Convulsive Seizures in Infancy and Childhood

By BARRY S. RUSSMAN, M.D.

One of the most dramatic medical emergencies that confronts the pediatrician is the convulsing child. In this situation, etiology should be thoroughly understood before treatment is considered. And it is imperative that the physician develop a well-organized diagnostic and therapeutic approach to the problem. Table 1 provides an outline that we have found successful.

The first step in managing the con-

TABLE 1

ACUTE MANAGEMENT OF SEIZURES

GENERAL
1. Maintain airway
2. Prevent shock
3. Protect child from injury
4. Step back

History
- trauma?
- pre-existing disease?
- toxins?

Physical
- trauma?
- focality
- (eye deviation)?

TREATMENT
1. Start IV
2. Draw blood — metabolic studies, toxins
4. Diazepam (Valium®): 0.1-0.3 mg./kg. at 1 mg./min.
5. If seizure stops, follow with phenobarbital, 5 mg./kg. IM
6. If seizure continues, place 240 mg. phenobarbital in 100 cc. D5W; drip in at 10-20 cc./hr.

PROBLEM
Barbiturates in combination with Valium® may produce apnea.

ALTERNATIVE TREATMENT
1. Paraldehyde — 4% solution — 0.1 cc./kg. (IM, slowly IV, or rectally in oil). Do not exceed 5 cc.
2. Diphenylhydantoin (Dilantin®), IV — 8 mg./kg. at 50 mg./min.; repeat every 30 minutes.
Because of potential cardiac arrhythmias, use ECG monitor. Absorption of IM Dilantin® is not reliable.
vulsing patient is to ensure that the airway is adequate and the patient is not in shock. We then ask everyone to step back. An understanding of what may cause a seizure allows specific questions to be asked and a pertinent physical examination to be performed quickly so that emergency action that might prevent a catastrophe can be taken.

Table 2 categorizes the causes of seizures under seven general headings. The immediate history should continued

TABLE 2

ETIOLOGY OF SEIZURES

1. Trauma
   Subdural
   Battered child
2. Infection
   Meningitis
   Encephalitis
   "Febrile convulsions"
3. Metabolic
   Hypoglycemia
   Electrolyte imbalance
   Renal failure
   Hepatic disease
   Aminoacidurias
4. Toxins
   Lead
   Phenothiazines
   Thallium
   Stimulant medication (camphor, amphetamines)
   Narcotic withdrawal
5. Vascular
   Arteriovenous malformations
   Sickle cell disease
   "Strokes"
   Collagen diseases
6. Structural lesions (congenital and acquired)
   Porencephalic cysts
   Agenesis of corpus callosum
   Microcephaly
   Brain tumors
7. Genetic

DESCRIPTION VERMOX (mebendazole) is methyl 5-benzoylbenzimidazole-2-carbamate.

ACTIONS VERMOX exerts its anthelmintic effect by blocking glucose uptake by the susceptible helminths, thereby depleting the energy level until it becomes inadequate for survival. An insignificant amount of mebendazole is absorbed from the gastrointestinal tract. Most of this is excreted in the urine within three days either as metabolites or unchanged drug.

INDICATIONS VERMOX is indicated for the treatment of Trichuris trichiura (whipworm), Enterobius vermicularis (pinworm), Ascaris lumbricoides (roundworm), Ankylostoma duodenale (common hookworm), Necator americanus (American hookworm) in single or mixed infections. Efficacy varies in function of such factors as pre-existing diarrhea and gastrointestinal transit time, degree of infection and helminth strains. Efficacy rates derived from various studies are shown in the table below:

<table>
<thead>
<tr>
<th>Trichuris</th>
<th>Ascaris</th>
<th>Hookworm</th>
<th>Pinworm</th>
</tr>
</thead>
<tbody>
<tr>
<td>cure rates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean (range)</td>
<td>98% (91-100%)</td>
<td>99% (90-100%)</td>
<td>99.7% (90-100%)</td>
</tr>
<tr>
<td>egg reduction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean (range)</td>
<td>99% (70-99%)</td>
<td>99.9% (90-95%)</td>
<td>99.9%</td>
</tr>
</tbody>
</table>

CONTRAINDICATIONS VERMOX is contraindicated in pregnant women (see Pregnancy Precautions) and in persons who have shown hypersensitivity to the drug.

PRECAUTIONS PREGNANCY: VERMOX has shown embryotoxic and teratogenic activity in pregnant rats at single oral doses as low as 10 mg/kg. Since VERMOX may have a risk of producing fetal damage if administered during pregnancy, it is contraindicated in pregnant women.

