for all. Its very emergency nature may at times add trouble since the tendency may be to overdose. It should be kept in mind that harm can be done by overtreating a patient in status epilepticus, particularly when using the longer-acting drugs. The comatose state frequently accompanying status epilepticus can be worsened by the cumulative effect of too frequent injections of large doses of barbiturates or other hypnotic agents.

Patients become so thoroughly drugged with phenobarbital and Dilantin that vital processes and consciousness are reestablished only with difficulty. One should also not ignore the disquieting, though still uncertain reports suggesting that large doses of these drugs may exert more than a transient effect upon neuronal structures, particularly those still undergoing vigorous ontogenetic development. This should not be construed as favoring a more nihilistic approach, but only as a reiteration of the opening statement in this discussion: namely, that management of status epilepticus taxes the ingenuity of the physician in establishing a proper balance between undertreatment and overtreatment.

**Psychomotor Epilepsy in Childhood**

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The most poorly understood and most frequently misdiagnosed seizure state of childhood is psychomotor epilepsy. Difficulties in diagnosis are related to the variety of possible clinical manifestations which characteristically differ from one child to another. In addition, psychomotor epilepsy can occur at any age, even during infancy. Therefore, the child's ability to verbalize the perceptive and affective sensations of this seizure state is obviously limited by his chronologic age.

The diagnostic dilemma is further complicated as physicians tend to confuse psychomotor seizures and petit mal epilepsy.

**INCIDENCE AND ETIOLOGY**

The terms psychomotor and temporal lobe epilepsy are often used synonymously and interchangeably. At times the seizure state may also be called uncinate epilepsy, epileptic automatisms or epileptic fuges. However, not all psychomotor seizures are associated with temporal lobe lesions, nor is temporal lobe pathology always productive of psychomotor epilepsy. Abnormalities and electrical foci from areas other than the temporal lobe can produce this seizure state. For these reasons, the term psychomotor epilepsy is preferred, and temporal lobe epilepsy, if used, should be restricted to those psychomotor seizures that result from primary temporal lobe pathology.

Ten to 20% of children in most pediatric seizure clinics have psychomotor epilepsy. Focal lesions are often considered to be the responsible etiologic factor, but diffuse encephalopathies, above all in children, are more commonly encountered. Prolonged febrile convulsions, perinatal trauma and hypoxia, craniocerebral trauma or meningoencephalitis can be the specific etiologic condition. Expansive lesions including neoplasms, vascular malformations, cysts and abscesses must be considered, especially when there is clinical or electrical evidence of a focal lesion. Genetic factors can also be responsible, even when focal temporal lobe electrical discharges are seen, as similar electrical abnormalities and/or seizures may be observed in parents and siblings.

**CLINICAL MANIFESTATIONS**

Psychomotor seizures have a variable occurrence in that one or more seizures can occur daily with an intervening seizure-free state of days or weeks. The episodes are generally brief; they may be as short as a few seconds or as long as 10 to 15 minutes,
but most of them last two to three minutes. The seizure has three essential components: (1) premonitory phase and aura, (2) ictus or seizure components, and (3) postictus.

Premonitory phase or prodrome can precede the actual seizure by hours or days. Personality changes, above all, irritability and hyperactivity, headaches, vasomotor disturbance with palid or flushed facies and changes in appetite with unusual hunger or anorexia are the most frequently encountered. An alert parent can recognize these manifestations of an impending seizure. The aura precedes the seizure by seconds or minutes. Rarely does the child describe the unusual sensory perceptions of déjà vu, déjà veçu, macropsia or micropsia, or olfactory and gustatory hallucinations which are common in the adult patient. Typically the child presents with a more primitive but nevertheless a highly consistent recurring aura. Examples of these are a “funny feeling” in the head or abdomen; a tight feeling; a rising feeling in the throat; a fear reaction with the child running to a parent or hiding in the bedroom; a headache or abdominal pain; the desire to urinate or defecate; or an inappropriate speech pattern; and (5) autonomic vasomotor disturbance with salivation, vomiting, flushing or pallor. Defecation or enuresis are rarely encountered.

Sensory manifestations result from involvement of the somatic and special sensory nervous system. Somatic sensory symptoms include numbness, tingling, or sensations of hot and cold. Special sensory involvement produces impairment, distortion or loss of vision, as well as hearing loss and dizziness. Rarely does the child express experiences of olfactory or gustatory phenomena.

Postictal manifestations, lasting minutes to even hours, include headache, lethargy, vomiting, hunger, thirst, impaired speech and focal or generalized paresis.

