Symposium

NEUROLOGICAL CONDITIONS IN CHILDREN

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INTRODUCTION

HERBERT H. JASPER, M.D.

It is indeed with great pleasure that neurologists of Canada have accepted the invitation of the Academy to participate in its meeting in Toronto and we take this occasion to express our sincere welcome to all our colleagues from south of the border.

When Dr. Orr asked me to organize this Symposium, he added that he would like it to be an "all Canadian show." I objected seriously to usurping the program in this manner and not inviting several outstanding speakers from among our colleagues in the United States who would have added greatly to this program. But finally we have yielded to Dr. Orr's persuasiveness in giving you a sample of some of the work being done in this country. We have the good fortune of possessing, in Canada, a Neurological Institute which is rapidly becoming a Canadian National Institute, established in Montreal under the direction of Dr. Wilder Penfield, who is to participate in the Symposium today, speaking on his favourite subject, epilepsy, with particular emphasis on a new conception of focal epilepsy in children. Dr. William Hawke, of the Hospital for Sick Children in Toronto, whom we like to claim as an adopted member of the staff of the Montreal Neurological Institute, will present some of the results of his long studies of behaviour problems in children. Dr. McEachern, who was unable to attend this meeting because of ill health, has been replaced by Dr. Rabinovitch, who will present a summary of his extensive experience in the study of neuromuscular diseases, a subject which presents puzzling problems to most pediatricians.

Following Dr. Penfield's presentation of a neurosurgical approach to epilepsy in children, I shall try to present a somewhat different point of view concerning the use of electroencephalography in the diagnosis and management of epileptic patients and Drs. McNaughton and Robb will give us their experience in the medical management of epilepsy.

We sincerely hope that you may gain something of interest and value to pediatric neurology from the experience of your Canadian colleagues who are to participate in this Symposium.

NEUROMUSCULAR DISORDERS OF CHILDHOOD†

DONALD MCEACHERN, M.D.,* and REUBEN RABINOVITCH, M.D.

Disorders of muscular function which may make their first appearance in infancy or in childhood include conditions which affect the anterior horn cell, the nerve fibre, the neuromyal junction or muscle fibre.

Congenital myotonia in newborn infants manifests itself in sucking difficulties, Graefe's sign, and protracted contraction of the orbicularis oculi when washing the face with cold water. The involuntarily protracted muscular contraction following voluntary, mechanical or electric stimulation persists after nerve block, indicating a peripheral mechanism. Myotonic muscle is abnormally sensitive to potassium, and epinephrine reduces myotonia in goats and in humans. Desoxycorticosterone


* Deceased.

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abolishes myotonia in the goat. The curarizing action of quinine diminishes neuronal impulses reaching the muscle fibre and produces clinical improvement. Cortisone abolishes the abnormal contraction.

Myasthenia gravis can occur in the newborn and should always be considered when gross muscular weakness or inability to suck is noted. Differential diagnosis should include amyotonia congenita and cerebral birth injury. The possibility that the thymus elaborates a curare-like substance which acts as a barrier at the neuromyal junction is worth investigating, as are modifications in myasthenic symptoms noted during pregnancy or with change in thyroid function.

The hereditary progressive muscular dystrophies detected at the walking age appear to be improved by prolonged daily intake of wheat germ oil concentrate in doses averaging 60 mg. alpha-tocopherol. Creatinuria, indicating a marked degree of breakdown of muscle tissue, is the most characteristic biochemical finding. The difficulty in establishing a human counterpart to the neuromuscular disorder seen in animals with vitamin E deficiency may be due to faulty digestion or absorption of the vitamin in children suffering from a dystrophy. Thus, vitamin E is relatively ineffective when given parenterally to animals with nutritional muscular dystrophy, but recovery occurs when this vitamin is given by mouth, indicating that it is conjugated or changed in some way during digestion to form the antidystrophic principle. There is regrettable confusion in clinical reports as to whether pure alpha-tocopherol, mixed tocopherols, whole wheat germ, wheat germ oil or one of its fractions has been used.

