Current Practice

CHILD CARE IN GENERAL PRACTICE

Convulsions in Infancy and Childhood—Part II

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In Part I of this article (published in the *B.M.J.* last week) the facts that should be elicited from the parents and the points to be noted by the family doctor were discussed. The control of attacks, general management, and prognosis are considered in this week's article.

Treatment of Attacks

In the case of symptomatic epilepsy treatment is of the underlying condition.

Status Epilepticus

Status epilepticus involves risk of death from anoxia and requires energetic treatment. The necessity for ensuring a clear airway must not be forgotten. Paraldehyde should be given intramuscularly. Response takes place within minutes, but the paraldehyde may have to be repeated in slightly smaller doses within one to two hours. Where response is lacking anaesthesia is indicated, chloroform having the advantage over ether of being non-inflammable, and the child should be transferred to hospital. Intravenous sodium thiopentone (0.01 g. at 1 year) is advocated by some. Sodium phenobarbitone is contra-indicated because of the respiratory depression that follows large doses.

Convulsions in Infants

Chloral hydrate is the drug of first choice in dealing with convulsions in the newborn and older infants. It is safe, quick in action, and without toxic effects, and should be given where the convulsions are thought to be due to injury or anomaly of the central nervous system or to be idiopathic. In the exceptional event of chloral hydrate proving only partly effective a change should be made to phenobarbitone. Seizures recurring without explanation despite anticonvulsant therapy may be due to pyridoxine dependency and respond to intramuscular (2 mg.) or oral pyridoxine (10 mg.).

Where pyelonephritis, otitis, or meningitis is present sedatives are best reserved for cases in which convulsions do not cease with antibiotic therapy. True febrile convulsions differ in that they are not an indication for sedative or anticonvulsant therapy. Where there is a tendency to recurrence the possible effects of pyrexia can be prevented by tepid sponging and antipyretic or antibiotic therapy.

Asymptomatic Epilepsy

Drugs constitute the basis of treatment of asymptomatic epilepsy. It must be recognized that only trial and error will determine the choice and desirable dose of drug for the individual case; that first choice should be based on the character

of the convulsions to be controlled; that the decision to try another drug or a combination of drugs should await proved ineffectiveness of the maximum tolerated doses of the first drug; and that therapy of established efficacy must be continued conscientiously for a minimum period of two years. Where convulsions tend to occur at a particular time of day, the drug of choice should be given in larger doses than at other times a few hours before the expected seizure. Parents can help by keeping a record of fits. A return of convulsions or a change in a rapidly growing child's behaviour may indicate the need for adjustment of the dose of the drug in relation to the body weight. Whatever therapy is employed a watch must be maintained for toxic signs. (See Table.)

Major Seizures

If major seizures predominate phenobarbitone should be given; if this is not wholly successful it should be replaced by phenytoin. It should be remembered, however, that phenytoin may cause hypertrophy of the gums (Fig. 2). If phenytoin in turn does not achieve the desired results a combination

FIG. 2.—Hypertrophy of gums following treatment with phenytoin sodium (Epanutin).

of phenobarbitone and phenytoin should be tried. This combination is particularly useful where control of seizures by phenobarbitone alone necessitates doses resulting in excessive drowsiness. Because it tends to worsen attacks of grand mal troxidone (Tridione) should be avoided. Primidone (Mysoline) or alternatively ethosuximide (Zarontin) is sometimes

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effective where phenobarbitone and/or phenytoin do not control major attacks which are not focal in character. It is important to remember that status epilepticus may follow too rapid withdrawal or substitution of drugs.

Failure of these various forms of therapy justifies resort to the use of bromide, using a mixture of the sodium and potassium salts in equal proportion. Bromide is well tolerated by children under 10 years of age in whom there is no evidence of renal inefficiency, and in the circumstances under consideration anxiety concerning bromism need not discourage a trial of bromide therapy provided that periodic checks of the blood concentration are made. A bromide level of 150 mg./100 ml. (19 mEq/l.) is safe at this age.

