1963, 1967) proposed that the criterion for a score free of initial level should be that the score has no correlation with initial level. Therefore the data from the present study was exposed to a Pearson

Product-Moment correlation analysis. If the prestimulus (baseline) heart rate is called X, and the poststimulus P-V (the response) is called Y, then the relevant correlation for detecting the relevance of LIV to the present data is given by r_{XY} . Correlation coefficients for each subject and for each condition are shown in table 2A. It can be seen that the correlation values did not reach significance levels, and it was therefore concluded that a significant linear dependency between initial and response levels did not exist in the current data, and statistical manipulation to 'free' such a dependency was thus considered unnecessary.

The second stage in the analysis of results consisted in establishing which of the experimental conditions produced significant heart rate responses. For each condition, analyses were conducted on the prestimulus (baseline) P-V valley measures and the poststimulus P-V values, using 't'-tests (for related samples) on differences between these scores ('d' scores). These results are shown in table 2B, where it can be seen that all seven conditions produced a significant heart rate response as defined by significant differences between baseline and poststimulus P-V scores.

Programme H.R.ANALYZER (display) enabled a visual display of the heart rate for any condition to be presented. A selection of these displays were photographed, and examples are shown in appendix C.

Heart rate recordings during periods of behavioural stereotypy were unfortunately almost all impossible to analyze, since the concurrent body movements yielded 'noise' levels that prevented any accurate separation of the heart rate signal.

The relative magnitudes of the heart rate responses were then analysed. Specific comparisons were of particular interest. These consisted of the responses to familiar stimuli compared with unfamiliar stimuli, primary reward compared with social reward, primary reward and

Measures For	Mean Base- line H.R. in b.p.m. X	Mean H.R. response (P-V)IBI in m.secs Y	correl. coeff. rXY	df	sig.
subject 1	87.5	203.3	-0.28	34	ns
subject 2	103.5	121.9	+0.18	34	ns
subject 3	90.6	289.3	-0.31	34	ns
subject 4	96.4	223.7	-0.22	34	ns
subject 5	87.4	196.4	+0.28	34	ns
condition 1	94.3	214.5	-.026	34	ns
condition 2	92.5	206.0	-0.14	34	ns
condition 3	86.9	222.8	-0.23	34	ns
condition 4	94.5	208.3	-.01	34	ns
condition 5	94.1	199.0	-0.25	34	ns
condition 6	95.9	208.0	-0.24	34	ns
condition 7	94.2	189.0	-0.37	34	ns

Table 2A: Correlations between mean baseline heart rate and heart rate response P-V scores

Condition	Mean Baseline P-V	Mean Response P-V	'd'	't'	df	signif.
condition 1	154.0	214.5	60.5	4.05	24	0.1%
condition 2	145.8	206.0	60.2	3.81	24	0.1%
condition 3	174.8	222.8	48.0	2.37	24	5%
condition 4	151.4	208.4	57.0	3.35	24	1%
condition 5	146.0	199.0	53.0	2.96	24	1%
condition 6	136.8	208.8	72.0	5.95	24	0.1%
condition 7	142.0	189.0	47.0	3.22	24	1%

Table 2B: Comparison of baseline and response measures

social punishment, and conditions involving social stimulation ("verbal demand" and "adult approach") in contrast with non-social stimuli ("familiar stimuli" and "unfamiliar stimuli"). These comparisons were conducted using 't'-tests for related samples on the P-V response measures, and the results are shown in table 2C.

None of the comparisons reached significance levels however, and indeed a condition x order x subjects analysis of variance yielded an insignificant value for the "conditions" term (see table 2D). The order of presentation for the five repetitions of each condition also had no significant effect upon the relative magnitude of the heart rate response.

Thus whilst no statistical differences appeared between the heart rate responses in the different treatment conditions, inspection of individual and mean scores suggested that novel stimuli produced a larger mean response than familiar stimuli, (mean IBI's: 208.8 : 189.0), 'social' stimuli resulted in a larger mean heart rate response than non-social stimuli (mean IBI's: 210.3: 198.9) and social reward a larger response than primary reward (mean IBI's: 222.8 : 208.4). However, it must be emphasised that these were only trends, and were not statistically significant.

Discussion

The results from the present study suggested that autistic children responded markedly to each of the separate experimental conditions presented to them. The mean response magnitude (mean P-V value) for all conditions consisted of a heart rate variance 38% above the mean baseline variance, and all individual conditions produced significant responses. It may therefore be concluded that the autistic subjects in this study perceived and responded to each of the varieties of stimuli and reinforcers presented to them.

Comparison	Mean H.R.R. (P-V) IBI in m.secs.	Mean H.R.R. (P-V) IBI in m.secs.	't'	df	sig.
"Novel" Vs "familiar" stimulus	208.8	189.0	1.10	24	ns
"Social" Vs "Primary" reward	222.8	208.4	0.65	24	ns
"Primary reward" Vs "Social punishment"	208.4	199.0	0.67	24	ns
"Adult approach" Vs "Novel stimulus"	214.5	208.8	0.37	24	ns
"Adult approach Vs "Familiar stimulus"	214.5	189.0	1.32	24	ns
"Verbal demand" Vs "Novel stimulus"	206.0	208.8	0.15	24	ns
"Verbal demand"Vs "Familiar stimulus"	206.0	189.0	0.91	24	ns

Table 2C: Comparison of relative magnitudes of heart rate responses

Source	df	SS	MS	F	P
Order	4	15351.01	3837.75	0.51	ns
Conditions	6	17501.51	2916.92	0.68	ns
Subjects	4	495927.58	123981.89	27.99	0.1%
Order x subjects	16	119758.88	7484.93		
Conditions x subjects	24	102289.46	4262.06		
Order x conditions	24	106276.03	4428.17		
Order x conditions x subjects	96	406899.28	4238.53		
Total	174	1264003.75			

Table 2D: Analysis of variance for cardiac responses (P-V)
to experimental conditions

During the course of the present study it was noted that the autistic subjects displayed minimal overt behavioural signs of interest, surprise, recognition, or other signs of responding. They typically fitted a common clinical description of indifference, aloofness, and behavioural unresponsivity to many environmental events. However, the autonomic data suggested that quite clearly such inferences from behavioural descriptions may be misleading. Whilst autistic children may have failed to develop or learn the social conventions of verbal, facial, and behavioural expression, autonomic responses are essentially involuntary and therefore potentially most enlightening with these children.

The cardiac response to conditions of both social reward and social punishment suggest that autistic children have not totally failed to learn secondary reinforcement, for they induced significant autonomic responses. If traditional secondary rewards are not 'meaningless' or neutral stimuli for the autistic child, it is suggested that their ineffectiveness in terms of behavioural control may be a function of the inappropriateness, rather than the absence, of values that they have acquired for autistic children. The results of the present study suggest that this is indeed the more plausible explanation of secondary reinforcement control in these children.

Statistical analysis of the relative magnitude of the cardiac responses failed to differentiate between the seven experimental conditions under study. There appear to be three possibilities resulting from this negative finding. First, it is possible that the conditions did in fact produce differing autonomic responses, but the response index selected (the P-V difference) did not reflect these differences. Thus for example, the responses may not have differed in absolute magnitude but may have differed in their time course and their shape. Inspection of randomly selected IBI plots for the conditions did not however suggest that responses to some conditions were consist-

ently monophasic whilst others were biphasic. In fact the sample selected suggested that all conditions tended to yield a biphasic response over time.

A second hypothesis that may be considered is that indeed the conditions did not produce differential responses, and that autistic children responded throughout in a uniform manner, perhaps responding to stimulation per se rather than responding differentially to the precise nature of the stimuli used in this study.

A final consideration is that wide individual differences appeared in the response magnitude to the experimental conditions, and in the light of the small subject sample under study, such individual differences may have effectively 'masked' underlying differential responding.

It is unfortunately not possible to accept or reject conclusively any of these three hypotheses. The inspection of a sample of IBI plots makes the first an unlikely possibility, and it may also be noted that for all but one subject the response data showed two clear differential trends: subjects displayed a larger cardiac response to novel stimuli compared with familiar stimuli, and larger mean responses to 'social' stimuli in contrast to non-social conditions.

If the autistic child is subject to transient excitatory and inhibitory physiological states, resulting in a clinical state of perceptual inconstancy, certain predictions may be made with regard to the heart rate response to the experimental conditions utilised in this study. During inhibitory states many environmental stimuli will fail to impinge upon the autistic child, and therefore during excitatory periods the child may fail to recognise stimuli that appeared during inhibitory states. The perceptual inconstancy model thus predicts that there will not be consistently larger heart rate responses to novel stimuli than to familiar stimuli, for the autistic child will have

difficulty in recognising those features in the environment that are familiar. There will be difficulties in associating events that are temporally related, and recognition failures will also impede the process of secondary reinforcement control. Social rewards, which become reinforcing through temporal association with primary rewards, will therefore have little value for the autistic child, and will produce smaller heart rate responses than primary rewards. The perceptual inconstancy model does not predict that social stimuli will produce larger heart rate responses than non-social stimuli. Similar predictions concerning the heart rate responses to the stimuli used in this study may be made from an under-arousal model of childhood autism.

In contrast to predictions from these models an over-arousal hypothesis states that autistic children are extremely sensitive to stimulation levels since they increase arousal levels beyond an acceptable threshold. Social stimuli appear to be especially potent stimuli for autistic children (Hutt and Vaizey, 1966), and this may be predicted to result in larger heart rate responses to social stimuli and social reward. Novel stimuli may similarly be predicted to have high arousal value and thus produce heart rate responses larger than that of familiar stimuli. The data from the present study is therefore most consistent with an over-arousal model of childhood autism when considering current physiological hypotheses.

It may be stated in conclusion therefore, that whilst statistically significant differential responses to the experimental conditions were not found, some interesting trends and significant absolute responses to the conditions were found. These trends were most consistent with an over-arousal model of childhood autism. Further research using autonomic indices may yield other important data on the responsivity of the autistic child to his environment.

CHAPTER FIVE:

BINARY SEQUENCE LEARNING WITH NORMAL AND AUTISTIC CHILDREN

Introduction

A number of interesting studies have yielded data that support a model of childhood autism as a disorder of cognitive functioning (Frith, 1968; Hermelin, 1972; Hermelin and Frith, 1971). Deficits in the information processing of the sensory input are consistent with several of the characteristic behaviours of the autistic child, including stereotyped mannerisms, resistance to environmental change, and the failure to acquire social and linguistic skills. Both stereotypy and a resistance to change may be seen as attempts by the autistic child to impose internally generated rules upon the environment, a mechanism for 'making sense' of a world in which the 'normal' environmental stimuli hold no meaning for the autistic child. Failures to develop linguistic and social skills are seen as a result of the inability to extract explicit rules from the environment, and to generalise and use these rules in a flexible fashion (Frith, 1972).

On the basis of errors made during the learning of binary sequences, Frith (1968, 1970a, 1970b) claimed that autistic children may be characterised by an inability to "feature extract" from patterned input (i.e., a failure to reflect the dominant features of the presented pattern in the errors made). She also suggested that they have a consistent tendency to utilise their own input-independent, internal 'rules' (a mechanism of "pattern imposition"). It is suggested that this behaviour is independent of modality, and is therefore a function of a broad cognitive defect.

Most of the errors made by autistic children whilst learning binary sequences, were due to the imposition of simple perseveration or alternation strings, independent of the given patterns. When spontaneous sequence production was studied, it was found that similar imposition tendencies were displayed by normal children as well as autistic children, although the "pattern imposition" tended to be more rigid with autistic subjects. It was concluded that autistic children are

insensitive to differences in the structures presented, and tend to impose their own stereotyped patterns whilst normal children only do so in the absence of structured sensory input (Frith, 1968, 1970a, 1970b; Hermelin and Frith, 1971).

This research has been reviewed at an earlier point (chapter 1, pp.81-87) where it was also noted that the cognitive dysfunction model does have certain limitations. If the autistic child does indeed treat the world as if it were an environment consisting of random unstructured stimuli, thereby imposing stereotyped patterns upon it and failing to "extract" salient features from it, it does not logically follow that the autistic child will display bizarre attachments or avoidance of eye contact and social stimuli (e.g., Hutt and Vaizey, 1966). A cognitive defect theory is not consistent with findings of strong negativistic behaviour and the importance of motivational variables on the performance of autistic children on experimental learning tasks (Cowan, Hoddinott and Wright, 1965; Wallace, 1975). Performance on structured learning tasks at less than chance level, and response patterns dependent upon the precise nature of the stimulus materials used, are not findings that are predictable from a deficiency in cognitive functioning.

Nevertheless, autistic children clearly display marked perceptual difficulties (Hermelin and O'Connor, 1970), language retardation and deviance (Bartak, Rutter and Cox, 1975), and pronounced problems of learning (Lovaas and Schreibman, 1971; Stevens-Long and Lovaas, 1974). Whether these are a function of a broad abnormality in cognitive functioning or are seen as specific learning difficulties (perhaps this distinction is one of terminology rather than semantics or concept), experimental investigation of the characteristic behaviour of autistic children on performance tasks is a highly relevant and important area for study. Both Bryson (1970) and Kovattana and Kraemer (1974) place particular emphasis on how the most effective methods of treatment and education

can only be designed when we fully understand the ability of autistic children to process incoming stimuli, and have identified patterns of cognitive, perceptual, and linguistic dysfunction. The present study attempted further investigation of the proposed cognitive defects.

Pattern perception is a process directly related to almost all cognitive activity, since reducing the size of the sensory input by coding techniques is the only way of perceiving such large inputs. Binary sequences have the advantage of being abstract, yet unambiguous patterns, and are therefore suitable for use in the study of cognitive behaviour. An analysis of the errors made during binary sequence learning was conducted, as used by Frith, but in addition a number of other features were also studied.

Frith (1968, 1970b) concluded from her data collected on the errors made by autistic and normal children during colour sequence learning, that the failure to feature extract and the imposition of patterns displayed by autistic children, is representative of other information processing in the visual modality. Whilst this may be so, it seems important to be cautious in extrapolating from behaviour on one visual dimension and one type of learning task, to assertions concerning a generalised defect in the modality. It may be that the failure to feature extract and the display of pattern imposition tendencies is related to the nature of the stimulus materials used. There is evidence that different performance results from the use of differing stimulus materials on certain discrimination tasks (e.g., Cowan, Hoddinott and Wright, 1965; Serafica, 1971; Morrison, Miller and Mejia, 1971). In the present study, a comparative analysis was conducted using normal and autistic children on binary colour sequence learning and also shape sequence learning. A central cognitive defect model predicts that similar error types will be found in performance on these two dimensions, for it implicates not simply an overall abnormality in structure of

sensory channels, but rather the availability of codes for organisation of the sensory input, which is independent of the modality in which the stimuli are presented (Hermelin, 1972). The binary sequences used in the current study contained 4 units, and each sequence consisted of 3 units of one colour/shape (labelled 0's) and one unit of another (labelled 1). Thus sequence 0100 might consist of the pattern red(0)-blue(1)-red(0)-red(0), or square (0)-circle(1)-square(0)-square(0). The subject had no way of knowing which colour/shape in the sequence would correspond to "1" or which to "0", until the first sequence unit was placed in front of him.

The present study also considered the possibility that these binary sequence learning tasks produce error data that may be classified into types other than those simply related to the maintenance of the dominant feature of the pattern and the imposition of internal "rules". Indeed, it is proposed that the latter system of classifying errors is not unequivocal. It is assumed by Frith that the dominant feature of patterns 0100 and 0010 is "alternation", and of patterns 1000 and 0001 "perseveration". It may be however, that subjects recall such sequences correctly by first learning the correct ratio of the elements in it, and then the position of the element that characterises each sequence (the single colour, labelled "1" in the above pattern examples).

Therefore it is suggested that subjects may make errors on the basis of errors in positioning and also errors in the ratio of the component elements. For example, if a subject produces a sequence 0001 whilst attempting to learn the sequence 0010, Frith concludes that such an error is due to the application of a perseveration rule, whereas the dominant feature of the pattern is alternation. It is concluded that the subject has imposed his own 'rule' upon the sequence. Thus he is said to have failed to feature extract. If however, one classifies errors according to whether they maintain the correct ratio

of the two components of the sequences (and are therefore errors of position), or whether they fail to display the correct ratio (and are therefore errors of omission or commission of one of the sequence elements), then inferences made on the basis of errors produced are somewhat different.

With reference to the example cited earlier, the error 0001 (in response to the pattern 0010) may be seen as an illustration of a position error. The subject has however recalled the correct ratio of the elements in the sequence (i.e., three elements of one colour, and one of the other), but has made an error in the relative positioning of them. It may be suggested at this point that in fact such a response sequence does reflect the dominant feature of the pattern, for the latter consists of the feature or 'rule' "three of one colour, one of another".

Similar discrepancies exist between inferences concerning feature extraction and pattern imposition when analysing other errors committed by subjects in terms of perseveration and alternation rules, or in terms of ratio and position rule learning. Thus in the present study, in addition to analysing error data according to Frith's concept of feature extraction and pattern imposition, error data was also classified into 'ratio errors' and 'position errors'.

A final consideration in the present study concerned experimental control designed to eliminate the possibility of learning through short-term visual memory. This is most important when investigating cognitive behaviour in contrast to perception. Frith (1968, 1970b) ensured that the pattern learning tasks in her studies were not dependent on immediate visual matching, by covering the completed pattern at the end of each trial so that direct copying was impossible. However, she kept constant the two colours used throughout the learning

trials administered for each pattern. The order of the two colours was reversed only in successive patterns, and not in the trials within any one pattern. It is therefore conceivable that subjects were able to utilise visual short-term memory to produce correct sequences. The experimental task may therefore have been one involving delayed visual sequencing. Whilst this yields more information on specific perceptual performance rather than broad cognitive functions, the situation is further complicated by our knowledge of specific perceptual deficiencies in autistic children. Bryson (1970) conducted a study with autistic children that involved visual-visual and auditory-visual matching and sequencing tasks, under conditions of simultaneous, successive, and delayed presentation. She concluded that autistic children display severe cross-modal coding deficits, but also particular deficiencies in short-term visual memory. She remarked that for these children, the situation may be quite literally "out of sight, out of mind". This implies that any visual matching or sequencing tasks involving delayed presentation may be especially difficult for autistic children.

Whilst it is noted that no overall performance differences (in terms of percentage of correct responses) existed between autistic and normal children in the pattern learning tasks reported by Frith, it is suggested that the methodology used makes exact definition of the task presented to subjects rather problematical. Specifically, it is suggested that the binary sequence learning tasks (as presented to subjects by Frith), were delayed visual sequencing tasks, dependent upon visual short-term memory and therefore perceptual abilities rather than, or in addition to, central cognitive functioning. For autistic children, who appear to have marked deficiencies in visual short-term memory (Bryson, 1970), the tasks may also have been especially penalising. In this context interpretation of differential performance between

autistic and normal children in terms of direct cognitive defects, may therefore be of questionable validity.

In the present study, short-term visual memory effects were properly controlled through the use of several pairs of colours in the trials within each pattern. This enforced learning of the dominant feature or rule of each pattern for successful performance, and was therefore more likely to be a cognitive task.

Method

a) Subjects

Eight autistic children took part in this study, together with eight control normal children selected from a sample of children attending a local infant school. The autistic children fitted the criteria described in chapter 3 (p.112-113) displaying severe difficulties in social relationships and communication, obsessional or ritualistic behaviour, and profound retardation and abnormalities of language. Five of the autistic children did display limited verbal ability. The EPVT (English Picture Vocabulary Test) was administered to both groups, and was selected for its suitability of administration for autistic children (who have short attention spans and are non-verbal in many cases). The mean chronological age of the autistic children was 8.8 years (range 5.6 - 11.10), with a mean mental age equivalent score on the EPVT of 5.1 years. The normal children had a mean chronological age of 5.4 years (range 5.00 - 5.7) and scores yielding a mean mental age equivalent of 5.7 years. The autistic and control group scores were thus similar on the EPVT, although it was recognised that this was only an approximation to the strict mental age matching one would conduct with other types of experimental groups.

b) Procedure

Sessions were arranged in pairs such that each child took part in two sessions, separated by at least one day from each other. The first experimental session commenced with an elementary sorting task to demonstrate that each subject was able to discriminate between the four colours used in the pattern learning tasks (red, blue, yellow, brown). Each subject was asked to sort coloured counters into separate boxes. Having successfully discriminated between the colours, the subject was then presented with a rectangular tray partitioned into 12 sections. He was also presented with two piles of coloured counters, and asked to place one counter into each section to make a row of twelve counters. The subject was simply told to: "make a pretty pattern, using any of the counters you wish". The two colours selected for this spontaneous sequence production were rotated from one subject to the next, and balanced for autistic and control groups.

