their normal caloric requirement. It seems there must be some mechanism that normally maintains the body weight at a remarkably constant level, but this is not as yet understood. Where obesity and slimness are undoubtedly related to caloric intake, and this is in the vast majority of cases, the theory that suggests that appetite control may be a combined function of the hypothalamus and cortex is the most satisfactory. Damage to or dysfunction of the hypothalamus leaves only cortical control, and man being what he is, that control may break under various stresses and result in anorexia or more often in over-eating.

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CONVULSIONS IN INFANCY AND CHILDHOOD

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To a mother the word 'convulsion' seems to mean almost any sudden temporary aberration from what she believes to be normal childish behaviour. The complaint of 'convulsions' in a child may cover simple starts and stares, crying spells, tooth-grinding, colic pain, temper tantrums, epilepsy in any of its forms, nightmares in a school child, or the Moro start reflex of a baby. The first task in such a case is to elucidate a description of the event and so define the term. For our present purpose no better definition could be found than that given by Hughlings Jackson: 'A convulsion is the product of sudden temporary nervous discharges.' Jackson was at pains to emphasize the importance of each of the adjectives he chose. Some 50 years later the electroencephalogram, whilst increasing our knowledge of epilepsy, served to substantiate the validity of Jackson's

concept so that the definition serves equally well today.

The definition 'sudden temporary nervous discharges' adequately describes the observed phenomena of epileptic seizures; it may also be said to cover the psychogenic discharges resulting in hysterical fits, breath-holding attacks and temper tantrums; it covers the state of increased irritability of nerve cells during pathological alterations of the physiological *milieu interieur*—tetany, for example—or affected by toxins either endogenous (uraemia), exogenous (lead encephalopathy) or bacterial (tetanus). Without stretching the point too far, even simple faints are covered by the definition, for the modern use of the term vaso-vagal syncope indicates that we recognize overaction of the parasympathetic as the aetiological factor.

Fits and Faints

A fainting attack may be defined as an alteration in the state of consciousness brought about by an alteration of cerebral blood supply or by a diminution of oxygen to the brain. The circumstances of the onset, the gradual beginning and the patient's good memory of the event help to distinguish a faint from a fit. It should be remembered that jactitations and incontinence are not confined to fits, but may occur in severe faints. Young children seem rarely to faint, but in the author's experience 'convulsions' and queer turns in older children, particularly about the age of puberty, are more often faints than fits. Enquiry into the full circumstances of these attacks and examination of the cardio-vascular system should always be undertaken: clear-cut differentiation may occasionally require the provocation of a typical seizure and such ancillary investigations as electroencephalography.

A Classification of Fits

A classification of fits may be offered on these lines:

Psychological fits: Hysteria, breath-holding attacks and temper tantrums.

Biochemical fits: Uraemia, tetany and hypoglycaemia.

Epilepsy, symptomatic and idiopathic: Febrile convulsions, *grand mal*, *petit mal*, *psychomotor seizures* and *myoclonic fits*.

Besides forming the framework for discussion of the problem as a whole, such a classification is a pertinent reminder of the breadth of the differential diagnosis. In the approach to the individual case it is convenient to bear in mind some such list of possibilities. Though each case of convulsions in a child deserves a complete physical examination, with particular emphasis on the central nervous system, positive findings are rare, though on occasion some special investigations may give a conclusive result, it is a careful history that provides the diagnosis in the majority of cases.

Psychological Fits

It is sometimes said that hysteria is rare in children; paediatric experience is contrary to this, for something like a quarter of paediatric out-patients present with minor behaviour problems that can fairly be termed hysterical. Frank hysterical fits may occur in childhood and are comparatively common in adolescence. The writer recalls a girl of five with diabetes whose hysterical fits exactly mimicked hypoglycaemic reactions and which were so regarded until the evidence of several blood sugar determinations could no longer be denied. The child who holds his breath till he becomes cyanosed and falls un-

conscious can be recognized by the circumstances of his seizure: the provocation by being thwarted, the presence of an audience, an understandable need on the part of the child to seek his mother's attention, and so on. Common-sense advice from the doctor to mother and resolute refusal on her part to show alarm or even concern, though conclusively effective, are easier to recommend than to put into practice, for it is often the nature of such a case that the mother cannot accept good advice. Fear that the temper tantrums or breath-holding attacks may develop into a 'real epileptic convulsion' is often the root of the mother's anxiety.

