PAPERS AND ORIGINALS

Epilepsy in childhood: findings from the National Child Development Study

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Summary and conclusions

By the age of 11 years 1043 children (6.7%) in an unselected national sample had a history of seizures or other episodes of loss of consciousness; 322 (20.8/1000) had a history of febrile convulsions without other epileptic problems. A clear-cut diagnosis of non-febrile epilepsy was established in 64 children (4.1/1000) by the age of 11 on the basis of confirmatory information supplied by family doctors and paediatricians. A further 39 (2.6/1000) were reported as having epilepsy but did not fulfil the study criteria. The progress of 59 of the 64 children with established epilepsy was reviewed again when they were aged 16. Of the 37 educated in normal schools eight (22%) had one or more seizures in their 16th year compared with 13 out of 22 (59%) who received special education.

A possible cause for epilepsy was found in 17 of the 64 (27%) children, but for the majority there was no obvious reason.

Introduction

Determining the natural history of epilepsy in childhood poses problems because doctors usually delay making the diagnosis until the condition has established itself beyond reasonable doubt. Deciding whether convulsions in young children are due to febrile or non-febrile incidents can be very difficult even

with the benefit of hindsight. These problems bedevil the accurate epidemiological study of children's epilepsy.

Many cross-sectional studies of children's epilepsy have been carried out on a variety of samples drawn from medical or educational sources, including community hospitals,^{1,2} general practice,³⁻⁵ island communities,^{6,7} and cities.^{8,9} Others have been directed to parents by door-to-door inquiries¹⁰ or through the post.¹¹ From the United States two large cohort studies have followed children with epilepsy up to 6¹² and 7 years of age.¹³ Cooper¹⁴ followed a much smaller British cohort, including 27 children with epilepsy, to the age of 15 years.

The need for more information about the long-term development of children with epilepsy remains.¹⁵ We review here the histories of 64 young people in the National Child Development Study who were identified as having epilepsy during their first 11 years and discuss their seizure patterns, possible causation, and progress up to the age of 16 and assess their condition in relation to the many other types of seizure found in childhood.

Method

The National Child Development Study (NCDS) includes all children in England, Scotland, and Wales who were born in one week in March 1958. The children were followed up at the ages of 7,¹⁶ 11, and 16.¹⁷ We studied the available medical, social, and educational questionnaire forms completed by school doctors, health visitors, and teachers on 15 496 children for details of seizures. The children participated in the study at either 7 years or 11 years, or both. The 423 children who did not survive to the age of 7 were excluded, as were those who had fits only in the first week of life. Birth histories were recorded during the 1958 Perinatal Mortality Survey.¹⁸

Of the 15 496 children with information gathered at 7 or 11 years, or both (most of them at both), 1043 were reported by their parents or school doctors to have had at least one fit, faint, or turn. In 346 cases we asked for further details from the child's general practitioner, hospital consultant, and other medical sources. These were children in whom there was evidence of recurrent episodes, an onset after 5 years, the prescription of anticonvulsant drugs, electroencephalographic (EEG) investigations, or any reference to epilepsy or where the child was attending or awaiting placement at a special school for any reason. Information was sought about the nature of the seizures, diagnostic category, type of treatment, EEG findings, aetiology, and

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prognosis. Completed documents were returned from 86% of all known sources and we obtained at least one reply from 98%. Information on events in the years from 11 to 16 was obtained from the 16-year follow up of the NCDS and was not verified by the children's own doctors.

Results and comment

Scrutiny of the records of 1043 children (6.7% of the cohort by the age of 11 years) who had experienced at least one episode of altered consciousness showed that 606 (58%) had had episodes which included single spontaneous or afebrile seizures of many types, brief faints, temper tantrums, breath-holding attacks, or behavioural problems; this group is not discussed further. The remaining 437 children included 12 who had had fits in the course of acute meningitic illness without recurrence, 322 with one or more febrile convulsions unassociated with later afebrile seizures, 39 with an unsubstantiated diagnosis of epilepsy, and 64 children who had had two or more definite afebrile seizures, which we called "established" epilepsy (see table).

