Epilepsy: A Major Health Problem

An Honors Thesis (ID 499)

By

Kathy G. Jack

Louise Prage

Ball State University
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INTRODUCTION

Epilepsy has been defined as "a paroxysmal and transitory disturbance of the function of the brain which develops suddenly, ceases spontaneously, and exhibits a conspicuous tendency to recur." An attack of epilepsy, called an epileptic seizure, occurs when the brain cells suddenly release a large burst of electrical energy. Normally these cells produce some electrical energy, which flows through the nervous system and activates the muscles. The brain of an epileptic patient sometimes fails to limit or to control this release of energy. The recurrent nature of the episodes that characterize epilepsy has resulted in special problems which are often as important to the patient as his medical care. The patient usually approximates the "normal," but on occasion he has an attack which temporarily incapacitates him, both as an individual and as a member of society. This insecurity greatly disturbs the patient and his family and often impairs his role as a student, worker, and family member. This has produced psychological frustrations, conflict, and dependency, and complications of which in turn have been greatly compounded by the attitudes of society toward epileptics. Serious limitations of education, employment, marriage, and self-development have resulted, as well as special legal restrictions.

Children who are subject to repeated seizures quickly become aware of the social (and sometimes physical) hazards and insecurity their condition imposes. In particular, they become acutely conscious of the attitudes of their family, friends, and others in their environment (Ireton, 1969). The situation is especially difficult if the parents
react with feelings of shame or guilt, if family discord develops, and if the child senses neglect or rejection. At the other extreme, an overly solicitous and overly protective parent also may have an adverse effect in exaggerating the dependency of an epileptic child (Henderson, 1953; Green and Hartlage, 1971).¹

Such problems become even more acute when the child enters school and is exposed to a broader environment beyond the home circle. The teacher is the key to the school situation in much the same way as the parents are the key to the family situation. "The attitude of the public has changed over the past centuries to the point that most individuals feel children with epilepsy should be educated and well-treated, but not near them."³²

It is estimated that one child in fifty has epilepsy. This means almost every teacher at sometime will have an epileptic student in his or her classroom. Additionally, teachers may not be aware of an epileptic child in the classroom since there are many hidden epileptics. The possibility of epileptic seizures in the classroom indicates teachers have a responsibility to be informed about the subject of epilepsy. It would seem imperative that teacher preparation for the management of epileptic students be comprehensive, that teacher attitudes be unprejudiced, that all available services be promoted by school nurses, and that guidance procedures meet the needs of epileptic students. A capable teacher who can handle a seizure in the classroom with composure and who can calmly explain in simple terms the situation to the other children may contribute enormously in determining attitudes, which are basic to successful adjustment. Also, the teacher is often in the best position to evaluate the educational needs of those epileptic children with whom he has dealt.
It is generally agreed that the epileptic child should be raised in circumstances, both at home and at school, which approximate the normal as nearly as possible. Extremes of neglect or overprotection should be avoided.

The employment of an epileptic presents several special problems. The precautions usually imposed on patients with epilepsy, such as restrictions on driving, handling machinery, and climbing into high places, often limit the work activities available to them. A far greater limitation, however, is the unwillingness of most employers to hire epileptics. Recently many epileptics have found themselves making headlines. They have not sought publicity—merely their rights as human beings. Steven Frazier, of Renton, Washington, had worked for the Container Corporation for five years when he was abruptly fired late in 1974 following a minor seizure at work. He had, the firm claimed, "falsified his job application" by failing to mention his epilepsy.49 Frazier learned his lesson and now duly notes his epilepsy on all job applications. No one has refused him a new job outright; he simply has heard nothing at all. His suit against his former employer for reinstatement under Washington's antidiscrimination statute is still pending.

In 1956, seventeen states in the U.S. prohibited marriage of epileptics.5 Today, no state denies a marriage to an epileptic merely on that basis. "Eugenic laws are intended to prevent marriages that would produce children having defects or diseases which would render the children of the marriage public charges."5 The widespread change in the law was made possible due to increased understanding of the genetic factor in epilepsy and the effective control of seizures in 80% of cases. It is now recognized that the genetic factor and the disabling effect
of epilepsy are not sufficiently great to justify laws which prohibit epileptics from marrying and bearing children. Possibly the practical approach for the epileptic is to obtain the advice of a specialist in the treatment of epilepsy. In most cases, the nature of the seizure and the extent of its control may well render marriage an acceptable course. In some cases, the underlying cause of the seizure pattern and difficulty in controlling seizures may suggest that marriage and child-bearing are not feasible. However, control of such decisions by law is both impractical and unsound. The decisions are best left to the individual guided by the best available opinion.
CAUSES OF EPILEPSY

Cerebral Causes

The tissue contained within a region designated as an epileptic focus will contain both normal and abnormal histological components. The abnormal tissue structure may be obviously necrotized and contain inflamed areas of degenerating tissue (as in cobalt foci) or local gliosis and scar formation (as in electrolytic and surgical lesions). In human neuropathology the foci include sclerosis of specific regions or local cellular changes related to such events as neoplasia, hamartoma infection, and meningeal adhesion. These widely ranging tissue changes undoubtedly disrupt local tissue organization, alter local vascular supply, and change the nature and dimensions of the local extracellular spaces and cellular relationships. The degree of cellular damage sustained will vary from cell death, with replacement by scar tissue and glial growth, to partial impairment from changed tissue organization and vascular supply. In many foci there would be a gradient of decreasing impairment from the center to the periphery. This is in accordance with the finding that neuronal hyperactivity is usually associated with the region at the periphery of the focus (Koyama; Prince and Futamachi; Schmidt et al.). In some cases, neurons would simply be absent from the central region and in others too damaged to be able to sustain hyperactivity. Where neuronal organization allows it, therefore, foci are likely to consist of a central inactive region surrounded by an
annulus of hyperactive cells at the boundary juxtaposed with the normal cells.

