Brit. J. Psychiat. (1963), **109**, 84-89

Childhood Psychosis

A REVIEW OF 100 CASES*

By E. MILDRED CREAK

A working party (*B.M.J.*, 1961) recently formulated nine points which they felt distinguished the early childhood psychosis or schizophrenic syndrome of childhood, and this paper describes some features of 100 such patients seen at Great Ormond Street. The nine criteria, which were strictly applied, can be summarized as follows:

1. gross and sustained impairment of emotional relationships with people;
2. apparent unawareness of his own personal identity to a degree inappropriate to his age;
3. pathological preoccupation with particular objects or certain characteristics of them, without regard to their accepted functions;
4. sustained resistance to change in the environment and a striving to maintain or restore sameness;
5. abnormal perceptual experience (in the absence of discernible organic abnormality);
6. acute, excessive and seemingly illogical anxiety as a frequent phenomenon;
7. speech either lost, or never acquired, or showing failure to develop beyond a level appropriate to an earlier age;
8. distortion in motility patterns;
9. a background of serious retardation in which islets of normal, near normal or exceptional intellectual function or skill may appear.

Organic Factors

Are childhood psychotics a homogeneous group or do they include cases arising from varied aetiologies? Is the syndrome often or ever primarily due to organic causes affecting presumably the central nervous system and preventing normal integrative cerebral function? The definition in the nine points is intended to include the possibility of this condition arising from multiple causes, among them organic factors. Point 5 specifically excludes psychotic withdrawal as it is sometimes seen in blind and deaf children, where the failure to maintain communication stems from a defect in the sensory (afferent) in-put mechanism. Much speculation is taking place at the present time as to possible organic factors of a more subtle kind in the form of biochemical deficits, as in phenylketonuria and chromosomal abnormalities, where we may in the future succeed in exposing the abnormality even if the way in which the child's capacity is affected is not understood.

The following case (not from the present 100) illustrates this:—

Case 1

When first seen at the age of 3 years 9 months, this was thought to be an autistic child, but she was primarily admitted because of backwardness in speech and vacant spells in which she adopted an odd posture, sucking her thumb and screwing up her eyes. Development throughout the first year of life had been normal. It seemed certain that the attacks, which were petit mal, had begun during this year. There was also a history of screaming, self-whirling, and extreme jealousy of a younger sibling. Skull X-ray and air-encephalogram established the correct diagnosis of tuberose sclerosis, confirmed at autopsy. For a brief period only, before the attacks grew more frequent and the child more apathetic, she could have been mistaken for an autistic child. There were no certain localizing signs in the C.N.S. when first seen.

There were 12 cases of epilepsy among the 100 psychotics, against an expected incidence of around 1 per cent. of established epilepsy. Five of these could be classified as infantile

* Based on a paper read at a meeting of the Mental Deficiency Section of the Royal Medico-Psychological Association, February, 1962.

https://doi.org/10.1192/bjp.109.458.84 Published online by Cambridge University Press

convulsions, one following the pattern of frequent salaam spasms. Another had a so-called teething convulsion with no subsequent attacks, and here the association may be quite irrelevant. Two girls each had a single convulsion between one and two years of age; one made a good recovery from her psychosis but the other did not.

The remainder form an interesting group about which speculation must continue, checked by repeated EEGs and long-term oversight. In all 7 children (2 girls and 5 boys), the onset of fits came long after the onset of psychosis. Four had their first fit before puberty at 7, 7½, 8 and 10 respectively. Two had fits starting around the age of puberty, at 13, and one boy, whose psychotic illness remitted to the point where he could do simple factory work, had his first fit at 20. All began their psychotic illnesses in early childhood, mostly between two and three, while two were lifelong autistic children. One, a girl, died in her first fit aged 10, and post-mortem showed a normal brain. Epileptic cases within the psychotic field may give us clues to aetiology, but here they significantly failed to do so.