Each subject was then presented with another tray, of smaller dimensions ($8'' \times 1\frac{1}{2}'' \times \frac{1}{4}''$ deep) and partitioned into 4 sections. The binary pattern learning tasks now began, there being a total of four patterns to be learnt which were as follows: 1000, 0100, 0010, 0001. The order of the patterns to be learnt was rotated across subjects, and balanced for the experimental and control groups.

The subject was instructed to place the coloured counters one by one in the tray, so as to make a row of 4 counters. The experimenter always selected the first counter to start each trial, and the subject selected the other three. When the subject selected an incorrect colour, that counter was removed and the subject was told to select the other colour. All responses were noted. For each trial, a new combination of two sets of coloured counters were used (the remaining two sets being removed for the duration of the trial), and there were a total of twelve counters of each colour. The order of the two colours

was thus rotated from trial to trial, but the rotation order was kept constant for all subjects. Because each trial involved the use of a new pair of colours, the experimenter always selected the first counter as a cue for the subject. For example, if on one trial the correct sequence was "red, red, blue, red" and on the next trial the subject was given red and brown counters, a knowledge of the colour of the first member of the sequence would be necessary to distinguish between the required sequence "brown, brown, red, brown" and the non-required "red, red, brown, red".

At the end of each trial (always consisting of four colours in a row), the counters were immediately removed to prevent any immediate visual matching on subsequent trials, and the rotation of the pairs of colours after each trial was considered to be a good control for the effects of visual short-term memory. Subjects were required to complete the same pattern on each trial until a total of nine consecutive trials were completed for each pattern, or the subject had reached a learning criterion. This criterion consisted of three consecutive error-free trials. Short breaks were held between patterns, to emphasise the end of trials with that particular pattern.

This procedure was repeated in a second session, where coloured counters were substituted with wooden shapes. There were four types of shape (triangle, square, circle, star), of a size very similar to that of the counters, but the shapes did not vary in colour. Shape discrimination tests were conducted and spontaneous sequences were recorded. The same four binary patterns used with the colour sequences were also to be learnt with the shapes. Identical methods were adopted to rotate paired combinations of shapes from trial to trial.

Experimental and control groups were each split into two halves, one half completing colour pattern learning before shape

sequences and the other half the reverse order. This procedure controlled for any overall influence upon results of transfer of learning from trials utilising one dimension to trials on the other dimension. In addition to this control, colour and shape pattern learning sessions were separated by at least 24 hours.

Results

a) Performance and Learning

An initial analysis was conducted to assess the overall level of performance of autistic and normal children, and to demonstrate significant pattern learning. All subjects selected three colours/shapes per trial, and were therefore capable of making a maximum of 27 errors per pattern and a total of 216 errors on all shape and colour patterns. The total number of actual errors made was expressed as a function of the total number of possible errors, and this demonstrated that autistic children made 78% correct responses over all patterns and normal children 86%.

To assess whether significant pattern learning occurred, mean error scores in the first three trials of each pattern were compared with scores on the last three trials of each pattern. The means of these scores are shown in Table 3A. A Groups x Patterns x Dimensions x Learning analysis of variance was conducted (Table 3B), and this confirmed that both groups demonstrated highly significant learning over trials ($F=240.17$; $df=1,14$; sig. 0.1% level). In all patterns, errors decreased markedly in the later trials. However, autistic and normal children differed in the mean number of errors produced. Despite the initial matching of experimental and control groups on the EPVT, normal children produced significantly fewer errors ($F=5.61$; $df=1,14$; sig. 5% level). For subsequent analyses of error 'type', it was therefore considered necessary to contrast

Table 3A: Errors in first 3 and last 3 trials in each pattern
(colour and shape patterns)

(max. errors = 3 per trial)

Patterns	1000		0100		0010		0001	
Trials	1-3	7-9	1-3	7-9	1-3	7-9	1-3	7-9
Autistics								
\bar{X}	3.5	1.1	3.4	1.6	3.9	1.3	2.8	0.5
Normals								
\bar{X}	3.3	0.4	3.6	0.6	2.6	0.0	1.9	0.0

TABLE 3B: Learning Scores Analysis of Variance

Source	df	SS	MS	F	p
Groups	1	32.35	32.35	5.61	5%
SWG	14	80.74	5.77		
Patterns	3	35.92	11.97	4.13	2 $\frac{1}{2}$ %
Groups x Patterns	3	8.54	2.85	0.98	ns
SWG x Patterns	42	121.85	2.90		
Dimensions	1	0.47	0.47	0.30	ns
Groups x Dimensions	1	0.19	0.19	0.12	ns
SWG x Dimensions	14	22.02	1.57		
Patterns x Dimensions	3	2.29	0.76	0.49	ns
Groups x Patterns x Dimensions	3	0.45	0.15	0.10	ns
Learning	1	377.82	377.82	240.17	0.1%
Groups x Learning	1	1.41	1.41	0.90	ns
Patterns x Learning	3	3.64	1.21	0.77	ns
Dimensions x Learning	1	2.44	2.44	1.55	ns
Groups x Patterns x Learning	3	5.54	1.85	1.17	ns
Patterns x Dimensions x Learning	3	5.51	1.84	1.17	ns
Groups x Dimensions x Learning	1	0.88	0.88	0.56	ns
Groups x Patterns x Dimensions x Learning	3	6.32	2.11	1.34	ns
Total	255	928.15			

autistic and normal children on percentage scores (as a function of total errors made by the group) rather than on absolute error scores.

There were no significant differences in the total error scores on the two dimensions of shape and colour, and learning did not differ significantly between shape and colour patterns. The Patterns term was significant however ($F=4.13$; $df=3,42$; sig. $2\frac{1}{2}\%$ level).

A posteriori comparisons were made using Scheffe contrast analyses. This is a conservative test, and is more rigorous than other multiple comparison methods with regard to Type 1 errors (Ferguson, 1966; pp.296-297). The Scheffe analyses showed that both groups produced fewer errors on pattern 0001 than on the other patterns ($F=2.88$; sig. 5% level). The groups x patterns interaction term was insignificant, and thus whilst normal children produced fewer errors in total than autistic children, the patterns were of the same relative difficulties for the groups.

b) Error 'type' analyses

Error 'types' were first scored according to the methods outlined by Frith (1968, 1970b), who analysed errors made during binary sequence learning in terms of errors reflecting the dominant input and those reflecting the imposition of patterns. She claimed that patterns 1000 and 0001 contained the dominant feature "perseveration", and patterns 0100 and 0010 "alternation". She also proposed that feature extraction and pattern imposition tendencies could only be unequivocally inferred from errors made in the third and fourth positions of any trial. Errors occurring in the first trial of any pattern were discarded as the required pattern had not been seen by the subject at this point, and such errors may be considered to be a function of guessing or preferred sequences. The remaining errors were then summed and expressed as a

percentage of total errors committed.

To study feature extraction, the error types shown in Table 3C were selected. Frith proposed that these errors have the property of producing strict alternation or perseveration strings, and only these four types are sufficiently unambiguous for studying feature extraction. Two of these error types are seen as a result of feature extraction and two are not; whilst two are perseveration errors and two due to alternation. Table 3C shows the mean percentage of these four error types expressed as a percentage of total errors committed.

A Group x Feature Extraction x Error (alternation or perseveration) x Dimension analysis of variance was conducted on these data (see Table 3D). The groups did not differ in terms of feature extraction errors, but the feature extraction term itself was significant ($F=7.57$; $df=1,14$; sig. $2\frac{1}{2}\%$ level). Both autistic and normal children produced more errors of the type that did not display feature extraction, and this was particularly marked with the normal children. There was a significant interaction between error type and task dimension ($F=5.36$; $df=1,14$; sig. 5% level). With shape patterns there were significantly more errors resulting in alternation strings in contrast to perseveration strings. With colour patterns this difference did not exist (Figure 3/i).

To study pattern imposition processes, the four error types shown in Table 3E were selected, as Frith considers that they are the only ones sufficiently unambiguous and not overlapping with feature extraction errors. Two of these errors display pattern imposition and two do not. A Groups x Pattern Imposition x Error x Dimension analysis of variance was conducted on this error data (see Table 3F). The pattern imposition term was significant ($F=12.57$; $df=1,14$; sig. 1% level) and this demonstrated that, for both groups, there was a higher percentage of errors that resulted from pattern imposition than from

Table 3C: Feature Extraction: Means of Error percentages

Pattern:	0001	0100	0010	1000
Response: (error)	0000	0101	000-	101-
Error due to:	Persev.	Altern.	Persev.	Altern.
Dominant Feature of Pattern	Persev.	Altern.	Altern.	Persev.
Feature Extraction	+	+	-	-
Autistic Children	16.67%	17.26%	18.04%	25.47%
Normal Children	0.00%	20.36%	15.63%	45.11%

Figure 3/i: Error Types x Dimension (Both Groups)

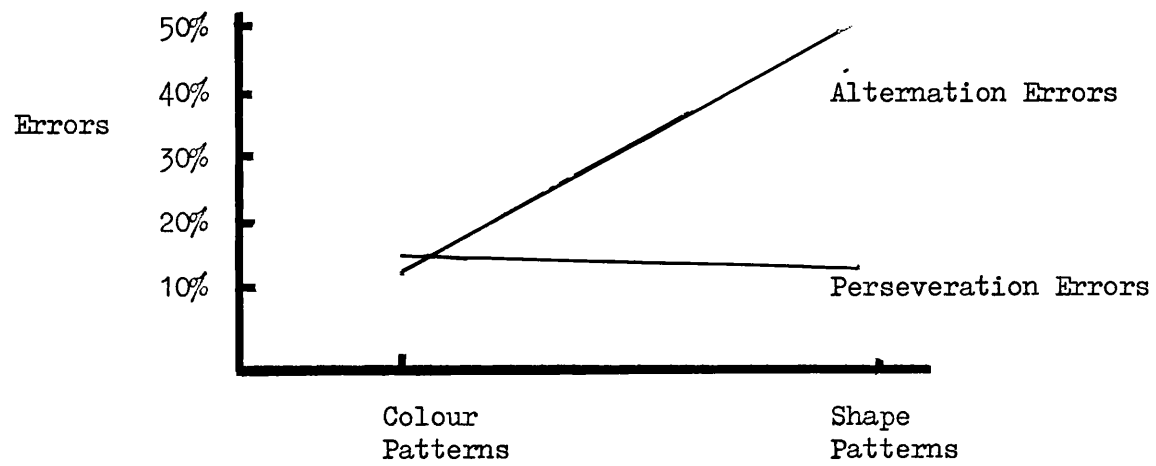


Table 3D: Feature Extraction Analysis of Variance:
Groups x Feature Extraction x Error x Dimension

Source	df	SS	MS	F	p
Groups	1	26.64	26.64	0.02	ns
SWG	14	21742.43	1553.03		
F.E.	1	4990.00	4990.00	7.57	2 $\frac{1}{2}$ %
Groups x F.E.	1	1895.74	1895.74	2.88	ns
SWG x F.E.	14	9228.60	659.19		
Error	1	6696.14	6696.14	4.43	ns
Groups x Error	1	3498.66	3498.66	2.32	ns
SWG x Error	14	21153.20	1510.94		
F.E. x Error	1	508.81	508.81	0.34	ns
Groups x F.E. x Error	1	10.47	10.47	0.01	ns
Dimension	1	4743.38	4743.38	3.14	ns
Groups x Dimension	1	0.05	0.05	0.00	ns
F.E. x Dimension	1	1421.78	1421.78	0.94	ns
Error x Dimension	1	8102.65	8102.65	5.36	5%
Groups x F.E. x Dimension	1	115.52	115.52	0.08	ns
F.E. x Error x Dimension	1	1243.76	1243.76	0.82	ns
Groups x Error x Dimension	1	10.47	10.47	0.01	ns
Groups x F.E. x Error x Dimension	1	87.78	87.78	0.06	ns
Total	127	150023.52			

Table 3E: Means of Four Error Types Demonstrating Pattern Imposition

Pattern:	1000	0010	10000	0010
Dominant feature:	Persev.	Altern.	Persev.	Altern.
Response (error)	101-	000-	1001	0011
Error due to:	Altern.	Persev.	?	?
Pattern Imposition	+	+	-	-
Autists	25.77%	18.04%	13.87%	14.10%
Normals	45.11%	15.63%	8.33%	5.2%

Table 3F: Analysis of Variance for Pattern Imposition Data:
Groups x Pattern Imposition x Error x Dimension

Source	df	SS	MS	F	p
Groups	1	12.38	12.38	0.01	ns
SWG	14	17457.46	1246.96		
P.I.	1	7947.45	7947.45	12.57	1%
Groups x Pattern Imposition	1	1965.65	1965.65	3.11	ns
SWG x P.I.	14	8848.9	632.06		
Error	1	3216.02	3216.02	4.36	ns
Groups x Error	1	1261.28	1261.28	1.71	ns
SWG x Error	14	10319.43	737.10		
P.I. x Error	1	2354.70	2354.70	3.19	ns
Groups x P.I. x Error	1	677.12	677.12	0.92	ns
Dimension	1	1504.26	1504.26	2.04	ns
Groups x Dimension	1	30.23	30.23	0.04	ns
P.I. x Dimension	1	4481.68	4481.68	6.08	5%
Error x Dimension	1	5745.92	5745.92	7.80	2 $\frac{1}{2}$ %
Groups x P.I. x Dimension	1	17.40	17.40	0.02	ns
P.I. x Error x Dimension	1	2365.00	2365.00	3.21	ns
Groups x Error x Dimension	1	358.45	358.45	0.49	ns
Groups x P.I. x Error x Dimension	1	671.61	671.61	0.91	ns
Total	127	132075.11			

error types. The groups x pattern imposition term did not reach significance level ($F=3.11$), but it was the normal children who displayed a higher percentage of pattern imposition errors in their responses (see Figure 3/ii).

There was a significant interaction between pattern imposition and dimension ($F=6.08$; $df=1,14$; sig. 5% level). Pattern imposition was displayed more frequently with patterns on the shape dimension than with colour (Figure 3/iii).

Errors were also classified into those types consisting of errors of position (but maintaining the correct ratio of the elements in the pattern), and those errors in which the correct component ratio was not present. A Groups x Error type (ratio error/position error) x Pattern x Dimension analysis of variance was then conducted on these data (see Table 3G). Both autistic and normal groups made more 'ratio errors' than 'position errors' ($F=13.73$; $df=1,14$; sig. 1% level). The Patterns term was significant ($F=5.29$; $df=3,42$; sig. 1% level), and a Scheffe Contrast analysis showed that both groups produced fewer ratio and position errors on pattern 0001 ($F=3.18$; sig. 5% level). This is in agreement with the earlier finding that both groups produced fewer errors in total, of all types, on pattern 0001.

c) Spontaneous Sequence Production

The spontaneous sequences produced with the coloured counters and shapes were analysed in two ways. First, they were classified into those with a consistent rule throughout (e.g., a strict alternation sequence), in contrast to irregular sequences. Second, spontaneous sequences were categorised into those displaying a predominantly alternation string and those with predominantly perseveration strings. The criterion for a sequence to be classified as a predominantly alternation/perseveration sequence was that at least 50% of the pattern

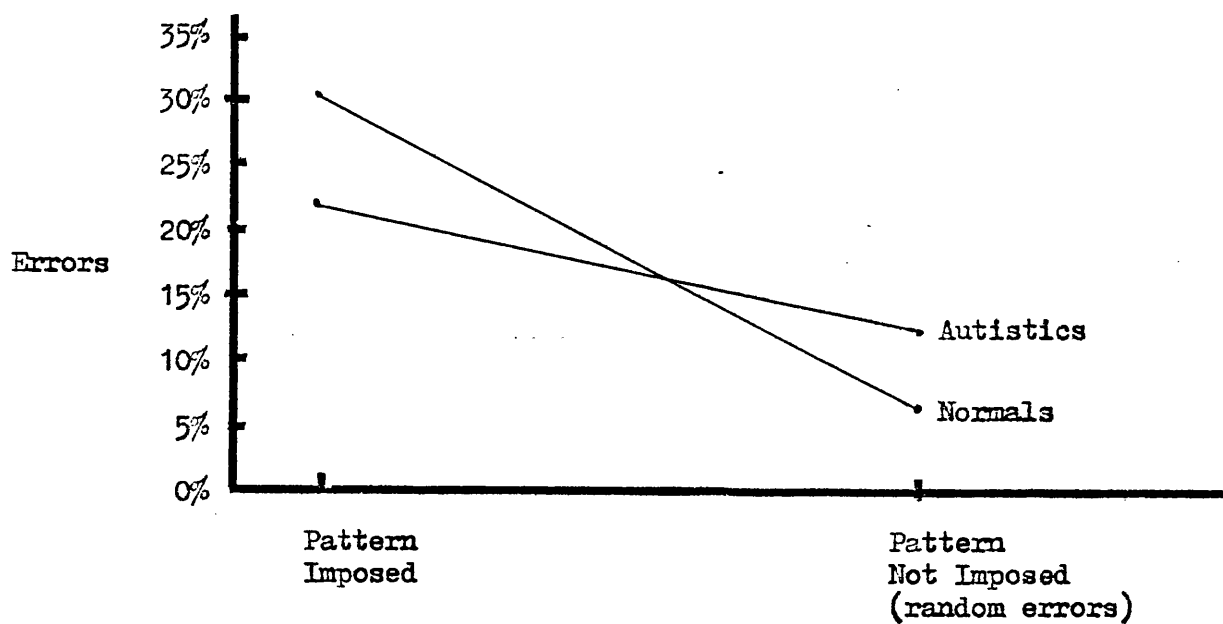
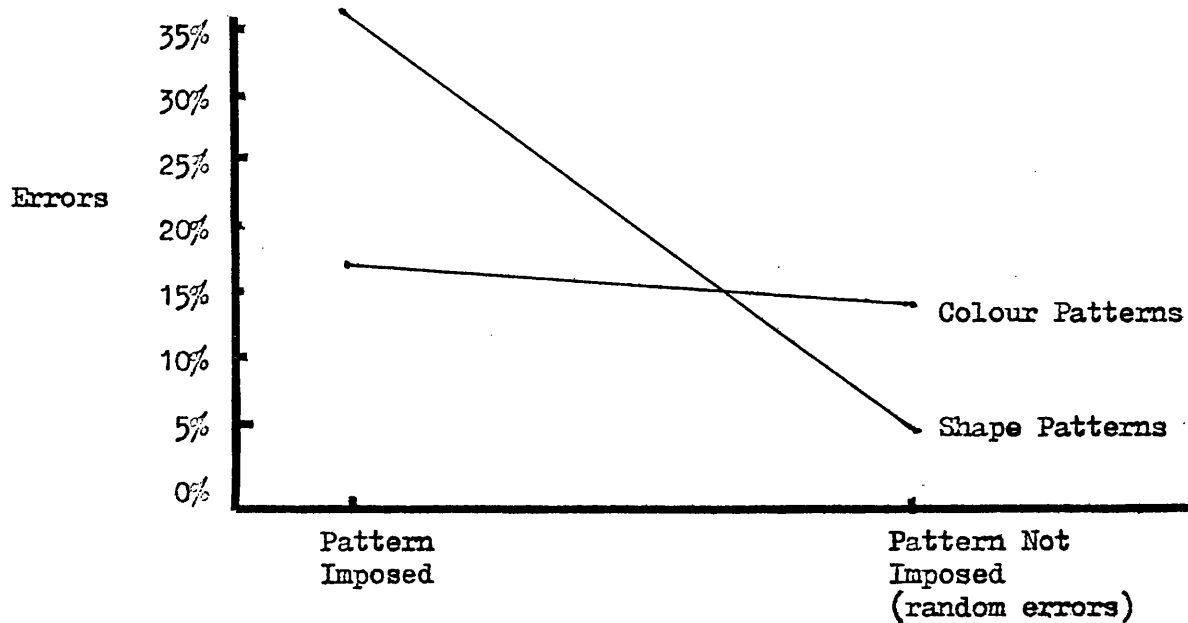
Figure 3/ii: Groups x Pattern ImpositionFigure 3/iii: Pattern Imposition x Dimensions (Both Groups)

Table 3G: Error Analysis of Variance:
Groups x Patterns x Error (Ratio/Position)
x Dimensions

Source	df	SS	MS	F	p
Groups	1	8212.89	8212.89	5.24	5%
SWG	14	21935.59	1566.69		
Patterns	3	10107.42	3369.14	5.29	1%
Groups x Patterns	3	2373.05	791.02	1.24	ns
SWG x Patterns	42	26738.28	636.63		
Errors (Ratio Vs Position)	1	56602.36	56602.36	13.7	1%
Groups x Error	1	1961.38	1961.38	0.48	ns
SWG x Error	14	57711.54	4122.25		
Patterns x Error	3	6897.31	2299.10	0.56	ns
Groups x Patterns x Error	3	3243.47	1081.16	0.26	ns
Dimension	1	2822.27	2822.27	0.68	ns
Groups x Dimension	1	87.89	87.89	0.02	ns
Patterns x Dimensions	3	2373.05	2791.02	0.19	ns
Errors x Dimensions	1	1.47	1.47	0.0	ns
Groups x Patterns x Dimensions	3	1044.92	348.31	0.08	ns
Patterns x Errors x Dimensions	3	7750.66	2583.55	0.63	ns
Groups x Errors x Dimensions	1	4568.07	4568.07	1.11	ns
Groups x Patterns x Error Dimensions	3	2858.50	952.83	0.23	ns
Total	255	429724.69			

produced consisted of one of these strings. All spontaneous sequences produced were amenable to this classification. The distribution of the sequence types is shown in Table 3H.

