RECURRENT CONVULSIONS IN CAPE TOWN CHILDREN*

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A layman who is present when a child has a convulsion is usually considerably distressed by the experience—the more so if the child is his own. Something of this anguish is reflected in the *New English Bible* account of a father's petition to Christ: 'Have pity, Sir, on my son: he is an epileptic and has bad fits. He keeps falling about, often into the fire and often into water. I brought him to your disciples, but they could not cure him.' (*Matthew* 17: 14). The modern parent with a similarly afflicted child is no less anxious to obtain assistance and it is therefore scarcely surprising that convulsion is a common presenting complaint in paediatric practice.

At Red Cross Children's Hospital, in Cape Town, patients with a history of convulsions are referred to a special clinic when physical examination and investigation show no evidence of an active disease process. Figures reported here are based on a study of the records kept at this clinic. Children who attended for the first time after 1 January 1970 have been excluded from the study, as have those whose convulsions were invariably a concomitant of fever, and in whom no clinical or EEG abnormality could be detected. The age of the children studied and the length of time that they had attended the clinic were calculated as on 1 July 1970.

*Date received: 3 November 1970.

RESULTS

There were 225 children who fulfilled requirements for inclusion in the study and these fell naturally into 6 groups (Table I):

Idiopathic epilepsy. In this, the largest group, recurrent convulsions had occurred in children with a normal perinatal history. There had never been any disease or trauma involving the central nervous system and clinical examination disclosed no abnormality.

Perinatal brain damage. In this group there was a definite history of prematurity, obstetric complication or cerebral disturbance in the neonatal period. On examination many of these children had clinical abnormalities.

Postnatal brain damage. The birth and initial development of children in this group were normal. Recurrent convulsions dated from a specific illness—most commonly meningitis (Table II).

Mental retardation. Here, retardation had become evident at an early age, and remained the most striking feature about the child. Perinatal history was normal and there had been no subsequent significant illness.

Petit mal. These children had typical 'absence' spells and diagnostic electro-encephalogram. There was no other abnormality.

Infantile myoclonic epilepsy. These 3 children had manifest

typical 'salaam' spasms in infancy, and subsequently showed significant mental retardation.

TABLE I. CLASSIFICATION INTO GROUPS

	No. of cases	Race %			
		Coloured	Bantu	White	Asiatic
Idiopathic epilepsy	105	76	18	5	1
Perinatal brain damage	53	72	17	11	—
Postnatal brain damage	44	77	18	5	—
Mental retardation	15	67	13	13	7
Petit mal	5	80	—	20	—
Infantile myoclonic epilepsy	3	67	—	33	—

TABLE II. POSTNATAL ILLNESSES CAUSING BRAIN DAMAGE

Condition	%
Meningitis	57
Encephalitis	25
Head trauma	7
Cerebral vein thrombosis	5
Cardiac arrest	5
Cysticercosis	2

In each of these groups the children had attended the clinic on average for 2 years.

Race

Distribution of cases between the 4 racial groups reflects the racial distribution of patients attending Red Cross Hospital. In 1969 there were 183 452 outpatient attendances by Coloured children, 26 359 by Bantu children and 11 397 by White children.

Sex Distribution (Table III)

Boys and girls were found in equal numbers in the idiopathic epilepsy group, and in the group who had suffered postnatal brain damage. There were more

TABLE III. SEX DISTRIBUTION

Condition	Male %	Female %
Idiopathic epilepsy	50	50
Perinatal brain damage	57	43
Postnatal brain damage	50	50
Mental retardation	73	27
Petit mal	20	80
Infantile myoclonic epilepsy	67	33

boys in the group who had suffered perinatal brain damage, and in the group with mental retardation there was a striking male preponderance. Girls far outnumbered boys in the petit mal group.

Age of Onset (Table IV)

The earliest onset of convulsions occurred among mentally retarded children. In the two groups of brain-damaged children the onset was on average much earlier than in the idiopathic group. True petit mal does not occur before the age of 3 years, and this is reflected in the higher mean age of the children in this group. Infantile myoclonic epilepsy characteristically starts in the first year of life.

TABLE IV. AGE AT ONSET

Condition	Mean age (years)	Mean age at onset of convulsions (years)
Idiopathic epilepsy	9	4
Perinatal brain damage	7	2
Postnatal brain damage	7	2
Mental retardation	6	1
Petit mal	11	5
Infantile myoclonic epilepsy	3	0·5

Milestones (Table V)

Motor milestones were commonly delayed in children with overt mental retardation and in those who had

TABLE V. MILESTONES

Condition	Mean birth-weight (lb)	Motor milestones % delayed	Family history of convulsions % positive
Idiopathic epilepsy	8	5	38
Perinatal brain damage	6	44	28
Postnatal brain damage	7	25	15
Mental retardation	7	47	18
Petit mal	7	—	75

suffered perinatal brain damage or intracranial disease in infancy.