Minor Seizures

Despite reports to the contrary, phenobarbitone not infrequently diminishes the incidence of, if it does not completely abolish, minor epileptiform incidents. On the other hand, it may aggravate the condition; if so ethosuximide and troxidone should be tried in that order. When other forms of therapy are unsatisfactory it is sometimes helpful to use a combination of phenobarbitone and ethosuximide or of acetazolamide (Diamox) alone. The beneficial results of troxidone are

sometimes evident within 48 hours and may sometimes persist after reduction of the dose.

Myoclonic motor seizures ("lightning attacks") of infancy present a special problem in that even partial response to any of the above forms of treatment is problematical, and it is customary to combine anticonvulsants with A.C.T.H. therapy.

General Management

Attention to general management, environment, and personal hygiene is important. Constipation should be guarded against. Fluid consumption at any one time should be moderate and habitual taking of fluids in excess of normal requirements discouraged. Activities should be reviewed with the object of securing as full a life as possible compatible with the safety of the patient and the interests of the community. Everything should be done to avoid imposing on the child a life different from that enjoyed by his companions. Nevertheless swimming should be permitted only when a relative or instructor with a knowledge of the facts is present; climbing of ropes and wall-bars in the gymnasium is undesirable, and in the interests of others the child should not cross busy thoroughfares unescorted. Bicycling is a difficult problem. It should certainly not be indulged in on traffic highways, but can probably be allowed

Convulsions in Infancy and Childhood: Drugs Suited to Use in General Practice

Indications	Name of Drug		Administration			Side-effects and Toxic Reactions
	Official	Other	Age (Years)	Dosage	Route	
Status epilepticus	Paraldehyde	—	1 5 10	1 ml. (15 min.) 2 ml. (30 min.) 4 ml. (60 min.)	Oral	
	Paraldehyde injection	—	—	0.15 ml. (2½ min.) per kg. (2.2 lb.) body weight	Intramuscular	
	Paraldehyde enema	—	—	0.3–0.6 ml. (5–10 min.) per kg. (2.2 lb.) body weight	Rectal	
	Phenobarbitone sodium injection (B.P.)	—	10	120 mg. (2 gr.)	Intramuscular	Depression of respiratory centre
Grand mal Petit mal	Phenobarbitone tabs. (B.P.) (barbiturate)	Luminal	0–4 wks. 5 10	7.5 mg. (¼ gr.) b. or t.i.d. 15 mg. (½ gr.) b. or t.i.d. 15–30 mg. (¼–½ gr.) b. or t.i.d.	Oral	May aggravate petit-mal attacks Refractoriness and irritability Morbilliform rash Drowsiness Ataxia
Grand mal Petit mal (but may be ineffective)	Phenytoin sodium tabs. (B.P.) (hydantoin)	Epanutin Dilantin Eptoin	5 10	50 mg. (½ gr.) b. or t.i.d. 100 mg. (1½ gr.) b. or t.i.d.	Oral (capsule)	
Grand mal Petit mal Psychomotor seizures	Primidone tabs. (B.P.) (barbiturate)	Mysoline	3 5 10	125 mg. } b. or 250 mg. } t.i.d. 500 mg. }	Oral	Drowsiness Abdominal pain Ataxia Macrocytosis
Petit mal	Troxidone (oxazolidine)	Tridione	—	300 mg. b. or t.i.d.	Oral	Agranulocytosis Skin rashes Nausea Hiccup Nephrosis
Petit mal	Ethosuximide (succinimide)	Zarontin	5 10	250 mg. 500 mg.	Oral	Drowsiness Nausea Vomiting
Minor epilepsy resistant to other therapy. Restlessness in a child subject to occasional grand-mal attack not responding to other therapy	Acetazolamide	Diamox	5 10	125 mg. 250 mg.	Oral	
Major and minor seizures (symptomatic and asymptomatic) in the neonatal period and later infancy. Occasionally in older children after isolated convulsion	Chloral hydrate	—	0–4 wks. 1 5 10	30–60 mg. (½–1 gr.) 3- or 4-hourly 60–180 mg. (1–3 gr.) b., t., or q.i.d. 180–540 mg. (3–9 gr.) b. or t.i.d. 600 mg. (10 gr.) b. or t.i.d.	Oral	
Grand and petit mal where other therapy has proved ineffective	Potassium bromide (with simultaneous low-salt diet and 1- to 3-monthly check on blood-bromide level)	—	5 10	670 mg. (5 gr.) t.i.d. 1,000 mg. (10 gr.) t.i.d.	Oral	Acne Apathy Depression Gastric disturbance