PEDIATRIC USE: The drug has not been extensively studied in children under two years; therefore, in the treatment of children under two years the relative benefit/risk should be considered.

ADVERSE REACTIONS Transient symptoms of abdominal pain and diarrhea have occurred in cases of massive infection and expulsion of worms.

DOSED AND ADMINISTRATION: The same dosage schedule applies to children and adults.

For the control of pinworm (enterobiasis), a single tablet is administered orally, one time. For the control of roundworm, whipworm (trichuriasis), and hookworm infection, one tablet of VERMOX is administered, orally, morning and evening, on three consecutive days. If the patient is not cured three weeks after treatment, a second course of treatment is advised. No special procedures, such as fasting or purging, are required.

HOW SUPPLIED VERMOX is available as tablets, each containing 100 mg of mebendazole, and is supplied in boxes of twelve tablets.

VERMOX (mebendazole) is an original product of Janssen Pharmaceutica, Belgium, and co-developed by Ortho Pharmaceutical Corporation.

†Because VERMOX has not been extensively studied in children under 2 years of age, the relative benefit/risk should be considered before treating these children. VERMOX is contraindicated in pregnant women (see: Pregnancy Precautions) and in persons who have shown hypersensitivity to the drug.
determine whether trauma has occurred. A child with convulsions secondary to head trauma might show skin manifestations of such trauma. Further, retinal hemorrhages should lead to the diagnosis of subdural or subarachnoid hemorrhage; nuchal rigidity should be found in the latter. Obviously, neurosurgical intervention has to be considered in these cases. Observation of eye deviation is extremely important, as this may be the only manifestation of a focal lesion, deviation to the right suggesting a left-sided focus. The pupils are usually dilated while the child is convulsing; asymmetry of pupil size will occasionally be noted. However, this does not necessarily establish the existence of an expanding intracranial lesion.\(^1\) Pupillary constriction, on the other hand, would suggest a narcotic overdose.

The presence of fever should arouse suspicions of bacterial meningitis. While the seizure is being stopped, a temperature should be obtained and preparations made for a spinal tap. This article will not discuss the controversies that surround the diagnosis and management of the simple febrile convulsion.

Metabolic causes of a convulsion also require immediate and very specific action. This category of seizure can also be suspected on the basis of a brief history and rapidly performed physical examination. Questions regarding pre-existing illnesses are very helpful, as seizures may occur in patients with chronic renal disease. A pale-appearing child with a cold sweat should arouse suspicions of hypoglycemia. The presence of sunken eyes would suggest dehydration.\(^2\) Blood should be drawn for sugar, electrolyte, calcium, and urea nitrogen analysis before initiation of anticonvulsant treatment. In fact, some recommend routine intra-

venous administration of glucose before treatment with anticonvulsants.

Toxic causes of seizures do not require immediate, specific action, although stomach aspiration should be considered. However, an appropriate history and laboratory studies should be obtained to determine the presence of lead intoxication (pica, flat plate of the abdomen, blood lead level). Ferric chloride testing of the urine is an adequate screening test for phenothiazine intoxication. Thallium is used in rat poison. Usually, there will be a history of vomiting for a few days before the convulsion. The treatment of camphor-induced seizures is similar to that for other central nervous system stimulant-induced seizures — namely, with anticonvulsants.

Vascular lesions constitute the fifth general category in our etiologic approach to the convulsing child. Unless nuchal rigidity is present, suggesting subarachnoid bleeding, symptomatic treatment is provided in advance of a more definitive evaluation. Sickle cell disease must be considered in a black child.\(^3\)

Structural lesions — such as porencephalic cysts, agenesis of the corpus callosum, microcephaly, and brain tumors — have been associated with convulsions. The initial approach consists of stopping the seizure; this is followed by the more definitive evaluation.

Genetic epilepsy, on the other hand, is characterized by the absence of a demonstrable structural or metabolic problem and implies that an inherited hyperexcitable population of neuron exists. Metrakos and Metrakos\(^4\) have shown that genetic epilepsy most commonly occurs between the ages of five and 12.

Table 3 outlines the most likely causes of seizures, depending on the
age of the child. During the first two years of life, metabolic, infectious, and congenital lesions are the most common causes of seizures. During childhood, trauma, toxins, and genetic predispositions are the most likely causes. Acute or chronic renal disease is a rare cause of seizure, but it must be considered. The most common causes of convulsions in the adolescent age group are trauma, genetic predisposition, and occasionally brain tumors.