The high incidence of epilepsy may be due to the inclusion of organic cases in this psychotic group; but several instances of this schizophrenic-like psychosis in early childhood offered no other evidence of physical brain damage. As the above case of tuberose sclerosis shows, a sufficiently severe disorganizing process will tend to reproduce, in young children, just that quality of detachment and emotional confusion, which we tend to associate with psychotic states. Add to that the perseverative tendencies, the failure in speech and communication and vegetative disorders such as disturbed sleep and vagaries in appetite, and it is clear that the differential diagnosis may prove very difficult. Where we do not know quite what to look for in the field of physical disturbance, the distinction between a psychosis, an inherent mental retardation and an on-going structural disorder is only possible on a long-term basis.

Two of the 100 cases recently died in hospitals, adding to our fairly complete knowledge of their lives some quite unexpected findings at autopsy.

Case 2

J.S. (13.10.33), male, was seen at the age of 9 years. He was the only living child of elderly parents whose first had died at birth. Labour was induced because of pelvic contracture and resulted in a forceps birth: weight 7 lbs. 3 ozs. He was breast fed, easily weaned at 8 months, and sat, stood and walked early. Speech began at 14 months but is said to have made no progress after 4 years, indeed at about 3½ years he appeared to lose interest and to withdraw somewhat. He had been what his mother called a "practical child", interested in the working of toys. This ceased between 3 and 4 years. Screaming fits, tantrums and fears became frequent. He was abruptly awakened by a firework and thereafter began to be afraid of falling asleep. By the time he reached school age he was unable to attend school because he clung terrified to his mother. By this time he was speaking very little and was regarded as backward. When first seen at Great Ormond Street, treatment was attempted, as he showed no physical signs or stigmata. It was further evident that his mother completely identified with him and was herself suspicious almost to the point of being paranoid. We tried to discover whether rapport could be established, and speech, at first non-existent, began again with occasional whispered remarks. He was physically restless, pursued no constructive or sensible activity and tantrums were frequent. Treatment was soon abandoned since it appeared to arouse excitement, making him more difficult to manage and control at home. As he grew older this impulsiveness made him a danger to himself and others, and he was admitted to a mental hospital. His behaviour in the ward was more like a defective than a chronic schizophrenic. He was mute except for occasional echolalia, and rather noticeably "escaped" from social contact by leaping away to the other side of the ward. He was greedy and would respond to the crackle of a sweet wrapping; it was indeed his greed in swallowing unchewed food that led to his sudden death in a choke, aged 28. At autopsy, Dr. Bodian found: Externally normal brain; showed widespread neurolipidosis on section; most neurones contained a glycolipid staining positive for fat with P.A.S. Secondary changes such as loss of neurones, gliosis and loss of myelin were quiescent and not actively progressive.

The neuro-lipidosis was a process which must have been going on for many years and which in all probability was the start of his mental illness. With the background of abnormal family life, it had been easy to suspect a psychogenesis: there were certainly no physical signs in the C.N.S. when he was first seen and at no time were there any fits or any paresis. It is interesting to wonder at what point this diagnosis could have been made by means of brain biopsy.

The other boy, T.B., seemed truly schizophrenic. His physical appearance was that of an alert, sensitive and intelligent looking child. His terminal illness was a slowly progressive

https://doi.org/10.1192/bjp.109.458.84 Published online by Cambridge University Press

deterioration, but his appearance never coarsened.

Case 3

T.B. (16.2.36) the second of 4 children was born to healthy parents. T's elder brother was severely spastic and cared for by maternal GM since the patient's birth. While this situation certainly caused additional worry in the parents' minds, this elder brother's death seems to have had only an indirect connection with the patient's illness around the age of 2½ years. His was a normal birth in the breech position following a short labour. He, too, was breast fed, and his early development appeared normal in every way. He spoke well by 2 years though not yet in sentences. At 2½ he suddenly stopped speaking and became babyish in habits. He started to wet his bed; sleep was disturbed often for hours when he would sing in bed or talk to himself and sometimes wander round the house. He was mostly silent but easily upset by changes in routine or attempts at control. He was excessively timid, but when demonstrated to a roomful of students he paid no heed to them, walking across the class to the wall, where he grimaced, whirled himself about and muttered unintelligibly. Stereotyped movements such as rocking and spinning were often seen. Feeding was also a problem. By 1951, around 15, he became too big, unpredictable and noisy to manage at home with two younger sisters, and he was moved to St. Audry's Hospital, where he died, aged 25. He remained solitary, moody and unoccupiable. On one occasion a severe haematemesis was thought to be due to his having possibly eaten a poisonous plant in the grounds. He would stuff things in his mouth, but even terminally his condition was regarded as schizophrenic rather than defective. At autopsy Dr. Bodian found: Externally normal brain, showed evidence of widespread neurolipidosis on section, with glycolipid staining positively for fat with P.A.S. Evidence of neuronal loss, past demyelination and acute demyelination notably in the left posterolateral tract of the spinal cord.

