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Childhood Autism: An Investigation of Aetiological Factors in Twenty-five Cases

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Introduction

In 1943 Kanner described a disease entity which he referred to as 'Infantile Autism'. Creak and Ini (1960) found no evidence of psychogenic aetiology in a study of 200 parents of autistic children. Bender (1955) retrospectively studied children seen at Bellevue Hospital for behavioural or developmental disorders and who had been diagnosed as schizophrenic before seven years of age. No preponderance of the sophisticated, intelligent, well-to-do parents as described by Kanner was found; every racial and religious group was represented, Jews predominating slightly. A strong genetic predisposing factor was indicated—40 per cent of the parents were schizophrenic. Stroh (1962) insists that the child's condition cannot be attributed to maternal mishandling, and Wing (1966) has said 'It is possible to interpret the data to support both environmental and biological theories of primary causation, but on the whole the former argument is very difficult to sustain.'

The present study, made in Western Australia, was designed to examine the possible aetiological factors and to compare autistic children selected on the nine criteria of Creak (1964) with a control group of clinically normal children. The parents of each group were also compared.

Method

The experimental sample.—Children, aged between four-and-a-half and seventeen-and-a-half years, whose parents were accessible and willing to co-operate in the testing, were studied (N = 25).

The Control sample.—The headmasters of State schools situated near the hospital where physical investigations were undertaken submitted lists, and parents willing to participate were chosen. Normal children without a history of neurological disorder were accepted, and each autistic child matched for age, sex, parents' socio-economic level and culture group.

Investigations

I: Children: (D J and G below omitted for controls).

A. History of illness.
   Ante-natal, natal, neo-natal; intercurrent illnesses.
B. Developmental milestones.
   Age of onset.
C. Social history.
   Birth order, size of family, sibs affected, marital status of parents, demographic origin.
D. Neurological examination.

Particular attention was given to bodily stature, mental state, head circumference, cranial nerve function and the condition of motor and sensory systems.

E. EEG studies.

Appropriate artifacts were induced by the recordist in the tracings of the control children as they were generally more co-operative than the experimental subjects. It was felt this procedure would reduce bias during interpretation.

Tracings from both groups were interpreted blind in random order, only the age of each child being known by the interpreter.

F. IQ, Vineland social maturity scale, and present placement.
G. Rating on the Rimland diagnostic check list form E2. (Rimland 1965.)
H. Speech assessment.
I. Height and weight.

II: Parents:

A. Assessment of IQ by progressive matrices (1938), educational and occupational level.

B. Personality assessment by the Maudsley personality inventory.

C. Cornell medical index.

D. Family history.

Results

A. Thirteen babies out of 20 with known natal histories in the autistic group were post-mature, as against 1 control, significant at $p = \cdot01$, $\chi^2 = 7\cdot999$. Post-maturity was defined as 'those babies born to mothers with regular menstrual cycles (25–30 day) whose pregnancies had reached 287 days after the start of the last menstrual cycle.'

One quarter of the post-mature babies had birth weights two standard deviations below the mean for their gestational age.

There were more complications of labour in the experimental group than the controls ($p = 0\cdot001$, $\chi^2 = 13\cdot56$). The complications included uterine inertia, false labour, obstructed second stage, protracted labour, maternal transfusion, anaesthetic problems, etc.

The incidence of forceps and otherwise assisted delivery was also greater in the patient group (13 cases) than in the controls (2 cases) $p = \cdot01$, $\chi^2 = 10\cdot68$. Abnormal conditions of the child noted at delivery occurred significantly more frequently in the experimental group, e.g. difficulty with resuscitation, cord around neck, fractured skull, cyanosis, head moulding, bruising, jaundice ($p < 0\cdot0004$, $\chi^2 = 10\cdot96$).

There were significantly more neo-natal complications in the patient group (13 cases against 0 in controls) including cyanosis, child in humidicrib, etc., ($p = 0\cdot00001$ $\chi^2 = 16\cdot34$).

B. *Developmental milestones:* The age of onset was obtained from the parents' account and presents a problem of validity; it may have been that an insidious onset passed unnoticed. Two groups emerged: The first (15) consisted of children who had demonstrable cerebral impairment with early delay in reaching milestones; the second group (10) did not cause concern until their third year. (Sitting, crawling, and walking had occurred normally.)

Twenty-two subjects had been identified as ill by the end of the third year. In the remaining three cases there was uncertainty as to age of onset.

Sixty per cent of the autistic children were walking by the age of 15 months compared with 84 per cent of the controls. This difference is significant at the ·005 level ($\chi^2 = 2\cdot48$). Words had been spoken by 52 per cent of the autistic children by two years, and by 96 per cent of the controls ($p = \cdot0001$).

C. There were four children in the autistic group who had siblings with positive psychiatric histories. Two of these were from the same family of five children. There were no affected sibs in the control group.