The specific causes of seizures that require immediate action have been considered. Diazepam (Valium®), 0.1-0.3 mg./kg. administered intravenously over a two-to-three-minute period, is now given. This can be repeated 20 minutes later. Once the seizure has been terminated, an intramuscular injection of phenobarbital, 5 mg./kg., is then given, as the effect of Valium® lasts no more than 30 to 45 minutes. Potential respiratory distress must be anticipated at all times.

A detailed history, with special attention to potential causes, can now be obtained. Not only should the causes outlined in Table 2 again be considered, but a history of personality change over the past several months or a drop in school marks should alert the physician to the possibility of degenerative brain disease, such as subacute sclerosing panencephalitis.

The physical examination most commonly reveals a normal child. However, one should look for some very specific abnormalities. Head circumference should be a routine part of the evaluation, and cranial bruits should be listened for. An examination of the skin might reveal a port-wine stain of the face (Sturge-Weber disease), depigmented spots on the trunk (tuberous sclerosis), or café-au-lait spots (neurofibromatosis).

LABORATORY EXAMINATION

The EEG is absolutely necessary as part of the total evaluation. Frequently, the EEG will help differentiate between petit mal and psychomotor epilepsy. Further, the EEG can provide clues as to whether there is a metabolic basis for the problem or whether a degenerative brain disease might be present. It is uncommon for a brain tumor to manifest itself as a cause of seizures in children; the possibility certainly exists, however, and an EEG is a valuable screening test for this diagnosis. Finally, it must be realized that an abnormal EEG does not establish the diagnosis of epilepsy unless the EEG happens to be obtained during the actual seizure.

Unless an infection, such as encephalitis or meningitis, is suspected, a spinal tap is not a routine part of the evaluation in our hands.

Skull films are routinely obtained in all children with a diagnosis of epilepsy. This is the only way calcifications can be found that might

continued

TABLE 3

<table>
<thead>
<tr>
<th>ETIOLOGY OF SEIZURES BY AGE</th>
<th>0-2 Years</th>
<th>5-12 Years</th>
<th>2-5 Years</th>
<th>12-20 Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracranial birth injuries</td>
<td>Infection</td>
<td>Trauma</td>
<td>Infection</td>
<td>Trauma</td>
</tr>
<tr>
<td>Congenital defects</td>
<td>Infection</td>
<td>Toxins</td>
<td>Genetic</td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>Fever</td>
<td></td>
<td>Genetic</td>
<td>Tumors</td>
</tr>
<tr>
<td>Metabolic</td>
<td></td>
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<tr>
<td>Fever</td>
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<tr>
<td>Metabolic</td>
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</tbody>
</table>
lead to a more specific diagnosis. Technetium scanning is not routinely ordered unless the seizure had a focal onset or was focal in nature or the EEG shows a definite focus.

**CONVULSION NOT WITNESSED BY THE PHYSICIAN**

Unfortunately, more often than not, the physician does not have the opportunity to witness the actual convulsion. In this case, the problem is not only to determine the cause but also to decide whether the paroxysmal event was, in fact, a seizure that might be recurrent or another type of paroxysmal behavior that would not require anticonvulsant therapy. A carefully obtained history is the only way to establish the diagnosis of a seizure under these circumstances. The description of the seizure should be obtained in such detail that one would conclude that the physician was the actual witness.

Before discussing the questions to ask, it is necessary to establish a working definition of the term "seizure" and to describe the different types of seizures.

The term **seizure** refers to a sudden onset of abnormal behavior resulting from an excessive discharge of a hyperexcitable neuronal population. This behavior should be disorderly and transient. Other manifestations of a seizure include some, but not all, of the following:

1. Loss of or derangement in consciousness
2. Excess or loss of muscle tone or movement
3. Alteration of sensations
4. Alterations of autonomic nervous system function, including palleness, sweating, etc.
5. Emotional changes, including fright, anger, and aggression.

As it is commonly understood, the term **epilepsy** refers to chronic, recurrent seizures; the term **convulsion** is synonymous with grand mal epilepsy.

Table 4 lists the five types of seizures. **Grand mal epilepsy** usually starts with an aura. The **aura** most frequently consists of dizziness and/or abdominal pains. If the patient is of an appropriate age, he should be asked about unusual sounds, smells, visions, or feelings prior to "passing out." The **ictus** follows shortly and usually starts with the tonic phase, which consists of increased tone of the extensor muscles of the four extremities as well as the neck muscles. This is followed by the clonic phase: contraction of the antigravity muscles, the biceps of the upper extremities, and the hamstrings of the lower extremities causes the patient to go into flexion. Commonly, with muscle relaxation, there is incontinence of urine or stool. After the patient goes through a series of tonic and clonic phases, fatigue occurs. The fatigue or **postictal** state can last from five minutes to several hours.