Penfield states that the cortico-diencephalic connections are very important and that epileptic discharge spreads in the cortex and reaches the higher brain stem along these connections. Certain subcortical regions are of importance from the epileptic point of view—the basal ganglia, thalamus, and hypothalamus may form the site of the epileptic discharge. The cerebral hemispheres seem to play no part in the origin of the seizures, although the gray matter of the brain stem and spinal cord may do so on rare occasions. The highest level seizures are a result of the discharge within the gray matter of the upper brain stem.

There are essentially three subdivisions of responses or three areas where convulsions may be produced. Each of these areas is sensory and motor: 1) the Rolandic or precentral and postcentral sensorimotor, 2) a second somatic sensory area, 3) a supplementary somatic motor area. Only the Rolandic can be subdivided clearly into two parts, a sensory somatic and a motor somatic. The character of the sensation produced by stimulation or by local epileptic discharge in the Rolandic cortex is described as numbness, tingling, or a feeling of electricity. Seventy-five percent of experimental stimulations which produced sensations were postcentral and twenty-five percent precentral. The secondary sensory area seems to occupy downward extensions of the precentral gyrus. Stimulation in the second sensory area produces tingling in the contralateral and ipsilateral extremities. The supplementary motor area is in the cortex just anterior to the upper end of the sensorimotor strip. It has a motor function that differs from that of the precentral gyrus and is a separate mechanism for the production or inhibition of movement.
Stimulation or local epileptic discharge in the supplementary motor area produces: 1) body posturing in which the contralateral arm raised with the head turned toward that arm, 2) vocalization, 3) slowing or complete inhibition of voluntary action or speaking without motor effect, 4) general body sensation, 5) pupillary reaction.

The electrical activity of the brain is of particular interest to the problem of epilepsy and is derived from the clusters of neurons in the gray matter of the cerebral cortex, especially from the subcortical ganglionic structures. Although the cortex is considered the site of discharge in epilepsy, the maintenance of the spontaneous rhythms of the cortex is dependent upon connections with the subcortical structures. Experimental studies (Penfield and Jasper 38) have made it clear that epileptiform discharge may originate in subcortical structures and be projected to the cortex. Even a local cortical epileptic discharge confined to specific areas of our hemisphere may be of subcortical origin.

Kreindler and Crighe 7 studied the effects of direct electrical cortical stimulation. Their investigations concerned the mechanism of the onset of neocortical convulsive activity under conditions of long-lasting direct electrical stimulation by supralaminal repetitive stimuli at low rates of ten to fifteen cycles per second. This long-lasting repetitive stimuli was used to best approach an epileptic focus model found in humans. The convulsive mechanism was studied by modifying the neocortical reactivity with topical application of drugs. Observations were carried out on cats immobilized with Flaxedil and maintained on artificial respiration. Clonic convulsions were obtained by stimulation of a wide zone of reticular formation extending from the medulla to the
mesencephalon. The excitability of the pyramidal neurons is modulated through the dendrite system. The imbalance between the inhibitory and excitatory mechanisms of the neuronal complex and the prevalence of excitatory mechanisms lead to the onset of convulsive activity in the neuronal complex. Under normal conditions, this activity is limited in time and space by inhibitory mechanisms that become active again. Depolarization of the superficial structures of the neocortex causes a loss of balance between inhibitory and excitatory mechanisms with the latter's prevalence. This leads either to a lowering of the epileptogenic threshold or to the appearance of spontaneous epileptic discharges. All of this data is based on the fact that there is an intrastimulatory tendency to reorganization of a previous disorganized activity and to reappearance of the initial aspect of the depressed direct cortical response. At the beginning of the stimulation there are alterations in the balance between inhibitory and excitatory mechanisms in favor of the inhibitory ones. But when continuing the stimulation, the inhibitory dendritic-internuncial superficial system intervenes and there is a reorganization of the activity.

Voinescu, Voiculescu, and Kriendler set out to verify, by means of penicillin epileptogenic foci, the possible role of the midbrain reticular formation as a pace-maker in epilepsy. Paroxysmal activity was studied in cats following penicillin injection into the midbrain reticular formation. Discharges were recorded from the midbrain, but in no instance did the midbrain assume the role of pacemaker. The paroxysmal activity spread to hemispheric structures only after secondary hippocampal involvement, with the hippocampal discharges imposing their own rhythms on the other formations.
Aquino-Cias and Bures examined the possibility of using the functional ablation technique for observing the role of thalamic nuclei in different forms of epileptic activity. The effects of thalamic spreading depression on various forms of epileptic discharges were studied in unanesthetized curarized rats using electrophysiological techniques. The rate of spikes generated in a cortical metrazol focus is reduced during thalamic spreading depression, probably because excitability of the de-afferented cortex is decreased. After-discharges evoked by cortical or hippocampal stimulation applied during the functional elimination of the thalamas are considerably shorter than those evoked under control conditions. This effect is mainly due to interference with the mechanism of seizure generalization at the thalamic level. On the contrary, cortical after-discharges evoked immediately after recovery of thalamic function are facilitated. A fully developed seizure is not stopped by functional elimination of the thalamus although the electroencephalogram patterns in different forebrain regions may be modified. It is concluded that the thalamus plays an important role in triggering a generalized epileptic seizure, but that it is not indispensable in maintaining it.