Familial periodic paralysis may appear in infants. It can be detected by low serum potassium which appears to be due to a fault of potassium metabolism. Rapid improvement occurs following adequate intake of this salt. Attacks can be provoked by administration of epinephrine, glucose or adrenal cortical extract, suggesting involvement of the carbohydrate cycle. The T-wave change in the ECG is also a rapid test for changes in serum potassium level.

ORGANIC FACTORS IN BEHAVIOUR DISORDERS

WILLIAM A. HAWKE, M.D. (Toronto), M.R.C.P. (London), F.R.C.P. (Canada)

This is a brief discussion of the methods by which organic diseases affect behaviour. To illustrate these methods, one disease, cerebral palsy, is selected because it seems to demonstrate clearly the various mechanisms involved.

In general, organic lesions affect behaviour in three ways: I. Through the direct effect of the organic lesion upon the central nervous system of the individual. II. Through the emotional reaction of the individual to the clinical symptoms produced by the disease. III. Through the emotional reaction of the environment (parents, siblings and community) to the clinical symptoms. These reactions in turn affect the behaviour of the individual with the disease.

I. The direct effects of the organic lesions upon the central nervous system are multiple, but of these 3 are particularly important.
   a. The reduction of intelligence by a disturbance of function of the cerebral hemispheres. This may be confused with "pseudo-retardation" produced by the limited environment of these children and the reduction of the normal stimuli for intellectual growth. It may also be confused with specific disabilities of perception, particularly sensory or motor aphasie, word blindness, alexia, etc.
   b. The production of convulsive attacks. These attacks may produce associated mental deterioration, confusion through constant subclinical attacks or through periodic disturbances of behaviour of the psychomotor pattern. In addition to this excessive medication may produce disturbances of behaviour.
   c. An organic or physical disturbance of the emotional control of the individual. This is probably due to involvement of the basal ganglia and/or interference with the cortical controls of these areas. It is shown by marked emotional lability and excessive reaction to external situations and stimuli.

II. The emotional reaction of the individual to the clinical symptoms is varied, but includes the following patterns of behaviour. Feelings of
   a. Inferiority
   b. Frustration
   c. Anxiety
   d. Dependency
e. Emotional and physical isolation from other individuals.

There are other patterns of behaviour but these are the ones most commonly encountered. The outward reaction of the individual child to these attitudes depends upon the presence or absence in the child of such qualities as aggression, drive and energy. For example, the shy passive child frequently tends to withdraw into a shell away from personal contacts and surrenders to his physical limitations. On the other hand the aggressive child attempts to ignore his limitations and forces himself into close, often undesired, relationships with other individuals. Quite frequently basic feelings of inadequacy and inferiority in these children become overcompensated and appear to the casual observer as attitudes of superiority.

III. The environment of the individual child reacts to the physical symptoms in various ways. For example, the parents frequently show the following reactions:

a. Fatigue, irritability, exhaustion or depression may be produced by the constant physical care of the child and the uncertain prognosis.

b. An intensification of previous difficulties between the parents may be produced by the above factors.

c. Rejection of the child may occur either in open or hidden form. This may occur because of the physical unattractiveness, the presence of mental deficiency, or the gross restrictions imposed upon the parents by the child.

d. Marked anxiety and concern over the welfare of the child may develop and ultimately lead to overprotection of the child with negligible discipline and absent training.

e. Feelings of guilt frequently develop because the parents feel in some way responsible for the condition which has crippled their child.

f. An inability to accept reality is frequently present and evidenced through their inability to accept a factual appraisal of the child or a poor prognosis in terms of ultimate development.

g. Neglect of the normal children in the family may occur because of the time and energy focused upon the handicapped child.

h. A disturbed relationship with the medical profession is frequently seen. The parents blame the physicians for the production of the condition through faulty obstetrics, for erroneous diagnoses in infancy and for their inability to successfully treat the child.

There is frequently an effect upon the siblings of the child. These siblings often have a deep pity for the handicapped child. However, the constant focus of the family upon the paretic child often leads to frank resentment and jealousy. Since these attitudes are unacceptable, the siblings find themselves in constant emotional turmoil with feelings of affection, rejection and guilt at varying times.