Both autistic and normal children produced more 'regular' than 'irregular' sequences, and this was particularly marked with sequences produced with shapes. For both colour and shape sequences, normal children tended to produce predominantly alternation patterns but autistic children produced mostly perseveration patterns. A Fisher Exact Probability test on the distribution of the alternation/perseveration predominant sequences over the two groups showed that this difference was statistically significant ($p=0.005$).

Discussion

Autistic children clearly demonstrated their ability to learn binary sequences in the dimensions of form (shape) and colour. Autistic children produced more errors than normals during learning trials, and it is possible that this is related to the relative mental ages of the subjects. The EPVT was selected as a test for initial matching of experimental and control groups because it does not require verbal responses from subjects and is a short test within the attention (and cooperation) span of the autistic child. However, it is a test that is most strongly related to comprehension and vocabulary age. It may be that were more comprehensive measures of global aspects of intellectual functioning possible, the autistic group would have yielded lower mean mental ages. In the light of the language retardation and deviance displayed by autistic children however, EPVT scores seem unlikely to overestimate substantially the intellectual performance of autistic children. An alternative consideration, and one that is considered to be more feasible, is that the autistic children were of approximately equal

Table 3H: Spontaneous Sequence Types

Dimension	Subject Groups	Regular Sequence	Non- Regular Sequence	Alter- nation Sequence	Perseveration Sequence
Colour	Autistic	50%	50%	37.5%	62.5%
Colour	Normal	50%	50%	87.5%	12.5%
Shape	Autistic	87.5%	12.5%	25%	75%
Shape	Normal	75%	25%	75%	25%
Colour	Autistic	68.8%	31.2%	31.2%	63.8%
+					
Shape	Normal	62.5%	37.5%	81.3%	18.7%

mental age to the controls group and their larger error scores are related to some characteristic behavioural deficit or abnormality that is fundamental to the syndrome of Childhood Autism.

Frith (1968; 1970b) reported that when learning binary patterns of 4 elements in length, normal children produced significantly fewer errors on patterns 1000 and 0001, whereas autistic children showed equal performance on all patterns. She related this to the observation that these particular patterns contain 2 "runs per cycle" in contrast to the 3 "runs/cycle" of the remaining patterns. Thus pattern complexity is envisaged in terms of the number of changes of the elements in that sequence. The data from the present study do not support this finding. Normal and autistic children did not differ in the way they found the patterns to be of differential complexity. Autistic children did not show less sensitivity to input structure than normals.

Both groups produced fewer errors with pattern 0001. Pattern 1000 is of equal complexity to 0001 in terms of number of "runs/cycle". However, the groups did not also find pattern 1000 to be easier to learn than the other patterns. It is suggested therefore that the number of "runs per cycle" may not be the primary determinant of pattern complexity in terms of ease of recall. Rather, the position of the characteristic element of the binary pattern (where such sequences consist of 3 elements of one unit and one of the other), may be a crucial factor in determining pattern complexity. The present data reveal that both autistic and normal subjects find recall easier when this position is at the terminal end of the sequence.

The data relating to errors demonstrating feature extraction, did not support Frith's conclusions that normal children make most errors that are consistent with the dominant 'rule' of the given pattern whereas autistic children show little evidence of such feature extraction. The present study suggests that both normal and autistic children make errors of the type that do not demonstrate feature extraction, and that this was particularly apparent with the normal

children. For both groups, most errors were found to be a function of alternation sequences rather than perseveration sequences. The Error type x Dimension interaction demonstrated that this was pronounced with patterns in the shape dimension rather than with colour patterns.

Discrepancies were also found between Frith's conclusions concerning Pattern Imposition and the present data. Frith (1968; 1970b) inferred that autistic children frequently impose simple patterns on the sequences recalled, regardless of the structure of the given input. This process of consistent pattern imposition apparently was not found in normal children. This proposal was made on the basis of 't'-tests calculated on mean scores from an insignificant interaction term in the analysis of variance. This is an unsound statistical procedure for these data. First, the use of 't'-tests for a posteriori multiple comparisons leads to an unacceptably high probability of a Type 1 error (Winer, 1962). More rigorous and conservative methods for making selected a posteriori and complete sets of comparisons exist (e.g., Scheffe, 1957; Duncan, 1955, 1957). Second, the use of multiple comparisons upon insignificant F-tests is only a legitimate procedure if the comparison is an a priori one. A priori comparisons are formulated prior to, and quite apart from, an inspection of the data. It is unlikely that we can cite sufficiently strong grounds for the use of an a priori prediction that autistic children will display pattern imposition significantly more so than normals.

The data from the present study revealed that both autistic and normal children produced a higher percentage of errors that are seen as exemplifying pattern imposition, than errors of the other selected types. Inspection of the group means showed that in fact the normal children produced the higher percentage of pattern imposition errors. For both groups, these errors were more frequent on the shape patterns.

These error 'type' results may be more meaningfully interpreted within the framework of errors in the ratio of the pattern elements ('ratio errors') and errors in the position of the characteristic element ('position errors'). Analysis of data relating to errors classified in this manner, demonstrated that 'ratio errors' were committed more frequently than 'position errors'. It is suggested that children learn these binary patterns by first correctly recalling the ratio of the binary members in the sequence, and then learning the correct position of these elements in the sequence. Having learned the ratio ("3 of one colour/shape, one of the other"), the position learning consists of recalling that position in the pattern in which the single binary member (and therefore the characteristic element) is placed. The much greater frequency of ratio errors in the present study, for both groups, suggests that once the correct ratio has been learned, the correct positioning of the sequence elements is a comparatively simple task, for fewer position errors are produced. However, the precise position of the characteristic pattern element may also be an important factor in pattern learning. Both the initial learning analysis and the ratio/position error analysis in this study suggested that pattern 0001 was significantly easier to learn than other patterns. The terminal position for the characteristic pattern element therefore appears to be the easiest to learn.

If one re-examines feature extraction and pattern imposition errors in terms of the position of the characteristic element in the sequences, one finds that both the selected errors displaying pattern imposition and the errors that do not exemplify feature extraction, involve errors other than in the terminal position in the pattern. Conversely, errors that have been related to feature extraction and errors that are not related to pattern imposition, all involve errors

only in the terminal position. The current data demonstrated that subjects make more errors that have been related to pattern imposition than the other selected errors, and also more errors that do not demonstrate feature extraction. Thus they make fewer errors in the terminal position of the pattern. This is in agreement with the finding that subjects make fewer errors overall with pattern 0001, because the characteristic element in this pattern is in the terminal position (where fewer errors are made).

It is proposed therefore that it may be more fruitful to examine errors made in the learning of binary sequences in terms of the ratio of the elements in the pattern and the position in the sequence at which the error is committed. This is in contrast to the conceptual model forwarded by Frith (1968, 1970b) in which selected errors are classified in terms of pattern imposition and feature extraction. If autistic children do indeed impose stereotyped patterns upon their recall sequences (independent of the given pattern), and also fail to extract the salient dominant 'rule' of the pattern, it is unclear how autistic children demonstrated significant learning in Frith's studies and why they produced no more errors in total than normal children over learning trials. It may now prove interesting to examine further the performance of autistic and normal children on patterns involving a number of binary unit ratios (in addition to 3:1), and to analyse data in terms of ratio errors and position errors.

Autistic and normal children found colour and shape patterns to be of equal complexity during learning trials. Errors that have been related to pattern imposition occurred more frequently with the form (shape) dimension, and there were also more errors resulting in alternation strings in this dimension. It is not entirely apparent why this should be so, although it may be related to a preference for the

form dimension. Research has shown that there is a developmental transition, for normal children at 3-4 years, from a preference for the colour dimension to form as a dominant dimension (Kovattana and Kraemer, 1974). One may predict therefore, that on the dominant dimension, errors will more strongly reflect the type of learning processes (and therefore error 'types') that typically operate than on the non-dominant dimension. Thus whilst pattern imposition errors for both normal and autistic children, occurred more frequently than the other types of error selected for comparison, this was particularly marked with shape patterns because this is the dominant dimension for children of the developmental age used in this study.

The spontaneous sequence data suggested that autistic children preferred to produce perseveration sequences, whereas normal children produced predominantly alternation sequences. These preferences were consistent in shape and colour sequences, and may be considered as generalised preferences, at least in the visual modality. Such preferences appear strongly related to mental age (Goulet and Goodwin, 1970; Gerjuoy and Winters, 1968). The perseveration tendencies of autistic children appear to be characteristic of a lower mental age, and related to their developmental delay and intellectual retardation. Frith (1968) reported that autistic children produced more 'rigid', regular, spontaneous sequences than normal children. This was not found to be the case with the subjects used in the present study.

To summarise, the data from this study do not lend support to the hypothesis of a cognitive dysfunction in childhood autism. The only differences found between experimental and control subjects concerned spontaneous sequences and a greater number of total errors (and slower learning). These differences appear to be most plausibly attributed to the intellectual retardation characteristic of the autistic group.

Methodological differences between the present study and those experiments reported by Frith (1968, 1970b) may account for differences in the results found, but it is also possible that the selection of subjects may be an important factor in this context. The present study involved autistic children of a higher mean mental age than the sample included in Frith's studies, and ~~most~~ were "verbal" autistic children. Differences in perceptual and learning performances between "verbal" and "non-verbal" autistic children have been reported in the literature (e.g., Hermelin and O'Connor, 1970; Kovattana and Kraemer, 1974), and it has been suggested that there may be two 'classes' of autism with the degree of retardation being one of the classifying criteria (Rutter and Bartak, 1971). It may be that data from subjects used in the present study is applicable only to higher mental aged, "verbal" autistic children and that more severely retarded, "non-verbal" autistic children do show evidence of cognitive pathology on binary sequence learning tasks whereas less severe cases do not. Symptoms of cognitive pathology on these tasks may be a function of the degree of linguistic and intellectual retardation. However, it is also suggested that the methodology adopted with the administration of binary pattern learning tasks, and the method of classifying errors, will be an important determinant of the precise function being assessed and the inferences made concerning cognitive pathology.

Learning processes and cognitive skills require further investigation, for they are of undoubted importance for an understanding of the precise locus of the disorder, in addition to educational and treatment considerations. Future studies might involve other tasks, directly related to cognitive functioning.

CHAPTER SIX:

"CONCEPT ATTAINMENT TASKS WITH AUTISTIC, RETARDED,
AND NORMAL CHILDREN"

Introduction

It has been suggested that autistic children are suffering from marked and characteristic abnormalities in their cognitive functioning (Frith, 1970a, 1970b; Hermelin and Frith, 1971). It was further proposed that these abnormalities are reflected in the perceptual deficiencies (Hermelin and O'Connor, 1970; Bryson, 1970, 1972), language delay and deviance (Rutter, 1968, 1975), and response stereotypy (Frith, 1972) which have been reported. Whilst clear abnormalities and deficiencies exist in these areas of functioning, substantial evidence is necessary before we can conclude the presence of an overall cognitive deficit which can account for these characteristic deficits and behaviours as expressions of one common central dysfunction in cognition. It is proposed that this criterion has not yet been reached, and further research in this area is necessary.

Methodological criticisms have already been made concerning the experimental findings with regard to visual pattern learning, and some contradictory findings have been reported earlier (chapter 5). In addition, it would seem that word recall data suggesting a cognitive defect via the auditory modality (Frith, 1970a) could be explained in terms of the severe impairment in language, which is undoubtedly present in autistic children and may well be characteristic (Bartak, Rutter and Cox, 1975). It may be argued that tasks requiring memory of word and nonsense syllable strings, or short binary sequence learning, are not directly measuring those very processes that we may refer to as 'cognition'. Such tasks may be utilising perceptual skills, which we believe to be impaired in autistic children (Hermelin and O'Connor, 1970; Bryson, 1970, 1972), but not necessarily cognitive functioning per se. Nevertheless, if we did accept such experimental evidence as support for a cognitive defect hypothesis, we should in addition predict that such impairments would be revealed in other tasks quite unequivocally demanding cognitive activity.

It is now necessary to investigate the behaviour of autistic children in areas that have been more traditionally related to cognition. This requires tasks involving problem-solving, hypothesis formation and testing, and strategy development, which are fundamental aspects of what has typically been labelled cognitive activity.

One such task measuring behaviour in this area is that of class concept attainment, an area investigated in some detail by Bruner, Goodnow and Austin (1956). Class concepts are the basis of classification and categorisation schemes, which are essential for reducing the complexity of incoming sensory information, and function as coding units. Concept attainment is achieved through learning the defining attributes of that concept, and this involves the use of perceptual discrimination, classification or coding, and the development of strategies. Such tasks seem well suited for the analysis of cognitive functioning. In this study therefore, the performance of autistic children was compared with that of controls on the attainment of three selected class concepts.

Method

a) Subjects

Ten diagnosed autistic children took part in this study, and they displayed the required symptoms that have been outlined in earlier chapters. Two control groups were used in this study, one consisting of normal children selected from an infant class in a state primary school and the other a group of severely subnormal (SSN) children. The SSN children were all institutionalised, and consisted of five with Down's syndrome, two suffering from phenylketonuria (PKU), and three with suspected brain damage of uncertain origin.

The autistic and control groups were matched on scores achieved on the Raven's Coloured Progressive Matrices test. This test was selected because, like the EPVT, it requires no verbal responses from subjects. Unlike the EPVT, the Raven's is relatively independent of purely verbal intelligence and measures skills of feature extraction that are fundamental to performance on concept attainment tasks. The Raven's scores and chronological age of the experimental and control groups are shown in Table 4A.

Table 4A: Test Data for Autistic and Control Groups

Group	N	Chronological Age		Raven's Raw Scores	
		Mean	Range	Mean	Range
Autistic	10	9.1	4.5-15.3	10.9	0-17
Normal	10	5.2	4.7- 5.7	10.7	5-14
SSN	10	15.4	13.9-16.11	9.0	0-12

b) Procedure

The procedure adopted for the study of concept attainment was based on the reception paradigm for class concept attainment, as used by Bruner, Goodnow and Austin (1956). The reception paradigm involves the presentation of exemplars and non-exemplars of the concept to subjects, in an order over which they have no control.

All subjects were individually tested with three class concepts, and the method required no verbal responses from subjects. Each subject sat at a table on which were placed two cylindrical boxes. These boxes were coloured and designed to represent 'Post' boxes, each containing a narrow opening in the upper region on the side facing the subject. The experimenter held a pile of 50 concept cards, consisting

of 3" x 3" cards on each of which was a picture. 25 of the concept cards consisted of positive instances of the concept to be attained (exemplars), and the other 25 consisted of negative instances (non-exemplars).

The subject was then told that he would be given some pictures to look at, and some of them must be "posted" into one box and some into the other. His task was to find the relevant box for each concept card. He was given a demonstration of how to 'post' cards, and was then presented with concept cards one at a time, and allowed to 'post' the cards at his own pace. The cards were arranged so that in every block of ten cards presented to the subject there were 5 exemplars of the concept and 5 non-exemplars. If the card was placed in the correct box the experimenter verbally praised the subject and gave him the next card. An incorrect placement however, resulted in the experimenter removing the card from the box (the rear of the box, facing only the experimenter, was open), and the card was then returned to the subject with the information that the box was not the correct one for that card. The box into which exemplars of the concept were to be placed, and the box for the non-exemplars, were alternated between different concepts to balance for left/right subject preferences.

All subjects' responses were recorded. The experiment continued in the manner described until either the 50 concept cards had all been 'posted' by the subject or until the subject reached the concept attainment criterion. This criterion consisted of placing 7 consecutive cards into their correct boxes with no errors. The probability of seven consecutive errorless trials by chance is less than 0.01, and therefore this was considered a satisfactory criterion of concept attainment.

At the end of trials for one concept, the subject was given a distinct break before returning to repeat the procedure for the next concept. There were three concepts in total, these being:

i. flowering plants ii. animals iii. foods.

The negative instances of each concept consisted of an heterogeneous collection of pictures, although they specifically did not include any exemplars of the other concepts to prevent any negative transfer effects (interference) from one concept to the next. An attempt was made to ensure that pictures of both exemplars and non-exemplars of the concept were within the everyday experience of subjects. It was considered that without such control the autistic subjects would be unduly penalised in the light of their handicap.

Each concept card varied in form and colour so that exemplars and non-exemplars overlapped in these dimensions. Therefore successful concept attainment could not be achieved through simple perceptual discrimination learning. Concept attribution learning involves skills of feature extraction and cognitive activity rather than perception per se.

The order of presentation of the three concepts was rotated and balanced between experimental and control groups. The order of presentation of positive and negative instances within each concept was also changed from subject to subject (although balanced between groups). Within any 10 consecutive cards however, there were always 5 exemplars and 5 non-exemplars of the concept. The within-concept card rotation was designed to prevent the possibility of any order-of-presentation effects unduly penalising either experimental or control groups.

Results

Subjects' responses were classified into four categories:

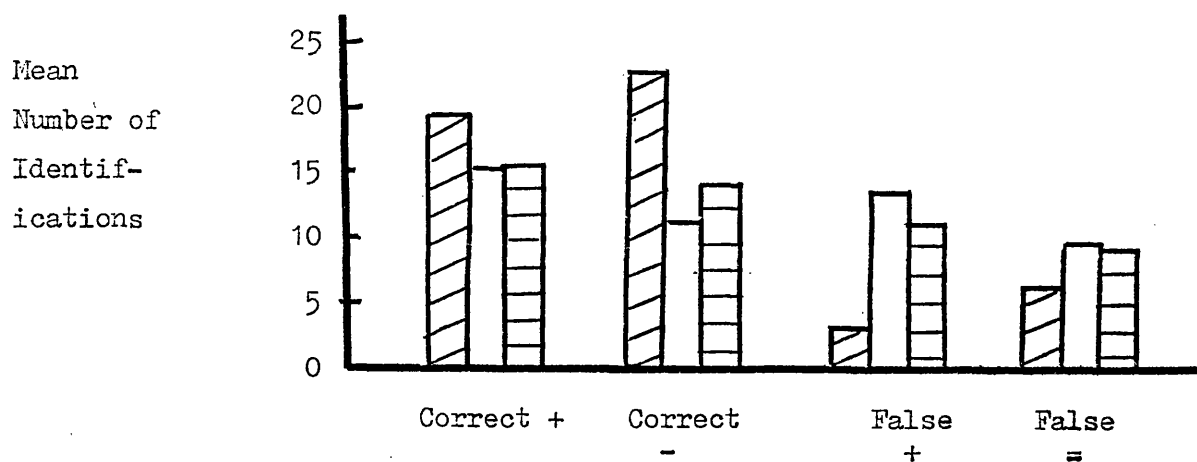
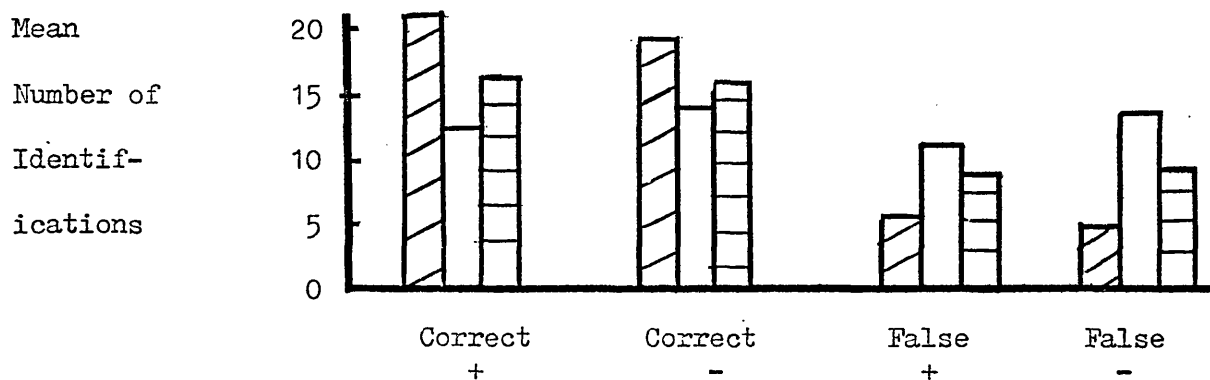
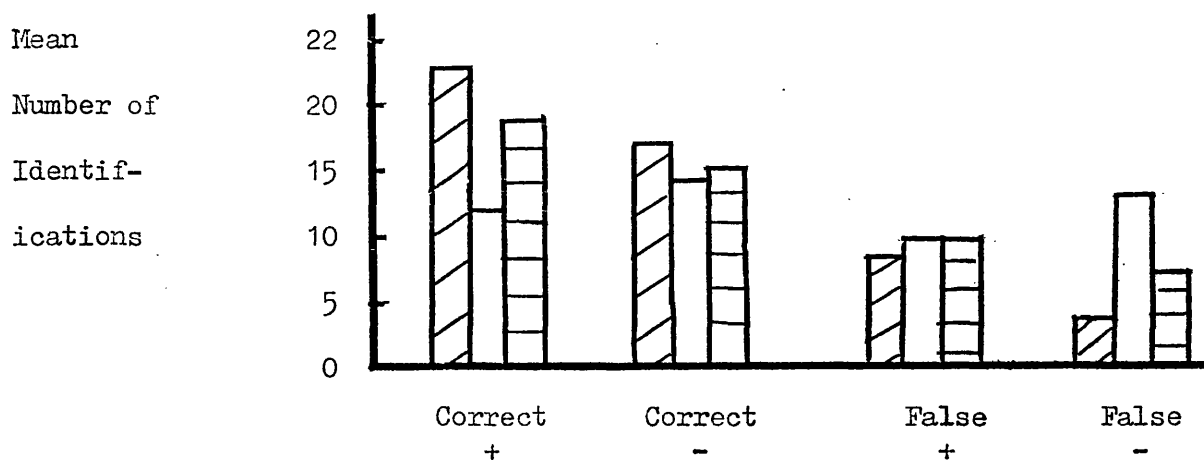
- i. Correct identifications of concept exemplars,
- ii. Correct identification of non-exemplars,

- iii. Identifications leading to "false positive" instances, of the concept,
- iv. Identifications leading to "false negative" instances.