Sometimes naughtiness at school or at home, particularly if stereotyped, may be mistaken for *petit mal* epilepsy; this differentiation will be discussed later on.

Biochemical Convulsions

Hypoglycaemia in a diabetic needs no comment; idiopathic hypoglycaemia, though an uncommon cause of convulsions, occurs in children and deserves mention. Amongst the cases of convulsions and coma admitted to a children's ward there will be two or three every year that are cases of acute nephritis: to call these *uraemic* convulsions is perhaps slipshod terminology—though sanctioned by use—for the blood urea is not necessarily much elevated and one thinks of them as more in the nature of attacks of hypertensive encephalopathy than as a manifestation of uraemia. Rickets is now a rarity in Great Britain; even so rachitic spasmophilia is not likely to escape notice, the age incidence (second half of first year) is well marked and the signs of rickets obvious in such a case, but hypocalcaemic tetany is often latent and the convulsions may then be provoked by a fever or teething. Hysterical hyperventilation produces tetany. It may be necessary to witness an attack to distinguish between this form of alkalotic tetany and *petit mal* epilepsy, which may readily be produced by hyperventilation. Neonatal tetany is a rare form of hypocalcaemic tetany occurring in the first week of life; it is usually ascribed to birth trauma to the parathyroid glands. The author has not seen a case in which the biochemical evidence was unequivocal and cerebral birth trauma could be excluded with certainty. In children lead poisoning usually takes the form of encephalitis; lead paint is the common source in children and lead nipple shields in infants.

Tetanus in children is now seen chiefly in those whose injuries are so trivial that medical attention has not been sought or anti-tetanus serum not been considered; one should not, therefore, be misled by the absence of a suggestive wound when considering the diagnosis.

TABLE I
CAUSES OF CONVULSIONS IN CHILDREN AT DIFFERENT AGES

Cause of Convulsion	Comparative incidence of different causes at different age points in childhood			
	1 week	2 years	10 years	16 years
Idiopathic epilepsy	2%	10%	45%	50%
Infections	5%	60%	25%	10%
Organic brain damage	90%	25%	20%	30%
Tetany	1%	—	—	—
Other	2%	5%	10%	10%

From McQuarrie's data for the years 1925 to 1940 (Mitchell-Nelson, 1950).

Symptomatic Epilepsy

The adjective symptomatic in connection with epilepsy has two meanings: it connotes both the convulsion that denotes demonstrable neurological pathology and in a more general sense, it refers to the fit that occurs as a symptom of some illness or generalized infection—the convulsion in a child that is the equivalent of a rigor in the adult. This ambiguity is no disadvantage, for it places the symptomatic epilepsies in a sharp contrast to idiopathic epilepsy. Encephalography and advances in neurosurgery in recent years have increased the former group at the expense of the latter.

The investigations of a child with fits having carried the case beyond matters already discussed would next be concerned with differentiation between symptomatic and idiopathic epilepsy, the last diagnosis being reserved for the case in which no general systemic or neurological pathology can be demonstrated to account for the seizures. The symptomatic epilepsies of infancy and childhood might be discussed in relation to the age at which they occur (see Table 1), but may be more conveniently considered under broad aetiological headings.

Organic brain damage. Some degree of intracranial birth injury and slight haemorrhage appear to be the rule and changes in the cerebro-spinal fluid of the newborn reflect this. Gross haemorrhage, as from a tentorial tear, usually results in death within a few days at most. Less severe haemorrhage may give rise to a variety of generalized signs; localizing signs are rare. Shock may be evident as asphyxia pallida; meningeal irritation may be manifest by the 'cerebral cry'; drowsiness, vomiting and failure to feed satisfactorily or to thrive, marked hypo- or hypertonia and, particularly, convulsive movements or generalized convulsions suggest intracranial injury. Treatment should at first take precedence over investigation.