The community has always been affected by these children. The adults are often unduly curious or critical of the parents; the children, cruel and mocking. The parents of the handicapped child are particularly aware of these attitudes and often unduly sensitive to them.

These disturbed attitudes of parents, siblings and community will affect the behaviour of the crippled child, frequently increasing previous attitudes and patterns of behaviour. There is often an exaggeration of such attitudes as inferiority, inadequacy, frustration, dependence, isolation, etc.

This article attempts to outline the mechanisms in which organic diseases, in this case cerebral palsy, affect the behaviour of many people, the patient, the parent, the siblings and the community, through various mechanisms, some of which are obvious and easily understood, and some of which are less obvious and less easily understood.

ROENTGENOGRAPHIC FINDINGS IN CEREBRAL LESIONS OF BIRTH AND INFANCY

DONALD L. MCRAE, M.D.

When the brain is growing the cranial vault is thin and develops brain markings. When brain growth ceases the vault thickens and brain markings fade. When intracranial pressure is elevated the sutures are separated and the head enlarges. An approach to the interpretation of skull radiographs in infants and children based on comparison of the width of the cranial sutures with the thickness and texture of the cranial vault is described.

When a cerebral infarct occurs in early life it causes smallness of the ipsilateral cerebral hemisphere
with smallness of that cranial chamber. The underlying lateral ventricle enlarges. When an angioma is present, it may mask some of the signs of atrophy, or even show some signs of a space-occupying lesion. In less severe damage leading to microgyria the normal convolutions may grow over the small ones, pushing them inwards, thus preventing ventricular enlargement. The ipsilateral hemisphere is small. A picture of a small cranial chamber with symmetric lateral ventricles will result.

An approach to pneumoencephalograms based on comparison of symmetry or lack of symmetry of the skull with symmetry or lack of symmetry of the lateral ventricles is described.

The following practical classification of cerebral lesions of birth and infancy is used.

A. Acute Lesions
   Average Head: Expanding lesions
   Traumatic lesions
   Inflammatory lesions
   Developmental abnormalities

B. Chronic Lesions
   Small Head: Microcephaly (small brain)
   Premature closure of sutures
   Average Head: Seizures
   Choreaathetosis
   Diplegia and paraplegia
   Cerebellar ataxias
   Large Head: Subdural hygroma
   Macrocephaly
   Expanding lesions

THE EPILEPSIES OF CHILDHOOD
WILDER PENFIELD, M.D.

The first objective of treatment is obviously to stop the seizures. But when it comes to surgical therapy—the radical ablation of epileptogenic areas of cortex is more easily carried out, and with greater accuracy after the age of 10 years when boys and girls will cooperate and help during craniotomy under local anaesthesia.

It has been our policy in the past, therefore, to wait for the dawn of understanding that comes, as a rule, between 8 and 12 years of age. But at present we are being forced to reconsider the wisdom of this attitude.

There are young children and infants who have received brain injury due to ischaemia or trauma, lesions usually acquired at birth, who develop focal seizures and whose intelligence fails to develop or even disappears upon the advent of the seizures. There are such patients who become increasingly hemiplegic with the continuance of attacks. Every pediatrician of any experience must have seen this sad progression more than once. For lack of better explanation he may assume encephalitis or some un-named degenerative disease of the brain.

There may be another explanation. In the patient who receives a brain insult and who is destined to develop epileptic seizures, there follows a gradual change in injured gray matter that eventually results in seizures years after the lesion was established. That focus of epileptogenic activity may interfere with the function and the evolution of the uninjured portions of the brain if there are any. Local seizures become generalized. Localized electrographic abnormality is succeeded by generalized abnormality.

When such a train of events is established in infancy and the child arrives at years which should be years of understanding there may be little left to save. Thus we have been forced to reconsider our policy in regard to radical ablation of epileptogenic cortex in those cases in which there is evidence of progressive intellectual degeneration or progressive weakness.

In the treatment of epilepsy, periods of 5 to 10 years should be allowed to elapse before summary and conclusion is made of a series of adequate size.