The mean group scores for responses categorised in this manner are illustrated in figures 4/i - 4/iii. From these figures it can be seen that normal children produced the largest number of correct identifications and the least number of false negatives and false positives. Subnormals also produced more correct than incorrect identifications of exemplars and non-exemplars of each concept, but autistic children appeared to be responding randomly and produced equal numbers of correct and incorrect responses.

The total number of correct identifications and total number of incorrect identifications were calculated for each subject. The mean number of correct identifications of exemplars and non-exemplars of each concept for the groups is shown in figure 4/iv. This shows that normal children produced the largest number of correct identifications, with subnormals making more correct identifications than the autistic group.

A Groups x Concept x Subject analysis of variance was conducted on the total number of correct identifications made by each subject (Table 4B). The Groups term was significant, and a posteriori multiple comparisons were carried out on the means, using the method outlined by Scheffe (1957), which is conservative with regard to type 1 errors. This confirmed that normal children produced more correct identifications than autistic children, ($F=16.8$; sig. 0.1% level), and more than subnormals ($F=4.92$; sig. 2 $\frac{1}{2}$ % level). Subnormals produced significantly more than autistic children ($F=3.56$; sig. 5% level). The Concepts term was insignificant, suggesting that the concepts were of the same relative difficulty of attainment. The Groups x Concepts interactions was also insignificant, demonstrating that group differences were consistent across the three concepts.

Figure 4/i: Concept I (Animals)Figure 4/ii: Concept II(Plants)Figure 4/iii: Concept III (Foods)




Subnormals = 
 Autists = 
 Normals = 

Figure 4/iv: Mean Number of Correct Identifications (max = 50)

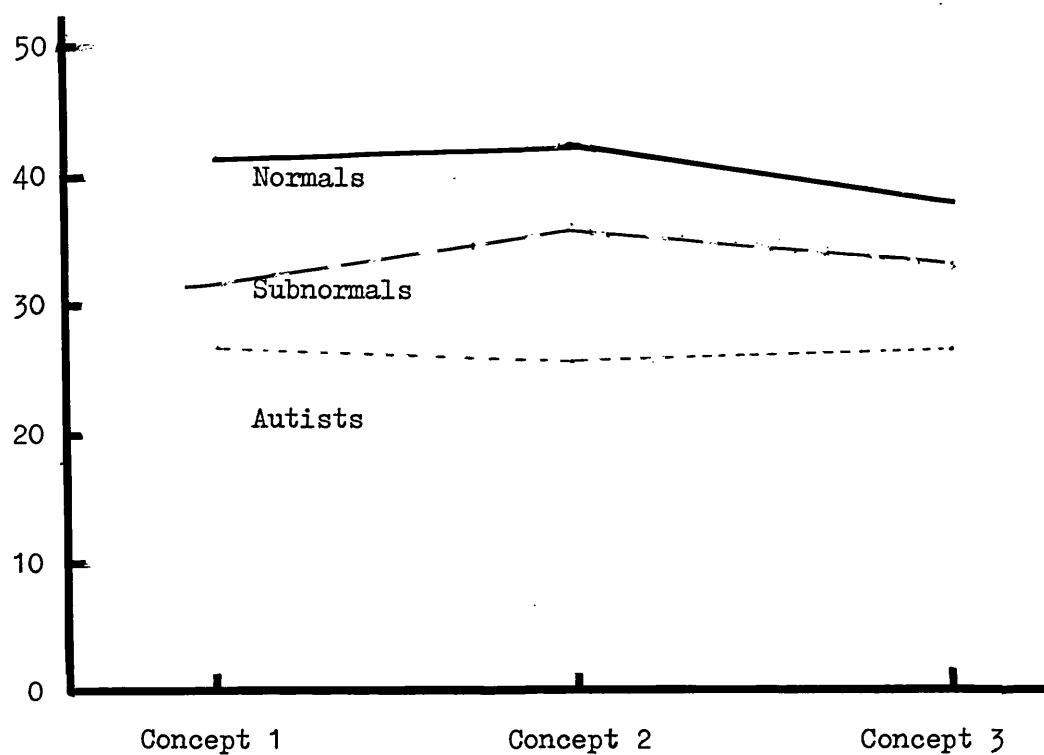


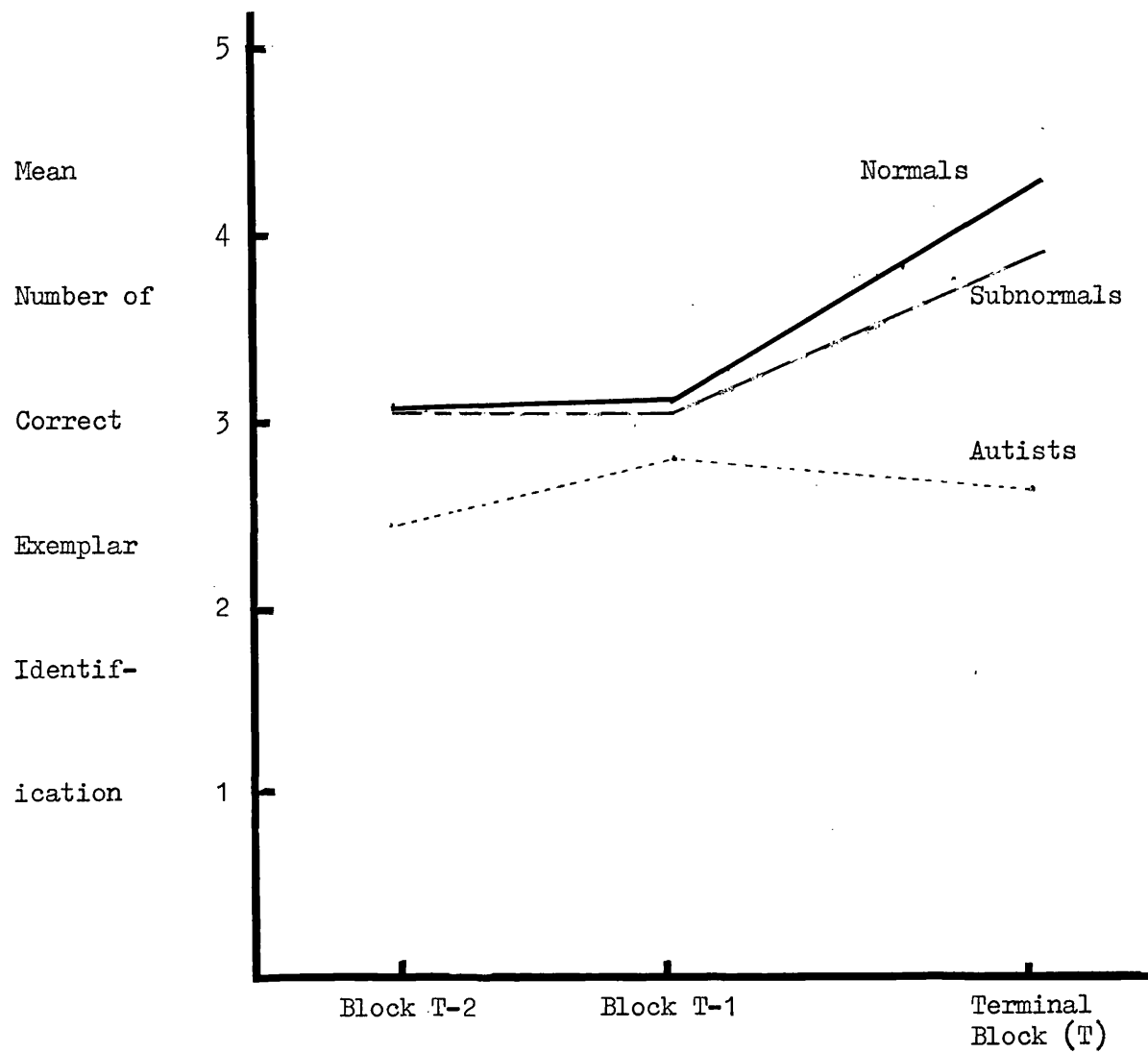
Table 4B: Total Correct Identifications Analysis of Variance

Source	df	SS	MS	F	Sig.
Groups	2	2729.09	1364.54	24.5	0.1%
Concepts	2	55.09	27.54	0.98	ns
Subjects	9	784.54	87.17		
Groups x Concepts	4	95.98	23.99	0.55	ns
Groups x Subjects	18	1002.69	55.70		
Concepts x Subjects	18	503.36	27.96		
Groups x Concepts x Subjects	36	1544.91	42.91		
Total	89	6715.66			

Data analysis also focussed on the identification of exemplars of the concept as a function of the number of instances encountered. Subjects' categorisations into the box relevant for exemplars of the concept were split into blocks of 5 responses. A terminal block was identified as those 5 trials during which the subject reached the criterion for concept attainment, or if he failed to reach this criterion, the terminal block was defined as the final 5 categorisations into the exemplar box. Having thus identified a terminal block (labelled "T"), the 5 concept cards placed in the exemplar box prior to the terminal block were identified (labelled "T-1"). Similarly, the trial block preceding this penultimate block was also identified (labelled "T-2"). The number of correct identifications of exemplars of the concept in these blocks was then calculated for each group. This data is illustrated in Figure 4/v, where it can be seen that both normal and subnormal children demonstrated rapid rises in the number of correct identifications of concept exemplars in the terminal block. These sudden rises in the number of correct identifications in the terminal block, in contrast to a gradual increase over the 3 blocks, is consistent with a 'conceptual change' model rather than a reinforcement model. The latter model predicts a continuous learning curve in which subjects gradually learn the reinforcement contingencies. A conceptual model predicts a sudden increase in correct identifications when the subject tests the hypothesis that proves to be continuously successful and leads to concept attainment. This model's predictions best fit the data obtained in this study.

It may be noted that the data for subnormals resulted in a less steep rise in correct identifications in the terminal block than that for normals. This is due to the fewer concepts attained overall by this group. The subnormals' mean score is thus depressed in relation to that of normal children. No significant rise in correct

Figure 4/v: Number of Correct Identification of Exemplars



identifications appeared in the terminal block for autistic children since they failed to reach the concept attainment criterion in all cases.

The rate of concept attainment was studied through the analysis of data from those concepts in which a subject did reach the attainment criterion. The mean number of concept cards placed in the box relevant for exemplars of the concept, (before the attainment criterion had been reached) was then analyzed for the groups. Since autistic children all failed to reach the criterion for concept attainment on any of the concepts, they did not appear in this analysis. Normal children reached the criterion within a mean of 13.3 cards placed in the exemplar box, whereas for subnormals this mean was 20.2 cards. The difference between the two distribution of scores was significant ($t=2.56$; $df=30$; sig. 2% level). Therefore in addition to learning fewer concepts in total, subnormal subjects required more encounters with exemplars and non-exemplars to do so.

Analyses were also conducted to assess the possibility that some groups were able to correctly identify more exemplars of the concept than non-exemplars, or more non-exemplars than exemplars. This may have arisen from subjects repeatedly preferring to place all concept cards in one box (i.e., a position response bias), or from a strategy that resulted in an over-inclusion of cards in either the exemplar or non-exemplar box. For example, with the concept "foods", at some stage a subject may have decided to test the hypothesis that the concept was "fruits". However, "fruits" is only a subset of the correct concept "foods", and so this subject would correctly identify many exemplars of the actual concept while maintaining this hypothesis, but would also produce a significant number of 'false negatives' (e.g., foods other than fruits being incorrectly placed in the non-exemplar box). This

kind of hypothesis may therefore produce an abnormally high number of 'false negative' responses in relation to the number of 'false positives'.

't'-tests were conducted separately for each group on the scores for 'false positive' categorisations and 'false negatives' (Table 4C). These however revealed that for neither groups nor concepts did the ratio of these two error types differ significantly.

Table 4C: False Positives Vs False Negatives

Groups	Concept	Mean No. False +	Mean No. False Negs.	df	t	sig
AUTISTIC	I	13.4	9.4	9	1.48	ns
	II	10.9	12.8	9	0.70	ns
	III	10.1	13.1	9	0.93	ns
NORMAL	I	3.3	5.7	9	1.49	ns
	II	5.1	3.7	9	1.95	ns
	III	7.9	4.0	9	2.40	ns
SSN	I	10.7	9.3	9	0.46	ns
	II	9.4	7.6	9	1.83	ns
	III	9.4	9.4	9	0.01	ns

Discussion

The data from this study of class concept attainment suggests that for autistic children the tasks proved to be beyond their current ability. All autistic subjects failed the specified criterion for concept attainment, and displayed overall performance no better than chance level. This failure does not appear to be a function solely of global intellectual retardation however, for SSN controls displayed a significantly higher performance level on the experimental tasks. There is evidence therefore, that autistic children are more severely impaired than other retarded children in cognitive functions required to achieve class concept attainment.

There are two points that must be considered in relation to this conclusion however. The first is that SSN controls produced fewer correct concept identifications than normal children, and needed more encounters with relevant instances of the concept to reach the specified attainment criterion where indeed they did so. This may be considered evidence that at least part of the poor performance of autistic children is related to their retardation. But this does not account for the complete failure of autistic subjects to achieve any of the class concept attainments, for they produced a markedly inferior performance to that of the subnormals. It is clear that despite their intellectual retardation, SSN controls were able to reach the specified concept attainment criterion in a substantial number of cases.

We must therefore infer that either the autistic children were in fact more severely globally intellectually retarded than the SSN controls (which seems unlikely in view of the Raven's scores and the profound subnormality of the control group), or that autistic children have a specific deficit that is not present in the control groups, and which markedly impairs their performance on tasks of class concept attainment.

It may be suggested that the results of the present study, and in particular the performance of autistic children, simply reflect the negativistic behaviour of the autistic child. The assumption that the autistic group were not learning on the concept attainment tasks and that their performance was "random", is of course based upon a statistical notion of chance level response patterns. It is possible that given more trials the autistic group would have reached the concept attainment criterion levels. However, there was no evidence of this from inspection of the data. If the autistic children were failing to cooperate in the study and displaying negativism, then their performance would have been at less than chance level, as was noted in one experimental demonstration of negativistic behaviour (Cowan, Hoddinott and Wright, 1965). This was not the case in the present study.

It may also be suggested that the performance of autistic children on the current tasks resulted from the ineffectiveness of the social rewards used in this study. However, the performance of normal and subnormal subjects was not consistent with a simple learning curve but rather a cognitive model of "conceptual shift". Thus social reward was unlikely to be fundamental to successful performance. In addition, it may be noted that the autonomic data reported in chapter 4 suggests that social reward is responded to by the autistic child.

A more probable and relevant variable to consider is the role of language as a mediator in the development of class concept attainment in tasks such as those used in this study. Furth (1961, 1966) concluded after studying deaf and normal children, that language experience may increase the efficiency of concept formation in certain situations, but is not a necessary prerequisite for the development of the basic capacity to abstract and generalise. There are others (e.g., Piaget) who propose that cognitive development is essentially independent of language, but several others who believe that thought is

dependent upon the language available to the child (e.g., Slobin, 1971). Most reviews on the relationship between language and cognition conclude that the arguments and evidence to date are both complex and controversial (e.g., Cromer, 1974). However, it seems difficult to deny that language skills, and in particular the use of 'inner' language, may facilitate the attainment of class concepts.

In the present study, the SSN children displayed distinctly limited language skills, in both production and comprehension. However, they may be considered as deficits related to language delay and retardation in contrast to deviance. Autistic children have been found to display both pronounced delay and deviance in language development, even when compared with children severely impaired with receptive language disorders (Bartak, Rutter and Cox, 1975).

The performance of autistic children in the present study was markedly inferior to both subnormal and normal children, and is consistent with a serious deficiency in cognitive tasks requiring conceptual thought. It may be debated whether this deficiency is a result of the severe language deficits of the autistic child, precluding the use of verbal labels as coding units and mediators in cognitive tasks. An alternative hypothesis is that the language deficits themselves, in addition to the deficiencies found in cognitive tasks like class concept attainment, are a function of a common deficit in cognition and conceptual thinking. To a large extent, the adoption of one of these alternative explanations may depend upon the way one envisages the relationship between language and thought.

What may be concluded from the current data is that autistic children appear to display deficits in conceptual thinking over and above those found in SSN children. This supports a model of childhood autism in which both profound language deficits and a dysfunction of the normal processes related to cognition, are implicated.

CHAPTER SEVEN:

"CHOICE BEHAVIOUR IN PROBABILITY LEARNING TASKS WITH
AUTISTIC, SUBNORMAL AND NORMAL CHILDREN"

Introduction

Probability learning is a variety of problem-solving that appears well-suited for an analysis of cognitive functioning and possible deficiencies in autistic children. These tasks are substantially independent of language skills, and learning requires the development and assessment of the effectiveness of strategies used. The considerable length of prediction response sequences that are produced yields much detailed data for the analysis of strategies adopted, the way in which information feedback is used, and the shape of learning curves. There are many variables in probability learning tasks that can be manipulated experimentally. These include the magnitude of the probabilities of event occurrences, the reinforcement schedule, the size and direction of event ratios, and the type of feedback given.

Probability learning under conditions of partial reinforcement shows clear developmental trends with children, important variables affecting responses being chronological age, mental age, and expectations of reinforcement (Goulet and Goodwin, 1970; Stevenson, 1972). It is a generally accepted finding that children younger than five adopt near maximizing strategies on such schedules, as do much older children of about 15 years. Five to 15 year olds however adopt matching strategies, both of these types of strategy becoming stable and clearly visible well within 100 trials (Goodwin, 1969; Weir, 1964). If the event schedule is itself changed during the course of the learning experiment, younger children (below four) have much greater difficulty in changing their response bias established by previous trials (Kessen and Kessen, 1961). On the basis of response patterns produced and also verbal reports from subjects, it has been concluded that the more readily children can generate complex hypotheses, the less likely they are to maximize reinforcement in initial blocks of

trials. Only later, when a full appraisal of the effectiveness of these hypotheses occurs do the levels of reinforcement attained (number of predictions correct) reach the level of the pre-school child. It seems therefore that although older children (above about 14), and nursery-school age children, both use near-probability maximising strategies at the terminal stage of learning, it is only the younger subjects who adopt this strategy in the early trials. Older subjects adopt a number of complex strategies before finally adopting near-maximising. Five to 14 year olds adopt many simple strategies in a trial and error fashion but fail to recognise the optimal strategy of maximising, and this depresses their overall learning performance on these tasks (Weir, 1964; Goodwin, 1969). This appears to be one of those rare instances where younger subjects produce more correct responses on a learning task than children of much greater chronological age.