Diagnostic categories of children with reported fits. Total number of study children (15 496) with information at 7 or 11, or both

Category	Total	No per 1000
"Established" epilepsy	64	4.1
Epilepsy reported by doctor but unsubstantiated	39	2.5
Febrile convulsions without later afebrile seizures	346	22.3
Febrile convulsions with later spontaneous afebrile seizures	20	1.3
Convulsions with meningitis or encephalitis	12	0.8
Breath-holding attacks, faints without convulsions, temper tantrums	280	18.1
Non-epileptic blank spells (confirmed by general practitioner or hospital)	7	0.5
Transitory afebrile convulsive episode not occurring after age 5 years	307	20.6
Convulsions reported by parent but not to general practitioner or hospital	12	0.8

UNESTABLISHED EPILEPSY

The 39 children with an unsubstantiated diagnosis of epilepsy were regarded, and reported to us by their general practitioner or consultant, as having "epilepsy," but the detailed additional medical information supplied did not support the study's definition of epilepsy as "recurrent paroxysmal disturbance of consciousness, sensation, or movement, primarily cerebral in origin, unassociated with acute febrile episodes."

Of these 39 children 24 had febrile convulsions but were treated as epileptics with long term anticonvulsants; they were also included in our febrile convulsions group. Three of the remaining 15 had single afebrile grand mal episodes and by definition were not considered to have epilepsy. Two had recurrent breath-holding attacks; two had behavioural problems or hysteria; others had less clear-cut problems including abdominal pain with an abnormal EEG (so called abdominal epilepsy), temper tantrums, seizures solely in the course of bacterial meningitis, and acute dehydrating illness without recurrence. Nine of the 39 attended a special school. All had been prescribed anticonvulsants, 38 of them phenobarbitone either singly or in combination. By the age of 16 three had had further afebrile seizures, and the others remained free of seizures although some were still prescribed anticonvulsants.

FEBRILE CONVULSIONS

By the age of 11, 366 (2.4%) children had a history of one or more seizures in the course of an extracranial febrile illness or upset. Of these, 24 were regarded and treated by one or more doctors as having epilepsy in view of the length, severity, or repeated nature of their febrile fits. A further 20 initially had febrile seizures but went on to have spontaneous fits.

Of the 346 who did not develop afebrile seizures by the age of 11, there were no excess perinatal problems such as abnormal presentation, fetal distress, unattended delivery, or low birth weight. School attainment at age 11,⁹ judged by tests of general ability, reading

comprehension, and arithmetic, was similar to that of those with no history of seizures. The prevalence of febrile convulsions was the same in all social classes with an insignificant ($p > 0.05$) excess among boys.

Over half the children (202) with febrile convulsions were treated entirely by their general practitioners, and only one (0.5%) later had seizures without fever. The remaining 164 were admitted to hospital overnight at least once. They had a much worse prognosis: 19 (12%) later had spontaneous seizures.

CHILDREN WITH ESTABLISHED EPILEPSY

By the age of 11, 64 children (0.4%) had a clear-cut history of two or more spontaneous afebrile seizures and were accepted from information supplied by general practitioners and hospital consultants as having an established epileptic disorder. There were slightly more boys (35) than girls (29), with an excess of fatherless children but no bias towards any particular social class.

Type of fits—Of the 64 children 35 had only grand mal seizures. In 18 the nature of the attacks changed with time and it was not possible to classify the fits into a single category. One child had petit mal attacks alone with classical EEG changes, and a further eight started with petit mal but later developed grand mal. Although two were reported as having temporal lobe seizures, they had relatively mild problems of giddiness and transitory confusion. Their EEGs were reported as abnormal without consistent temporal lobe changes. Eleven had at least one bout of status epilepticus lasting an hour or more.

Family history—Although family histories of epilepsy were recorded, this information is inherently subject to error because of the impossibility of obtaining contemporary medical records on parents and relatives to confirm their diagnoses. Since histories of epilepsy tend to be suppressed, adults may never know that they had fits during their childhood. Moreover, it is difficult to gather control information on the epileptic histories of parents of children without fits, because their family histories are even less likely to be known. Sixteen children had parents with a history of fits (10 fathers, 7 mothers). Four parents had had fits before the age of 10 years. The severity and type of parental fits varied greatly: one mother died in a fit, and one child had both parents affected. The fact that his fits began only after a head injury highlights the difficulty in ascribing seizures to genetic or acquired factors.

