Management of Psychomotor (Temporal Lobe) Seizures

The term "psychomotor epilepsy" was introduced in 1937 to designate a heterogenous group of seizures which did not conform to the classical descriptions of grand mal, focal or petit mal seizures. This has proved useful in emphasizing to the physician that the term "petit mal" should be reserved only for a characteristic disorder which develops in childhood or adolescence (onset rare, if ever, after the age of 20), with highly specific clinical features. Petit mal attacks are characterized by transient clouding of consciousness, an absence, lasting usually 5 to 30 seconds, with or without minor movements of the head or extremities (myoclonic jerks), following which the patient is alert and continues his usual activity. Occasionally loss of postural tone may occur, and the youngster may drop to the floor in an akinetic petit mal attack. Often the child is unaware of having an attack and it may be dismissed by parents as momentary daydreaming or inattentiveness. The delineation of psychomotor attacks from petit mal has important implications with regard to the etiology of the seizure, the correlation with focal brain pathology, the need for diagnostic complete-
nature of the aura and of the behavior following the attack must be sought. Diagnostic errors commonly occur when the physician cannot obtain and clarify the history from an observer other than the patient.

The occurrence of a seizure disorder in a child or adult may have major consequences upon their general behavior apart from the seizure disorder itself. The behavioral problems vary considerably, but are most likely to occur in patients with frequent psychomotor attacks. A tendency toward easy excitability, irritability, aggressive behavior, and “schizophrenic reactions” has been commonly noted. Similarly, depressive reactions are also common. Some of these appear to derive from difficulties in social and occupational adjustments. The intensity of the behavioral problems commonly makes evaluation of the severity and frequency of the seizures difficult to ascertain. The emotional consequences of the attacks may be more disabling than the seizure disorder itself. Patients, and their families, must be educated concerning the nature of the disorder, and, at times, formal psychotherapy is an essential adjunct to drug therapy.

Falconer and Taylor (Arch. Neurol. 19, 353, 1968) have reviewed the pathologic findings in 100 cases of surgically treated cases of temporal lobe epilepsy, 53 percent of which had “medial temporal sclerosis” or other scarring, 22 percent had small cryptic tumors, and only 25 percent revealed no structural lesion. Thus, the occurrence of small angiosas or gliomas must be considered in the diagnosis, particularly with the onset of the seizure disorder after the age of 20. A detailed neurological examination should always include a careful plotting of the visual fields to detect small lesions in the temporal lobe. Routine skull films are essential in detecting the occasional occurrence of a calcified lesion in the temporal lobe. While electroencephalography has contributed greatly to the delineation of these cases, the presence of a normal electroencephalogram need not detract from the diagnosis in a patient with a characteristic history. The occurrence of an elevation in the cerebrospinal fluid protein is generally indicative of a neoplastic or other space-taking lesion in the temporal lobe and usually warrants radiologic contrast studies.

The key to the successful management of such patients is the ability of the physician to prevent the occurrence of seizures with drugs. Diphenylhydantoin and phenobarbital, in combination, are most useful in the management of psychomotor seizures. Primidone (Mysoline®) and a wide variety of other anti-convulsant drugs play a supplementary role. Specific anti-petit mal drugs, which include ethosuximide (Zarontin®) or trimethadione (Tridione®) are not indicated. The most common error in the treatment of such patients is the failure to use enough medication. Diphenylhydantoin and phenobarbital should be increased to the point of tolerance in an effort to control the seizure disorder. If drug dosages are gradually increased in time, frequently the depressant effects of the medication may be minimized despite high dosages. Diphenylhydantoin is slowly excreted from the body and need never be given more than twice a day. When diphenylhydantoin is begun, at 300 or 400 mg daily, about five days are required to obtain satisfactory blood and tissue levels. Thus, the “Dilantinizing” dose is approximately 1,500 mg.

Factors responsible for the failure of drug therapy include (1) improper classification of seizure type and with failure to use appropriate drugs, (2) failure to administer an adequate dosage, (3) the frequent shifting of drugs and premature withdrawal of drugs, (4) poor education of the patient with regard to his illness and the need to take medication, and (5) the lack of recognition of social and economic needs of the patients. Perhaps the single most common cause for poor control of seizures is the failure of the patient to take the prescribed dosage! The monitoring of the blood diphenylhydantoin level with periodic laboratory tests has been demonstrated to result in improved control, because this supervision induces many patients to greater conscientiousness in taking the amount prescribed. The absence of nystagmus in a patient presumed to be taking large amounts of diphenylhydantoin and phenobarbital should suggest to the physician that the patient is not taking the designated dosage. When an anticonvulsant or a combination of anticonvulsants give indications of being effective, the medications should be maintained. The pressures from the patient and others to utilize newer drugs should be reserved for those resistant or allergic to therapy with adequate dosages of well-established compounds.