on country lanes and byways. Participation in team games and youth clubs should be encouraged, and youth leaders should be instructed in the possible needs of epileptics.

Every endeavour should be made to ensure that the family accept the child as a virtually normal member of their circle. Any sense of inferiority must be dispelled. Neither at home nor at school should an unnecessarily protective attitude implying any form of stigma be adopted. It is in the interests of the child that the local education authority should be informed when a pupil is diagnosed as suffering from epilepsy. It is desirable that the child's condition should be discussed at a professional level between family doctor, school medical officer, and teacher, if only to ensure that the possible sedative effects of drug therapy are not misinterpreted and that the educational opportunities offered neither underestimate nor overestimate the child's abilities. Sometimes a child's limited scholastic ability, restless behaviour, or liability to frequent convulsions may necessitate enrolment at a special school. Parents can readily be distressed if the first intimation of an Education Authority's proposals is given in a terse official communication, but, after discussion with the education-authority staff, the family doctor can often by kindly explanation help parents to accept the proposals as offering the best prospects for their child.

When Should a Child be Referred to Hospital ?

There is no one answer. Many factors are involved, including age, nature of the convulsions, general physical state, and the facilities within the home. In general it may be said that among indications for reference to hospital for immediate admission are status epilepticus, frequent recurrence of convulsions unresponsive to therapy, convulsions suspected of being due to intracranial infection or haemorrhage, myoclonic motor convulsions of infancy, any suggestion of encephalopathy—for example, uraemia, lead-poisoning (Fig. 3)—extreme physical debility, and significantly inadequate homecraft. Depending on the patient's clinical signs and symptoms, special hospital examinations may consist in haematological and serological studies ; microscopic and chemical examination of the urine and cerebrospinal fluid ; electrolyte studies ; radiographs of the cranium and contents ; electroencephalographic records ; and in the rare case where the possibility of neurosurgery arises pneumoencephalographic and arteriographic investigations.

FIG. 3.—Radiograph of child with lead encephalopathy showing increased density of bones and "specks" of radio-opaque material in colon.

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Of other children only a proportion call for a second opinion. The reason may be a need to confirm the diagnosis ; the fact that the convulsions appear to be focal in nature ; resistance to treatment ; or a wish on the part of the parents to be reassured that no detectable organic disease is present. With the approach of puberty some epileptic children give rise to new problems of management and a second opinion is required for reassessment. Electroencephalography is not essential for diagnosis in the majority of cases and often does not add significantly to knowledge derived from a thorough history and neurological examination. Indeed, a normal recording can be obtained from a patient known to have convulsions, and what would appear to be abnormal recordings are sometimes found in healthy children with no history of seizures.

In the event of a specific cause for recurrent convulsions not being found the diagnosis remains one of idiopathic epilepsy irrespective of whether the E.E.G. is normal or abnormal. "The electroencephalograph is not neurology's diagnostic machine" but a valuable aid with certain limitations in infancy and childhood (Bucy, 1956). The E.E.G. can assist diagnosis by differentiating syncopal and breath-holding attacks from convulsions ; major motor, petit mal, and hypsarrhythmic convulsive incidents from one another ; and focal abnormalities of different types from widespread abnormalities of asymptomatic epilepsy. The E.E.G. is sometimes but not always useful in assessing the value of treatment—response to a particular drug being reflected by changes in the electroencephalograph. While it is true that cessation of minor epileptiform seizures is frequently accompanied by the simultaneous disappearance of corresponding abnormalities in the E.E.G. record, electroencephalography has as yet only a limited contribution to make to prognosis.