Outcome

Though the process seems sometimes to burn itself out as in adult schizophrenics, and in some cases a rescue operation aimed at re-awakening the capacity to form relationships may avert the slide-off into complete isolation, yet the overall results are far from encouraging. I have assessed them on the simplest possible criteria: it is obviously difficult to judge a child who is quietly living at home and attending a training centre. They are vastly "improved" on the initial hyperkinetic state, but probably moving into a chronic and irrecoverable position. Being at home as against being in an institution may well depend on the parents' intelligence, their means and their circumstances. I have not attempted to assess these cases, but have singled out for comment those who have significantly improved or recovered, relating this to treatment, and those, by contrast, who are permanently institutionalized.

Out of 100 children, 17 have recovered or significantly improved. That is to say they attend ordinary schools, cope with an ordinary curriculum or are working. (Two of these are in schools for maladjusted where they present no particular problem.) Of these 17 cases, 9 have improved in relation to treatment (analytically-oriented psychotherapy) and 8 without treatment, other than occasional consultations and counselling. Two of these 17 were reactive psychoses to a known trauma.

Forty-three children are permanently institutionalized as severely mentally retarded persons.

This leaves 40 children living at home, attending Rudolf Steiner or other E.S.N. schools or training centres.

Early Development and Outcome

With few exceptions, these children were first seen in their early childhood. The mother's history of early development was, of course, retrospective. She had, perhaps, an emotionally-determined stake in persuading us, and herself, that "he was all right at first". She may have hung her explanation of when things went wrong on some distressing episode, and there were very few detailed early observations made on an infant thought to be normal but later proving to be psychotic. Tramer's classical case (1934, 1935) furnishes us with possibly the best example. The history of early milestones was classified here as "normal", "delayed", and "inconsistent". "Delayed" meant consistently delayed in all aspects of development, while "inconsistent" covered those cases where, for example, a child was noted to be very early sitting and crawling, but late in walking. The development of normal speech was excluded from this judgment, as a high proportion became deviant before speech had really got under way —and in some it never did. The milestones referred to were sitting, standing walking, grasp, playing with objects, noticing, meaningful babble, single words or cries such as "dada"—

https://doi.org/10.1192/bjp.109.458.84 Published online by Cambridge University Press

"mama", often a start to habit training, and the ability to feed normally.

Of the 100 children, 46 showed development which from these aspects could be regarded as fully normal; here and there development was even quite precocious. Seven of these have recovered or significantly improved.

In 27 children milestones were delayed and, interestingly enough, two of these have made good recoveries. One is now adult, pursues a skilled profession and drives his own car—admittedly socially an odd personality. The other is an anxious, rather obsessional, child whose latest test gives an I.Q. of 95, who is doing well in an ordinary school. One of this "delayed" group was slow in walking because of a congenital hemiplegia, but his psychosis did not manifest itself until the age of 7 years when it seemed to be only indirectly related to his handicap. At least 2 others were almost certainly brain-damaged children. The remaining 30 children fall into the "inconsistent" group, 5 of whom have made good recoveries. Four of them hold their own in normal schools. On the basis of early history it is therefore hard to judge who is going to do well.