**Focal seizures** may be sensory or motor (Jacksonian), depending on the location of the abnormal discharge. Focal epilepsy is considered in a separate category, as a focal

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**TABLE 4**

**CLINICAL CLASSIFICATION OF SEIZURES**

1. Grand mal
2. Focal
3. Psychomotor
4. Petit mal
5. Minor motor
6. Akinetic
7. Myoclonic

**continued**
Before prescribing, please consult complete product information, a summary of which follows:

**INDICATIONS:** Tigan is indicated for the control of nausea and vomiting.

**CONTRAINDICATIONS:** The injectable form of Tigan in children, the suppositories in premature or newborn infants, and in patients with a known hypersensitivity to trimethobenzamide are contraindicated. Since the suppositories contain benzocaine they should not be used in patients known to be sensitive to this or similar local anesthetics.

**WARNINGS:** Tigan may produce drowsiness. Patients should not operate motor vehicles or other dangerous machinery until their individual responses have been determined. Reye's Syndrome has been associated with the use of Tigan and other drugs, including anesthetics, although their contribution, if any, to the cause and course of the disease hasn't been established. Tigan is characterized by an abrupt onset shortly following a nonspecific febrile illness, with persistent severe vomiting, lethargy, irritability, behavioral changes, or encephalopathy leading to coma, convulsions, and death.

**Usage in Pregnancy:** Trimethobenzamide hydrochloride was studied in reproduction experiments in rats and rabbits and no teratogenicity was suggested. The only effect observed was an increase in the percentage of embryonic resorptions or stillborn pups in rats administered 20 mg and 100 mg/kg and increased resorptions in rabbits receiving 100 mg/kg. In each study these adverse effects were attributed to one or two dams. The relevance to humans is not known. Since there is no adequate experience in pregnant or lactating women who have received this drug, safety in pregnancy or in nursing mothers has not been established.

**PRECAUTIONS:** During the course of acute febrile illness, encephalitis, gastroenteritis, dehydration and electrolyte imbalance, especially in children and the elderly or debilitated, CNS reactions such as opisthotonos, convulsions, coma and extrapyramidal symptoms have been reported with and without use of Tigan or other antihistamine agents. In such disorders caution should be exercised in administering Tigan, particularly to patients who have recently received other CNS-acting agents (phenothiazines, barbiturates, belladonna derivatives). It is recommended that severe emesis should not be treated with an anticholinergic drug alone; where possible the cause of vomiting should be established. Primary emphasis should be directed toward the restoration of body fluids and electrolyte balance, the relief of fever and relief of the causative disease process. Overhydration should be avoided since it may result in cerebral edema.

The antiemetic effects of Tigan may render diagnosis more difficult in such conditions as appendicitis and obscure signs of toxicity due to overdosage of other drugs.

**ADVERSE REACTIONS:** There have been reports of hypersensitivity reactions and Parkinson-like symptoms. There have been instances of hypotension reported following parenteral administration to surgical patients. There have been reports of blood dyscrasias, blurring of vision, coma, convulsions, depression of mood, disorientation, dizziness, drowsiness, headache, jaundice, muscle cramps, and opsiphotonos. If these occur, the administration of the drug should be discontinued. Allergic-type skin reactions have been observed; therefore, the drug should be discontinued at the first sign of sensitization. While these symptoms will usually disappear spontaneously, symptomatic treatment may be indicated in some cases.

**HOW SUPPLIED:** Suppositories, Pediatric, 100 mg. Boxes of 10. Suppositories, 200 mg. boxes of 10 and 50.

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**Psychomotor epilepsy** is the third clinical category. Clinically, this is one of the most difficult types of epilepsies to diagnose. If one approaches it according to the schema outlined for grand mal seizures, it can be more readily understood. The aura for the psychomotor seizures is quite varied but does not differ significantly from that of grand mal seizures. It precedes the seizure by a few seconds to minutes. The ictus, on the other hand, is quite complex. There are sensory, psychic, and motor phenomena, although not all three are necessarily present. The seizure may consist merely of a feeling of numbness or paresthesia in the extremities. Psychic symptoms include hallucinations, partial or complete loss of awareness, and feelings of depression, anger, and fear. The motor component may consist entirely of purposeless movements of the extremities. The postictal state includes complaints of headache, fatigue, and hunger.

**Petit mal epilepsy** is the fourth clinical type. This is an uncommon type of epilepsy, occurring in only 5 per cent of children with epilepsy. Further, petit mal does not occur under the age of three. The attacks start suddenly and without warning. The episodes last less than 15 seconds. The patients are unaware of their environment during the episodes, and the attacks stop suddenly. Occasionally, purposeless motor movements of the extremities and face are noted.