Prognosis

As to long-term prognosis opinions differ from decade to decade, from authority to authority, and from country to country. Many studies have been based on the experience of hospitals or colonies, and lack of a wholly reliable overall prospective knowledge of the life history of an adequate number of cases. Two facts make it extremely difficult to venture long-term prognosis in epilepsy in childhood. Firstly, there is the frequent uncertainty concerning diagnosis, and second, while there is a tendency to remission and possible recovery in children it has not been established that the trend persists beyond childhood.

Certain generalizations can, however, be made. Contrary to popular belief, the prognosis of patients with a family history of epilepsy is better than that of those whose seizures are probably related to undisclosed organic lesions. Seizures in babies between 1 and 6 months old are usually due to brain damage and carry a poor prognosis : those occurring between 6 months and 3 years are mainly of febrile type and have a good prognosis ; and idiopathic seizures with onset after 3 years of age are associated with a mixed prognosis depending on the type of seizure, response to drugs, and discernible pathology.

An occasional seizure is not justification for giving a bad prognosis, and it is exceptional for chronic epilepsy to develop in a child with a history of an isolated febrile convulsion. Remission is more likely where seizures are infrequent and more particularly when seizures are of grand-mal and not petit-mal type. The prospects of a normal life are poor where frequent attacks persist without remission. To a large section of the general public epilepsy implies mental deficiency. In actual fact the majority of epileptic children are normal mentally, but it is important to recognize that mental impairment is an almost invariable sequel in infants with a history of myoclonic motor seizures, including those in whom convulsions ceased with A.C.T.H. treatment. Among children with cerebral palsy of antenatal or natal origin a relationship appears to exist between the degree of mental handicap and the incidence of convulsions.

The family doctor may be asked for advice concerning the future employment of a child nearing school-leaving age. Intelligent adolescents with well-controlled attacks will have no great difficulty in entering an academic career, but the parents of a less well-endowed child may experience frustration in placing their child in manual work. There are opportunities for such adolescents, however, and the practitioner can often obtain helpful advice from Youth Employment Officers, and

from medical social workers and social workers in the employ of hospitals and local authorities.

Dr. Alexander Wilson, formerly of Meanwood Park Hospital, gave us valuable advice, for which we are grateful.

REFERENCE

Bucy, P. C. (1956). *J. Amer. med. Ass.*, **160**, 1232.

ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Branchial Cysts

Q.—What can be done for a branchial cyst that surgical excision on three occasions and injection of sclerosing agents have failed to cure?

A.—Much depends on the exact location of the external opening. Lateral cysts and sinuses arising from the branchial-cleft system require complete extirpation of the track leading to the pharynx. These remnants are often associated with the presence of cartilaginous nodes, infection of which maintains the discharge. Persistent sinuses at the level of the hyoid bone frequently turn out to be thyroglossal in origin, although they have pointed away from the midline. Excision of the body of the hyoid bone is essential. There is therefore no alternative to further surgery in either case.

Stress and Coronary Thrombosis

Q.—What is the evidence in support of the view that physical or mental stress are precipitating causes of acute coronary thrombosis?

A.—There is little evidence to show that any particular event actually precipitates coronary occlusion. However, for what it is worth, Pell and D'Alonzo's careful study¹ demonstrated that myocardial infarction was less common when subjects were on vacation than when they were at work. When not on vacation the infarction occurred at rest or during only moderate physical activity in 95% of patients.

Nevertheless, in patients with established coronary-artery disease it is probable that emotion may lead to sudden death, possibly via circulating adrenaline causing ventricular fibrillation. John Hunter's demise after a stormy committee meeting at St. George's Hospital is the classic example of this.

REFERENCE

¹ Pell, S., and D'Alonzo, C. A., *J. Amer. med. Ass.*, 1963, **185**, 831.