DIAGNOSIS

The diagnosis of psychomotor epilepsy is primarily dependent on its clinical recognition. Once the diagnosis is established, ancillary testing may be helpful in confirming it and delineating atrophic or expansive focal lesions. For these reasons the child with psychomotor epilepsy is frequently subjected to more detailed testing than children with other seizure types. Skull radiographs are usually normal, but hemiatrophy of the skull or middle cranial fossa and focal calcifications may be of diagnostic importance. In addition to skull radiographs, an electroencephalogram (EEG) is routinely performed and must include the routine tracing, as well as that in sleep. Under special circumstances a nasopharyngeal recording may be necessary. The EEG may be normal, nonspecifically abnormal, or may show focal abnormalities, particularly at the temporal electrodes. Focal abnormalities include independent spike discharges at the anterior and mid-temporal electrodes, especially during light sleep, and unilateral slow wave foci.

Further testing is indicated to delineate and distinguish an atrophic from an expansive lesion when the seizure state is associated with focal clinical, radiological, and/or electrical abnormalities, and in certain instances refractory to anticonvulsant medication. This may include cerebrospinal fluid analysis, tangent screen visual fields in the older child, radioisotope brain scan, and, in selected children, a cerebral angiogram and pneumoencephalogram.

Psychomotor epilepsy is often confused with other seizure types, above all petit mal epilepsy. Petit mal never occurs before 2 years of age, rarely before 4 years of age, and is most commonly seen between 4 to 10 years of age. The seizure is never preceded by an aura nor is it followed by postictal phenomena. Petit mal is characterized by its (1) brevity, duration less than 30 seconds; (2) fre-
quency, up to 100 times daily; (3) specific electrical abnormalities, with bilaterally synchronous 2½ to 3½ cycles per second spike and wave discharges; and (4) the ability to precipitate a seizure with hyperventilation. Postural tone changes, with falling to the ground never are observed in petit mal epilepsy. Complex automatisms, frequently observed during a psychomotor seizure, are rarely seen in petit mal where seizure activity consists primarily of staring or twitching movements of eyelids, eyebrows or the head, recurring at the rate of about three jerks per second.

TREATMENT

Treatment of childhood psychomotor epilepsy is primarily medical, and surgery is reserved for the rare child with an expansive lesion or the child with an atrophic lesion that is refractory to anticonvulsants.

Control of seizures is more difficult with psychomotor epilepsy than with most other seizure types, though the principles of medical management are no different than with grand mal or major motor epilepsy. Seizure control without untoward effects is the ultimate goal. The recent introduction of anticonvulsant blood levels (see page 557 of this section) has been invaluable, particularly for children who show toxic signs or poor control of seizures. Phenobarbital, the drug of choice, is administered in the initial dosage of 5 mg/kg and therapeutic blood level is usually between 10 to 30 mg/100 ml. When phenobarbital is not tolerated or seizures are not controlled, diphenylhydantoin (Dilantin) sodium is added or substituted in the dosage of 10 mg/kg and the therapeutic blood level is 10 to 20 mg/100 ml. Recently carbamazepine (Tegretol) has proven to be highly effective in selected cases of psychomotor epilepsy. Infants and young children are started on a daily dose of 100 mg twice daily, whereas older children can tolerate 200 mg three or four times daily. Tegretol is potentially toxic, and both blood counts and liver function must be monitored at monthly intervals. Other compounds necessary to achieve seizure control may include primidone (Mysoline), 15 to 25 mg/kg; bromides in the daily dose of 500 to 2,000 mg in older children; and only as a last resort, the potentially hepatotoxic phenacemide (Phenurone). If this must be used it can be prescribed in the dosage of 250 mg three times a day; if necessary it may be increased by 250-mg increments at weekly intervals until seizures are controlled or an approximate total dose of 1,500 mg is prescribed. Children receiving this potentially toxic compound must have monthly blood counts and liver function studies.

Since psychomotor seizures are often difficult to control, it is suggested that anticonvulsant medication be maintained for at least four years after the last reported seizure, and in the presence of continued electrical abnormalities drugs should be administered for an indefinite period of time.

Diagnosis and Treatment of Childhood Myoclonic Seizures

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Epileptic seizures in children occur in a variety of forms. While it may be difficult to classify some attacks, most seizures may be broadly divided into five groups: major motor (grand mal), petit mal, psychomotor (temporal lobe), myoclonic and autonomic. This writing is devoted to a discussion of the clinical manifestations, EEG findings, etiology, prognosis and treatment of myoclonic epilepsy of childhood. The data presented are based on approximately 1,500 children with myoclonic seizures who have been studied at The Johns Hopkins Hospital Epilepsy Clinic. In 1,150 patients, myoclonic seizures appeared during the first two years of life, most commonly between 3 and 9 months of age; and in the remaining 350 children, after the first two years of life, usually between 3 and 7 years of age. We classify myoclonic epilepsy into two types on this basis of age at onset.
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