Drug therapy is less successful in controlling psychomotor seizures than the other seizure disorders and, in appropriately selected patients, surgical therapy is indicated. Criteria for surgery include: (1) psychomotor seizures of sufficient...
frequency that the patient is incapacitated socio-
 economically; (2) intensive drug therapy has been
 unsuccessful; (3) availability of facilities for de-
tailed clinical and electrophysiological analysis
 essential for neurosurgical exploration. Surgical
 excision is of value in a limited number of patients
 with epilepsy and search must be continued for
 newer drugs, without serious toxicity, to reduce
 our current failures in drug therapy.

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Phenformin and Lactic Acidosis

Since the earliest days of its clinical use in the
 treatment of diabetes, there has been published
 concern about the possible role of phenformin in
 the causation of lactic acidosis.1 Elsewhere in this
 issue of California Medicine is the twenty-sev-
 enth published case report of lactic acidosis occur-
ing in a diabetic known to have been treated with
 phenformin. We are aware of reports of 63 di-
 abetic patients with lactic acidosis, of which 54
 cases have been published and the remaining nine
 cases are unpublished,2,3,4 known to us in our own
 experience. Of this number, 38 were receiving
 phenformin at the time they developed lactic aci-
dosis, five patients were known to have been taking
tolbutamide, two chlorpropamide and six insulin.

This very high incidence of use of phenformin
 among reported and known cases of lactic acidosis
 in the diabetic leads naturally to the question of
 cause and effect. Since lactic acidosis is known to
 have occurred both in diabetic patients who were
 taking no drug therapy and in diabetics on therapy
 other than phenformin, the relationship is not clear.
 On the other hand one of the more convincing ob-
servations is the fact that at least ten of the reported
 cases of lactic acidosis in which phenformin was
 being taken occurred in patients who were taking
 no other medication and who were previously in
 metabolic balance and who had no other identifi-
able cause for the lactic acidosis.

The subject of lactic acidosis has been reviewed
 in detail elsewhere.3 The accumulation of quanti-
ties of lactic acid sufficient to create severe and life-
 threatening metabolic acidosis is a very common
 event in patients who are in shock from a variety
 of causes, including hemorrhage, myocardial in-
 farction and septicemia. A significant association
 with gram-negative infection has been noted. A
 few patients, however, have been demonstrated to
 develop lactic acidosis apparently spontaneously,
 and in the absence of other recognizable disease.
 It is noteworthy that among reported diabetic
 patients who have developed "spontaneous" lactic
 acidosis, in whom there was not identifiable addi-
tional cause for lactic acidosis, all were receiving
 phenformin.

The latter factor we believe to be of major sig-
nificance, since virtually every other case of lactic
 acidosis in the diabetic has been preceded by an
 acute illness whose characteristics are such as to
 be related to lactic acidosis in the non-diabetic as
 well. In other words, in at least ten diabetics who
 developed lactic acidosis there was no other identifi-
able cause than phenformin ingestion. One case
 of attempted suicide by ingestion of 1,500 mg of
 phenformin did not result in an elevation of blood
 lactate;5 while a second case of ingestion of 850
 mg resulted in an unidentified metabolic aci-
dosis.6 Furthermore, it seems clear that many thou-
sands of patients have taken phenformin as therapy
 for diabetes mellitus and have not developed lactic
 acidosis.

Part of the confusion in our ability to assign a
direct relationship of phenformin ingestion to an
 incidence of lactic acidosis lies in our incomplete
 knowledge of the mechanism of action of this drug.
 Recent evidence that phenformin is rapidly metab-
 olized, probably by the liver, to para-hydroxyphen-
 ethylbiguanide casts much doubt upon the results
 of in vitro examinations of the mechanism of ac-
tion of the drug.7 Furthermore, there is a significant
 relationship between the in vitro effects of phen-
 formin and its concentration in experimental situa-
tions. Evidence exists which supports the idea that
 normally achieved blood levels of phenformin are
 not capable of producing the metabolic effects
 which have been directly associated with the in-
 creased production of lactic acid by tissues exposed
to phenformin. These effects are, however, possible
 with higher concentrations of phenformin, and such
 levels have only been demonstrated in diabetics
 who develop lactic acidosis while taking phenfor-
 min.8

In the absence of definitive evidence, however,
it is essential to have a rational approach to the use