We have in the past made 3 follow-up studies of the results of surgical ablation of focal atrophic epileptogenic lesions of the cortex. The first (which was carried out with Theodore Erickson) included all cases of this type operated upon at the Montreal Neurological Institute between 1929 and 1939 (115 patients).
They were followed from 1 to 11 years. In this series 43% were placed in the success group, which means that each of them either had no postoperative attacks, or had 1 or 2 attacks before cessation.

The second study carried out with Harry Steelman covered the period of 1939 to 1944 (Penfield and Steelman, 1947) with a follow-up period of 1 to 7 years—59 cases of cortical excision with an operative mortality of 1.2%. Of these 55.8% were placed in the success group. That is to say that about half this group had had no attacks after operation and the other half had 2 or 3 attacks before cessation.

The third report carried out in 1950 with Flanagan consisted in a follow-up of 60 cases of radical operation for temporal lobe epilepsy which Gibbs and Lennox call psychomotor. The results were about the same, 55% of undoubted success.

Now these were for the most part adults in each series. But it may be pointed out that of the 59 cases reported in the second series 17 patients were suffering from seizures produced by unquestioned birth injury. In many other cases birth lesions were a possible cause of the lesions found. The incidence of success as the result of operation rose in these 17 cases from the general average of 56% to 76% success. Thus it may be seen that birth injury provided us with our most favorable group and the attacks usually began in childhood.

**ELECTROENCEPHALOGRAPHY**

**HERBERT H. JASPER, M.D.**

You will recall the original description of Gibbs and Lennox of 3 principal types of epileptic seizure, namely, petit mal, grand mal and psychomotor, and that each of these kinds of attack were manifest by characteristic and distinctive forms of electroencephalogram taken during the seizure.

It should be made clear from the beginning that the petit mal form of attack is not just a minor seizure but it is the momentary, sudden loss of consciousness, lasting a few seconds with immediate recovery so commonly seen in children with epilepsy. There is usually no warning, except in rare instances. This type of seizure should not be confused with minor attacks of a different form which are not associated with immediate sudden loss of consciousness and which may be merely minor attacks of focal cortical epilepsy.

During these 3 forms of seizure the characteristic EEGs, namely, the familiar 3/sec. spike-and-dome discharge for petit mal, the rhythmic 4 to 6/sec. activity during the so-called psychomotor seizure and the more rapid multiple spike or rhythmic spike discharge associated with the major convulsion of attack, have received abundant confirmation from hundreds of investigators throughout the world. There has not been such general agreement that all epilepsies can be adequately pigeon-holed into these 3 types. Furthermore, the attempt to diagnose these 3 forms of epilepsy on the basis of the form EEG alone has been often misleading.

In the electroencephalographic examination of the majority of epileptic patients the diagnosis is not made on the basis of an electric discharge occurring during a clinical seizure, but it is made on the basis of the form and localization of epileptiform waves which occur during intervals between clinical attacks, since the patients do not frequently enough favor us with attacks during the recording of the EEG. On the basis of epileptiform discharges in the EEG an attempt is made to make a diagnosis of the type of epilepsy in any given patient.

In a recent survey of the records from over 2000 epileptic patients carried out with the late Dr. Kershman and myself, it was soon apparent that any form of paroxysmal discharge, including the spike-and-dome, the so-called psychomotor patterns and so forth, could be derived from a focal cortical epileptogenic lesion and that this lesion might be present in most any part of the cortex from the frontal to the occipital or temporal regions. The logical deduction from these facts is that the wave form alone of the EEG is not a reliable index for classification of the epilepsies.

For example, if one finds a spike-and-dome pattern in the EEG and it is localized to, let us say, the postcentral gyrus of one hemisphere, are we to call this petit mal epilepsy because of the characteristic spike-and-dome complex? The patient does not have attacks of sudden loss of consciousness, but on the contrary, he has Jacksonian sensory motor seizures.

It should be obvious that to call these electrographic discharges "petit mal," or even petit mal variant, because they are of the spike-and-dome form, only leads to confusion and mismanagement of the patient. I could cite numerous other examples, especially of the so-called "psychomotor"
electroencephalographic pattern being recorded from focal areas of the cortex in the parietal or frontal region and not associated with any seizures which, by the greatest stretch of the imagination, could be classified as psychomotor in type. When this form of electroencephalographic abnormality is localized to the temporal region, it may be related to psychomotor attacks, but even then we find this true in about one half of patients with temporal lobe seizures.