Responding on probability learning tasks is also clearly affected by expectancies of reinforcement, either from life experiences or pretraining. Several studies have shown that institutional children and working-class children tend to respond to the more frequent stimulus to a greater extent than other children (thereby being rewarded more in fact); and it is suggested that this is because they do not expect to get continuous reinforcement, unlike home-reared and middle-class children (Stevenson and Zigler, 1958). This effect is perhaps more convincingly demonstrated by experimentally inducing certain expectations of reinforcement by pretraining subjects on varying levels of reinforcement. It may be predicted that middle-class children, expecting to find a solution yielding consistent reinforcement, should vary their responses more widely than lower-class children and thereby show a lower frequency of response to the most frequent stimulus. Gruen and Zigler (1968) found these predicted results in a study using

pretraining. For middle-class children, pretraining with low frequencies of reinforcement increased the incidence of choices of the reinforcing stimulus over that found following pre-training with high degrees of success. Lower-class children's performance was not influenced by pretraining. Apparently, the short-term manipulation of success was insufficient to overcome the expectancies of lower-class children regarding their expectation for success. Similar results have been obtained using teacher's ratings of achievement and children's levels of aspiration (Kier and Zigler, 1970; Gruen, Ottinger and Zigler, 1970). It would appear to be quite plausible to suggest that we might expect a variable like retardation to operate in a similar manner to lower-social class and institutionalisation, in its effect upon expectancies of reinforcement and thus response strategies (with relevance here to childhood autism and subnormality).

Another variety of probability learning design is that of transfer of training. This represents an extremely sensitive method for determining the effects of past experience (Goulet and Goodwin, 1970). Weir (1964) suggested that very young children (of about three years C.A.) do not respond in probabilistic tasks by formulating strategies and testing hypotheses. Rather, he suggested they respond on a purely reinforcement basis. Thus it was predicted that nursery school children would perform in a similar fashion in transfer tasks where the designs were solution identical, response identical, and solution different, response identical, since the solution change would not produce any 'negative transfer'. Similarly, they would find solution identical, response different a difficult transfer as they are responding on a purely response reinforcement basis, and here performance would be much poorer than for older subjects. Evidence for this hypothesis has been reported (Stevenson and Weir,

1959; Jones and Liverant, 1960), although this effect was only found during initial trials on the second schedule. The difficulty with decreasing age in such transfer of response, and for older children with transfer of solution (Crandell et al., 1961), is a failure to conform to a new schedule due to difficulty in rejecting a well-learned strategy (Odom and Coon, 1966).

Finally, there are developmental trends in the complexity of strategies that can be generated, and much evidence suggests that the characteristic pattern is one of a change from perseveration up to five, to alternation up to nine, to increasingly more complex strategies (Goulet and Goodwin, 1970). These authors also suggest that the failure to find maximising behaviour in probability learning tasks between the ages of six and about 15, is due less to the actual inability to generate more complex hypotheses than to inadequate memory for past events and their outcomes, resulting in interference among competing, partially reinforced, response patterns generated during the subject's search behaviour.

It would thus appear that in the light of developmental studies of probability learning, we know something of the kinds of strategies children use in this situation, and the effects of reinforcement schedules, transfer designs, and expectancies of reinforcement. We therefore have a body of knowledge in which to evaluate any differential performance of autistic and normal children on such tasks. In view of the effects of developmental (and thus cognitive) changes, it is important to match subjects on a measure of mental age, and to control for the effects of retardation (e.g., on expectancies of reinforcement) by using a subnormal control group.

Method

a) Subjects

Ten autistic children, ten SSN children, and ten normal subjects took part in this study. The autistic children displayed the symptom groups of severe language impairment, failure in social development, and ritualistic and obsessional behaviours that were described in chapter three. The normal controls were from a state primary school, and the retarded children from a subnormality institution. The latter group consisted of two suffering from PKU, five with Down's Syndrome, and three with suspected brain damage.

The autistic and control groups were matched on the Raven's Coloured Progressive Matrices test, and these scores, together with chronological ages, are shown in Table 5A.

Table 5A: Test Data for Autistic and Control Groups

Group	N	Chronological Age		Raven's Raw Scores	
		Mean	Range	Mean	Range
Autistic	10	9.3	4.5-15.3	10.4	0-17
Normal	10	5.3	4.9- 5.7	10.8	5-14
SSN	10	15.4	13.9-16.11	9.0	0-12

b) Procedure

Experimental and control groups completed a pre-test task and two probability learning tasks. The learning tasks were separated by a complete day to prevent significant interference effects from learning on one task to the other. Each subject sat at a table on which was positioned a large model of a house, and at the base of this there was

a subject control panel. The experimenter sat behind the 'house', which contained an open compartment at the rear. The model house was constructed in large dimensions (24" x 26") in order that the relevant parts should be of sufficient size to maintain the attention of subjects. Details of the structures of the 'house' were painted in bright colours, and some components were constructed in 3 dimensions (e.g., the doorway, roof, and chimney). Above the doorway of the 'house' there was an orange bulb, electronically operated, and in the chimney-pot a concealed buzzer similarly operated.

Behind the model house, a series of switches and relays electronically operated the release of either a figure of a boy, or a girl, which could drop into the open doorway of the house, directly in front of the subject. The control panel consisted of a rectangular board, on which were positioned two large wooden keys (2" x 4"). On the left hand key a picture of a boy was painted in one colour, and on the right hand key a girl was painted in a different colour. These pictures directly matched the boy and girl figures that appeared in the doorway of the house.

The subject's task was to predict whether the boy or the girl would appear in the doorway on each trial. This two-choice prediction was however made non-verbally, the subject pressing either the key with the picture of the boy or the girl in accordance with his specific prediction. A pre-test matching task had shown that all subjects were able to match the pictures to the relevant figures, and could also make this discrimination when verbally requested to do so.

The experimental procedure began with the experimenter selecting whether the boy or girl would appear in the door at the next trial. He did this on the basis of a predetermined schedule throughout, and made the selection by operating a switch at the rear of the house. This also automatically activated the keys on the subject

control panel. The subject was then instructed to predict which child would appear in the door on the next trial by pressing the key relevant to his choice. He was told to: "Show me whether you think the boy or the girl will come to the door this time". The action of pressing the response key triggered the release mechanism, and either the boy or girl (as pre-selected by E), appeared in the doorway. If the subject predicted correctly, the orange bulb above the doorway illuminated, and the buzzer sounded (for 3-4 seconds). If, during demonstration trials, a subject showed any signs that he found the buzzer aversive, it was immediately switched off and only the light operated on further trials. (This was necessary for one autistic and one subnormal subject). If the subject was incorrect in his prediction, the pre-selected boy/girl appeared in the doorway, but the buzzer and the light were not triggered. Thus contingent feedback was given to subjects after each trial in the form of the visual display of the boy or girl, and the light and buzzer on correct trials. Subjects were also verbally praised after each correct trial, and encouraged to make as many correct predictions as possible.

Trials continued in this manner, with the subject making predictions at his own preferred pace. The predictions for each trial were recorded.

A set of demonstration trials were shown to each subject before task one commenced, and there was also a pre-test practice sequence of 30 trials. Here subjects made predictions and the experimenter did not follow a predetermined sequence of events. Rather, the experimenter ensured that 90% of these 'spontaneous' predictions were correct, through the use of an electronically operated "all correct" switch. This sequence of 30 prediction responses was analysed to determine any apparent event preferences or biases, and to observe any pre-experimental strategies that subjects may have brought with them to the tasks. A response bias (either boy/girl or left/right)

was defined by predictions totalling more than twenty girls or more than twenty boys. Exact probabilities using the binomial expansion were calculated, and these showed that more than twenty predictions of one member of a binary choice (out of a total of 30) occurs by chance with a probability of less than 0.05. Any such response biases were recorded.

Task 1: The experimenter-defined event sequences in Task 1 consisted of a schedule in which there were 80 consecutive trials on an event ratio of 75% event A: 25% event B. After these 80 trials the event ratio was reversed to 25% event A: 75% event B, and there was no interruption or any cues to indicate to the subject that the event ratio was changing at this point. There were 80 further trials on this second ratio, making a total of 160 trials on Task 1.

The event ratios were constructed in such a manner that within every block of 20 trials the overall ratio of the events was always 75% of one event and 25% of the other event. However, within this constraint the precise order of the events varied from block to block. Event A and Event B were alternated from boy: girl to girl: boy from one subject to the next, and balanced across groups. However, if subjects showed a significant response bias on the pre-test 'spontaneous' sequence, they always commenced the task with their preferred figure as the least frequent event.

The exact order of events within the constraint of a 75%:25% event ratio was of course not predictable with 100% success. The task was thus a probabilistic one in which subjects could not be correct on every trial and could only assign subjective probabilities to the occurrence of each event. The event presentation was successive, and determined by the experimenter. Task 1 was designed to measure and

compare the learning curves for experimental and control groups, and to assess the effect of a reversal in the most frequent event (i.e., the most frequently reinforced event).

Task 2: The second task involved a schedule designed to demonstrate the effects of changing the magnitude, but not direction, of the event ratio. Thus the most frequently reinforced event did not change in this task, but its probability of occurrence decreased.

The first 60 trials of Task 2 consisted of an event ratio of 80% event A: 20% event B. At trial 60 this ratio changed to 60% event A: 40% event B. The event ratio thereby decreased in trials 60-120. As in Task 1, the events A and B were alternated from one subject to the next. Performance on Task 1 had demonstrated that by 60 trials the subjects' event predictions had reached an acceptably stable level. Therefore on Task 2, the two ratios under observation were restricted to 60 trials each. This was considered necessary in order that subjects' attention span should not be exceeded. Probability learning tasks such as those used in this study can be extremely monotonous after a period of initial novelty. The attention span of both autistic and subnormal subjects was clearly limited. It was therefore considered necessary to reduce the length of tasks to the minimum number of trials needed to ascertain stable performance levels. The somewhat elaborate apparatus used in the study (in contrast to packs of playing cards for example, that are frequently used in studies with other experimental samples), was also an attempt to maximise the attention span of the subjects used.

Task 2 was conducted in the same manner as Task 1. The experimenter determined the event on every trial according to the predetermined schedule, and feedback was given to subjects after every trial prediction, verbal encouragement after each correct response, and every

prediction was recorded. Similarly, there was no interruption or cue given when the event ratio changed at the mid-point of the task.

Results

All subjects' predictions were recorded, and the percentage predictions of the most frequent event was plotted as a function of trials. Trials were grouped into blocks of 20 since these blocks had been designed to contain the same overall event ratio, although intermediate trials were also plotted where necessary and at points within a block where the event ratio was commensurate with the overall ratio. The data for the group means for Task 1 is shown in Figure 5/i.

The event predictions for Task 1 suggested that both normal and subnormal children increased their predictions of the most frequent event over the blocks for trials 12-80. By the 80th trial these groups produced prediction levels that approximately matched the relative frequency of the occurrence of the two events. This change in event predictions over trials blocks was not apparent in autistic children.

An empirical analysis of the trends was calculated using a Groups x Trial Blocks x Subjects analysis of variance (see Table 5 B). The Groups and Trials terms were both significant, demonstrating that the predictions of the most frequent event increased with trials and the groups differed in their prediction levels. However, the interaction between groups and trials was also significant ($F=3.35$; sig. 1%). Trend analyses were conducted across trial blocks on the means for the groups. This confirmed that for both normal and subnormal children, the percentage predictions of the most frequent event increased significantly with trial numbers ($F=9.97$; sig. 0.1% level; and $F=2.25$; sig. 5% level respectively). There was no significant trend for the autistic group ($F=0.53$).

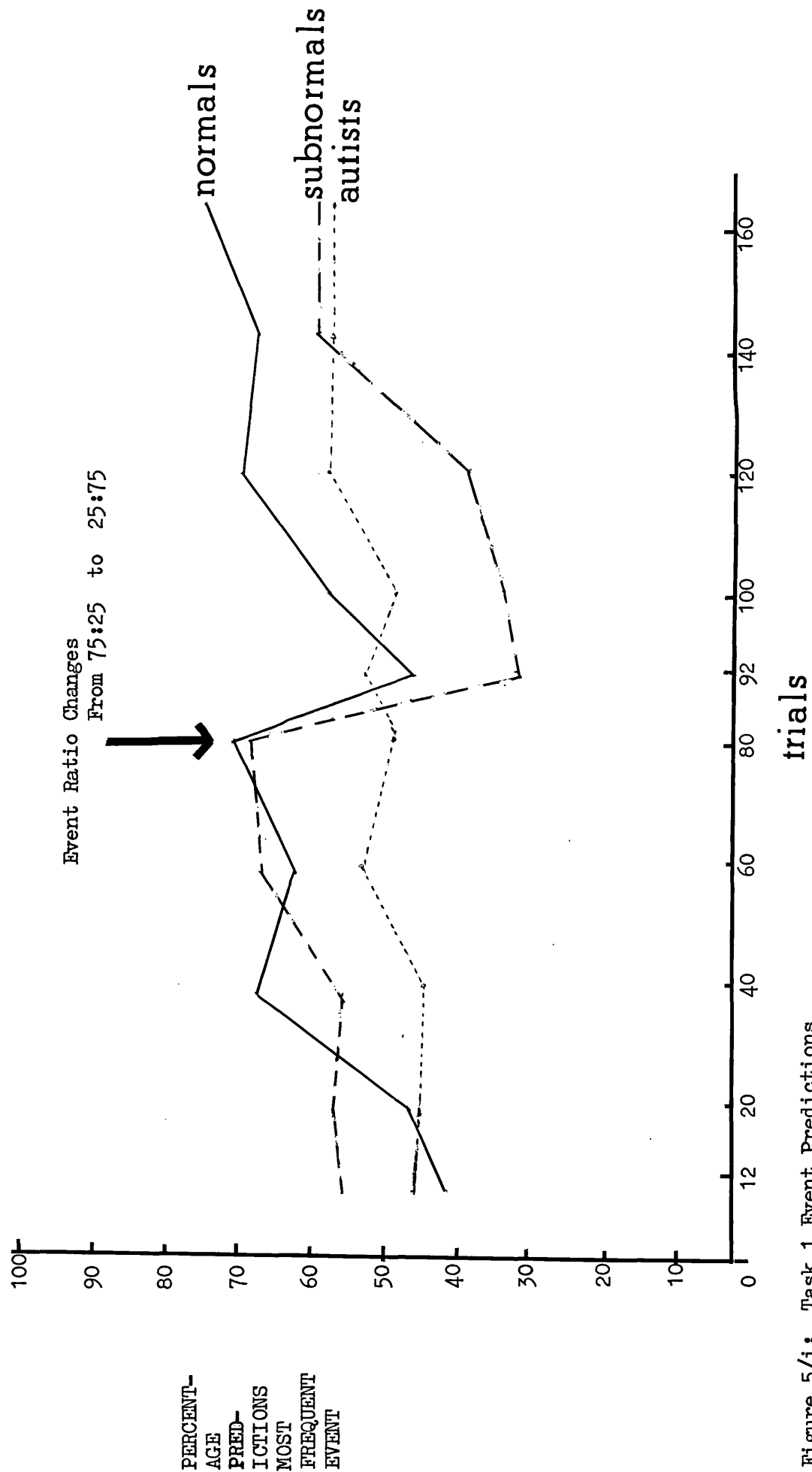


Figure 5/i: Task 1 Event Predictions

Table 5 B: Groups x Trials 12-80 (Task 1) x Subjects
analysis of variance

Source	SS	df	MS	F	p
Groups	5817.72	2	2908.86	3.86	5%
Trials	4130.56	4	1032.64	9.37	0.1%
Subjects	4990.83	9	554.54		
Groups x Trials	3997.68	8	499.71	3.35	1%
Groups x Subjects	13547.21	18	752.62		
Trials x Subjects	3968.77	36	110.24		
Groups x Trials x Subjects	10735.39	72	149.10		
Total	47188.16	149			

The group data in Figure 5/1 also suggest that during the trial blocks following the reversal in the event ratio, there was a rapid drop in predictions of the most frequent event made by normal and subnormal children, but this was not so for autistic children. A Groups x Trials x Subjects analysis of variance was conducted using the mean data from trials 80 and 92 (Table 5 C). The Groups x Trials interaction was significant ($F=5.07$; sig. $2\frac{1}{2}\%$ level), and this confirmed that whilst the performance of autistic children did not change significantly following the reversal in the event ratio, normal and subnormal children made significantly fewer predictions of the most frequent event when this changed after trial 80. Subnormal children continued to respond at a level commensurate with their predictions at later stages of the first event ratio. Thus after the event ratio reversed at trial 80, they produced an especially low percentage of predictions of the most frequent event.

A Groups x Trials x Subjects analysis of variance was carried out on the group data during trials 92 to 160 (Table 5 D). The groups differed significantly, and there was also a significant effect of trials on predictions. However, the Groups x Trials interaction was also significant ($F=2.45$; sig. $2\frac{1}{2}\%$ level). Trend analyses were conducted on the group means for the trial blocks under consideration, and this revealed that both normal and subnormal children increased their predictions of the most frequent event over trials in response to this second event ratio ($F=10.92$; sig. 0.1% level; and $F=14.40$; sig. 0.1% level respectively). Autistic children did not show such an increase however ($F=0.75$).

On Task 1 therefore, autistic children responded with approximately an equal frequency to both events throughout, irrespective of the event ratio change. Both normal and subnormal children however,

Table 5 C : Groups x Trials 80-92 (Task 1) x Subjects
analysis of variance

Source	SS	df	MS	F	p
Groups	673.63	2	336.82	3.05	ns
Trials	5900.42	1	5900.42	11.69	1%
Subjects	1378.82	9	153.20		
Groups x Trials	4110.23	2	2055.12	5.07	2 $\frac{1}{2}$ %
Groups x Subjects	1011.03	18	110.52		
Trials x Subjects	4543.75	9	504.86		
Groups x Trials x Subjects	7291.10	18	405.06		
Total	24908.98	59			

Table 5 D : Groups x Trials 92-160 (Task 1) x Subjects
analysis of variance

Source	SS	df	MS	F	p
Groups	8059.72	2	4029.86	9.96	1%
Trials	10484.11	4	2621.03	14.39	0.1%
Subjects	3649.04	9	405.45		
Groups x Trials	3732.61	8	466.58	2.45	2 $\frac{1}{2}$ %
Groups x Subjects	7280.68	18	404.48		
Trials x Subjects	6553.63	36	182.05		
Groups x Trials x Subjects	13711.65	72	190.44		
Total	53471.44	149			

developed prediction patterns approaching 'probability matching' on the first event ratio (75:25) and demonstrated changes in their response patterns in the direction of the second event ratio (25:75) after the event ratio change at trial 80. Normal children changed their prediction levels in response to the event ratio change rather quicker than subnormals. By trial 160 the normal children approached 'probability matching' of the second event ratio, whereas subnormal were still predicting the most frequent event in only 60% of their responses.

The group data for mean predictions in the trial blocks on Task 2 are displayed in Figure 5/ii. A Groups x Trials x Subjects analysis of variance was conducted on data for trials 10-60, where the event ratio was 80:20 (Table 5 E). Both the Groups and the Trials terms were significant ($F=4.58$; sig. $2\frac{1}{2}\%$ level; and $F=7.95$; sig. 0.1% level respectively). All three groups increased their predictions of the most frequent event on this event schedule, and trend analyses confirmed that for each group this increase was significant (for autists $F=9.71$; sig. 0.1% level; for normals $F=20.27$; sig. 0.1% level; for subnormal $F=3.19$; sig. 1% level). However, only normal children demonstrated prediction patterns approaching a probability matching level. Subnormal subjects produced fewer predictions of the most frequent event, although more so than autistic children.