Although it seems unlikely that a single kind of pathophysiology with an associated specific neurochemistry underlies all forms of epilepsy, the existence of a number of specific features in a wide range of experimental foci does indicate some degree of similarity in their mechanism. First of all, there is the epileptiform electroencephalogram which is always present. The physician makes the diagnosis by considering the type of brain wave pattern together with the patient's symptoms and the results of other laboratory procedures. In addition, neurons in many acute and chronic experimental foci display a characteristic large
depolarization shift called the paroxysmal depolarization shift (PDS). These recurrent shifts of depolarization take the neuronal membrane through the critical firing level causing increased spike discharge and subsequently gross depolarization with inactivity due to cathodal block. This is followed by a period of hyperpolarization before the onset of the next PDS. The cause of the PDS is unknown, but all evidence points to their being synaptically generated and graded in nature, appearing to be greatly enhanced post-synaptic potentials of an excitatory nature. Recordings from neurons in these foci (between the PDS events) do not indicate any intrinsic hyperexcitability, suggesting that the properties of the neuronal membrane itself are not altered (Ajmone-Marsan; Prince). Therefore, the PDS must arise as the result of an abnormal increase in synaptic activity. Such excitatory activity could result from increased excitatory synaptic input, decreased inhibitory synaptic input, or direct stimulation of synaptic receptors on the membrane of the cells showing the PDS by agonists appearing locally in the interstitial fluid. In addition to these electrical changes, some foci show characteristic structural changes in neurons. These changes involve the loss of dendrite spines and the smoothing out of the dendrite surface, reduced dendrite arborization, and the appearance of varicosities. This implies a substantial loss in the number of dendrite synapses, resulting in a reduction of excitatory input to the neurons (partial deafferentiation). Some researchers believe that partial deafferentiation in the focus could readily lead to the augmented spontaneous neuronal activity which is observed, and increased synaptic receptor sensitivity may be part of the mechanism. Convulsive and anticonvulsive influences interact in an additive fashion in both normal and epileptic animals. Most experimental foci are activated by chemical convulsants at doses well below the level
precipitating generalized seizures. The motor activity evoked under these conditions takes a form which is characteristic for the focus concerned, and the parallel increase in electrical discharge remains focal (Hommes and Obbens\textsuperscript{25}). In the same way, anticonvulsants control the motor effects of these seizures and greatly raise the threshold for the convulsants. The exact mechanism for making the foci susceptible to the activating effects of these convulsant agents is not clear. It is possible that there is a breakdown in the local blood-brain barrier in the focal area, allowing a more rapid and effective build-up of convulsant concentration in that region. Certain kinds of scar may develop this increased permeability to agents in the blood and become of central importance in controlling epileptic conditions in man. Also, certain compositional changes in biochemical factors consistently emerge in the wealth of data on this aspect of epilepsy. The most significant of these are changes in cation and amino acid content. Sodium could be expected to show an increase and potassium a decrease in tissue samples from central regions of foci which are necrotized and show a greatly increased cerebrospinal fluid space. The relatively normal cells of the primary electrically-active focus and its associated mirror focus should show a smaller change in the same direction since sustained excitation taxes the ability of the sodium pump to release sodium and recover potassium. The ability of ouabain to create a temporary epileptogenic focus is probably due to partial paralysis of the sodium-potassium-ATPase activity which constitutes the sodium pump. It follows that partial inhibition of this enzyme in scar tissue or in chronic experimental epilepsy could lead to a similar but more chronic condition than that produced by ouabain. Research indicates that some primary deficiency in ion transport might be present in the pre-synaptic
membrane which could have a direct effect on neuronal excitability. (It is significant that the important anticonvulsant Phenytoin appears to accelerate the activity of the sodium pump). Calcium involvement in excitability is through its role in transmitter release and in control of membrane stability (Shanes, 1959). Neural tissues "in vitro" show increased respiration and glycolysis in calcium-free media. This is interpreted as being due to a change in membrane structure leading to increased sodium influx, and therefore to sodium pump stimulation. A number of simple and widespread amino acids which are involved in a variety of metabolic pathways in the central nervous system are also likely to function as major synaptic transmitters in the brain and spinal cord. Much research focused on the excitatory properties of glutamic acid. Its high tissue content seems to stimulate high-affinity transport systems, thus ensuring a very low concentration of glutamate in the region of synapses operated by this amino acid. Changes which reduce the efficiency of this transport activity through inhibition or over-saturation will lead first to raised levels of extracellular glutamate, and then to neuronal hyperactivity followed by cathodal block, depending on the levels reached. Dendritic de-afferentiation is present in both human and experimental epilepsy. If such de-afferentiation involves the widespread appearance of active glutamate receptors on the dendrite surfaces of epileptogenic cortical neurons, super-sensitivity to glutamate would occur and might explain the abnormally high synaptic activity which seems to generate the PDS commonly observed. The ability of non-synaptic regions of nerves to release glutamate suggests that this ion may be held within neurons by the membrane potential. It could then be briefly released from both
non-synaptic and synaptic regions during the depolarizing wave of the action potential. If this were true, the apparently random and episodic occurrence of many epileptic seizures might be explained by the glutamate-releasing effect of certain activity patterns in the region of the epileptic focus. Damaged neurons present in the active focus and displaying lowered membrane potential would more readily reach the threshold level at which glutamate was released.

EXTRACEREBRAL CAUSES

Strictly speaking, there are no extracerebral causes of epilepsy, for epilepsy is a tendency toward recurring seizures produced by excessive neuronal discharge in the central nervous system. It is permissible to speak of extracerebral causes only because the brain is united to every other part of the body by the circulation of blood and by the conduction of nervous impulses along the peripheral nerves. These causes must all produce changes, either in the blood or in the nerves, which in turn are transmitted to the site of epileptic discharge in the brain. Although epilepsy culminates in a dramatic and excessive neuronal discharge, it represents series of physiological events, some of which occur outside the central nervous system. There is a great variety of extracerebral pathological states which occasionally act as causes of epilepsy through interference with cerebral circulation or through an effect on cerebral metabolism or function.