Family History of Convulsions (Table V)

This was recorded as positive when a close relative had experienced a convulsion of any sort, including 'febrile' convulsions. The familial predisposition to idiopathic epilepsy is reflected in this study but the incidence of positive family history in children with brain damage is surprisingly high. This finding will be further discussed.

Perinatal Brain Damage (Table VI)

In the majority of cases brain damage appeared to have resulted from asphyxia neonatorum or prematurity. In

TABLE VI. PERINATAL BRAIN DAMAGE

Condition	%
Asphyxia neonatorum	43
Prematurity	21
Caesarean section	13
Hypoglycaemia	6
Breech delivery	4
Forceps delivery	4
Vacuum extraction	2
Antepartum haemorrhage	2
Twins	2
Subdural haematoma	2
Hyperbilirubinaemia	2

13% of cases caesarean section had been necessary following complicated labour. There were a number of miscellaneous causes.

Clinical Findings (Table VII)

By definition the presence of clinical abnormalities excluded idiopathic epilepsy. Hemiplegia was the commonest clinical abnormality in both groups of brain-damaged children. Mental retardation was common in children whose convulsions dated from a postnatal illness. Among those regarded primarily as mentally retarded,

TABLE VII. PRINCIPAL CLINICAL FINDINGS

Condition	Normal	Hemiplegia	Microcephaly	Mental retardation	Cerebral palsy
Perinatal brain damage	53	17	15	4	11
Postnatal brain damage	41	32	—	21	5
Mental retardation	27	20	27	—	13

microcephaly was the commonest clinical abnormality.

Type of Convulsion (Table VIII)

The majority of children had grand mal convulsions. In each group there were a few who had both grand mal

TABLE VIII. TYPE OF CONVULSION (%)

Condition	Grand mal	Grand mal and partial attacks	Partial attacks	Myoclonic	Psychomotor
Idiopathic epilepsy	91	4	5	—	1
Perinatal brain damage	81	15	—	2	2
Postnatal brain damage	86	7	—	5	2
Mental retardation	73	7	7	—	13

and partial attacks. Partial attacks alone and myoclonic attacks were rare and only among those with mental retardation did psychomotor attacks occur to any degree.

Electro-encephalogram (Table IX)

Records compatible with epileptic dysrhythmia were found in all the major groups. EEG evidence of focal or generalized cortical damage occurred commonly in children whose history suggested brain damage. The incidence of focal abnormalities in mentally retarded children is high. In each group there were a significant number of children whose EEG was normal.

Drugs Used (Table X)

Phenobarbitone was more effective than any other drug

TABLE IX. THE ELECTRO-ENCEPHALOGRAM

Condition	Epileptic dysrhythmia	Focal abnormality	Multiple spiking	Interictal	Normal
Idiopathic epilepsy	39	24	4	16	17
Perinatal brain damage	20	31	10	12	27
Postnatal brain damage	21	33	10	21	15
Mental retardation	8	46	—	8	39

Petit mal All showed spike and wave pattern, 3 per record

or combination of drugs in all children except the mentally retarded. The addition of phenytoin often secured control in idiopathic epilepsy when phenobarbitone alone proved inadequate. In each group there were a significant number in whom control could only be achieved by using a combination of drugs. This applied particularly to the mentally retarded children. The incidence of side-effects was 11%. Most commonly encountered was hyperactivity due to phenobarbitone, and ataxia due to phenytoin.

Control Achieved (Table XI)

Adequate control (less than 4 seizures per year) was achieved in 75% or more of the children with idiopathic epilepsy and in those whose convulsions were the result of brain damage. In each group satisfactory control was never achieved in about 10% of cases despite the exhibition of a number of drugs. All the cases of petit mal were well controlled. The convulsions of mentally retarded children proved most difficult to control and in 10% of cases control was never satisfactory.

DISCUSSION

The separation of cases into groups is based on history and clinical criteria, and is therefore somewhat arbitrary. It is possible that children classified as cases of idiopathic epilepsy may in fact have suffered perinatal brain damage. A child with true idiopathic epilepsy may have suffered an incidental episode of meningitis, without any permanent brain damage, but leading again to incorrect classification.

In this study 49% of children had idiopathic epilepsy

TABLE X. DRUGS USED (%)

Condition	Phenobarbitone	Phenytoin	Primidone	Phenobarb and phenytoin	2 drugs	3 drugs
Idiopathic epilepsy	51	5	1	16	10	16
Perinatal brain damage	47	8	9	—	13	19
Postnatal brain damage	30	7	14	7	18	21
Mental retardation	13	—	—	13	27	47

TABLE XI. CONTROL ACHIEVED (%)

Convulsions per year: Condition	Excellent (0 - 1)	Good (2 - 3)	Reasonable (4 - 5)	Poor (6 - 10)	Little control (10+)
Idiopathic epilepsy	61	18	11	6	5
Perinatal brain damage	51	30	9	6	4
Postnatal brain damage	52	23	14	5	7
Mental retardation	47	20	13	13	7
Petit mal	40	60			

and in 43% convulsions were due to assumed brain damage. These figures may be compared with figures from Boston, quoted by Lennox.[1] Among the children he studied who were between 5 and 9 years old, only 29% had history or clinical findings suggestive of brain damage. In another study Abernathy[2] found that 31% of children with recurrent convulsions had evidence of organic brain damage. The much larger proportion (43%) of brain-damaged cases found among Cape Town children is probably related to the low socio-economic standard of the population, with attendant increase in incidence of obstetric complications, prematurity and postnatal infection.