A Groups x Trials x Subjects analysis of variance was conducted on prediction data for trials 80-120, the period during which the event ratio had dropped to 60:40 (Table 5F). The Groups and Trials terms were both significant, but the interaction between them also reached significance level ($F=3.16$; sig. 5% level). This suggested that whilst the percentage of predictions of the most frequent event initially dropped for each group following the reduction in magnitude of the event ratio, normal children subsequently increased

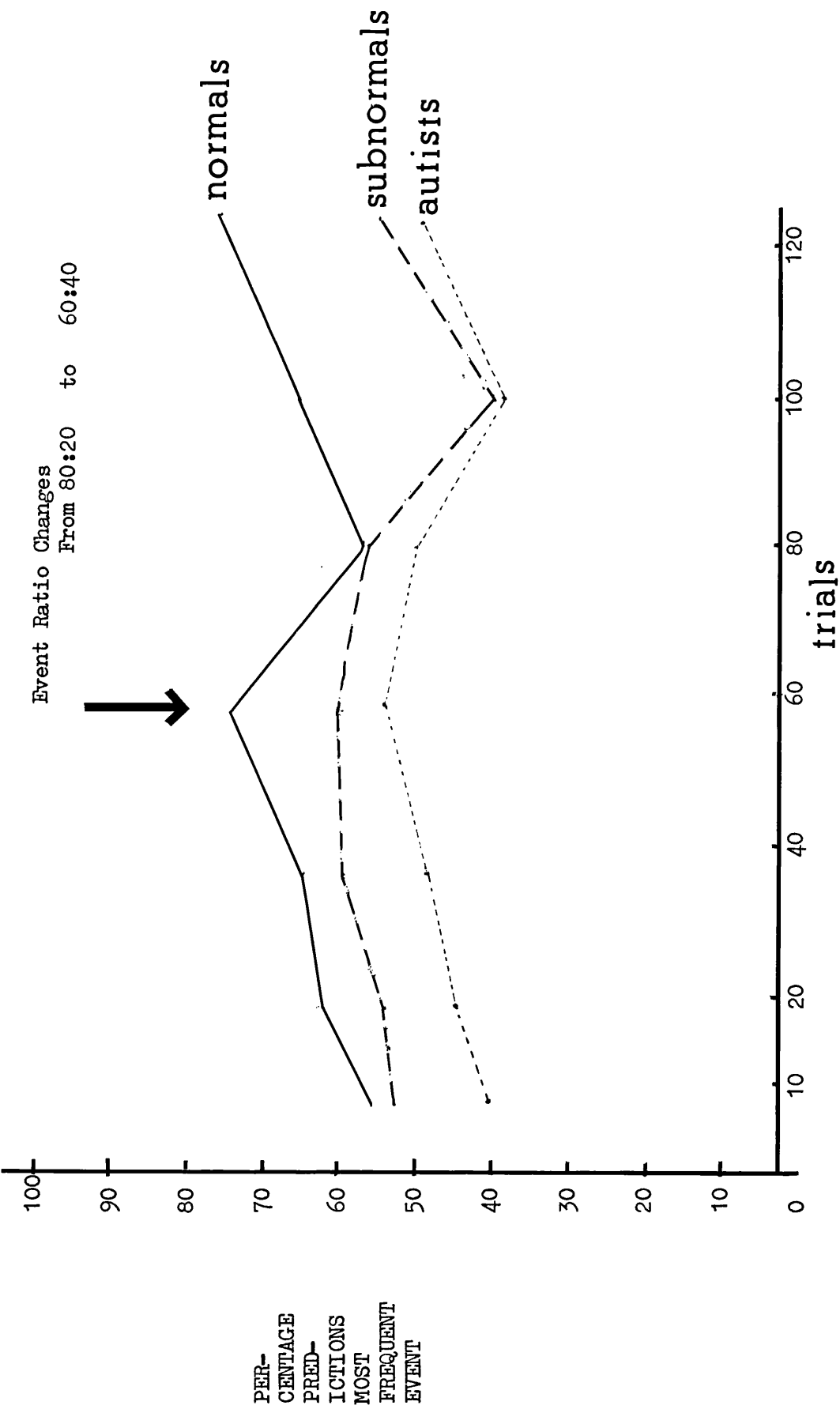


Figure 5/ii: Event Predictions for Task 2

Table 5 E : Groups x Trials 10-60 (Task 2) x Subjects
analysis of variance

Source	SS	df	MS	F	p
Groups	5661.82	2	2830.91	4.58	2 $\frac{1}{2}$ %
Trials	2463.37	3	821.12	7.95	0.1%
Subjects	4431.87	9	492.43		
Groups x Trials	497.78	6	82.96	0.60	ns
Groups x Subjects	11185.18	18	621.40		
Trials x Subjects	2788.47	27	103.28		
Groups x Trials x Subjects	7375.88	54	136.59		
Total	34404.37	119			

Table 5F: Groups x Trials 80-120 (Task 2) x Subjects
analysis of variance

Source	SS	df	MS	F	p
Groups	6600.60	2	3300.30	8.36	1%
Trials	1965.60	2	982.80	9.83	1%
Subjects	2698.04	9	299.78		
Groups x Trials	2016.40	4	504.10	3.16	5%
Groups x Subjects	7104.29	18	394.68		
Trials x Subjects	1799.96	18	100.00		
Groups x Trials x Subjects	5740.71	36	159.46		
Total	27925.60	89			

the frequency of their predictions of the most frequent event. A trend analysis confirmed that this increase was significant ($F=3.34$; sig. $2\frac{1}{2}\%$ level).

Having assessed overall performance levels and the percentage predictions of the most frequent event, analysis was then focussed upon the strategies that subjects adopted from trial to trial, and their use of information feedback concerning predictions and the actual events that occurred. The binary predictions (boy/girl) were classified into four categories on the basis of their relation to the previous prediction. These were:

- i. Win-Shift (preceding prediction correct, but the subject switched his current prediction to the alternative event).
- ii. Win-Stay (preceding prediction correct, current prediction remained the same).
- iii. Lose-Shift (preceding prediction incorrect, but subject changed his current prediction to the alternative event).
- iv. Lose-Stay (preceding prediction incorrect, subject maintains the same prediction on current choice).

Subjects' predictions were classified in this manner, and absolute numbers were also converted to percentages. The number of 'shifts' and the number of 'stays' following a correct prediction were expressed as a percentage of the total number of correct predictions. Similarly the number of 'stays' and the number of 'shifts' following incorrect predictions were expressed as a percentage of the total number of incorrect predictions. Percentage data for the three groups is shown in Figure 5/iii.

In order to determine if the proportions of the 'stay' and 'shift' predictions, following 'wins' and following 'losses', differed from chance, groups means of the absolute number of each of the prediction types were taken and exact probabilities using the binomial

Figure 5ii: Strategies Used During Probability Learning Tasks 1 and 2

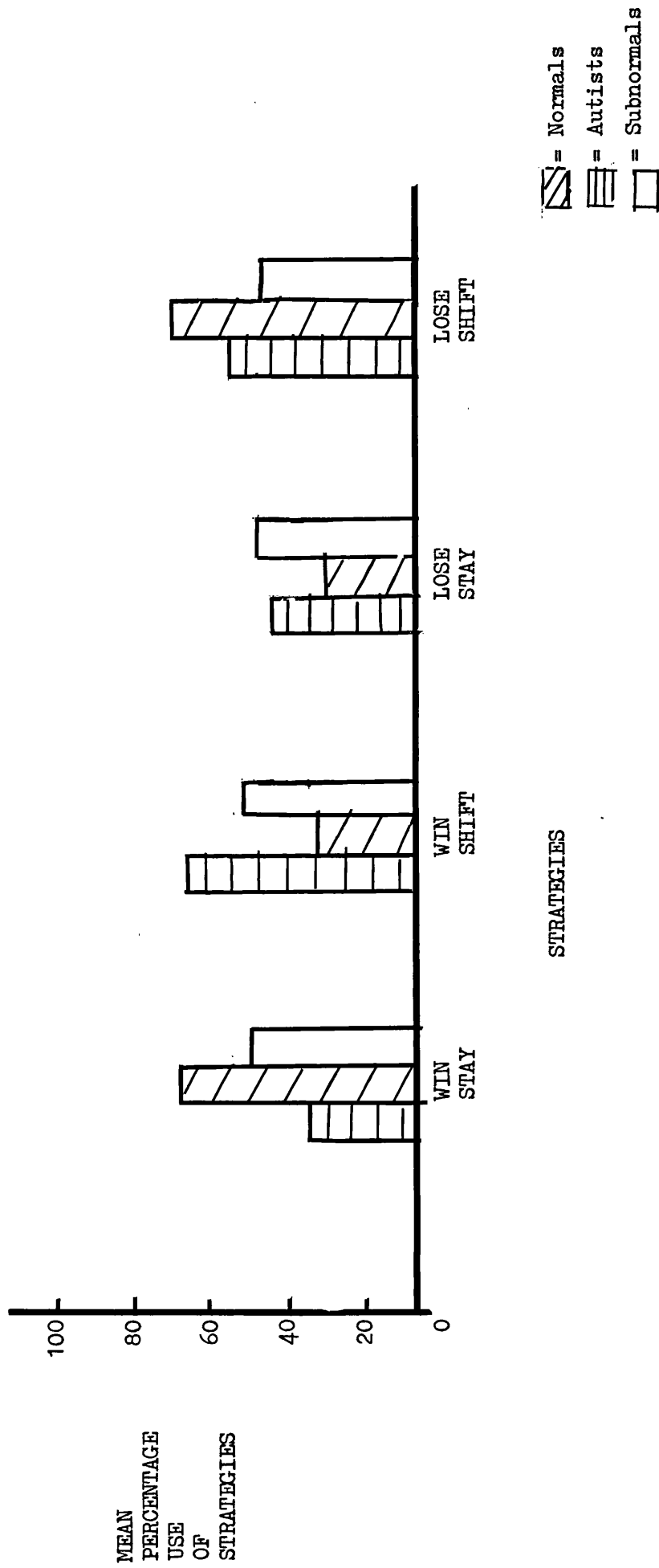


Table 5/G: Group mean data for strategies used in probability learning tasks

Groups	No. Win- Stays	No. Win- Shifts	Prob. W.Stay > W. Shift	Prob. W.Shift > W.Stay	No. Lose- Stays	No. Lose- Shifts	Prob. L.Stay > L. Shift	Prob. L.Shift > L.Stay
Autists	50.4	90.0	-	sig. 1%	60.9	73.9	-	ns
Normals	103.7	49.6	sig. 1%	-	38.8	87.9	-	sig. 1%
Sub- Normals	72.3	70.3	ns	-	63.2	67.3	-	ns

expansion were calculated (Table 5/G).

These probabilities revealed that for normal children the probability of a win-stay strategy was significantly greater than a win-shift, and the probability of a lose-shift strategy was greater than a lose-stay. However, for autistic children the probability of win-shift was greater than win-stay, and the probabilities of lose-shift and lose-stay were not significantly different from each other. For sub-normal subjects, 'stay' and 'shift' strategies did not differ in their frequency following either wins or losses.

Therefore the current data suggests that normal children adopt clear win-stay, lose-shift strategies on probability choice-learning tasks, whereas autistic children use win-shift, lose-?(i.e., shift or stay) strategies. Inspection of probabilities calculated for data from each subject confirmed that these strategies were representative of the individuals in the autistic and normal groups. The group mean values for subnormal children however, suggest that they do not adopt a consistent strategy of 'shift' or 'stay' following either wins or losses. Data for individual subnormal subjects however suggests that the group mean figures are compounded from two groups of subjects, one of whom

adopt a win-stay, lose-stay strategy and the other a win-shift, lose-shift strategy. Thus subnormals appeared to use either repeated alternation or repeated perseveration strategies on the experimental tasks, consistently sticking to predictions of one event or consistently switching from one trial to the next.

Discussion

The choice prediction behaviour recorded in the probability tasks selected for this study revealed a number of differences between group data for autistic, normal and subnormal children. During the first task, normal and subnormal children displayed prediction patterns consistent with probability 'matching' strategies. This may be seen in relation to the general finding that normal children between the ages of approximately 5 and 15 years adopt matching strategies in such tasks (Weir, 1964).

Following the change in the most frequent event (when the event ratio reversed), normal and subnormal children differed in their ability to adapt to the modified schedule. Both groups were 'sensitive' to the change in the event schedule, for both normal and subnormal subjects were able to modify their behaviour so that their prediction levels corresponded more closely to the new event probabilities. However, for normal children this modification was rapidly achieved, and indeed these children probability 'matched' on this second event ratio. Subnormals found much greater difficulty in changing their response bias established from the first event schedule. The difficulty in rejecting a well-learned strategy has been found to be characteristic of young normal children (Kessen and Kessen, 1961; Odom and Coon, 1966), and on the current task it may be considered that subnormal subjects performed in this manner due to a developmental delay.

Autistic children did not change their prediction levels throughout Task 1, suggesting an apparent 'insensitivity' to the event schedule and the marked change in the direction of the event ratio (and thus the most frequent event) after trial 80. They failed to develop 'matching' or 'maximising' strategies, and indeed failed to predict the most frequent event at more than chance levels, even at the terminal stages of the event schedule. If autistic children responded on a purely reinforcement basis, the prediction level of the most frequently occurring event would be less than probability matching levels but significantly above chance levels. The data from the autistic group may be indicative of the operation of stimulus-independent, 'internal' response patterns. This is consistent with a cognitive deficit model of childhood autism that has been proposed (Frith, 1968, 1970a, 1970b). The data is also consistent with a perceptual inconstancy model, where attention and recognition deficits may severely impair learning on tasks that require continuous monitoring of long sequences of stimuli. The autonomic data reported in chapter 4 do not support such a model, but whilst an over-arousal model also implicates learning performance, the relationship between the current choice prediction data and abnormalities in arousal function in Childhood Autism will be more fully discussed in chapter 8.

The second probability learning task involved a change in the magnitude of the event ratio, but not a change in the most frequent event. During the initial event ratio, normal children approached probability matching in their prediction patterns. Subnormal children appeared to have difficulty learning this event ratio, for their prediction level of the most frequent event fell short of matching levels. It is not clear why this should occur in the light of their performance on Task 1, where the event ratio was indeed marginally lower. It is possible that trials on this ratio were terminated somewhat prematurely,

and were it practicable to do so with the type of subjects used in this study, it would have been desirable to record predictions on each event ratio over 100 trials.

On this second task autistic children did display some increase in predictions of the most frequent event, although for the first two trial blocks these subjects were predicting the least frequent event with the higher probability and by trial 60 predictions of the most frequent event were only slightly above chance levels.

The reduction in the magnitude of the event ratio appeared to depress predictions of the most frequent event for all three groups, but by the end of the event schedule the normals were responding at a level exceeding probability matching. . This latter finding was not predicted, and it is uncertain why normals should show this increase in predictions of the most frequent event during the 60%:40% schedule.

The data taken from the probability learning tasks used in this study suggest that autistic children respond to trial-defined event ratios only when the ratio is highly pronounced (e.g., 80%:20%) although this is only marginally higher than the ratios used in Task 1 when there was no significant response change, and even on these schedules prediction levels did not approach a level predictable from responding to differential reinforcement from the two events. This could support a cognitive defect model in which it is proposed that autistic children are insensitive to temporal input and respond to any kind of patterned input as if it were random (Frith, 1970b). This model also proposes that the responses are not themselves "random", rather they reflect internal processing characteristics rather than external features. However, it is proposed that there are other viable formulations. Analysis of trial by trial strategies suggested that autistic children have difficulty in the use of task-relevant information feedback concerning their predictions. Thus they 'shifted' their

prediction choice following a correct prediction (win-shift), and were inconsistent in their response to incorrect predictions. These are inefficient strategies. The probability of the most frequent event is, by definition, higher than the least frequent event and therefore a correct prediction of the most frequent event is best followed by a repetition of that prediction. In other words a win-stay strategy leads to more correct predictions than win-shift. Similarly, when an incorrect prediction occurs, and this is most likely to follow a prediction of the least frequent event, the most efficient strategy is to consistently 'shift' to a prediction of the other (most frequent) event. This win-stay, lose-shift strategy was indeed found to characterise the behaviour of the normal children.

Subnormal children adopted either consistent win-stay, lose-stay (perseveration) strategies, or win-shift, lose-shift (alternation) strategies. Goulet and Goodwin (1970) suggest that there is a developmental trend in the complexity of strategies that subjects can generate, with perseveration preceding alternations in development. Perseveration strategies, in which the most frequent event is continually predicted, result in probability maximising. This is consistent with predictions that retardation may operate on probability learning task performance in the same manner that institutionalisation, lower social class backgrounds, and experimentally induced low levels of aspiration do. The latter factors have been related to low levels of expectancies of continuous reinforcement, where subjects do not seek or expect solutions that lead to continuous success, and therefore they maximise (Gruen and Zigler, 1968; Kier and Zigler, 1970). However, some subnormal subjects in the present study adopted alternation strategies. It seems that the group mean data for subnormal subjects is best explained in terms of a product of these two types of strategy displayed.

With regard to the autistic children, it may be that the analysis of trial-by-trial strategies used by autistic children suggests that their performance is more strongly related to inefficient and indeed inappropriate use of choice information feedback. This is in contrast to an explanation in terms of a total 'insensitivity' to the reinforcement and event contingencies in the probability learning tasks, and an alternative cognitive deficit hypothesis to the model proposed by Frith (1968, 1970a, 1970b).

The autistic children appeared unable to consistently utilise negative feedback upon making an incorrect prediction. However, where predictions were correct, they adopted a more inappropriate strategy by switching their next choice to the alternative event. It seems plausible to suggest that autistic children are infrequently consistently 'correct' in their everyday life experiences, and indeed may live in a world of "perpetual failure" (Churchill, 1971). They have profound problems in the area of language and communication and in social relationships, and these deficits are in addition to marked retardation in the vast majority of cases. As with subnormality, this may lead to low levels of aspiration and expectations of reinforcement. This predicts that such children will vary their responses less than others, since they do not expect to find a solution leading to continuous reinforcement. However, autistic children did not probability maximise in this study. It may be speculated at this point that there are two possible models to account for the behaviour of the autistic child on the current tasks. The first is that the autistic child has such a chronic set of behavioural deficits (e.g., in language, social relationships, perception), that he has not developed consistent strategies for dealing with positive feedback from his behaviours, such as choice predictions in the probability learning task. Thus whilst he may have

low levels of expectation of reinforcement, like the subnormal child, he has such severe behavioural deficits in addition to his retardation that he is unable to formulate and develop a strategy such as probability maximising.

The second possibility is that the very deficits in the ability to utilise positive feedback, themselves result in the characteristic deficiencies in social skills, language, communication etc., rather than being a result of them. The current data is naturally unable to distinguish between these two alternatives. It is of course also possible that deficits in both areas are themselves the result of a third factor, quite possibly organic in origin.

It may be considered relevant that some research suggests that inappropriate behaviours like negativism, stereotypy, and gaze avoidance, dissipate when autistic children are given tasks on which high levels of success are ensured by experimental manipulation (e.g., Churchill, 1971). This at least suggests that autistic children have developed 'styles' or modes of behaviour in response to demands beyond their ability level.

A failure to utilise positive feedback correctly would inevitably lead to severe learning problems. Much learning in educational contexts, and most social behaviours and language, require successful discrimination learning and concept attainment. But these depend upon correct utilisation of information concerning positive and negative feedback from prior responses.

Finally, it should be noted that the trial-by-trial strategy data analysed in this study, concerning 'shifts' and 'stays', was based on data summed over the four event ratios used in both probability learning tasks. We cannot assume that such strategies will be consistent across these trials and event ratios - they may be 'masking' strategy changes. The strategies used by subjects may be expected to change as

trials continue on a specific event schedule, and as subjects' levels of predictions of the most frequent event continue. Nevertheless, it may well be that the mean data for the autistic children is the most reliable, as their prediction levels of the most frequent event changed comparatively little over trials. It is therefore more likely to be characteristic of their choice behaviour in probability learning tasks than mean data for the other subject groups.

The hypothesis of a cognitive deficit in autistic children, in which failure to use information feedback in an efficient and appropriate manner is considered fundamental, is certainly speculative. Further research on the use of information feedback by autistic children, in different tasks and modalities, may be warranted.

CHAPTER EIGHT:

SUMMARY AND CONCLUSIONS

The experiments reported in this thesis focussed upon three major areas within the field of childhood autism. The first concerned features of the autistic child's environment, and specifically the behavioural content of mother-child interactions. The second involved autonomic responses to selected stimuli and reinforcers, and the third area consisted of studies investigating the possibility of specific learning deficiencies and cognitive dysfunction in the autistic child.

Whilst these three areas are superficially somewhat diverse, in reality they all relate to the observed behaviours and presenting symptoms of the autistic child. They must be evaluated in the context of information they may yield for an understanding of the psychopathology of the condition as a whole, together with any resultant treatment and educational implications.

The mother-child interaction analysis produced a number of interesting data, perhaps the least predictable being that autistic children tolerated mutual 'facing' for significant periods and indeed for longer periods than normal children, under the experimental conditions utilised. Whilst 'facing' included a broader category of behaviour than specific eye to eye contact, this finding lends no support to the hypothesis that gaze aversion is a characteristic symptom of the autistic child.