Diseases of the peripheral vessels are sometimes associated with convulsions. The fact that seizures occasionally occur in patients with cardiovascular disease may be explained on the basis of a direct cerebral vasoconstriction rather than on the basis of a defect in the general circulation. Vasoconstriction may also play a part in the convulsion
due to irritability induced by atherosclerosis. A variety of cardiovascular disorders are occasionally the cause of convulsions. However, the clinical features in these cases differ from the usual epileptic seizure. In the ordinary case of epilepsy, there are no consistent primary disorders of the general circulation in the interval between or immediately preceding seizures.

Primary respiratory changes have been demonstrated in some cases of petit mal. These seizures may be precipitated by decreased oxygen tension and tend to be prevented by increased oxygen tension. Oxygen under higher pressure may also cause convulsions.

There is no strong evidence that either the gastrointestinal tract or the liver play any important etiological role in epilepsy. Constipation has long been regarded as a precipitating factor for the epileptic attack. It is conceivable that a stasis in the large bowel induced by constipation might cause a certain amount of hydration and thus lower the convulsive threshold. Considering the prevalence of constipation among the non-epileptic population, it should not be ascribed any more than a very minor role.

Although occasional cases of anemia associated with epilepsy have been reported, neither primary nor secondary anemia has been shown to have any effect in producing seizures unless circulation fails acutely. Diseases of the blood-forming organs may induce convulsions only in so far as they are responsible for the production of destructive lesions in the central nervous system.

Renal function tests in epileptic patients have all given normal results except when there is coincidental renal disease. During the
course of acute glomerulonephritis, hypertensive encephalopathy may occur of which headaches, convulsions, and coma are most frequent. The pathogenesis of these convulsions seems to be closely associated with vascular hypertension.

Abnormalities in protein metabolism were regarded as possible etiological factors in epilepsy. This theory was put to use in the form of a low protein or of purine-free diets in the treatment of epilepsy. However, the total non-protein nitrogen, urea nitrogen, amino acid, nitrogen, uric acid, and creatinin have been determined in large groups of epileptic patients and were found to be within normal limits. At the present time, there are no established abnormalities of protein metabolism in epileptic patients.

The observation of convulsions resulting from hypoglycemia after injection of insulin has stimulated interest in the blood sugar level in epileptic patients. Insulin in sufficient doses to produce severe hypoglycemia and coma causes loss of alpha activity and a great increase in the slow waves of the electroencephalogram. Rapid changes in the tension of glucose within the nerve cell, anoxemia of nerve cells, and interference with afferent impulses from the labyrinths are some of the proposed theories. Increased irritability of muscle and nerve has been found to occur when blood sugar is lowered by insulin. Insulin diminishes oxygen consumption of brain tissue. This cellular anemic affect may account for the convulsions as well as other symptoms of hypoglycemia. Hypoglycemia is not the direct cause of convulsions but sets in operation another series of events including hydration, cellular anemia, and other unknown factors. These events in turn are responsible for causing an epileptic discharge of nerve cells.
There is reason to believe that changes in fat metabolism are of importance in epilepsy. Cholesterol injected parenterally in white mice produced a rise in the convulsive threshold for convulsive doses of cocaine hydrochloride. This was interpreted as indicating delayed absorption of the convulsive agent and additional evidence that vital lipids play a significant role in the permeability of cell membranes. This evidence suggests that this mechanism is an important factor in epilepsy.

Both experimental and clinical studies indicate that there is an intimate relationship between the occurrence of convulsions in epileptic patients and the water balance of these patients. Convulsions are a prominent feature of water intoxication produced by administration of large amounts of water by stomach tube to various mammals. Such convulsions, as well as those induced clinically, have been attributed either to an increase of intracranial pressure, to a disturbance of the salt and water equilibrium of the body and particularly of the central nervous system, or to a disturbance in the permeability of nerve cells. A close relationship between the water balance of the body (the ratio of the total amount of water entering the body and the total amount leaving the body) and the occurrence of seizures has been demonstrated. Since increased intracranial pressure and vasopressor action of the pituitary extract have been ruled out as primary factors in the causation of these convulsions, these experiments may indicate an inherent weakness in the mechanism for controlling the semipermeability of the brain cell membranes. The tendency of water to enter nerve cells produces a condition which the non-epileptic can resist or compensate for much better than the epileptic individual. Closely related to the varying degrees of hydration are other changes in metabolism, particularly
disturbances in electrolyte balances. As the extra water enters the cells, potassium diffuses out of the cells and is eventually excreted in the urine. An increase in the potassium to sodium ratio in the urine is manifested 12-36 hours before seizures occur. In some patients, seizures may be precipitated by an increase of body fluids, or a state of hydration, while they cease during periods of dehydration. There is no evidence, however, that abnormalities of water metabolism are invariable causative factors in all cases of epilepsy nor that they are the sole cause in any one case.

Hyperpnea, tetany, and the administration of bicarbonate, all of which tend to induce a state of alkalosis, may precipitate epileptic seizures. On the other hand, attacks may often be suppressed by fasting or by a high fat ketogenic diet, procedures which tend to produce an acidosis. Local changes in the pH of the cerebral cortex have been demonstrated to occur during experimentally induced epileptiform seizures. Increase of pH increases the electrical activity of the cerebral cortex. As the nerve becomes acid, the threshold for electrical stimulation is increased; an increased alkalinity is accompanied by a decreased threshold of excitability. When the threshold falls in alkaline nerves, a spontaneous electrical discharge develops which becomes visible at pH 7.6-7.7 in the phrenic nerve and increases in height and frequency as the pH further increases. The effects produced by an increase of pH are mainly attributable to deionization of calcium while the effects of a decrease of pH are mainly due to an ionization of calcium. It must be remembered that local changes in cortex pH may occur without accompanying changes in blood pH.
It is probable that most changes in mineral metabolism are secondary to the seizure itself. However, it is reported that serum calcium, potassium, and sodium were usually normal in epileptics but that serum calcium was high immediately before an attack. The increase of serum potassium and increased excretion of potassium have been observed to antedate the convulsion by as much as thirty-six hours. This phenomenon signifies a leakage of potassium from the cell and may indicate an increased permeability of the cell membrane, perhaps a primary etiological factor in the epileptic discharge.