D. (A detailed account of the neurological and electroencephalographic findings forms the basis of a separate report to be published.) Fourteen children had unequivocal evidence of organic neurological disease, but only 12 of these were so categorized on the basis of history and neurological signs alone. One of the other two subjects had phenylketonuria, and the other had a recurring temporal spike in the EEG. Seven other subjects had evidence of probable organic disease on the basis of equivocal physical findings. Thus there were only four children remaining in the group who had no evidence suggestive of neurological dysfunction.

Of the 12 children with definite evidence of organic neurological disease there were seven with epilepsy (five major, one minor and one mixed). Positive physical findings in the entire group of autistic children included microcephaly (1), hypotonia and hyporeflexia (5), corticospinal tract involvement (8), defects of ocular movement (2), dystonia (2), ataxia (5), abnormal movements (1) and dysarthria (1).

One head circumference measurement was at the 97th percentile. One at the third percentile was in a child who had other neurological abnormalities. The child with head measurement below the 0·5 percentile had a fractured skull at birth and manifested spastic quadriplegia and grand mal. All the other measurements (when assessed for age according to standards used

in this series (Vickers and Stuart, 1943) fell within normal limits.

E. *EEG Studies:* In the experimental group the EEG's of 13 subjects were abnormal, three children had tracings within normal limits, and three subjects were not tested. Five tracings were virtually unreadable through artifact and one showed borderline abnormality.

The abnormal findings were gross in one, with focal abnormalities and epileptiform discharges; moderate in four (2 diffuse abnormalities) (2 epileptiform discharges); and mild in 8.

In the control group 5 (20 per cent) had abnormal EEG's—three with mild diffuse abnormality and two with borderline diffuse abnormality—18 were normal, and two were not tested. The difference between the groups is significant at p = ·0001.

F. *IQ, Vineland social maturity scale and present placement:* It was possible to administer a complete WISC to only one autistic child and his were the highest scores obtained. Other results were based on an incomplete profile, or on the Vineland for the twelve children who were untestable. Scores were in the severely subnormal range for all children except three whose results were at the high grade defective level. All controls except four were at or above the 50th percentile and no children were below the tenth percentile.

None of the 25 autistic children were placed normally in a school. Eight in the age range 6–17 years were patients in a mental hospital, while nine of the remainder (ages 4–12 years) attended the 'autistic centre' daily from home. One of these, an eleven-year-old boy without speech, showed an islet of intelligence (Block design scales score = 17) and was reported to have acquired a few words during a course of operant conditioning.

TABLE I

Autistic children, their age and IQ according to placement

Placement	Age range	Number of children	Range of IQ
Sheltered workshop ..	15	1	Above 55
Occupational centre ..	9–17	4	45–55
Autistic centre	4–12	9	Below 55
Attending village school ..	14	1	43
Child minding centre ..	16	1	Below 30
Residential home for defectives	13	1	Untestable
Mental hospital	6–17	8	Untestable

G. *Rating on the Rimland check list:* The symptoms most commonly shared by the autistic group were those usually associated with 'autistic symptomatology' lack of awareness of other people, inability to tolerate change, and 'rare signs of pleasure'. All but one child had delayed speech development. Clumsiness and attacks upon the self were two further features which were demonstrated by more than half the group. These characteristics have been well documented by other writers.

The three children with IQ's above 50 differed from the group in that they could show pleasure, were less aggressive and less prone to injure themselves. One of these, with the islet of intelligence, differed from the other two in that he was well co-ordinated physically, did not indulge in spinning, and had very early acquisition of speech which was subsequently lost.

H. *Speech Assessment*: All children in the series had retarded speech development. Two boys (IQ 55–67) had useful speech at a concrete level, but with no attempt at conceptualization: Eight used phrased speech but with no attempt at communication. Two siblings using single words had a father who reported himself as unable to communicate efficiently before the age of six. No child was completely mute.

Parental Personality assessment: All but eight parents in each group completed the MPI, and personality characteristics were compared between the mothers and fathers within each

group as well as between groups. There was no significant difference in the personalities of the mothers of the two groups but the MPI indicated that the fathers of the autistic children were significantly more neurotically introverted than the fathers of the control group ($p = \cdot0001$). This was confirmed by the Cornell medical index, completed by the parents of 17 autistic and 16 normal children when the number of positive answers in the psychiatric sections was compared. It was seen that the fathers of the autistic children were highly neurotic compared with the fathers of the controls (the same trend, though less marked was present for the mothers).

A comparison of the mothers' and fathers' personalities within each group showed that both parent's personalities were more divergent ($T = 2\cdot025$ and $p = \cdot02$) in the control group than in the experimental group.

Parental family histories: Family histories were obtained from 23 autistic and control families. The histories differed as regards alcoholism (8 autistic: 1 control), psychiatric illness (8 : 3), and mental retardation (6 : 2). These occurred significantly more frequently in the autistic families ($p = \cdot001$). Epilepsy, diabetes, rheumatic fever, rheumatoid arthritis, coronary disease, Parkinson's disease, tuberculosis, ulcers, allergies and migraine occurred in both family histories.

Non-significant findings: Among the children, maternal illness during pregnancy, medication in ante-natal period, maternal age at birth, birth order, size of family, marital status of parents, demographic origins, height and weight did not correlate with autism. Among the parents there was a normal distribution of IQ in both groups and all occupations were represented, with no preponderance of parents at the professional or administrative level.