Present-day practice is to handle these babies as little as possible; warmth and quiet and oxygen are the cardinal requisites. The start of feeding should be delayed and even fluids are best withheld for 48 to 72 hours and longer in some cases. One waits for a lusty hunger cry before attempting the first feed and then first gives a trial feed of water; if this is swallowed safely, subsequent feeds may be of dilute milk mixture, the amount given being the amount the infant will take and the feeding interval being determined by evidence of hunger. (This method has now many names: gipsy feeding, natural feeding, on-demand, self-demand, etc.) Medication is largely a matter of personal preference, the author's being for sodium phenobarbitone by intramuscular injection, gr. 1/8 being the unit dose, repeated as necessary.

If improvement fails to follow these measures, the possibilities of a subdural haematoma and neonatal meningitis must be excluded by subdural exploratory needling and examination of the cerebro-spinal fluid respectively. *B. coli* is commonly the organism causing neonatal meningitis. Suggestive signs of meningitis are unusual, yet the diagnosis is important, for the condition may now be successfully treated with the newer antibiotics, terramycin perhaps being the drug of choice.

Infections. Toxoplasmosis is an example of a foetal encephalitis that may cause convulsions in a newborn infant. Several cases have been reported in this country; the typical case, showing microcephally, intracranial calcification, choroido-retinitis and convulsions, should not be missed. Confirmation can be obtained by seriological tests. Less typical cases, particularly where the diagnosis rests principally on serology, should be viewed with suspicion.

The important role of infection in the causation of convulsions during the earlier years of childhood is illustrated in Table 1. Any generalized infection

in a child may cause a convulsion; the prodromata of measles, chicken pox and exanthem subitum may be marked by fits, and fits may accompany the signs of pneumonia or pyelitis. Infections within or near the central nervous system (tonsillitis, otitis media and meningitis) seem particularly prone to cause convulsions. In a young child lumbar puncture should not be delayed if the cause of the fit is not obvious, for changes in the cerebrospinal fluid may precede other signs in influenzal and tuberculous meningitis and both have their maximum incidence in the first two years of life.

Febrile convulsions. Febrile convulsions deserve special mention regarding prognosis. That a convulsion in a child may signify no more than a rigor in an adult may be reassuring to the parents, but the physician should consider the possibility of latent epilepsy in each case. Electroencephalography has demonstrated epileptiform abnormalities in many such cases. Thus Lennox (1947), taking electroencephalograms on 153 children with febrile convulsions, found E.E.G. changes of latent epilepsy in 45 per cent. of them. She concluded, from both clinical and electroencephalographic evidence, that febrile convulsions may presage recurrent idiopathic convulsions. Before the days of the electroencephalogram Patrick and Levy (1924) had reached the same conclusion by careful history taking; they found that only some 4 per cent. of normal adults have a history of febrile convulsions in childhood, as against 20 per cent. of epileptic adults giving a similar history.

Neurological disease. Symptomatic epilepsy denoting neurological disease is not uncommon in childhood. During this period, for example, there occur some 10 per cent. of all primary brain tumours; brain abscess is relatively more frequent. In children a brain abscess is often, and a brain tumour, is usually situated in the posterior fossa. Though lumbar puncture has an important place when investigating a child presenting with convulsions, lumbar puncture should never precede examination of the optic discs.

Acute infantile hemiplegia deserves special mention. The condition was at one time regarded as a manifestation of polio-encephalomyelitis and is still often called Marie-Strümpell encephalitis. The present-day concept excludes encephalitis and polio-encephalitis and suggests cortical cerebral thrombosis as the pathological lesion in some cases; but (in the author's series) especially in those showing porencephalic cysts birth trauma cannot be excluded with certainty. The typical case is a child of less than six and usually less than three years who suffers a short febrile illness characterized by fever and coma and often convulsions; other signs are lacking in the acute stage and the cerebro-spinal fluid rarely shows ab-

normality. Upon recovering consciousness after a few days the child is left with a spastic hemiplegia. Considerable recovery of function is the rule, particularly in the leg, though growth of the affected limbs may be stunted. Many cases later develop epilepsy.