Discussion

In 1930 Heller described cases which presented in much the same way as psychotic children and gave his own evidence for this being a deteriorating organically determined illness. Yakolev (1948) published a further group of cases with rather less convincing evidence from brain biopsy. It may be remembered that Tramer's case, referred to above, appeared to deteriorate, and Tramer himself accepted an extensor response as evidence of complete physical and mental regression.

Cases such as my cases 2 and 3 tempt one to think in terms of a clear-cut aetiology, as yet unknown. However, a study of the whole group of psychotics does not allow such an easy solution. It might help to look at the group of non-communicating children as a whole.

Luria (1961) remarks that "speech enters integrally into the structure of mental processes and that it is a powerful means of regulation of human behaviour", and goes on to point out that without speech, the developing child lacks both understanding of the world around him, and any means of regulating his behaviour in terms of human relationships. It is exactly this undirected "floating" activity which creates such problems for the psychotic child and those caring for him. This is also the basis for the widely held view that the prognosis is invariably bad for the non-speaking autistic child who fails to develop speech. This is however one of the most difficult features in studying this condition, and perhaps it matters less than we think what is the starting point of the disorder. Obviously withdrawal can effectively prevent relationships, and understanding may be impossible without language as a means of reference. Therefore in the early diagnosis of these cases, and especially in selecting cases for treatment, the focal point for success or failure is likely to be the possibility or otherwise of establishing a relationship (if necessary preverbal as the earliest human relationship must be), and from this an effective means of communication. It then becomes possible to distinguish the child who is really without the means of communication from the child who seeks to communicate, but has no effective language with which to do so. As in other schizophrenic situations, communication can be indirect and much distorted by what Arieti has called paleosymbols (Arieti, 1955). That a child should use language in a primitive way, individual to himself, is a usual enough experience as a phase; in the normal child, personal ties are so strong and so badly needed, that his drive is always toward acquiring a means of self-expression which will be understood and which will achieve his ends. If this point in development is by-passed, then in purely biological terms, the child learns to exist without relationships and his physical development proceeds, lacking any directive pattern.

This surely is why successful therapy so often involves, not a blindly permissive situation, but certainly consciously permitted regression in the hopes that earlier and more primitive means of contact may afford a relationship which can be trusted and subsequently built on.

Earlier diagnosis is always important. Kanner (1942–3) has so well described those children

https://doi.org/10.1192/bjp.109.458.84 Published online by Cambridge University Press

who seem autistic from birth. (It is interesting that neither of the two undoubted organic cases belongs to this group.) This condition is sometimes thought a variety of mental defectiveness, but perhaps it is better to move away from this idea of a fixed degree of failure in mental development, and see in certain children a great proneness to live apart. All too readily they accept isolation—"pram at the bottom of the garden" and "so good I didn't know I had a baby in the house", or they fret and clamour excessively for a comfort they never seem able to derive from the mothering they get. There appears to be a group among them of not very intelligent children, neither quick nor responsive, who can be watched slipping into this state. Sometimes the regression is associated with intercurrent illness, or with the mother's withdrawal to have a new baby. Sometimes it seems to be no more than a failure to move on into the next stage often at just the point where language is expected to build up into a really adequate means of communication. Sometimes a quite acute episode of withdrawal may occur, as in one child with a sudden surge of anxiety, associated with father leaving home, who screamed a lot and was isolated in hospital as a suspected encephalitic. This resulted in a psychosis lasting six months during which the prognosis seemed just as bad as in the many other psychotics who do not recover. Such points need carefully considering by those who counsel young parents, not necessarily all of them psychiatrists.

Mental deficiency is the condition most readily confused with childhood psychosis, and indeed they have much in common. The psychotic child while in the early withdrawn stage of his illness is the most ineducable of any, and conversely one meets among retarded patients odd skills, obsessional drives to no very obvious purpose, active withdrawal from social contact, and many instances of bizarre behaviour. The distinction whether these are psychotic children, or examples of psychotic behaviour in severely retarded children, is almost an academic one. Such children function at a grossly retarded level, even if it often seems that it is their emotional isolation that closes the doors of learning for them.