Fumes from Fire-extinguishers

Q.—Some fire-extinguishers contain chlorobromomethane and the fumes are said to be dangerous, especially in a confined space. Is that so?

A.—Chlorobromomethane was introduced as a fire-extinguisher fluid because it was less

toxic than methylbromide or carbon tetrachloride. Thus dangers from leaking extinguishers in confined spaces would be reduced.

Comparative experiments on mice did not indicate any very great differences between the toxicity of the combustion products of various extinguishers, including chlorobromomethane.¹ A recent paper² describes three cases of men affected by chlorobromomethane and the products of its combustion. All made a complete recovery.

It should be remembered that the products of any combustion are toxic in a confined space mainly owing to carbon monoxide. There is no evidence that the use of chlorobromomethane introduces any significantly greater hazards when used in fire-fighting.

REFERENCES

¹ Comstock, C. C., and Oberst, F. W., *Arch. industr. Hyg.*, 1953, **7**, 157.
² Rutstein, H. R., *Arch. environm. Hlth*, 1963, **7**, 440.

E.C.G. after Infarction

Q.—What is the minimal time after myocardial infarction that E.C.G. changes can be expected?

A.—Naturally it is rare to get an E.C.G. done in the first few minutes after a cardiac infarct, but when a tracing is taken within an hour or so it may well show S.T.-segment elevation. Though not strictly analogous, I have observed a current-of-injury pattern on the E.C.G. within five minutes of a coronary air embolism being observed when the heart was exposed during cardiac surgery for a septal defect, so presumably cardiographic changes could occur this early after coronary thrombosis.

Routine Dental X-rays

Q.—Are there any general or local risks associated with the use of dental x-rays? Some dentists consider that x-ray examination should form a routine part of 6-monthly dental inspections.

A.—There is no risk associated with the use of dental x-rays so long as proper protection is provided. The local field is too small to do any damage to bone marrow. The position of the tube, however, which is pointed downwards for certain of the exposures, can involve some gonadal radiation. It is an easy matter to protect the gonads in

modern dental x-ray practice. It is doubtful if there is any justification for routine six-monthly dental x-ray inspections.

Resistance to Infection in Infants

Q.—A 15-months-old girl has suffered from recurrent severe chest infections since the age of 2 months. Extensive investigations (including tests for hypo- and dysgammaglobulinaemia) have shown no primary cause for this susceptibility. Nevertheless, the episodes have recently been found to respond to injections of gammaglobulin. Is there any explanation for this?

A.—Resistance to infection depends on many factors, humoral and cellular, innate and acquired, and the relative importance of these is difficult to assess. The relative freedom from infection of infants is probably at least partly owing to the mothers' gammaglobulin, acquired passively, in above adult concentrations. This provides protection against the organisms with which the mother has been infected, and lasts some weeks. As it decays it is supplanted by the child's own gammaglobulin, so that after the "physiological trough" for gammaglobulin has been passed the level rises towards adult values. Presumably this rise was occurring in this girl, since the questioner says that hypogammaglobulinaemia had been excluded. None the less, this gammaglobulin will contain antibodies only to organisms to which the child has been infected, and so presumably will not provide protection against first infections.

Occasionally, however, the antibody deficiency syndrome occurs in spite of relatively normal concentrations of gammaglobulin, which is presumably functionally ineffective. This condition is sometimes called dysgammaglobulinaemia. The other two immunoglobulins are frequently deficient in these patients, so their estimation provides a useful screening test, though proof requires a test of the patient's capacity to produce antibodies. It is not clear how this diagnosis was excluded in this case.

There is a strong clinical impression that gammaglobulin treatment is valuable for patients with hypogammaglobulinaemia, but there is no conclusive proof; such proof is difficult to obtain. It would be unwise certainly to ascribe this child's improvement to the gammaglobulin injections in view of other possible explanations such as maturation of other uninvestigated immune mechanisms, development of adequate quantities of the relevant antibodies, or changes in other environmental factors (therapeutic, nutritional, etc.). But there is a possible rational