Neither sexual behavior nor function bears any causal relationship to epilepsy. In some female patients whose seizures show a clear chronological relationship to the menstrual periods, there is apparently a lowering of the convulsive threshold by the hydration and the vascular changes associated with menstruation. Pre-menstrual edema with changes in membrane permeability appears to be the most important factor.

The ancient Hebrew literature gives evidence of a belief that epilepsy might be linked to sexual irregularities. It was thought that epileptic children would result from improper behavior during cohabitation, coitus immediately after defecation or after blood-letting, or coitus with the husband lying on his back. Other more recent folk beliefs have ascribed the disease to masturbation or to an oral and anal eroticism. Convulsions have occasionally been reported after castration, but there is no indication that epilepsy is more common among eunuchs than among others.

A large variety of chemicals and drugs have been reported to cause convulsions in isolated instances. The results of experimental pharmacology have revealed the fact that each of these substances has a characteristic site of maximum action in the central nervous system.
Convulsant drugs or poisons may be classified according to the portion of the neuromuscular mechanism upon which they exert their primary action, i.e. the portion that is most susceptible to the drug. Convulsions may be caused by direct action on the muscles (veratrine), on the efferent nerves (physostigmine, aconitine), on the spinal cord (strychnine, caffeine), on the medulla (camphor, picrotoxin, asphyxia), and on the cerebrum (absinthe, thujone, metrazol). The old opinion that alcoholism in the parents may be a cause of epilepsy in the offspring is no longer tenable. Chronic alcoholism is a rare cause of epilepsy although acute alcoholism is a common precipitating factor in the epileptic patient.

Unfortunately, too many investigations of epilepsy have been based on the assumption that it is a static condition. There is a great need for more information concerning the physiological events which immediately precede and initiate the seizure. These meager facts are the only indication as to what pulls the trigger to release the epileptic discharge.
SYMPTOMS OF EPILEPSY

The manifestations of epilepsy vary greatly in different individuals and even in the same individual at different times. Therefore, it is possible only to outline the general course of what might be considered a typical seizure and to point out certain commonly-occurring characteristics which are thought by some observers to be frequently associated with epilepsy.

GRAND MAL

Attacks of the more severe form of epilepsy, the grand mal seizure, differ greatly in detail but almost always pass through the same phases. Their frequency varies in different patients but is apt to remain approximately uniform for long periods of time in any given individual. Often seizures begin only during sleep or in the morning just after waking. The intervals between attacks may be hours, days, weeks, or months in length.

Only between one-third and one-half of all patients have any warning of the onset of a seizure. The rest suddenly lose consciousness and, uttering a loud cry, fall to the ground in convulsions. The "aura" or warning assumes so many forms that no attempt will be made to outline its characteristics. Suffice it to say that the patient comes to know that a certain unusual sensation, visual or auditory hallucination, pain, or psychic condition is apt to be followed either immediately or after an interval by the convulsive attack. The length of time that elapses
between aura and convulsion usually remains constant for each patient, as do the other details of the syndrome.

In the first or "tonic" stage of the attack, muscular contractions fix the limbs in irregular positions. The head may be positioned to either side and, like the trunk, is held stiffly. The features are distorted and the patient is very pale until rigidity of the muscles stops the movements of respirations. The pallor then gives way to suffusion and finally to lividity. Contraction of the masseter muscles frequently results in severe biting of the tongue. The eyes may be either open or closed and are insensitive. The pupils dilate as cyanosis increases. In most patients this tonic stage is very brief, lasting only thirty seconds or less.

The succeeding "clonic" stage begins when cyanosis is intense. All the muscles of the body become vibratory and undergo fixed rhythmic irregular sequence. In the clonic spasm, the limbs, head, face, and jaw are jerked violently, the eyes are rolled upward or to the side, the trunk is twisted, and the diaphragm contracts rhythmically. As the air is thus forcibly expelled from the lungs, the patient froths at the mouth, grunts, coughs, and makes stertorous noises. If the tongue or cheeks have been bitten, the frothy saliva is tinged with blood. The bladder, and sometimes the bowels, is emptied by the muscular contractions. If the attack is very severe, the patient becomes livid and appears to be at the point of death. Hemorrhages occur in the conjunctiva and other parts of the body, and very rare cases of heart rupture have been reported. The blood pressure rises at first and then suddenly falls and remains low. The pulse is rapid. As the intervals between spasms lengthen, the remissions are more complete, inspiration is less restricted,
and the cyanosis diminishes. However, the muscular contractions continue to be strong up to the last jerk. This stage of an attack may continue for only a few seconds or may persist for three to five minutes.

In the "stuporous" stage that follows, the subject relaxes completely and lies senseless and prostrate. This condition may persist for a few moments or for hours before the patient can be roused.

On wakening, epileptics have no recollection of what has occurred and occasionally do not even remember events which immediately preceded the onset of the attack. Some jump up ready to resume activities where they left off. More frequently these grand mal seizures leave behind a feeling of exhaustion, great fatigue, and a severe headache which may continue for several hours. When this is the case, the patient is irritable and wants to be left alone. Individuals who do not suffer from this unpleasant consequence are likely to have felt awful for several days before the seizure and to experience a sense of relief when it is over.