Anticonvulsant treatment in first eleven years—All but one of the children had been prescribed anticonvulsant drugs. The child who was not treated had a brief grand mal fit at 6½ years and again a year later. Of the remainder one started treatment with primidone, four with ethosuximide, and 58 with phenobarbitone, which was the sole drug prescribed for 22 children up to the age of 12 years. Thirty-six had their treatment changed from phenobarbitone—usually because of side effects rather than because of its failure to control seizures—and five had drug intoxication. Phenytoin was the most usual second drug; it was given singly or in combination to 24 children. Twenty-two children stopped drugs within 12 months of their last fit and a further 12 within 12-24 months, but anticonvulsant drugs were often continued for many years. Twenty-six were prescribed drugs for over three years after their last fit; three were still prescribed drugs over seven years after their last known fit.

Perinatal factors—Four children were members of twin pairs, but in no case was the other twin affected with epilepsy. When the twins and those with gross congenital abnormalities were excluded there was little evidence that perinatal factors caused epilepsy. Among singletons, maternal bleeding before 28 weeks of pregnancy was more common in pregnancies that resulted in a child with epilepsy ($p < 0.05$) than in pregnancies not resulting in a child with epilepsy, but low birth weight, unattended delivery, caesarean section, breech delivery, fetal distress, and maternal cigarette smoking were not found in significant excess. Ten children with epilepsy were low birth weight singleton infants, three had gross congenital abnormalities, and one had a skull fracture when 5 months old. Of the remainder, two weighed 1500 g-2000 g and four 2000-2500 g at birth. The mean age at the first fit was 42 months, which suggested that low birth weight was unlikely to be an immediate cause of epilepsy. Two had severe rhesus disease, which may have caused brain damage.

Congenital and acquired physical handicap—Although it is rarely possible to be certain that any insult or abnormality is the true cause of a child's seizures, 13 children had abnormalities compatible with secondary epilepsy. Three had cerebral palsy, presumably of congenital origin (one with cleft palate and microcephaly); two others had

cerebral palsy, one resulting from viral encephalitis, the other following a head injury thought to be due to child abuse; there were single cases of Down's syndrome and tuberose sclerosis. Three who were normal at birth later had bacterial meningitis; one of these required residential education for deafness. One had a first fit after measles and another after gastroenteritis. Another child started seizures after a road accident. The causes of two cases of epilepsy, one associated with severe mental retardation and the other with cerebellar ataxia, were unexplained.

EEG findings—Children were categorised as having epilepsy solely on the basis of clinical histories, though abstracts of EEG reports were obtained for the first 11 years. Ten children had not had an EEG, but the remaining 54 had had 101 recordings between them. Abnormal records were reported in 38, four were "inconclusive," and 12 were "normal."

School ability at 11—At age 11 43 children were in normal schools, 20 were at special schools, and one was at home. Ross and West²⁰ discussed the educational performance of these children and showed that of those at normal schools, non-academic tasks such as design copying were done well but their scores in reading comprehension and maths tests were inferior to those of children without epilepsy. Prolonged absence from school was common; 13% of those with epilepsy in normal schools and 23% of those at special schools (excluding those in whole-time residence) missed at least one month of school in their 11th year compared with only 6% of children without epilepsy.

Outcome at 16—Of the 64 children studied at 11, 59 took some part in the 16-year follow up. (Two were abroad, one was a gypsy, two were untraced.) Twenty-nine had had at least one further fit since previous contact with the study at 11, and 21 of them had had fits in their 16th year. Of those educated at special schools 13 out of 22 (59%) were still having fits in their 16th year compared with 8 out of 37 (22%) attending normal schools. Twenty-two children were receiving special education, including one in a special class of a comprehensive school, 15 were in special schools for educational backwardness (ESN (moderate) 11, ESN (severe) 4); two in schools for physical handicap; and four in special residential schools for epilepsy. Ten had been formally ascertained as ESN(M) and five as ESN(S). The remainder were ascertained on account of epilepsy (3), deafness and physical handicap (1), physical handicap (1), ESN(M) and epilepsy (1), and physical handicap and ESN(M) (1). Epilepsy appeared to be a major handicap in four children, contrasting with educational retardation in 18. Only three of those receiving special education were regarded as having enough ability to cope with everyday reading needs such as newspapers and official forms; none were taking any public examinations.

Of the 37 at normal schools 34 took the study's reading and maths tests and achieved mean scores of 20.8 and 9.9 respectively, which were not significantly different at the 5% level from the study norms of 25.3 and 12.3. Nine were taking O levels and 20 the certificate of secondary education, but their results were not available to us.