Finally, **minor motor seizures**, the most difficult type of seizure to control, are the fifth clinical type. These can be further subdivided into akinetic seizures, which consist of loss
of muscle tone (drop attacks), and myoclonic seizures, which consist of a sudden jerk of muscle groups (head drop, unilateral or bilateral extremity, or body flexion or extension). These seizures are usually observed in the younger child and very rarely occur after age 12.

As has already been stated, the history is the only way to establish the diagnosis. The history must be obtained from the person who observed the actual seizures. This may mean contacting a child’s schoolteacher or speaking to a sibling or a peer who observed the episode. The parent must be interviewed, and the child should not be neglected during this aspect of the diagnostic process.

Just as a seizure can be divided into the aura, ictus, and postictal phases, so the history should approach the problem with questions regarding each of these stages. Precipitating factors — such as fatigue, emotional upset, and nonspecific illnesses — should be determined, as seizure treatment might be altered depending on the presence of one of these variables.¹

**DIFFERENTIAL DIAGNOSIS**

When the history is taken, the differential diagnosis of unexplained episodic behavior must be considered (Table 5). Hysterical “seizures” can

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**TABLE 5**

**DIFFERENTIAL DIAGNOSIS OF PAROXYSMAL BEHAVIOR**

1. Hysterical “seizure”
2. Syncopal attacks
3. Breath-holding attacks
4. Sleep disturbances, including night terrors, sleep walking, and talking
5. Migraine headaches
Abnormal behavior during sleep is commonly confused with epilepsy

sometimes be very difficult to differentiate from grand mal or psychomotor seizures. The aura of hysterical "seizures" may consist of choking; consciousness is impaired but not lost. The motor movements are commonly bizarre rather than of the typical tonic-clonic type. The patient almost never hurts himself, and incontinence rarely occurs. The hysterical "seizure" usually stops almost as rapidly as it starts, with no postictal state; however, the patient will occasionally appear or act fatigued.

Syncope (fainting spells) must also be considered in the differential diagnosis of epilepsy. Syncopal attacks almost invariably occur in the upright position. The "aura" commonly consists of vertigo or light-headedness. During the attack, the patient may develop brief tonic-clonic movements, a result of transient cerebral anoxia. The postictal symptoms are minimal and consist of irritability or fright. In my experience, many children use the term "dizziness" when, in fact, they mean light-headedness.

Breath holding also has to be considered in the differential diagnosis of paroxysmal behavior with a loss of consciousness. These attacks always occur following a cry or a fright. In association with the apnea and perioral cyanosis, some clonic-tonic movements as well as opisthotonic posturing may be observed. Occasionally, pallor rather than cyanosis will be noted. Postictal symptoms, which may consist of irritability and fatigue, are short-lived.

Abnormal behavior during sleep is commonly confused with epilepsy. Night terrors consist of unexplained screaming, staring, and incomprehensible rambling associated with bizarre motor movements. The patient will have no recollection of the episode the following morning. Also, the patient may sleep-walk as part of the attack.

Migraine headaches are common in children and can be very difficult to differentiate from epilepsy. The major symptom in migraine is headache. The migraine phenomenon also consists of an aura that commonly comprises visual symptoms. Headache then occurs, followed by nausea, vomiting, and sleep. In epilepsy, the headache usually occurs when the patient awakens from the postictal state. However, a sudden or transient headache may be the only manifestation of epilepsy. If the history does not allow one to differentiate between these two entities, a trial of anticonvulsants may be the only alternative.

IMPORTANCE OF FOLLOW-UP CARE

Once the diagnosis of epilepsy has been established, a prescription is written. This should not be the end of the consultation, however. Whitehouse found that approximately 56 per cent of children with epilepsy had learning problems. Rodin et al. have established that it is very difficult for some people with epilepsy to obtain employment. This difficulty relates not to the frequency, severity, or type of epilepsy but, rather, to learning problems experienced by the child when in school. It has also been noted that the attitudes of school personnel are still archaic, suggesting that the physician should have some school contact once the diagnosis has been established.

SUMMARY

Epilepsy is a symptom and not a disease. An etiologic and therapeutic approach to the acutely convulsing child has been outlined. The necessity of attempting to determine the cause while in the process of stopping the seizure has been em-
phased. Suggestions as to what to evaluate on physical examination and what laboratory tests to order have been discussed.

An approach to the child whose seizure was not witnessed by a physician is also presented. Emphasis is placed on obtaining an accurate history. Finally, the treatment of a child with epilepsy involves more than prescribing appropriate medication. The child’s performance in school and the concerns of parents and patient must be considered.

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