PETIT MAL

These minor attacks of epilepsy are often so difficult to distinguish from non-epileptic conditions that their true character is revealed only by their periodic recurrence. In some patients they consist merely of momentary loss of consciousness, while in others they appear as fainting spells or periods of dizziness with peculiar motions. The patient rarely falls during these seizures, but the head may be dropped forward or turned to one side, the eyes may become fixed and expressionless, and the arms may be contracted. As time goes on petit mal tends to become more severe and to develop gradually into grand mal. The two conditions are frequently associated. Attacks of petit mal sometimes recur at very brief intervals; in the majority of subjects the number varies from two or three to a dozen a day.
**PSYCHOMOTOR**

The third type of seizure is the psychomotor attack which accounts for about one-third of adult epileptic attacks. It is the result of an irritative injury to the anterior portion of the temporal lobe. As the attack comes on, there will be a change from ordinary behavior, consisting of the person seeming to suddenly "switch off" from whatever is going on at the time. He will probably seem withdrawn and intently preoccupied with something going on within himself. While some affected by psychomotor attacks can seem aware of what is happening, as a rule they have no recollection of the episodes later on. Only if restrained physically do these persons appear angry or become unruly. As in other types of seizures, a psychomotor spell usually lasts only a few minutes.

**JACKSONIAN EPILEPSY**

Although one source classified Jacksonian seizures as a type of grand mal epilepsy, most authors consider it as a separate type. Jacksonian epilepsy results from abnormalities of the cerebral cortex and is frequently due to brain tumor. In most cases, convulsions are restricted to the region controlled by the abnormal part of the brain, but in some patients they become generalized before the end of the attack. If they are widespread from the beginning, this type of epilepsy is very difficult to recognize. The aura, when they occur, are apt to be motor in character. Jacksonian epilepsy has been described as localized spasms of different groups of muscles in the face, arm, or leg with the retention of consciousness. The epileptoid convulsions which occur in thirty-three to fifty per cent of children with cerebral palsy and
hemiplegia usually begin on the paralyzed side. As they become more frequent, however, they eventually become generalized.

**INTERVAlS BETWEEN ATTACKS**

If the intervals between attacks in all types of epilepsy are long enough to allow recovery, patients usually feel quite well. It is not uncommon, however, for them to suffer from headache for a while before the seizure begins or to have a strong sense of anxiety and fear. At all times they are very susceptible to fatigue.

**STATUS EPILEPTICUS**

In the condition known as "status epilepticus," seizures recur at such brief intervals that there is no opportunity for complete recovery from one attack before the next one begins. As many as 150 may occur within twenty-four hours. Because of the continued excessive muscular exercise involved in the convulsions and because the heat-regulating mechanism at the surface of the body is somewhat disturbed, the temperature may become very high. The pulse rate rises simultaneously; in some cases until it is very difficult to count. The body is covered in a cold, clammy sweat. If the patient is not fatally exhausted by the almost continuous convulsions, the intervals between attacks gradually increase in length so that recuperation is possible. Status epilepticus frequently ends, however, in cardiac dilatation, pulmonary edema, loss of reflexes, and death.

**ATYPICAL ATTACKS**

Although almost no epileptic attack corresponds exactly to the general descriptions already given, some differ so markedly from the standard
that they are considered to be "atypical." In some instances, the tonic stage is elaborated by turning, running, jumping, and other unusual activities. Patients may run amok. In petit mal the symptoms may be aborted, consciousness may not be lost, or the patient may carry out some complicated performance. In grand mal, the occurrence of the aura without other manifestations should be considered an aborted attack.

NOCTURNAL EPILEPSY

"Nocturnal epilepsy" is a term which may be applied to any form of epilepsy in which seizures occur only at night or during sleep. The condition is probably far more common than many observers think, and may exist for years before being recognized. Sooner or later, however, the occurrence of an attack during the day or in the presence of some onlooker leads to its discovery.
SUMMARY OF PRECIPITATING FACTORS OF SEIZURES

There is no doubt that epileptic seizures are produced by disturbances of nerve cell function in the brain. However, in some patients the epileptic seizures occur spontaneously on some occasions and at other times they occur in association with certain factors. These factors cannot be considered the basic cause of their epileptic disorder, but merely precipitating factors of some of the epileptic seizures.

Some factors which affect seizure threshold levels include:

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>RAISES THRESHOLD (decreases attacks)</th>
<th>LOWERS THRESHOLD (precipitates attacks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>oxygen</td>
<td>rich supply</td>
<td>poor supply</td>
</tr>
<tr>
<td>pH</td>
<td>acidosis</td>
<td>alkalosis</td>
</tr>
<tr>
<td>water</td>
<td>dehydration</td>
<td>over hydration</td>
</tr>
<tr>
<td>calcium</td>
<td>normal</td>
<td>decreased</td>
</tr>
<tr>
<td>glucose</td>
<td>normal</td>
<td>decreases (hypoglycemia)</td>
</tr>
<tr>
<td>body temperature</td>
<td>normal</td>
<td>increased</td>
</tr>
<tr>
<td>synaptic resistance</td>
<td>normal</td>
<td>decreased</td>
</tr>
<tr>
<td>GABA-gamma aminobutyric acid</td>
<td>increased</td>
<td>decreased$^{61}$</td>
</tr>
</tbody>
</table>

EMOTIONAL DISTURBANCES

Emotional disturbances such as marked excitement, fear, frustration, tension, and anxiety are probably the most common precipitating factors of epileptic seizures. This is particularly true in the case of the teenager and the younger adult. Emotional disturbances may increase the frequency of all types of epileptic seizures. The epileptic patient may
experience a variety of emotional difficulties relative to his fear of a recurrence of seizures. His fear of suddenly being thrust into a state of unconsciousness is one of the most disturbing problems with which he must learn to live.