These cases show that even with reasonable care and long-term oversight it is still possible to be in the dark as to the real diagnosis. Greater precision in diagnosis should be possible and the nine points (*supra*) were designed to help in this; but we will always probably be faced with the problem "is he mad because he's limited, or limited because he's mad?"—to put it crudely.

The Problem of Treatment

In the past the diagnosis of serious mental retardation has often been given to parents although they themselves have protested vigorously that their child is different from others so handicapped. From this may stem their great unwillingness to accept placement in training centres and in large hospitals for the severely retarded. Unfortunately, no good comes of implying that detailed and careful treatment will guarantee a cure, and it is essential that parents be alerted at an early stage to the grave turn which their child's development has taken. The tragedy is that so often parents notice and call attention to small and early signs; and they may be falsely reassured if the condition is not recognized, or given the idea that only very intensive treatment will help (when sometimes it doesn't), or will be told that this child is grossly defective.

Psychotherapy as an out-patient involves a lot of travelling, and can only be done by skilled and experienced therapists. Therapeutic in-patient units require a very high ratio of staff to patients: those there are must continue to publicize their successes and their failures. Day units such as the nursery class at the Marlborough Day Hospital and the small group which the L.C.C. have opened at the Natal Centre give further opportunities for exploring the child's latent capacity for building a relationship which may in time become therapeutic. Perhaps the good results obtained in some of the Rudolf Steiner groups may stem from their insistence on a family-based grouping with a warmly protective but carefully regimented programme carried out always by the same people. Something of this kind might be possible within a larger group, in the interesting work done by O'Gorman (1958) at Smith's

https://doi.org/10.1192/bjp.109.458.84 Published online by Cambridge University Press

Hospital. For these unstable isolated non-communicating children, any large group is likely to find them increasingly withdrawn since their difficulty in conforming is based not on lack of comprehension so much as a total failure in integration. Whether this is usually non-integration *ab initio* as seems to be the case in the primarily autistic child, or whether a disintegration, as it certainly proves to have been in the two cases cited, is something which only long-term observation and better diagnosis can tell us.

Summary

Some features of 100 cases of childhood psychosis are described with reference to epilepsy, early development, and outcome. Two such patients were found at autopsy to have an unsuspected cerebral lipidosis. The problems of therapy with the non-communicating child are briefly described.

Acknowledgments

In collecting this material I would like to acknowledge help given by a substantial grant from the Mental Health Research Fund in 1958. This enabled a more detailed study of the material collected. I am also grateful to my colleagues at the Hospital for Sick Children for their co-operation in allowing me to see and report on their cases. I am particularly grateful to Dr. Bodian and his department for help with producing slides from the autopsy material. Not least am I grateful to doctors in hospitals and special schools, and to parents of these children, who have endured my persistent visits and follow-up letters. But for this, the autopsy material described here could not have been obtained.

References

Arieti, S. (1955). *Interpretation of Schizophrenia.* New York, p. 294.

British Medical Journal (1961). Vol. *ii*, 889.

Heller, T. (1930). *Ztschr. f. Kinderforsch.*, **73**, 661. Translated by Hulse, C. W. (1954). *J. Nerv. Mental Dis.*, **119**, 671.

Kanner, L. (1942–3). *Nerv. Child*, **2**, 217.

Luria, A. R. (1961). *Role of Speech in the Regulation of Normal and Abnormal Behaviour.* Pergamon Press, p. 26.

Malamud, N. (1959). *Am. J. Psychiat.*, **116**, 215.

O'Gorman, G. (1958). "A hospital for the psychotic-defective child", *Lancet*, *ii*, 951.

Tramer, M. (1934–5). *Zeitschr. Kinderpsychiat.*, **1**, 91.

——(1935–6). *Ibid.*, **2**, 17.

Yakolev, P., Weinberger, M., and Chipman, C. (1948). *Am. J. Ment. Def.*, **55** (2), 318.

E. Mildred Creak, *Physician in Psychological Medicine, Hospital for Sick Children, Great Ormond Street, W.C.1*

https://doi.org/10.1192/bjp.109.458.84 Published online by Cambridge University Press