With regard to grand mal, we have also found rapid multiple spike discharge from a focal cortical epileptogenic lesion even present during the intervals between attacks. To speak of this as grand mal activity is meaningless for it simply represents the focal cortical epileptiform discharge and the form of the attack itself has no specific relationship to this form of electric discharge but is determined by the functional characteristics of that particular area of cortex in which the focus happens to lie. Regardless of the type of epilepsy, all seizures look alike when they have reached the stage of a major generalized convulsion, so that grand mal in this sense is also of no value for the differentiation of the epilepsies.

For many years we have been trying to discover why there seem to be relatively higher percentage of focal cortical epilepsies in Canada and so few true grand mal epilepsies by comparison with statistics from various laboratories in the United States. It is only recently, in discussing this with workers in the States, that we have discovered that it is the tendency in many laboratories to classify a patient as grand mal if he has major convulsive seizures, regardless of their manner of onset. It soon became apparent that a large proportion of the patients classified as grand mal in many laboratories were actually patients with focal cortical seizures, which proceeded into major convulsions. Without a careful analysis of the seizure pattern and extremely detailed localization studies in the EEG, the focus of onset may not be detected. This is largely because of a general lack of appreciation for the extremely varied forms that epileptic seizures may take, depending upon their cortical focus of onset, as described in the previous paper by Dr. Penfield. It is necessary, therefore, that we reorganize our thinking regarding the use of the EEG in our understanding and management of the convulsive disorders, if the use is not to become abuse and the management is not to become mismanagement.

The possibility of the misuse of electroencephalography is enhanced by the fact that it is an attractive and somewhat mysterious laboratory technic to most pediatricians, not having received basic training in electronics and electrophysiology. This is enhanced by the general trend towards mechanized medicine and specialization, which I presume applies to pediatricians as it does to other branches of the medical profession today. It is much easier for the pediatrician to order an EEG in the hopes of having it provide him with the correct analysis or diagnosis of a child with seizures than it is for him to take the trouble of carefully analyzing the history and pattern of the attack from the onset in an attempt to derive a conclusion on the basis of clinical evidence.

The EEG cannot be used to give an automatic nickel-in-the-slot diagnosis of epilepsy. In fact it may be misleading as often as helpful unless carefully correlated with other clinical observations. Perhaps it is now time that we had a general reconsideration of how best we can use the data furnished by the EEG in handling the convulsive disorders.

In the first place it seems well to abandon the conception that seizures of any particular form can be identified with any particular form or frequency of the EEG. A spike-and-wave is not equivalent to petit mal, nor is any other form of discharge equivalent to a form of seizure. The brain cannot be considered a homogeneous organ like the spleen for example which produces one form of attack when it beats slowly at 3/sec., for example, and another form of attack when it beats more rapidly.

The statement that "the brain acts as a whole" is a sterile notion for, from the preceding paper of Dr. Penfield, it is obvious that it is composed of a variety of interrelated parts with distinct functions which manifest themselves in different forms of seizure. Our first problem, therefore, in the analysis of any epileptic patient is to try to understand what part or what system in the brain is primarily involved in the onset of the seizure, just as in any other form of clinical diagnosis the first step is the attempt to locate the site of the disorder, before treatment can be intelligently applied or pathology understood.

When the brain as a whole seems involved we must decide whether this is a result of infectious or toxic disease—past or present anoxia or ischemia of birth—or a reaction to systemic disease of liver—endocrines, etc. Most important is to determine whether the generalized affection may not be a result of seizures, which may have been initially of focal cortical origin.
There are many factors which give a tendency towards epilepsy, factors which lower the threshold sufficiently for the patient to have seizures. These factors may be: hereditary, metabolic, infectious, traumatic, or even emotional.

What actually happens at the site of origin of a seizure is not known. We do know that these factors, acting on the site of origin, lower the threshold and render the brain more susceptible to abnormal electric discharges. Fortunately, also, we have things that raise this threshold.