Nine of those whose career intentions were known expected to take up non-manual work and 32 manual work. Thirteen were stated by their teachers to need sheltered accommodation and be unable to enter the job market.

Discussion

The prognosis of children's epilepsy is a matter of great complexity which includes the degree of structural brain abnormality, the influence for good and ill of anticonvulsant drugs, the appropriateness of education, school attendance, stigma, and family and personal esteem. The role of each of these factors in isolation is very difficult to unravel. Children's epilepsy should be viewed as much as an educational matter as a medical problem.²¹

Both the present study and the experience of the Mayo Clinic in 1935-74²² show that about 6% of all children with at least one febrile convulsion go on to have spontaneous afebrile fits later. Among those children in our study admitted to hospital during one of their febrile fits the rate for subsequent spontaneous fits was 13% in the present study, similar to the 12% found by Wallace²³ in her large hospital-based series of children with febrile convulsions followed till the age of 8-9 years. These different rates of recurrence underline the need for community-based studies of the natural history of febrile

convulsions. Children admitted to hospital seem to have a different pattern of illness from those who stay at home.

A knowledge of the size of the child population with epilepsy is needed to plan services. The absence of any reliable whole-community studies in the past makes it impossible to know whether these conditions are becoming any more or less common.

In the past 20 years eight cross-sectional prevalence studies based on populations of over 3000 10-20 year olds with non-febrile epilepsy each using medical sources have been carried out in USA,¹ Iceland,⁷ and North-west Europe.^{2 3 6 9 14} These studies reported rates varying from 3.1-7.1 per 1000. Each study, however, used different definitions and methods. The highest figures were derived from studies which included children considered to have epilepsy on the basis of continuing anticonvulsant therapy as well as the recent occurrence of fits. In combination, these studies reported 803 people with epilepsy in a population of 207 054, giving a rate of 3.9 per 1000. This is very close to the prevalence of substantiated recurrent non-febrile epileptic seizures by the 11th birthday of 4.1 per 1000 that we found.

Using the findings of the National Child Development Study as a guide and assuming that they would apply to children born in other seasons of the year, we calculate that 2000-2500 11 year olds in England, Scotland, and Wales have a history of non-febrile postneonatal epilepsy. Over half (2.5/1000) will have further fits between their 11th and 13th birthdays. In their 16th year about 1.5/1000 will still have fits. Most of this latter group attend special schools and 0.9/1000 (representing 650 young people per year) require continued sheltered accommodation after school leaving age.

Whatever the type of epilepsy, the third who receive special education do so because of backwardness rather than epilepsy. Those at special schools were much more likely to have clear-cut organic brain abnormalities than those in normal schools. These were multiply handicapped children whose seizures were often not their main problem. Children in special education for epilepsy require expert provision for a wide range of disorders, especially mental handicap and cerebral palsy.

Could the children educated at normal schools have an intrinsically different form of epilepsy from those at special schools? Heijbel *et al*²⁴ found benign focal nocturnal epilepsy in 15% of their outpatient sample, aged up to 15 years. This form of epilepsy often does not need drug treatment and recovers by puberty. Our data were collected before this condition was well recognised, but future studies should make a point of searching for this form of epilepsy.

Despite their complexity and expense, longitudinal studies are the only means of determining the natural history of epilepsy and should be repeated at intervals. Only two other long-term studies have been reported. Cooper¹⁴ followed 27 children who developed epilepsy to their 16th year. Harrison and Taylor²⁵ followed 207 young people from their first convulsion (of any type) for a period of 20-25 years and found that although two-thirds could be regarded as doing well, the remainder had gross problems "largely hid in remote institutions or in the grave." Although our study was carried out by different means the conclusions are broadly similar. The present study shows that children able to stay in the normal educational system are much less likely to have fits in their 16th year than those in the special education group. Although epilepsy has long been regarded as a sequel of birth injury and has been used to support the concept of a "continuum of reproductive casualty," our findings support Illingworth's²⁶ view that obstetric mishaps even back in 1958 did not contribute much to the aetiology of children's epilepsy.