This particular group of epileptic children is at present attracting much medical attention. Foci of abnormal electrical discharge can often be demonstrated by the electroencephalogram. It is in these cases that excision of cerebral scar tissue has been carried to the extreme of hemispherectomy, the removal of the whole cerebral hemisphere on the damaged side.

There are other paediatric conditions having symptomatic epilepsy as a part of the syndrome and which become evident in the early years of life; the Sterge-Weber syndrome, tuberous sclerosis, amaurotic family idiocy, Schilder's disease, and kernicterus complicating erythroblastosis foetalis may be mentioned.

Idiopathic Epilepsy

Since idiopathic epilepsy usually first becomes manifest in childhood, the condition is an important one in paediatric practice. Table 2 shows the age incidence in children.

TABLE 2
AGE OF ONSET OF EPILEPSY IN CHILDREN

	Per Cent.
Less than 1 year	23
" " 3 years	50
" " 6 "	75
" " 15 "	100
Age period 10 to 15 years	11

Note.—Cerebral birth trauma and its sequels are not separated here from idiopathic epilepsy, both are counted in a total group of 742 children representing some 40 per cent. of the Johns Hopkins Hospital Epilepsy Clinic figures. Data (arranged from Peterman, 1946.)

One would make the comment here that, contrary to the usual teaching, the physiological stress of puberty is not associated with a marked increase in the incidence of idiopathic epilepsy during the period of adolescence. Thus only a little over 10 per cent. of epilepsy in childhood first occurs between 10 and 15 years of age (Table 2) and, amongst causes of convulsions, idiopathic epilepsy is credited with an increase of 5 per cent. between the ages 10 years and 16 years (Table 1).

The frequency of epilepsy, compared with other diseases of childhood as judged by admission to an

important children's hospital, is shown by the admission to the children's wards of the Johns Hopkins Hospital in a recent three-year period: Convulsions, 978 cases; rheumatic fever, 510; asthma, 421. The cases of convulsions being classified as: Epilepsy, 309 cases; febrile, 350; unclassified, 328. (Bridge, 1949.)

Electroencephalography. The diagnosis of idiopathic epilepsy is necessarily one of exclusion, confirmation usually being obtained from a period of observation and, if necessary, by the deliberate provocation of a fit. This is not the place to discuss the value of electroencephalography (E.E.G.), but it may be stated categorically that an encephalogram is not essential for diagnosis. The procedure is technically difficult, the interpretation intensely complex and the reports often necessarily so equivocal that they are of little help to the clinician. As a tool of research, electroencephalography is at present confined almost entirely to neurological centres; there resort can be made with the few difficult and doubtful cases. Though of somewhat limited value in the individual case, electroencephalography has had the most important practical bearing on the problem of epilepsy by increasing understanding of the nature of the disease, not the least of these benefits being simplification of terminology. It is now usual to speak of three main varieties of idiopathic epilepsy, each distinguished by its own electroencephalographic pattern: (1) The term *pyknolepsy* becoming obsolete and the term *petit mal* being used to describe those minor seizures, usually without loss of consciousness, having the three per second spike-and-dome E.E.G. (2) *Grand mal* epilepsy, the classical major seizure, showing a crescendo of E.E.G. waves of greater frequency and greater amplitude than the normal delta rhythm. (3) Psychomotor-equivalent epilepsy (co-ordinated and apparently purposeful acts carried out whilst in a dazed state), showing a three per second square-topped wave pattern; in clinical practice psychomotor-equivalent epilepsy, like some of the visceral manifestations of epilepsy, may be difficult to diagnose without the help of an E.E.G. As an ancillary investigation to neurosurgery electroencephalography has an established place both in the identification of symptomatic epilepsy and in the localization of the focus of origin of the disturbance. But, as is shown by Table 3, there are definite limitations even to this application.