Hughlings Jackson divided chronic convulsions into 2 classes. (1) "Those in which the spasm affects both sides of the body almost contemporaneously—these cases are usually called epileptic, and sometimes 'genuine' or idiopathic epilepsy. (2) Those in which the fit begins by deliberate spasm on one side of the body, and in which parts of the body are affected one after the other."

As we learn more and more about the various wave patterns as seen in the EEG, and their site of origin within the central nervous system, the distinction between 'focal' and 'generalized' becomes somewhat artificial. We find increasing evidence of 'focal' mechanisms acting even in the case of widespread disorders. The day may come when all epilepsy will be understood in terms of anatomic-physiologic systems within the brain, and a term such as 'diffuse' or 'generalized' will be obsolete.

One year ago we set out to review the causes of focal epilepsy in children. We both had many fixed ideas, and we endeavoured to adjust these by a study of 100 children with focal seizures from the clinics and wards of the Children's Memorial Hospital and the Montreal Neurological Institute. Only patients in whom the seizures had started before the age of 10 years were considered. There were 65 patients from the Children's Memorial Hospital, and 35 from the Montreal Neurological Institute. All the EEGs were done in the Montreal Neurological Institute and were interpreted by different men under the supervision of Dr. Jasper. Pneumoencephalograms were done in both hospitals. There was a certain amount of overlap. That is, one patient might have attended both clinics, or have been in one or both hospitals. Ninety-three of the patients had had one or more EEGs. The rest were not done for various reasons. It may have been that it was felt they would not cooperate, that we did not feel it would help the patient, or maybe the patient might not have appeared for the test.

Cases were selected that showed one or more of the following features:

1. First, a definitely focal seizure.
2. Secondly, a focal electroencephalographic abnormality even if the description of the seizure could not be classed as focal.
3. A third criterion, evidence of cerebral damage, but all these patients fell into one or both of the first 2 groups. There was felt to be evidence of cerebral damage, if there were:
   a. Abnormal neurologic signs.
   b. Grossly abnormal psychometric evaluation in the presence of normal siblings and normal parents, or
   c. Abnormal pneumoencephalogram.

As well as an electroencephalographic localization, an attempt was made to localize the lesion either from the description of the seizure, or from the abnormal neurologic findings. In these patients, we charted all the available data. In many, the patients or their parents were called in to get additional information, and a more accurate description of the seizure and of the etiology was obtained. Frequently, either because the seizures had been infrequent or nocturnal, we were unable to get a good description. These had to be considered as nonfocal seizures, even though the EEG suggested a sharply localized focus.

In some of the younger children they were described as running to their parents as if they were afraid of something. Then, as they grew older, they were able to describe a definite aura. Presumably, they had an aura before, but could only express it as fear.

Focal seizures in infancy and childhood are most readily recognised when motor in type—including such features as head and eye turning, classic movements of face, arm and leg, either simultaneously or by a "Jacksonian March." Often the entire seizure takes place without loss of consciousness. Some-
times it is a postseizure hemiparesis, lasting hours or days, which will draw the attention of parent and doctor to the focal character of the attack.

Sensory phenomena—such as a visual or an auditory "aura"—parasthesias, vertigo, etc., will only be recognised in older children: also, such focal defects as postseizure aphasia.

Automatism—as a focal manifestation either during or following the seizure—has been uncommon in the childhood group we have observed.

We feel that focal manifestations are often missed through not taking adequate history from the parents, or through inadequate observation and recording of attacks occurring in the hospital or admitting office. Sometimes it is focal findings in the EEG which drive one back to complete the clinical history and confirm the diagnosis of focal epilepsy.

Precipitating Factors: We felt that more time should be spent in trying to determine what the precipitating factors were, but found that our own records were inadequate. There was a fairly large number whose seizures were either nocturnal or occurred in the early morning. In a large number, no reference was made to this, and we did not feel that the figures were of any value.

Such things as etiology, other neurologic disabilities, family history, skull roentgenograms, pneumoencephalograms, intelligence evaluation and social history, and the results of medication were reviewed.