**SLEEP**

Many patients only have their seizures in association with sleep. Epileptic seizures may occur at any time in the sleep cycle, but they occur most frequently at two specific times: 1) within the first or second hour after falling asleep, and 2) one to two hours before the usual time of awakening. Several investigators have determined that these are the times when the maximum depth of sleep is reached, suggesting that there might be some relationship between the depth of sleep and the occurrence of epileptic seizures.

The patient who is subject to sleep seizures should be instructed against taking daily afternoon naps unless it is absolutely necessary. Such patients should also definitely be warned against the possibility of dozing off while operating an automobile. They should not drive for long periods of time particularly on super-highways or turnpikes, which are known to be conducive to dozing off and falling asleep.

**MENSTRUATION**

The physiological changes which occur during the menstrual cycle, and which are responsible for the precipitation of epileptic seizures, are not clearly understood and remain a highly controversial subject. The retention of body fluids which occurs in association with the menstrual period is thought by some investigators to be the precipitant. Other physicians believe that the complex hormonal changes which produce
menstruation may play an important role in precipitating seizures.

WITHDRAWAL OF ANTI-EPILEPTIC DRUGS

The sudden withdrawal of antiepileptic medication is a very common cause of an increased frequency of a patient's seizures. It is also a very common cause for a return of seizures in a patient who has been taking medication for a prolonged period of time and who has been free of seizures for this period.

HYPERVENTILATION

Hyperventilation will precipitate a petit mal seizure in practically all cases. The primary importance of this procedure is that it provides a quick and simple means of activating seizures for diagnostic purposes. Since it is known that deep breathing will cause petit mal seizures, some patients are concerned about activities such as running, bicycle riding, and swimming, because they require deep breathing. However, ordinary exercise is nearly always harmless and usually does not precipitate petit mal attacks.

ALCOHOL

The ingestion of alcohol can cause convulsions in any individual. However, these convulsions almost always occur in association with an excessive consumption of alcoholic beverages. They generally occur either during the acute stages of alcoholic intoxication or immediately following an alcoholic debauch.

PHOTIC STIMULATION

Patients who suffer from any type of seizures while exposed to light are sometimes classified as having photogenic epilepsy. These patients
should have an electroencephalographic examination with photic stimulation by white and colored lights so that relevant sensitivity may be determined. Suitable colored eye glasses can then be prescribed if necessary.

**FLUID INTAKE**

Investigations have shown that the ingestion and retention of large amounts of water may, in some epileptics, disturb cerebral function and cause convulsions. However, the ingestion of a normal amount of fluid by patients who have normal excretory function usually has no adverse effect on the course of pre-existing epilepsy. Restrictions are required only in those patients whose seizures occur in association with the menstrual cycle and in those patients who are receiving the ketogenic diet for their epilepsy.
THERAPY FOR EPILEPSY

IMMEDIATE CARE DURING ATTACKS

Generally, most epileptic seizures are of short duration and do not require immediate specific care. In fact, most epileptic seizures are terminated before any assistance could be rendered. In the case of those seizures of prolonged duration, the patient should be allowed to remain, if possible, at the place where the seizure occurred until the active portion of the attack has subsided. The patient should be placed in such a position that he cannot hurt himself by knocking his body against hard objects. Tight clothing, especially around the neck, should be loosened or removed. If the seizure occurs while the patient is in bed, he should be observed so that he does not fall to the floor. All pillows should be removed from the bed. The patient should not be allowed to lie on the face into the mattress or other soft bed clothing. If possible, the patient should be kept on his side so that mucus, saliva, and vomitus will flow more freely from the mouth. Since patients are unable to swallow during convulsive episodes, material may flow from the mouth down into the lungs and cause respiratory distress. Therefore, the patient should not be permitted to lie flat on his back for any length of time. In those patients who bite their tongue or cheeks or show marked evidence of respiratory difficulty during a convulsive episode, some object should be inserted between the teeth. It is important to insert the object before the attack begins. This avoids
potentially broken teeth and/or bitten fingers. Any solid, blunt, non-damaging object of the right size and firmness can be inserted between the patient's teeth. Examples include a padded tongue depressor, a folded leather belt, or a leather glove. The object should be placed to one side of the mouth between the back teeth since it easier to insert an object in this area. Also, the front teeth are more easily broken and can cause respiratory difficulties if aspirated.

No effort should be made to stop the muscular contractions by forceful means such as holding down the patient's arms and/or legs. It is also not advisable to lift the patient or to carry him from place to place while he is in the active stages of the convulsion, unless absolutely necessary. No special efforts such as shaking the patient, applying cold applications to the face, or pinching should be made in an attempt to terminate the unconsciousness associated with an epileptic seizure. These efforts are of no avail, and consciousness will return automatically of its own accord. In most instances the unconsciousness is of relatively short duration, generally lasting no longer than five to ten minutes. If a patient manifests prolonged unconsciousness, a physician should be consulted immediately.

In the case of the known epileptic, it is generally not necessary to consult a physician for immediate treatment of the convulsive episode itself. However, the physician should subsequently be informed of the seizure so that he can make appropriate adjustments in the patient's regular anti-epileptic regimen. Exceptions to this general rule are as follows: 1) when the seizure lasts much longer than previous ones; 2) when the seizure is different in character than previous ones; 3) when the seizure occurs in association with circumstances different from previous ones; 4) when the patient experiences one seizure after
another without regaining consciousness. Each of these exceptions should be considered an emergency and the physician should be consulted or the patient should be taken immediately to the nearest hospital.