Diagnosis. Bearing in mind the necessity of electroencephalography in special cases, and realizing something of its limitations and the practical difficulty of lack of the requisite facilities for an electroencephalogram, one may briefly discuss the diagnosis of idiopathic epilepsy presenting

TABLE 3
LOCALIZING VALUE OF ENCEPHALOGRAM

Type of Epilepsy	Percentage showing Abnormality	
All types of epilepsy	Focal abnormality	14%
	Generalized abnormality	71%
	No E.E.G. abnormality	15%
Symptomatic epilepsy	Focal abnormality	50%
	Generalized abnormality	41%
	No E.E.G. abnormality	9%

Data from Gibbs (1947).

in the ordinary way as one of the more common diseases of childhood.

In the average case, history alone suggests the diagnosis, complete physical and particularly neurological examination carry the case a stage further, but do so largely in a negative sense by excluding 'symptomatic' epilepsy.

Satisfactory confirmation of the diagnosis is obtained by witnessing the patient's seizure. Factors making fits more likely may be listed as follows: Sudden cessation of treatment, fever, sleep, an increased fluid intake, emotional disturbance, fatigue, menstruation and, perhaps, constipation. A difficult case for diagnosis is best admitted to hospital, any anti-convulsant treatment previously given can there be stopped with the intention of producing a fit. In the author's practice the child is put on to a salt-free high-carbohydrate diet as a preliminary to McQuarrie's water intoxication test (McQuarrie and Peeler, 1931). This test makes use of the known fact that increase of body water without a corresponding increase in body salt makes epileptic fits more likely. After a preliminary period on a salt-free diet, forced water drinking is commenced (75 to 100 ml. of water per kilogram of body weight) and posterior pituitary extract given to inhibit a water diuresis (pitressin 0.3 to 0.6 ml. three-hourly by subcutaneous injection). Most epileptics will produce a *grand mal* seizure before the body weight has been increased by 5 per cent.; this increase is usually obtained in 24 to 36 hours. It is claimed that no false positive results are obtained from this test with the possible exceptions of hypoparathyroidism and Addison's disease. Suspicion of an intracranial tumour should contraindicate the procedure.

Petit mal epilepsy in a school child rarely presents diagnostic difficulty though patients are often referred for naughtiness or supposed deliberate inattention at school. Momentary lapses of consciousness, dropping things or a bizarre movement bring the child to the teacher's attention; punishment (misguided in this instance)

makes matters worse, for the child, being unhappy has more fits.

Hyperventilation may frequently be used successfully to produce a *petit mal* seizure in those subject to this form of idiopathic epilepsy, the technique may often be used in the out-patient clinic. Another valuable provocative test in cases of suspected *petit mal* epilepsy is the therapeutic trial of tridione. Tridione therapy often unmasks previously latent *grand mal* or makes *grand mal* more easily provoked. Major epilepsy is commonly the sequel to *petit mal*, the change often occurring at puberty.

Psychomotor-equivalent epilepsy is a more difficult problem. In-patient observation may yield the diagnosis but an E.E.G. may be necessary in such cases. Often some other type of seizure present in the same case elucidates the problem. A recent case of the author's had walked into a bicycle during a psychomotor attack, she also had little attacks when she looked dazed and raised her right arm above her head (in one of them at school dinner she had poured a cup of soup over her hair). A water intoxication test produced a typical *grand mal* seizure.

Epilepsy and mental deficiency. It deserves to be emphasized that the majority of epileptics are people with normal intelligence; that many mental defectives have epilepsy is undeniable, but it by no means follows that the majority of epileptics are mentally defective.

The paediatrician sees both types of case and meets them frequently. For during the first ten years of life mental deficiency, with or without epilepsy, will usually become apparent and, as has been shown already, most of those who are intellectually normal, but who have latent epilepsy, will have experienced convulsions within the age range covered by paediatrics.

The history of delay in development enables the clinician to suspect mental deficiency. Myoclonic jerks in addition to epileptic fits or psychomotor restlessness tend to confirm the suspicion. An intelligence quotient determined by an experienced worker confirms it.