Results: Our figures did not vary a great deal from those of others. Thirty-four could be attributed to difficulty at the time of birth. In 12 there was a definite history of anoxia at birth. In 10 there was a definite history of trauma, such as forceps marks, and 11 gave a history of a difficult birth.

Nine were considered to be due to cerebral vascular occlusion. Eleven gave a definite history of severe head injury, and were considered to be post-traumatic.

Thirteen were due to miscellaneous causes, including meningitis, congenital anomalies, tuberculosis, lead poisoning, arrested communicating hydrocephalus, encephalitis and tuberous sclerosis.

In 34 we were unable to establish a definite cause.

Of the 100 patients, 81 were considered to have had clinically a focal type of seizure.

With regard to cerebral damage, it is interesting and significant that 69 showed evidence of cerebral damage, as shown by abnormal neurologic findings such as hemiplegia, marked mental retardation in the presence of normal parents and siblings, or an abnormal pneumoencephalogram. Uniformly moderately enlarged ventricles were not considered as an abnormality.

Fifty-seven had some recognizable neurologic disorder. Of these, 33 showed evidence of hemiparesis of a varying degree. These figures were the most surprising thing of all, and we did not realise that the incidence of clinically recognizable brain damage would be so high.

Thirty-nine showed definite evidence of mental retardation. However, only 59 had a definite assessment. The rest were more or less normal.

We were surprised at the number of children who were previously classed as "idiopathic epilepsy," and who, on careful analysis, had focal seizures, had a focal electroencephalographic abnormality, and evidence of focal cerebral damage.

In 35 we were unable to establish a definite cause, and yet in 17 of these, there was evidence of cerebral damage. In many of these, the onset of the seizures was associated with a fever, or there was a previous history of a febrile illness.

Bailey and Hass (1937) pointed out the incidence of dural sinus thrombosis in early life in routine autopsies and suggested that the condition was more common than was suspected. Putnam and Alexander showed that the pathologic changes found in encephalitis, associated with infectious diseases, may have their origin in multiple small thrombi scattered throughout the brain. O'Brien has recently stressed the importance of cortical venous thrombosis. It is our impression that vascular occlusions probably play a large part in the etiology of focal seizures, but we have no way of proving it.

A good deal of the allotted time has been spent on a discussion of the etiology. Perhaps from an academic point of view this is the most interesting, but the patient is not interested in this. He wants to know how to get rid of his seizures.

The basis of the present-day treatment is drug therapy. It is true that one has to consider the whole picture. To have an accurate diagnosis, an EEG, and to know the social background of the patient are all part of the treatment, but still we must turn to drugs to control the attacks.

Dr. William Lennox recently laid down certain principles which bear consideration:

1. Treat the patient—not just his seizure symptoms.
2. Fit the treatment to the fit.
3. Dosage must be individualized.
4. Dosage must be adequate.
5. Persistent trial is essential.
6. Confidence and cooperation of the patient is essential.
For the purpose of treatment, seizures may be divided into 4 simple groups.
1. Generalized convulsions and focal seizures.
2. Petit mal convulsions.
3. Combined petit mal and grand mal.
4. Psychomotor seizures.
In children one usually starts the treatment with phenobarbital for all types. This is simple, safe and effective.

Generalized Convulsions and Focal Seizures: Start with phenobarbital. If this fails, add dilantin®. If this fails, try mesantoin® and phenobarbital— or any combination of these. Occasionally mebaral® may be substituted for phenobarbital. On rare occasions, phenurone® or thiantoin® may be tried.
Petit Mal: Here again, one starts with phenobarbital. If this fails, then add or switch to tridione®. This is frequently miraculous. If the photophobia bothers the child, paradione® may be used.
Combined Petit Mal and Grand Mal: Again start with phenobarbital and add dilantin® and/or tridione®.
Psychomotor Seizures: A psychomotor seizure is best treated like a grand mal attack.
In spite of these medications, patients still have seizures. It is our feeling that seizures secondary to vascular lesions may be resistant to treatment, and then slowly come under control, and will be easily controlled.
Although we emphasise the importance of drug therapy, in closing, I would emphasise the importance of treating the patient as a whole.
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