It is recognized that mental retardation occurs more frequently in male children.[3] Significantly more male children in this study had recurrent convulsions following perinatal brain damage. This is perhaps an expression of their larger size and consequent greater vulnerability during passage through the birth canal. It is also recognized that more male infants develop the respiratory distress syndrome of the newborn[4] with its attendant danger of cerebral anoxia, leading to subsequent convulsions.

Investigations by Metrakos and Metrakos[5] have shown the existence of a dominant autosomal gene of irregular penetrance responsible for the transmission of an epileptic disposition demonstrable on EEG. Gastaut[6] has confirmed their findings and in addition defined a simple convulsive predisposition, probably dependent for transmission on several genes. Possession of this latter predisposition is expressed in a very low convulsive threshold but no inherent EEG abnormality. In the present study a family history positive for convulsions was obtained in 38% of children with idiopathic epilepsy. This figure compares with the findings of Lennox[1] and others in studies of epilepsy and is an expression of the genetic effect demonstrated by Metrakos and Metrakos. A family incidence of 28% among children who had suffered perinatal morbidity seems high, as it is usually assumed that their convulsions are the result of focal or multifocal brain damage. However, it is suggested that Gastaut's concept of a genetically determined variable threshold to convulsions applies here. The child with minor focal damage but a normal or average threshold may never convulse and so is not brought to the paediatrician's attention on this account. On the other hand the child with comparable minor focal damage but a low threshold will convulse readily. Hence the positive family history recorded in children with perinatal and postnatal brain damage is a reflection of a genetically determined low convulsion threshold.

As expected, a majority of the children with clinical evidence of perinatal or postnatal brain damage showed EEG evidence of focal abnormality. Among those classed as idiopathic epilepsy according to clinical criteria, the incidence of focal EEG abnormality was 28%. If one adheres to Penfield's[7] concept of a centrencephalic origin for idiopathic epilepsy, then these children with focal EEG abnormalities must be reclassified. However, Penfield's hypothesis has been much modified and it now seems generally accepted that cortical lesions, by discharging along the diffuse projection paths of the reticular system, can initiate the bilaterally synchronous discharges of centrencephalic epilepsy. Goor[8] has suggested that this epilepsy be designated 'generalized cortico-centrencephalic'. Temporal lobe lesions, responsible for 50% of secondary epilepsies, may by direct contiguity discharge into the brain stem with similar result.

Treatment

All children in this study were treated initially with phenobarbitone except where there was an associated history of hyperactivity or behaviour disorder, when primidone became the drug of choice. If further convulsions occurred, the dose of phenobarbitone was increased to a maximum of 5 mg/kg/day. If this did not secure reasonable control phenytoin was added. If convulsions were still frequent on this combination, other drugs, such as primidone, carbamezepine, diazepam and sulthiame were used, singly or in combination. It is felt that complex medication is always undesirable and that the number of drugs administered should be the minimum necessary to maintain adequate control. Associated hyperactivity, most common in brain-damaged children, was treated variously with amphetamine, methylphenidate, dixyrazine and haloperidol. The last of these proved most successful, but, in general, results were disappointing.

The control of seizures achieved compares with that reported by Livingston,[9] Yannet,[10] Lundervold[11] and Fukuyama.[12] Livingston found that the seizures of 15% of his patients proved refractory to all forms of treatment. The comparable figure in Cape Town was 10%. While control of seizures is of great importance, the welfare of the child prone to recurrent convulsions is influenced also by his physical handicaps, social adjustment and school progress. These factors should engage the paediatrician's attention in addition to the manipulation of drug therapy, as considerable problems of adaptation may exist, especially in an underprivileged community. Parents and patients require continual encouragement, emotional support and empathy. It is felt that time devoted to these aspects of the children's management has contributed materially towards the attainment of a seizure control rate comparable with those obtained in sophisticated communities.

SUMMARY

A study has been made of 225 children with recurrent convulsions attending a special clinic at Red Cross War Memorial Children's Hospital in Cape Town. The underlying aetiology, clinical features, EEG, medication and degree of control achieved have been analysed and discussed. In each aetiological group good control was maintained in 75% of children. In 10% of cases satisfactory control was never achieved. These figures compare favourably with those from centres serving sophisticated communities.

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