Mothers terminated mutual facing more frequently when interacting with autistic children, despite spending longer overall periods in 'facing' the autistic child in contrast with normal children. It was suggested that these terminations were a function of the lack of verbal and non-verbal feedback given to mothers by the autistic child. This is consistent with the severe impairment in communication skills which is a fundamental characteristic of the autistic child, which may be seen to make him an unrewarding interaction partner.

Despite the periods of mutual facing between the autistic child and both familiar and unfamiliar mothers, it was apparent that these children are nonetheless most sensitive to 'demands' made upon them (of both verbal and non-verbal types). The majority of stereotyped behaviours, and over 75% of the movements away from the interaction partner, were displayed by the autistic child following such 'demands'. A number of possible explanations for this behaviour were proposed in chapter 3, including "cut-off" functions and a sensitivity to failure on the part of the autistic child. This finding is also consistent with an over-arousal model of childhood autism, although the hypersensitivity to social stimuli must be considered a selective process in the light of the data on mutual facing.

The autonomic data recorded from the response to 'social' stimuli in the second study, suggest that autistic children may be markedly responsive to such stimulation in contrast to 'non-social' stimuli. This is also consistent with a model of childhood autism in which the child is hypersensitive to particular social stimulation, including demands placed upon him. The profound failure to form social relationships, and the non-communicative behaviour of the autistic child, may also be related to the responses seen when 'demands' are made upon the autistic child.

Mothers of normal children elicited more cooperative responses from autistic children than mothers of autistic children, and yet did not reinforce such responses significantly more frequently. The autonomic data collected also suggested that autistic children may respond more to novel stimuli than familiar stimuli. It may be suggested then that autistic children produced more cooperative behaviour with mothers of normals because they were novel interaction partners. However, such cooperative behaviour levels were not observed when autistic children interacted with unfamiliar mothers of

autists, and it seems therefore that cooperation level differences are not solely related to familiarity of the interaction partners. It seems more probable that mothers of autistic children do not interact with their own, or another, autistic child in an optimal fashion in terms of eliciting cooperative responses.

Whilst the cooperative responses of the autistic child were frequently rewarded by mothers of all 'types', negativistic behaviours were infrequently punished (verbally or physically) or ignored, and often were reinforced with contingent attention. This is consistent with data reported by Moore and Bailey (1973) which demonstrated that "reinforcement errors" were consistently less frequent than "punishment errors" in pre-treatment baseline data for an autistic child and his parents. "Punishment errors" may be considered important in the maintenance of maladaptive behaviours like those included in the category of 'negativistic' behaviour. The latter behaviours cannot be considered unique to the autistic child, but were found to be common to those autistic children used in the studies in this research programme.

The mother-child interaction study also revealed that mothers directed more speech to autistic children, but this included more questions and commands rather than "guiding" statements. Marked speech modifications were made by mothers when discriminating between interactions involving autistic children and those involving normals, and therefore the content rather than the complexity of speech to the autistic child may be considered more relevant to analyses in terms of the most appropriate speech model for the autistic child.

The parental (or 'own') mother of the autistic child directed the least utterances to children. This is consistent with an hypothesis that predicts a partial extinction of mother's speech to the child as a result of the autistic child's failure to provide feedback to the mother. This is also seen in the characteristic non-communicative

behaviour of the autistic child and his failure to develop normal social skills and relationships.

It is recognised that the generality of these findings may be limited to the setting in which they were observed. However, these findings indicate a number of features which might be emphasised in a generalised treatment programme for autistic children in which parents are used as co-therapists: It may include an emphasis upon the recognition of the child facing the mother as indicative of a willingness to interact, despite a lack of eye-to-eye contact. Mutual facing should be encouraged and rewarded, and maintained for as long as the child permits, as a first stage in the shaping of eye-to-eye contact. In addition, emphasis may be placed upon the need to make "demands" upon the child only within his known capabilities, breaking down required activities into smaller sub-components such that the child learns to succeed. Consistent, contingent reward is naturally necessary to increase and maintain cooperative behaviour, but parents may need particular training in the use of extinction and "time-out" procedures for non-adaptive behaviours like negativism. Comprehension and intellectual ability appropriate modification of speech, although clearly made by mothers of autistic children, may be supplemented by a greater use of speech including "guiding" statements.

The autonomic data suggest that despite severe expressive and communicative deficits in the autistic child, these children do perceive traditional reinforcers and environmental stimuli in the absence of apparent overt responses. Whilst not statistically significant, trends found in the autonomic data at least suggest larger responses to novelty and 'social' stimuli, and this is not consistent with a perceptual inconstancy model in which the autistic child has severe recognition deficits. This model predicts recognition deficits in autistic

children, resulting from the failure of environmental stimuli to impinge upon him during the hypothesised inhibitory states. Thus novelty and familiarity may frequently be indistinguished, and discriminations between social and non-social stimuli may be impaired. (These points have been discussed in chapter 4).

The studies involving binary sequence learning, class concept attainment, and probability learning, were all designed to investigate potential learning deficits and cognitive functioning. It was concluded from the binary sequence learning tasks that the autistic children did not exhibit less sensitivity to input structure than normal children. The data found were not consistent with those reported by Frith (1968, 1970b) from which she proposed that autistic children display deficits in the information processing of the sensory input. Frith suggested that these children fail to extract explicit 'rules' from the environment and to generalise and use these rules in a flexible manner. They have a consistent tendency to utilise their own input-independent internal 'rules' (a process of "pattern imposition").

It was concluded on the basis of the experiment reported in chapter 5 that autistic and normal children did not significantly differ in their performance on these tasks, except in the rate of learning of the prescribed sequences. The larger number of errors produced by autistic children may be related to their intellectual retardation. Explanations were suggested for the discrepant findings between this study and those reported by Frith in terms of methodological procedures, and subject populations, although the formulation of error 'types' and inferences drawn from them may be particularly relevant. The data from the study reported in this thesis suggested that both autistic and normal children committed most errors that have been related to "pattern imposition" and to "non-feature extraction". But this seems not altogether surprising, for while subjects are committing errors, it

seems likely that they have not extracted the relevant features of the pattern and will therefore be testing hypotheses in their response sequences. The latter errors may be interpreted as reflecting a process of pattern imposition. However, it was concluded that it may be better to classify error 'types' into those involving "ratio errors" and those involving "position errors".

Autistic children displayed an outstanding failure to reach criterion performance on all three of the class concept attainment tasks. The data suggested that autistic children were significantly more impaired than normals and matched retardates in learning and in cognitive functions required to achieve class concept attainment. Intellectual retardation was considered insufficient to account for the performance of autistic children, in the light of the use of matched retardate controls. It was proposed that 'inner' language may facilitate performance on these tasks, and indeed may be a fundamental prerequisite for successful class concept attainment. It is also possible to suggest that the performance of autistic children reflects a profound deficit in cognitive tasks requiring conceptual thought. Whether the primary impairment is considered to be related to language delay and deviance, or to cognitive dysfunction, depends substantially upon the envisaged relationship between language and thought.

One may question why autistic children were capable of learning short binary sequences yet failed completely to make the required discrimination in the class concept attainment tasks. If the latter reflects specific language difficulties and the concept attainment tasks depend upon language mediation processes, it remains necessary to explain the performance of autistic children on the probability learning tasks. It is proposed that these tasks are essentially independent of language functions.

Data collected over trial blocks for predictions of the most frequent event on these probability learning tasks, demonstrated that autistic children did display an apparent insensitivity to the input structure when compared with normal and subnormal controls. They failed to develop 'matching' or 'maximising' strategies, or respond on a reinforcement basis. However, a trial-by-trial analysis of response sequences suggested that autistic children were not necessarily completely insensitive to probability contingencies, but rather were characteristically using feedback in an inefficient and inappropriate manner.

It is possible that the choice prediction data for autistic children reflects attention and recognition failures from perceptual inconstancy. However, the autonomic data reported in chapter 4 was inconsistent with a perceptual inconstancy model of childhood autism, since it appears that the autistic child does perceive social reward and make associations. In addition, a perceptual inconstancy model does not account for the "win-shift, lose?" trial-by-trial strategy predominantly used by autistic children.

An over-arousal model of childhood autism implies that the autistic child will have difficulty in habituating to incoming stimuli, and this will indeed present problems for him on probability learning tasks, particularly in changing strategies and response patterns when event ratios are changed. However, the specific inappropriate use of feedback by autistic children, revealed in chapter 7, is more consistent with a cognitive defect model.

Three possibilities appear therefore to follow from these findings: i. Autistic children may be unable to formulate and evaluate different global strategies as a result of severe cognitive deficits; ii. Deficits in social skills, language, and cognitive

functioning reflect the operation of generalised deficits in feedback utilisation; iii. It is also proposed that both may be a function of a common third factor, quite possibly of an organic origin.

If autistic children display a cognitive deficit in which there is a failure to use information feedback from their responses, then it may be predicted that they will have marked difficulties in discrimination learning. This will include severe difficulties in learning secondary reinforcers, and this is consistent with the observed deficits in language and social development of the autistic child. It is also consistent with experimental research that reports marked difficulties in establishing secondary reinforcement control (Hudson and DeMyer, 1968; Lovaas, 1971).

However, if a failure to utilise feedback in an efficient and appropriate manner is considered fundamental to childhood autism, the model must account for the ability of autistic children to learn short visual binary sequences despite the striking failure to learn on class concept attainment tasks. The latter data may be accounted for on the basis of severe language deficits and the role of language mediation in such tasks. It appears that autistic children are able to learn discrete binary sequences, but are unable to formulate efficient strategies for their choice prediction behaviour in probability learning tasks. The proposed cognitive deficit in the autistic child is not apparent in tasks where the stimuli presented, and the response sequences required, are constant during learning trials (e.g., in binary sequence learning). However, where the stimuli and correct responses vary continuously in a manner that is not totally predictable (e.g., in probability learning tasks), autistic children do not develop the strategies observed in normal children or even subnormals. Rather, they adopt inefficient and inappropriate strategies.

We must ask whether such a cognitive deficit can account for the observed behaviours of the autistic child. Certainly many real-life situations involve events occurring in a probabilistic fashion, and most reinforcers are delivered on an intermittent basis. The "insistence upon sameness" and ritualistic behaviours of the autistic child may be seen as attempts by him to maintain a static, 'controllable' environment.

Severe deficits in learning may be predicted from inappropriate use of information feedback (including reinforcers), leading to the intellectual retardation reported in the vast majority of autistic children. Normal language development, communication skills, and cognitive and social development all depend upon efficient, intact, learning processes. Indeed, it is difficult to conceive of an important area of behavioural functioning that does not depend upon appropriate and efficient utilisation of information feedback.

If such a deficiency is characteristic of the autistic child under certain conditions, then education and treatment could be structured such that the child is given consistent, continuous information feedback and reinforcement. It may be necessary to structure required tasks into small unit steps, facilitating continuous success for the child, but also ensuring that he learns to repeat responses that have proved to be successful.

These suggestions, together with those made earlier in relation to behaviour modification programming, with parents as co-therapists, are open to empirical investigation and evaluation in future research.

It is clear that the research reported in this thesis has not led to vast advances in our understanding of the aetiology of childhood autism: it was not anticipated that such advances would be made. Following earlier work on delineation of the syndrome and

differential diagnosis, and the more recent accumulation of knowledge on the psychological characteristics of afflicted children, progress now depends upon systematic investigation of specific deficiencies in the autistic child. It was intended that this research should contribute to this systematic investigation. Also, future research should be more clearly directed towards clinical intervention and evaluation in terms of treatment and educational effectiveness.

The acute problems of the autistic child are of considerably more than academic significance, as those who have witnessed the anguish and despair of parents of these children are well aware. Kozloff

(1973) most succinctly expresses this sentiment thus:

"If human life is sacred, then biological death is no greater tragedy than the social and psychological death involved in lifelong commitment to an institution for the chronically ill. In both cases, life has been wasted. For those of us working to help autistic children and their families, criticism of our theories and therapies is hard to take, perhaps because in the light of the above statement, such criticism implies that we are wasting not merely our own time, but others' lives."(p.vii)

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APPENDIX APARENTAL QUESTIONNAIRE

- 1) Name and age ...
- 2) Where educated ...
- 3) Parental occupation ...
- 4) Who has the clinical responsibility ...
- 5) How old when diagnosed and where ...
- 6) Age of mother at birth.
Length and any complications in pregnancy ...
- 7) Other children in family and their age ...
- 8) Earliest symptoms and age at appearance ...
- 9) Does he (she) have difficulties with relationships
with people ...

Avoid talking or looking at people ...

Experience difficulties in mixing and playing with
other children ...

Has the inability to relate to others been present from the
beginning of life ...

Prefer objects to people ...
- 10) Any abnormal posture or exploration with own limbs or face ...

Any aggression towards himself (herself) ...
- 11) Does he (she) become preoccupied with or attached to any
particular objects ...

- 12) Does he (she) try to resist change in any routines or surroundings ...

- 13) Is he (she) sometimes apparently unaware of pain, such as when touching very hot objects or bumping him (her) self ...

Does he (she) sometimes behave as if almost deaf ...

Does he (she) prefer to touch, taste and smell things rather than look at them ...

- 14) If you change your routine or way of doing things with him (her), does he (she) get upset or anxious ...

Is he (she) terrified of anything which is really in fact just commonplace ...

- 15) What speech does he (she) show ...

Are there lots of repetitions of the things that you say, and any odd phrases or words ...

- 16) Does he (she) have any unusual rituals or mannerisms like rocking or spinning or flapping the hands ...

Does he (she) walk and generally move in an ordinary fashion ...

- 17) Does he (she) have outstanding abilities, such as in music or memory, which you have noticed to stand out from his (her) usual level of ability ...

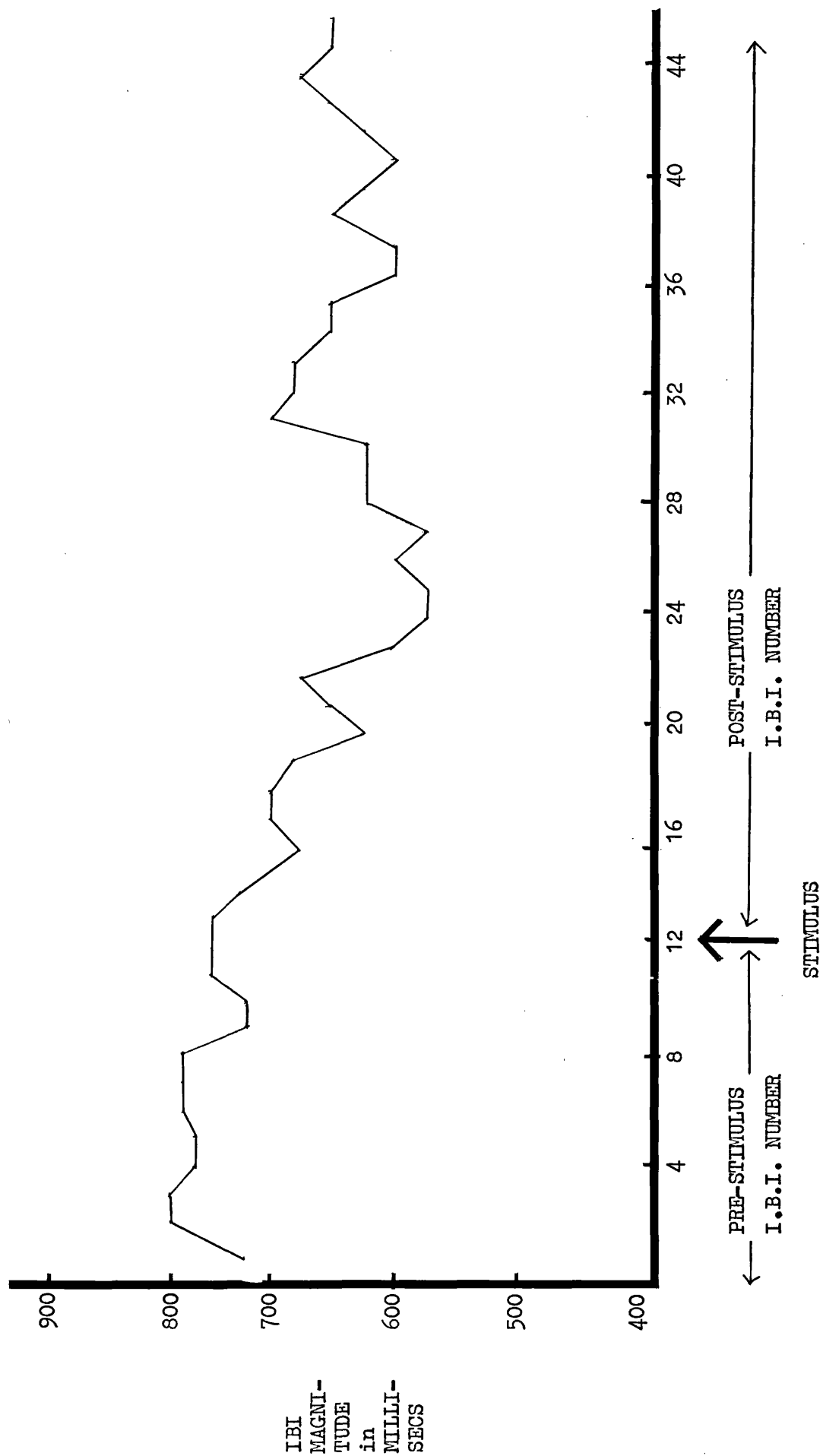
- 18) Does he (she) show particular stubbornness and refusal to cooperate at times?

```

LIST
5 DIM P(80)
10 FOR I=1 TO 80
15 P(I)=0:NEXT I
20 S=0:SI=0:X=0:Y=0
25 SET RATE 3.25
30 L=ADC(1)
35 WAITC\N=ADC(1)\N=D=P-L\N=L=P
40 S=S+D\SI=SI+D*D\X=X+1:IF X<1000 THEN 35
45 PRINT "X"\"X=X/1000\N1=SGR(SI/1000-N*10)
50 C=1.8\G2=.75\G3=1.25
52 INPUT \"SET RATE 3.25\"L=ADC(1)
55 WAITC\GOSUB 300:IF TST(0)<1 THEN 55
60 WAITC\T=TIN(0)\T=T+800
65 WAITC\GOSUB 300:IF TIN(0)<T THEN 65
70 PRINT "X"\"T=T-1200\X=X
75 FOR I=1 TO 80
80 IF P(I)=0 THEN 95 \P(I)=(P(I)-T)*.25
85 IF P(I)>0 THEN 90 \X=X+1\P(I)=0\GO TO 95
90 P(I-X)=P(I)\P(I)=0\N=I-X
95 NEXT I
100 FOR I=1 TO INT(L/5)+1
105 J=I*5\PRINT P(J-4),P(J-3),P(J-2),P(J-1),P(J)
110 NEXT I
115 H=0\X=0\Y=0\Z=2000
120 H=750
125 S=0:SI=0\N=0
130 FOR I=1 TO L-1
135 IF X>0 THEN 140:IF P(I+1)<10000 THEN 140:GOSUB 400
140 J=1\N=1
145 D=P(I+J)-P(I)\IF D<H*(0*00) THEN 175
150 IF D>H*(0*00) THEN 170
155 IF D>1 THEN 160 \PRINT ENS=S+D\SI=SI+D*D\N=0+1\GO TO 185
160 FOR K=1 TO N
165 PRINT D/N, "*"\"S=S+D/N\SI=SI+(D/N)*(D/N)\N=0+1:NEXT K\GO TO 18
170 N=N+1\GO TO 180
175 J=J+1
180 IF (I+J)>L THEN 195:IF J<6 THEN 145 \PRINT "LOST"\"STOP
185 H=L/N\IF H<Y THEN 190 \Y=Y
190 IF H>Z THEN 195 \Z=H
195 I=I+J-1
200 NEXT I
205 GOSUB 400 \STOP
300 P=ADC(1)\N=L=P-L\N=P\IF D<0 THEN 330
315 IF D<C*N1 THEN 330 \N=B+1\N(P)=TIN(0)
320 WAITC\N=ADC(1)\N=D=P-L\N=P\IF D<0 THEN 330
325 IF D<C*N1 THEN 330 \N(P)=TIN(0)\GO TO 330
330 RETURN
400 PRINT "X"\"X = ";X, "TIN = ";T
405 IF X>0 THEN 410 \X=X+1
410 G1=S/(N*G2=SGR(SI/1000-G1*01)
415 PRINT "HEAD IBI = ";G1;" SE IBI = ";G2
420 H=60000/0\PRINT "HEART RATE = ";H
425 Y=0\Z=2000\N=0\SI=0\N=0\N=0\N=0\N=0\N=0
500 END