More fruitful areas of prevention seem to lie in the provision of wise genetic advice, especially where there is a strong history of idiopathic disorder in several members of the family, the avoidance of head injury,²⁷ and the intensive early treatment of meningitis. Failure to detect possible causes for epilepsy in the majority of children means that new approaches to the study of causation are needed. These include intensive searches for

child abuse, especially "shaking" injuries, and firmer evidence concerning the role of cytomegalovirus and other infections in pregnancy. Subclinical herpes simplex²⁸ and toxocara²⁹ infestation are sometimes held to be common causes of epilepsy, but there is little firm knowledge of their prevalence in British children with and without epilepsy. An evaluation of routine computed tomography in children's epilepsy is needed to determine whether the high detection rates of structural brain abnormality reported from French³⁰ and German³¹ special centres would be repeated in unselected British patients. Review of the findings of medical examination on the present group at 16 did not suggest that previously occult brain diseases were now coming to light.

We acknowledge with thanks the many family doctors and hospital and community paediatricians who supplied data and our colleagues at the National Childrens Bureau, London, and the university departments of child health and community medicine at Bristol, Middlesex Hospital, and Charing Cross Hospital Medical Schools for help in preparing this report.

The National Child Development Study (1958 cohort) is supported by grants from the Department of Health and Social Security, the Department of Education and Science, and the Social Science Research Council. The epilepsy substudy was financed by the research fund of the British Epilepsy Association and additional monies donated by the pharmaceutical divisions of Abbott, Bayer, ICI, and Parke-Davis.

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(Accepted 19 October 1979)

ONE HUNDRED YEARS AGO A very large number of attacks from the parasitic disease known as Guinea-worm (*Dracunculus Medinensis*) has lately been officially reported by Mr Chunter Dutt, assistant-surgeon in medical charge of the Warora Colliery establishment in Chonda, Central Provinces of India. No fewer than 180 cases came under Mr Dutt's notice from one village named Saimbul, three miles south of Warora. It appears that the Hindoos obtain their water for drinking and washing purposes from a well (the only well) in the vicinity of the village. This well has a flight of stone-built stairs, down which the people descend to fill their pots with water. It contained a large quantity of mud and dirt at the bottom; and the water was muddy and of the appearance of weak gruel or rice-water. All those who obtained water from this well suffered more or less from the disease; whilst those castes that were never allowed to touch the well were almost totally exempt from it. All that could be gleaned as to the outbreak of the disease was that men from a neighbouring village, where the disease was very rife the year before, used to go down to the well for drawing water whilst suffering with mature Guinea-worm in their legs and feet, and dip their feet in the water. From this, the reporter infers, "that the germs of the mature worm might have access to the water; and, having found a germinating soil for propagation and development, on account of filthy and muddy water, these zoosperms soon developed and infected the well." In reference to this, it is to be observed that the field-labourers, to whom the disease was to a great extent limited, have a habit of drinking from this well on returning from their work, descending one or two steps into the water to dip their hands or water-vessels therein. Thus the hands and the feet are the parts attacked by the worm, and these were the members affected in all the patients but one, in whom the worm appeared in the abdomen.

Happily, in England, we need have no fear of this formidable parasite; but in the tropics its ravages are well known and greatly dreaded. On the subject of the worm, numberless treatises (ancient and modern) have been written; but, in the writings of Kuchenmeister, Davaine, Aitken, and Cobbold on the subject, will be found all that is historically and professionally interesting. The Guinea-worm has been known from the earliest times. It is quite evident that Plutarch spoke of dracunculi when, in the eighth book of his "Symposiacon," he quotes Agatharchidas as stating that the people taken ill on the Red Sea suffered from many strange and unheard-of attacks, amongst other worms, from "little snakes, which came out upon them, gnawed away their legs and arms, and when touched retracted, coiled themselves up in the muscles, and these gave rise to the most unsupportable pains." For these are the characteristic symptoms of the attack, which make it so greatly dreaded at the present day. As to treatment, every Oriental traveller is more or less familiar with the time-honoured methods employed in the process of extracting the adult Guinea-worm. The plan followed of old is essentially the same as the one most successfully employed at the present day. This is done by winding the exposed or protruded end of the parasite round a small stick of ivory, bone, or wood, or even round a portion of adhesive plaster, card-board, or stout paper. The great thing to be avoided is the rupture of the worm, which, if it once take place, is extremely liable to produce serious inflammatory action in the infected parts. From this condition, there usually follows a considerable amount of irritative fever, which is subsequently aggravated by the formation of abscesses, fistulous diseases, and more or less extensive gangrene. As a preventive measure, all persons travelling in Guinea-worm districts should be careful to protect their feet and legs during the rainy season. (*British Medical Journal*, 1880.)