EPILEPSY IN CHILDREN

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No doctor can be in practice for long before meeting with cases of convulsions in infants or young children. It is, of course, common knowledge that these have a special tendency to convulsions although the reason is obscure. Some authors have attributed this tendency to the lack of a properly developed myelin sheath in the brain tissue, others to greater permeability of the infantile cerebral capillaries leading, under appropriate conditions such as fever, to cerebral oedema. As the child grows this predisposition to convulsions appears to wane and is exceptional after the third year of life.

The commonest causes of convulsions at birth or shortly after are intracranial birth injury and congenital brain defects. From the 2nd month to the 3rd year febrile convulsions, that is, fits associated with acute feverish conditions, are frequent. Other and less common causes are uremia, tetany, brain tumour, intracranial injuries and infections and idiopathic epilepsy. It should be observed that in all these conditions *apart from idiopathic epilepsy*, the convulsion is merely an incident in the course of a more important disease, whereas in the latter condition it is the main if not the only symptom. However, one must not forget that convulsions occurring during fevers are not all "innocent" and that in a certain percentage of cases the fits tend to recur even in the absence of any pyrexia; provided no organic cause to account for the fits is found, these cases should be diagnosed as epileptics.

It is usually said that idiopathic epilepsy is a hereditary disease. Out of 24 cases met with in my practice this was so in only 4 cases. However Lennox and others claim that encephalograms of relatives of epileptics are abnormal in a higher percentage of cases than could be accounted for by pure chance. Gibbs states that the hereditary factor in epilepsy is, generally speaking, no greater than in diabetes and he thinks that what is inherited is not the actual disease but some kind of weakness which must somehow be activated for the fits to occur.

What is the essential nature of the malady cannot yet be understood. Since the myelin sheath, that presumably acts as an insulator, is well developed at the age when epilepsy makes its appearance, this anatomical factor can be discounted. During the last war a technique was developed to discover whether service personnel with histories suggestive of epilepsy were genuine. This consists in the administration of large quantities of water (2 to 6 litres per day), while salt was completely eliminated from the patient's diet. Simultaneously pitressin, the anti-diuretic hormone, was injected three-hourly. When the water-retention reaches 5 per cent of the patient's weight a fit occurs in the epileptic subject only. It is reasonable to suppose that the pitressin water-retention test indicates an increased permeability of the cell membranes of the brain tissue. Dehydration lessens the tendency of fits which explains the success of the "fasting cure" in the middle ages when epileptics were considered holy. So also does acidosis, whereas a shift of the pH of the blood towards the alkaline side increases the liability to epileptic fits. Hence the reason why hyperventilation will induce an attack of petit mal in a suitable subject.

SYMPTOMS: Irvine McQuarrie classifies epilepsy into three types: (1) Somatic motor; (2) Somatic sensory; (3) Psychic.

I do not propose to discuss the somatic motor seizures of which "grand mal" is the principal type nor the somatic sensory or "auras", as the former are familiar to all and the latter, in my experience, are rare in children. The third group, the psychic, sometimes presents great difficulties in diagnosis. Muscular spasms are absent or

only larval but the main feature is disturbance of consciousness. This may be lost for a short while as in "petit mal" when the patient may suffer from so called "fainting fits" or "dizzy attacks" often misdiagnosed as heart syncope or vaso-vagal attacks. In other cases consciousness is disturbed or clouded but is not abolished. The child may have *periodical* fits of destructiveness when otherwise he is a quiet obedient type, or, although naturally active he becomes at times apathetic, refusing to budge from his chair or bed. It is the periodical reversal of temperament or character which should make us suspicious and also the occasional irritational acts which the patient does not remember performing. It is especially in these cases that the encephalogram may be very useful.

TREATMENT: Too little regard is usually paid to the environment of the epileptic child. If the fits are not frequent and the mental powers normal the child should be sent to a normal school and kept in his home if this is pleasant and congenial. If the fits are severe and numerous, or if the intelligence quotient is much below normal, his education may be beyond the capability of his home or school. In Malta no proper institutions exist and it is doubtful if the limited number of cases would justify the creation of such a school. It would probably be better in these cases to educate the parents to carry out their added responsibility. It is to be hoped that in future arrangements may be made to create a special institution in England for the reception of difficult epileptics and other abnormal children from various small colonies, each incapable of catering adequately for such cases.

DRUG TREATMENT: Phenobarbitone is probably the most popular drug in "gran mal" or somatic motor epilepsy. A few years ago another drug was introduced and is known as phenytoin sodium (dilantin and epanutin). It has no narcotic or soporific action like phenobarbitone and this is a great advantage, but Peterman is not greatly impressed by this drug and states that the toxic manifestations, such as vertigo and disorientation were far more common and much more undesirable with this drug than are the occasional reactions of drowsiness met with phenobarbitone treatment. This is especially the case, states Peterman, in children, whereas the drug is stated to be more effective and better tolerated in adults.

"Petit mal" is often resistant to both phenobarbitone and to phenytoin sodium and it was only lately that an efficient drug has been prepared; this is trimethadione (tridione). I have used it recently in a case refractory to phenobarbitone and the fits, which numbered 2 or 3 per day disappeared within a few days. It is stated that it may precipitate attacks of "gran mal" in "petit mal" subjects that occasionally suffer from somatic motor siezures. Also, it is a bone marrow depressant and cases of agranulocytosis have been reported. Lennox in 1946 introduced "Paradione" a derivative of trimethadione and Peterman in 1947 reported his results of "Thyphenytoin" both said to be more effective and less toxic than trimethadione and phenytoin sodium in the treatment of "petit mal." All these drugs may be used together with phenobarbitone.

Finally we must not omit diet, which in the treatment of epilepsy may have a specific effect. According to Peterman 50% of all cases of epilepsy in children may be controlled by the use of a ketogenic diet. The basis of such a diet lies in the excess of fats given to the child coupled with a diminution of carbohydrates. The details can be found in many text-books on treatment. I have never used diet alone and cannot check the accuracy of these figures but 4 cases of "gran mal" in children of 10 to 14 years which were refractory to doses of phenobarbitone that did not produce sleepiness, were enabled to have reasonably small and effective doses of the drug when a ketogenic diet was instituted. On the whole a ketogenic diet, even if effective, is very difficult to carry out for long and is probably impossible in children under 6—7 years of age owing to lack of cooperation on the part of the little patients.