```

APPENDIX C/i: Heart Rate Response to Adult Approach: Visual Display



APPENDIX C/ii: Heart Rate Response to Adult Approach: Data

APPENDIX C/iii: Heart Rate Response to Social Reward: Visual Display

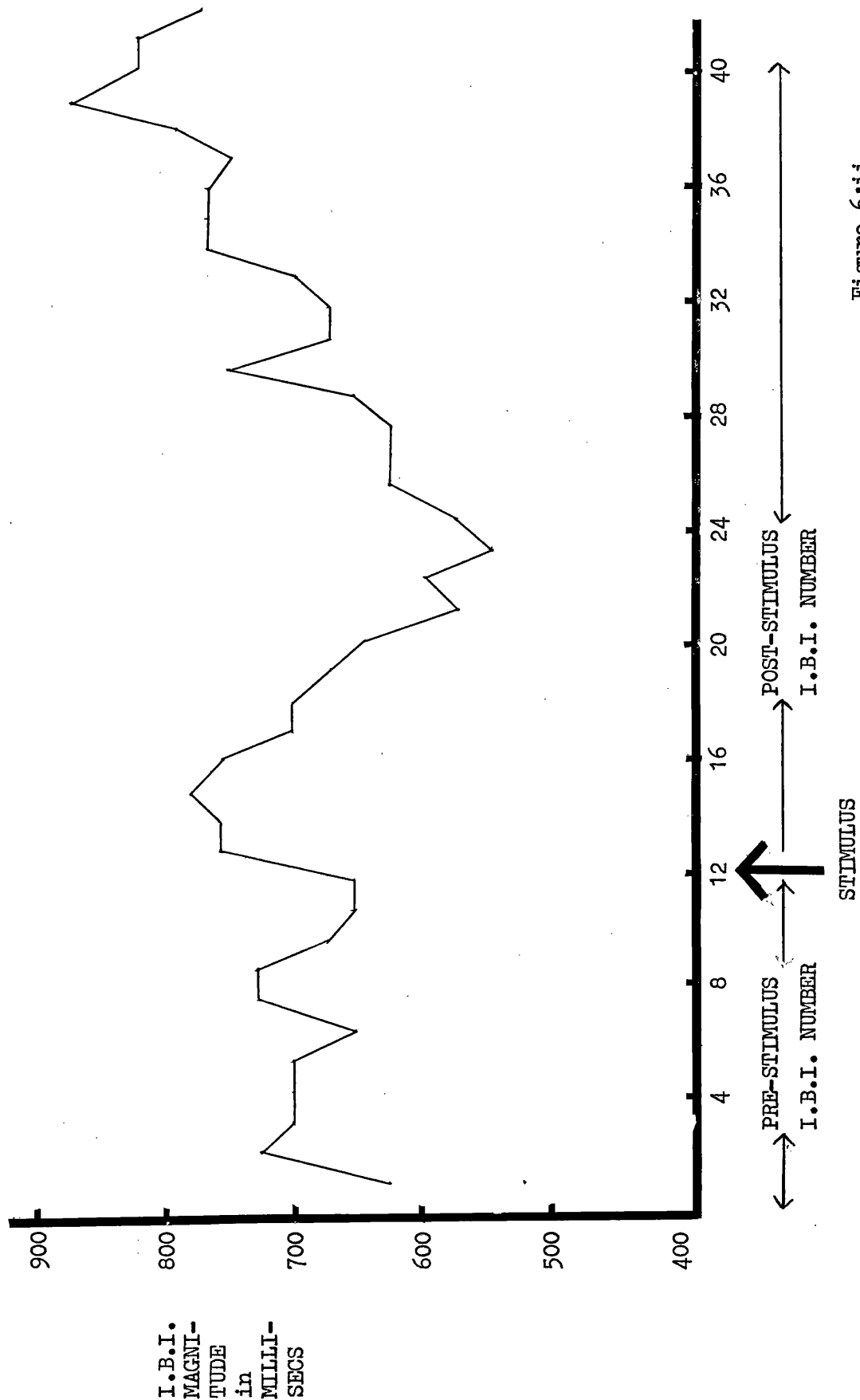


Figure 6:ii

APPENDIX C/iv: Heart Rate Response to Social Reward: Data

APPENDIX D

Mean scores for data analysed from mother-child interactions

Variable Type	Measure Taken	Mean score for autistic children	Mean score for normal children
FACIAL POSITIONS	Total time mutual facing	22.75 secs	12.08 secs
	number of occurrences of mutual facing	7.75	7.17
	number of adult terminations of mutual facing	2.50	1.01
	average duration before adult terminated mutual facing	1.92 secs	0.65 secs
	duration spent in position adult facing/child not facing	376.1 secs	286.0 secs
	percentage time in position adult facing/child not facing	62.6	48.3
	average duration before adult terminated adult facing/child not facing	14.50 secs	9.08 secs
	total time spent in neither facing position	187.3 secs	286.2 secs
	average duration before adult terminated neither facing	6.89 secs	9.74 secs
	total percentage time spent facing child by adult	66.3	50.3
STEREO-TYPED BEHAVIOUR	number of stereotyped behaviours	9.30	0.00
	total duration of stereotypy	43.6 secs	0.00
	number of antecedent NVDQ to stereotyped behaviours	4.43	0.00
	percentage of stereotyped behaviours preceded by NVDQ	47.7	0.00
TOY PLAY	total time spent in constructive toy play	311.9 secs	545.3 secs
	number of different toys used by child	4.47	3.57

Variable Type	Measure Taken	Mean score for autistic children	Mean score for normal children
TOTAL BODY MOVEMENTS (TBM)	number of TBM towards adult	2.00	0.00
	number of TBM towards adult preceded by NVDQ	1.40	0.00
	percentage of TBM towards adult preceded by NVDQ	70.2	0.00
	number of TBM away from adult	4.90	0.00
	number of TBM away from adult preceded by NVDQ	3.67	0.00
	percentage of TBM away from adult preceded by NVDQ	74.8	0.00
	number of TBM away from child	0.50	0.00
	number of TBM towards child	3.23	0.00
COOPERATIVE + NEGATIVISTIC BEHAVIOUR	number of cooperative responses rewarded	4.70	2.17
	number of negativistic behaviours	5.03	0.00
	number of punishment 'errors'	4.13	0.00
	total cooperation: number of verbal + non-verbal cooperative responses	14.33	19.30
MOTHER'S SPEECH	total number of utterances	132.63	101.03
	mean utterance length	4.10	5.09
	number of 'guiding' statements	6.27	9.20
	percentage of verbal statements	48.31	65.17
	number of questions	38.30	26.03
	percentage of questions	26.77	26.00
	number of verbal commands	27.01	6.73
	percentage of verbal commands	20.95	6.14
	type-token ratio (TTR)	0.34	0.42
	number of verbal rewards	12.97	5.90
	number of verbal punishers	0.73	0.00

Variable Type	Measure Taken	Mean score for autistic children	Mean score for autistic children
CHILD'S SPEECH	total number of words spoken	59.93	230.93
	total number of utterances	21.80	59.30
	mean utterance length	1.57	3.74
	number of verbal statements	13.80	41.27
	percentage of verbal statements	45.47	68.61
	number of verbal commands	0.37	1.10
	number of verbal replies to questions	3.73	9.93
	percentage of verbal replies in speech	8.98	20.52

Variable Type	Measure Taken	Own Mother (OM)	Mother of autistic (MA)	Mother of normal (MN)
'TYPE' OF MOTHER	mean percentage of punishment 'errors'	32.80	21.65	10.04
	mean utterance length	4.20	4.83	4.74
	number of questions used	22.70	30.50	43.30
	percentage of questions	20.23	27.52	31.40
	percentage of verbal commands	25.98	8.07	6.58
	total number of words spoken	426.65	521.40	621.00

APPENDIX E

From the data analysis in the mother-child interaction study 21 measures have been selected for simpler analysis using t-tests for dependent measures. These variables were selected because they are the ones that are cited in the text of the "discussion" section.

CHILD EFFECTS

(AUTISTIC VS NORMAL)

* a = interactions with autists n* = with normals

VARIABLE	MOTHER OF AUTISTIC CHILD						MOTHER OF NORMAL CHILD							
	X _a [*]	SD _a	X _n [*]	SD _n	t	df	p _{ns} or	X _a [*]	SD _a	X _n [*]	SD _n	t	df	p _{ns} or
total time in position AF/CF	28.3	21.88	11.6	6.47	2.26	9	0.05	17.34	18.77	12.55	23.06	0.49	9	ns
No. adult terminations of AF/CF	1.60	2.33	0.50	0.67	1.12	9	ns	2.70	1.17	1.30	0.55	2.45	9	0.05
mean duration before adult terminates AF/CF	5.29	4.54	0.42	0.59	1.93	9	0.10	1.11	2.14	0.78	0.71	0.47	9	ns
total duration AF/CNF	401.8	95.39	292.6	90.88	2.62	9	0.05	336.1	137.3	275.7	95.0	1.19	9	ns
mean duration before adult terminates AF/CNF	16.43	9.14	8.74	1.99	2.50	9	0.05	12.17	7.09	7.8	2.54	1.84	9	0.10
prob. transition ANF/CF to AF/CF	0.19	0.21	0.35	0.43	1.10	9	ns	0.32	0.38	0.05	0.09	2.07	9	0.10
prob. transition ANF/CF to ANF/CNF	0.70	0.31	0.52	0.44	1.06	9	ns	0.37	0.40	0.81	0.30	2.73	9	0.05
No. cooperative responses (non-verbal)	8.70	7.23	7.80	6.55	0.29	9	ns	14.20	6.20	10.70	6.70	1.23	9	ns
No. cooperative responses rewarded	4.00	4.75	1.40	2.57	1.57	9	ns	6.70	4.47	2.50	2.37	2.62	9	0.05
percentage punishment errors	43.30	44.41	0.00	0.00	2.91	9	0.02	20.01	29.90	0.00	0.00	2.49	9	0.05
total number mothers' words	514.6	199.4	528.4	182.5	0.15	9	ns	677.7	233.3	564.3	184.0	1.40	9	ns
total number of mothers utterances	124.9	49.8	99.5	38.0	1.15	9	ns	163.1	56.9	109.0	36.32	2.73	9	0.05
mothers' mean utterance length	4.22	0.76	5.44	1.07	3.00	9	0.02	4.20	0.65	5.27	0.83	3.56	9	0.01
No. "guiding" statements	5.30	3.31	7.40	5.33	1.07	9	ns	7.80	2.85	10.2	2.56	2.28	9	0.05
percentage mothers' statements	46.35	12.89	68.47	9.76	4.51	9	0.002	51.40	14.57	60.67	12.56	1.61	9	ns
No. mothers' questions	35.90	18.65	24.70	11.68	1.56	9	ns	55.8	30.24	30.80	12.21	2.56	9	0.05
percentage mothers' questions	29.73	9.68	25.32	8.33	1.06	9	ns	32.33	12.82	30.41	11.45	0.33	9	ns
percentage mothers' commands	21.40	15.37	5.26	6.71	2.88	9	0.02	10.89	8.29	6.52	5.44	1.41	9	ns
type-token ratio	0.33	0.06	0.38	0.06	1.88	9	0.10	0.35	0.07	0.44	0.05	4.70	9	0.02
No. verbal rewards	10.50	9.58	5.60	5.71	1.38	9	ns	15.80	8.5	6.60	4.27	3.07	9	0.02
No. child's verbal replies	2.00	3.34	12.70	7.59	3.59	9	0.01	2.90	5.00	9.60	6.37	2.70	9	0.05

* ma = interactions with mothers of autists
 * mn = interactions with mothers of normals

MOTHER EFFECTS (MOTHERS OF AUTISTS Vs MOTHERS OF NORMALS)

VARIABLE	AUTISTIC CHILDREN						NORMAL CHILDREN							
	X _{ma} [*]	SD _{ma}	X _{mn} [*]	SD _{mn}	t	df	P _{ns} ^{or}	X _{ma} [*]	SD _{ma}	X _{mn} [*]	SD _{mn}	t	df	P _{ns} ^{or}
total time in position AF/CF	28.33	21.88	17.34	18.77	0.93	9	ns	11.60	6.47	12.55	23.06	0.10	9	ns
No. adult terminations of AF/CF	1.80	2.27	2.70	1.17	0.63	9	ns	0.50	0.67	1.30	1.55	1.53	9	ns
mean duration before adult terminates AF/CF	3.29	4.54	0.87	0.71	1.49	9	ns	0.42	0.59	0.78	0.71	1.20	9	ns
total duration AF/CNF	401.8	95.4	336.1	137.3	1.21	9	ns	292.6	90.88	275.7	95.01	0.49	9	ns
mean duration before adult terminates AF/CNF	16.43	9.14	12.17	7.09	1.10	9	ns	8.74	1.99	7.80	2.54	1.03	9	ns
prob. transition ANF/CF to AF/CF	0.19	0.22	0.31	0.38	0.85	9	ns	0.35	0.43	0.05	0.09	2.31	9	0.05
prob. transition ANF/CF to ANF/CNF	0.70	0.31	0.37	0.40	2.35	9	0.05	0.52	0.45	0.81	0.30	1.75	9	ns
No. of cooperative responses (non-verbal)	8.70	7.23	14.20	6.02	1.92	9	0.10	7.80	6.55	10.70	6.70	0.99	9	ns
No. of cooperative responses rewarded	4.00	4.75	6.70	4.47	1.50	9	ns	1.40	2.57	2.50	2.37	0.96	9	ns
percentage punishment errors	43.30	44.40	20.01	29.90	1.36	9	ns	0.00	0.00	0.00	0.00	-	9	-
total number of mothers words.	514.6	199.4	677.7	233.35	1.66	9	ns	528.2	182.54	564.3	184.1	0.59	9	ns
total number of mothers utterances	124.9	49.87	163.1	56.93	1.56	9	ns	99.5	38.1	109.0	36.3	0.60	9	ns
mothers mean utterance length	4.21	0.79	4.20	0.65	0.03	9	ns	5.44	1.10	5.27	0.84	0.39	9	ns
No. "guiding" statements	5.30	3.31	7.80	2.85	1.89	9	0.10	7.40	5.33	10.2	2.56	2.28	9	0.05
percentage mothers statements	46.35	12.89	51.40	14.57	0.79	9	ns	68.47	9.76	60.66	12.57	0.58	9	ns
No. of mothers questions	35.90	18.65	55.80	30.24	1.97	9	0.10	24.7	11.68	30.80	12.21	1.12	9	ns
percentage mothers questions	29.73	9.68	32.33	12.82	0.54	9	ns	25.32	8.33	30.41	11.47	1.09	9	ns
percentage mothers commands	21.40	15.37	10.89	8.29	1.88	9	0.10	5.26	6.71	6.52	5.44	0.44	9	ns
type-token ratio	0.33	0.03	0.35	0.07	0.63	9	ns	0.38	0.07	0.44	0.05	2.31	9	0.05
No. verbal rewards	10.50	9.58	15.80	8.50	1.41	9	ns	5.60	5.71	6.60	4.27	0.46	9	ns
No. of child's verbal replies	2.00	3.34	2.90	5.00	0.48	9	ns	12.70	7.59	9.60	6.37	0.92	9	ns

* o = "own" mother familiar interactions * mn = interactions with mothers of normals (unfamiliar with child)
 * ma = interactions with mothers of autists (unfamiliar with child)

FAMILIARITY EFFECTS (FAMILIAR, "OWN" MOTHERS Vs MOTHERS UNFAMILIAR WITH THE CHILD)

VARIABLE	AUTISTIC CHILDREN							NORMAL CHILDREN						
	X _o [*]	SD _o	X _{ma} [*]	SD _{ma}	t	df	P _{ns} ^{or}	X _o [*]	SD _o	X _{mn} [*]	SD _{mn}	t	df	P _{ns} ^{or}
total time in position AF/CF	22.61	19.66	28.30	21.88	0.48	9	ns	12.09	10.79	12.55	23.06	0.05	9	ns
No. adult terminations of AF/CF	3.00	3.54	1.80	2.27	0.86	9	ns	1.20	1.40	1.30	1.55	0.15	9	ns
mean duration before adult terminates AF/CF	11.36	11.24	3.29	4.54	1.28	9	ns	0.75	0.99	0.78	0.71	0.07	9	ns
total duration AF/CNF	390.2	89.29	401.8	95.39	0.30	9	ns	289.7	71.75	275.7	95.01	0.38	9	ns
mean duration before adult terminates AF/CNF	114.89	8.65	116.40	9.14	0.37	9	ns	10.70	5.74	7.80	2.54	1.52	9	ns
prob. transition ANF/CF to AF/CF	0.36	0.28	0.20	0.21	1.57	9	ns	0.31	0.29	0.05	0.09	2.59	9	0.05
prob. transition ANF/CF to ANF/CNF	0.52	0.31	0.71	0.31	1.32	9	ns	0.67	0.29	0.81	0.30	1.14	9	ns
No. of cooperative responses (non-verbal)	8.90	6.65	8.70	7.23	0.06	9	ns	9.60	3.63	10.70	6.70	0.44	9	ns
No. of cooperative responses rewarded	3.40	4.17	4.00	4.75	0.30	9	ns	2.77	2.35	2.50	2.37	0.18	9	ns
percentage punishment errors	65.56	43.40	43.30	44.41	1.27	9	ns	0.00	0.00	0.00	0.00	-	9	-
total number of mothers words	428.7	189.3	514.6	199.4	0.97	9	ns	424.6	151.1	564.3	184.1	1.86	9	0.10
total number of mothers utterances	109.9	45.95	124.9	49.87	0.71	9	ns	94.6	35.83	109.0	36.32	0.98	9	ns
mothers mean utterance length	3.87	0.66	4.21	0.79	1.03	9	ns	4.54	0.48	5.27	0.84	2.28	9	0.05
No. of "guiding" statements	5.70	4.53	5.30	3.31	0.21	9	ns	10.00	4.53	10.20	2.56	0.08	9	ns
percentage mothers statements	47.19	14.89	46.35	12.89	0.13	9	ns	67.17	9.06	60.67	12.56	1.32	9	ns
No. of mothers questions	23.20	20.20	35.90	18.65	1.39	9	ns	22.2	13.05	30.8	12.21	1.50	9	ns
percentage mothers questions	18.22	11.31	29.73	9.68	2.33	9	0.05	22.24	6.68	30.41	11.45	1.88	9	0.10
percentage mothers commands	30.56	20.21	21.40	15.37	1.17	9	ns	6.65	4.51	6.52	5.44	0.06	9	ns
type-token ratio	0.34	0.06	0.33	0.06	0.16	9	ns	0.44	0.03	0.44	0.05	0.15	9	ns
No. of verbal rewards	12.60	11.57	10.50	9.58	0.47	9	ns	5.50	4.88	6.60	4.27	0.51	9	ns
No. of child's verbal replies	6.30	8.90	2.00	3.34	1.42	9	ns	7.50	5.85	9.60	6.37	0.78	9	ns

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ABSTRACTTHREE ASPECTS OF CHILDHOOD AUTISM:MOTHER-CHILD INTERACTIONS, AUTONOMIC RESPONSIVITY,
AND COGNITIVE FUNCTIONING.

A survey of the current literature published in the field of childhood autism suggested that despite a rapidly expanding body of research, we have little definitive understanding of the aetiology of the condition or a highly efficacious treatment model. However, there is a growing consensus that the autistic child suffers from a primary organic impairment, resulting in particular deficiencies in cognition and language, and consequently he displays pronounced learning difficulties.

The literature survey also indicated that there is a lack of empirical data on the manner in which parents interact with their autistic child, including the contingencies they deliver upon his characteristic responses, and the influence of the child upon the adult's behaviour. Analysis of mother-child interactions were conducted therefore, using diads with normal and autistic children and their mothers. Differences were found between the behaviour of mothers of normal children and mothers of autistic children on a number of verbal and non-verbal measures relating to their mode of interaction with these children. Such data may have potential utility for the design of generalizable behavioural treatment programmes for autistic children, with parents as the primary change agents.

The second study involved an analysis of psychophysiological data collected from a group of non-verbal autistic children who typically display infrequent, abnormal or unpredictable overt responses to important environmental events including traditional reinforcers, novelty and social stimulation. Autonomic data appeared suitable for determining the effects of such environmental stimuli, which also may have implications for treatment design and for an understanding of the aetiology of childhood autism.

The third area of investigation consisted of three studies designed to investigate further the apparent deficits and abnormalities in cognitive functioning that have been reported recently in experimental research literature. These studies involved binary sequence learning, concept attainment and probability-learning tasks, and contrasted the performance of autistic children with normal and subnormal control subjects. It was concluded from these data that there is evidence of abnormalities in functioning on cognitive tasks and that they may be particularly related to the nature of strategies adopted in such tasks and the failure to utilise task-relevant information feedback in an appropriate manner.