Discussion

Lotter (1966) estimated the prevalence of autistic conditions in Middlesex to be 4·5 per 10,000 children. Of these 2·1 per 10,000 showed 'behaviour closest to Kanner's syndrome'. Wing (1966) gives the number of autistic children in England and Wales as 3,000. There is no reliable information on the prevalence of early onset psychosis in Australia (Repin, 1966) but Barnett (1968) has suggested a total of 1,500 to 2,000 cases so that the incidence of autism in Australia may be approximately three times as great as in England and Wales. The present series is not a population survey, but it is thought to represent about half the autistic children in Western Australia, or an incidence of only 0·6 per 10,000.

This investigation shows that autistic children can be differentiated from controls by their longer gestation period and history of more complications during labour, delivery and the neonatal period. There is ample evidence to substantiate the increased risk to babies born after the 287th day of gestation. Walker (1954) has commented on the definite fall in the mean oxygen saturation of the blood in the umbilical vein after the 41st week in mothers in the age range 25–30, the babies being considered to have been at serious risk from intra-uterine anoxia. The present study included a high number of mothers in this age range. Our results are in line with the findings of Taft and Goldfarb (1964) who compared the birth records of schizophrenic children with two control groups and found more reproductive complications in the former.

The greater incidence of neurological and EEG abnormality and the history of epilepsy found in the experimental group may well be connected with this peri-natal history. The number of autistic children with a history of epilepsy is comparable with those reported by Schain and Yannet (1960).

The intellectual retardation of the autistic group is very marked. Only the occurrence of characteristic behaviour patterns seemed to justify their separation diagnostically from any heterogeneous group of retarded children. With current treatment methods there appear to be no grounds for optimism regarding general or specific potential ability.

A comparison of family histories revealed a significantly greater incidence of alcoholism, psychiatric illness and mental retardation for the autistic group. It calls for a further study to determine the extent of similar findings in a matched group of non-specific intellectually retarded children.

Interesting possibilities are opened up by the finding that mothers and more particularly fathers of autistic children have higher scores on tests purporting to measure neuroticism than parents of control children, and that the latter parents have more divergent personalities. It could be postulated that the normal child is reared in an emotional environment where the neuroticism or instability of one parent is balanced by the stability of the other; on the other hand the autistic child is reared in a setting where the characteristics of one parent's personality are similar to and reinforce those of the other. The cause and effect of these differences needs further exploration.

Summary

1. Twenty-five autistic children were investigated and compared, as were their parents, with a matched control group.

2. Mothers of patients had a significantly longer gestation period and more complications of labour at every stage than mothers of the control group.

3. 56 per cent of the subjects showed unequivocal evidence and a further 28 per cent probable evidence of organic cerebral nervous system disease on the basis of combined neurological and electroencephalographic assessment.

4. No subject scored above the high grade defective level on IQ testing and the patients were slow to develop speech when compared to controls.

5. Fathers of patients were significantly more neurotic than fathers of controls. Both parents of patients gave higher scores on the psychiatric section of the CMI and appeared to have less divergent personalities than parents of the control group.

6. Alcoholism, psychiatric illness, and mental retardation occurred significantly more often in the families of autistic children.

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References

Barnett, V. A. (1968). Personal Communications.

Bender, L. (1955). *Emotional Problems of Early Childhood*. New York: Basic Books.

Creak, M. (1964). 'Schizophrenic syndrome in childhood —further progress report of working party.' *Develop. Med. Child. Neurol.*, **4**, 530.

—— and Ini, S. (1960) 'Families of psychotic children.' *J. Child Psychol.*, **1**, 156.

Kanner, L. (1943). 'Autistic disturbances of affective contact.' *Nerv. Child.*, **2**, 217–50.

Lotter, V. (1966). 'Epidemiology of autistic conditions in young children.' *Soc. Psychiat.*, **1**, 124.

Repin, G. (1966). 'Early infantile autism.' Thesis for Diploma in Public Health.

Rimland, B. (1965). *Infantile Autism*. London: Methuen.

Schain, R. J., and Yannet, H. (1960). 'Infantile autism. An analysis of 50 cases and consideration of certain neurophysiological concepts.' *J. Ped.*, **57**, 560–7.

Stroh, G. (1962). 'Psychosis in childhood.' *Pub. Hlth.*, **77**, 21.

Taft, L., and Goldfarb, W. (1964). 'Prenatal and perinatal factors in childhood schizophrenia.' *Develop. Med. Child. Neurol.*, **6**, 32–43.

Vickers, V., and Stuart, H. C. (1943). 'Anthropometry in the pediatrician's office: norms for selected body measurements based on studies of children of North European stock.' *J. Pediat.*, **22**, 155–70.

Walker, J. (1954). 'Foetal anoxia.' *J. Obstet. Gynaec. Brit. Emp.*, **61**, 162.

Wing, J. K. (1966). *Early Childhood Autism: Clinical, Educational and Social Aspects*. Oxford: Pergamon Press.

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