Prognosis. The prognosis of children with epilepsy is difficult to discuss for the cases pass out of the hands of the paediatrician before it can be said with certainty that the epilepsy has been cured. It is common practice to try first to prevent the fits and then, when freedom from fits has been achieved for a period of two years, medication is reduced and, if symptoms do not recur, may finally be discontinued. This may be achieved in many cases; others require regular medication for many years, a few throughout life. But it may be said that the majority should, with proper treatment, be rendered free from fits and able to lead

normal lives. The author's experience would lead him to suggest that any patient failing to respond to adequate anti-convulsant therapy should be suspected of mental deficiency with epilepsy, for in these patients the seizures seem much more difficult to control. It must be said in this connection that few epileptics referred to hospital seem to have been treated adequately; many epileptics receive too small a dose to be effective; few seem aware of the factors in their lives most apt to provoke fits; many seem to have been denied even a trial of remedies developed since barbiturates replaced bromides.

Treatment. Treatment may be briefly discussed. In cases of *grand mal* it is the author's practice to start with phenobarbitone and to regard the correct dose as that dose which stops the fits. Initial drowsiness quickly passes off as tolerance is acquired. If drowsiness remains as an undesirable side-effect a change is made to methyl phenobarbitone which is more effective as an anti-convulsant and less sedative; its only disadvantages being cost and limited availability (few country chemists seem to stock it). The hydantoin derivatives are reserved for those who are not managed satisfactorily by barbiturates alone; phenytoin sodium ('Epanutin') being used, and usually in conjunction with phenobarbitone. Occasional resort is made in difficult cases to the ketogenic diet, the principle of which is to bring about a metabolic ketosis by substituting fat for carbohydrate in a large measure in the diet and severely restricting liquid intake. Acetone should be present on the breath and be shown in the urine as a strongly positive Rothera test. In prescribing a ketogenic diet one aims at the caloric requirement for age from a diet in which the amount of fat

protein
exceeds $2 \times \text{carbohydrate} + \frac{\text{protein}}{2}$. Even in the

U.S.A. the ketogenic diet has failed to achieve lasting popularity, but it has been used often enough and long enough to show that no harm results from it to the patient; moreover, it is undeniably effective in reducing the tendency to fits. The high cost of dietary fats is one serious drawback. Children seem to find the diet palatable enough but all too often the parents think it nauseating; parental propaganda brings the child to dislike it. Yet there is a place for the ketogenic diet, albeit a small one, in the management of epileptic children.

Tridione is the treatment of choice for *petit mal* attacks. A starting dose of 0.3 g. two or three times a day is usual for a child (adult dose 1.8 g. per day). The possibility of provoking latent *grand mal* must be remembered but is usually easily controlled with phenobarbitone. Phenobarbitone

alone usually makes *petit mal* worse. What used to be called pyknolepsy or 'myriad spells' (many *petit mal* attacks) used to be so resistant to treatment before the introduction of tridione that the slight danger of agranulocytosis and less serious side-effects cannot contraindicate its use provided proper precautions are observed.

Rarely emphasized but of paramount importance in the handling of the epileptic child are those ancillary measures that are of little use in themselves but need to be combined with pharmaceuticals if the latter are to be fully effective. A reassuring and optimistic attitude on the part of the physician, the removal of anxiety from the parents, relief of psychological stress in the home, the prescribing of a simple first-aid routine to be followed in the event of a fit, prohibition of a high and particularly of a sudden increase in fluid intake; these and similar measures need to be combined with a rational and reasonably lavish prescription of anticonvulsants. Such a combination is almost always successful in straightforward epilepsy without mental defect. As soon as freedom from fits has been achieved the family and the physician can begin to look forward to the day—two years hence—when reduction of anticonvulsant dosage can be attempted and even to the day when drugs are no longer needed and it

can be said that the epileptic child has outgrown his tendency to fits.

Conclusion

In conclusion one may quote a saying attributed to Hippocrates, 'a generalized convulsion is always to be regarded as a serious omen.' One would endorse that recommendation at least so far as the first approach to a patient is concerned. Initially it is a serious omen and as such has to be investigated. Once the diagnosis of idiopathic epilepsy is made it becomes necessary to emphasize both to the patient and his parents that the disease is not necessarily serious nor is it resistant in most cases to treatment. The outlook for a happy and useful life is excellent in the majority.

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