It is not unusual for a patient recovering from a grand mal seizure to exhibit bizarre activities such as talking incoherently, being extremely restless, or conducting himself in a generally confused state. Due to a lack of knowledge and understanding about seizures by the general public, the epileptic is frequently subjected to considerable ridicule and mockery by individuals witnessing the episode. In some instances, the patient is aware of and sensitive to the reactions of the bystanders. The patient's embarrassment can be greatly intensified by expressions of shock and horror by individuals witnessing the attack. Observers can greatly reduce a patient's feelings of shame by being nonchalant and casual about the episode. Perhaps the most valuable service that a bystander can render to a patient recovering from a seizure is an expression of encouragement, understanding, and a desire to give assistance.

MEDICAL TREATMENT

The ideal objective in the treatment of epilepsy is complete control of seizures and provision for normal physical, mental, and social development. (Seizure control includes reducing the frequency of seizures to the extent that they do not interfere with the patient's general well-being.) The degree of success in attaining this goal depends upon many factors such as the type of epilepsy, the duration of the illness, the absence or presence of brain damage, and the cooperation of the patient and his family.
Treatment should be instituted as soon as the diagnosis has been established. This is an important aspect in the treatment of epilepsy for the following reasons. First, in most cases, the degree of success in controlling seizures bears a direct relationship to the duration of epilepsy; the longer the duration, the less likely a satisfactory result will be obtained, regardless of the type of therapy employed. Second, it is exceedingly important to institute measures to prevent a recurrence of seizures, not only because of the attacks themselves, but also because of injuries, brain damage, and emotional disorders that are sometimes associated with them.

Some antiepileptic drugs are more effective in controlling certain types of seizures. On the other hand, some drugs often increase the frequency of certain types of seizures. The following table gives a list of the drugs useful for the control of various forms of epilepsy.

<table>
<thead>
<tr>
<th>GRAND MAL</th>
<th>PETIT MAL</th>
<th>PSYCHOMOTOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenobarbital</td>
<td>Benzedrine or Dexadrine Sulfate</td>
<td>Dilantin</td>
</tr>
<tr>
<td>Dilantin</td>
<td>Zarontin</td>
<td>Phenobarbital</td>
</tr>
<tr>
<td>Mysoline</td>
<td>Tridione</td>
<td>Benzedrine or Dexadrine Sulfate</td>
</tr>
<tr>
<td>Peganone</td>
<td>Paradione</td>
<td>Mysoline</td>
</tr>
<tr>
<td>Mebaral</td>
<td>Diamox</td>
<td>Peganone</td>
</tr>
<tr>
<td>Gemonil</td>
<td>Atabrine</td>
<td>Celontin</td>
</tr>
<tr>
<td>Bromides</td>
<td></td>
<td>Mebaral</td>
</tr>
<tr>
<td>Elipten</td>
<td></td>
<td>Elipten</td>
</tr>
<tr>
<td>Diamox</td>
<td></td>
<td>Tridione</td>
</tr>
<tr>
<td>Mesantoin</td>
<td></td>
<td>Mesantoin</td>
</tr>
</tbody>
</table>

\*Phenurone*
Treatment should begin with one drug. Other drugs should be prescribed, if necessary, only after it has been determined that the maximal tolerated dosage of the starting drug failed to produce a satisfactory clinical response. The rationale for using this method are as follows. First, since there are only a few "really good" drugs available for the control of seizures and since it is known that the dosage necessary to control epileptic seizures varies from patient to patient, the physician should give each drug a thorough trial before resorting to another drug. Second, if two drugs of completely different chemical structure were initially prescribed and the patient manifested some untoward reaction, it would be extremely difficult to determine which of the two drugs was responsible for the unfavorable effect and to decide what action to take.

The medication should be taken at times of the day which do not interfere with the patient's routine activities, such as with meals, upon returning home from school or work, and at bedtime. It is important that patients and their families realize that it is not necessary for the medication to be taken at the same minute each day. Sometimes when an individual is in the hospital, the medication is administered in this manner and no one instructs him to take it differently when he is discharged. The physician should emphasize the importance of taking the medication exactly as prescribed, as omissions are often shortly followed by seizures.

The dosage of anticonvulsant medication varies from patient to patient. The proper dosage for any given epileptic patient is that which controls his seizures without producing untoward reactions which interfere with his general well-being. However, the drug dosage necessary for
complete control of seizures may produce unpleasant effects which are more of a handicap to the patient than the seizures themselves. Some patients may be better off leading a normal life between occasional attacks than living seizure-free in a perpetual state of drug-induced drowsiness and confusion.

The medication should be discontinued very gradually. A sudden withdrawal of anticonvulsant medication is a frequent cause of a recurrence of seizures or status epilepticus. Therefore, when a physician decides to withdraw anticonvulsant medication in a patient who has been seizure free for a period of time, he should reduce the dosage gradually over an interval which is governed by the amount and type of medication the patient has been taking.

The only dietary regimen which one author has found to be of value in the control of epilepsy is the ketogenic diet. A starvation period of approximately five to seven days must be instituted prior to starting the diet. Careful observation, preferably in a hospital, is mandatory during this interval of fasting. The patient receives nothing but a limited amount of liquid by mouth.

A daily measured water intake of not less than 400 cc. and not more than 800 cc. is required to obtain the desired degree of dehydration. The presence of excess fluid in the central nervous system is thought to be responsible for seizures in some way; consequently, drying out the body should reduce the amount of fluid until mechanical pressure on the brain is relieved sufficiently to permit cessation of symptoms. This starvation results in ketosis which is then maintained with the ketogenic diet. To be effective, the diet must be rigidly controlled
and weighed. Other factors which influence its success are the age of
the patient, the type of epilepsy, and the willingness and capability of
both the patient and the parents to cooperate satisfactorily.

Medical treatment should be given an adequate trial in all cases
of epilepsy and considered unsuccessful only when all appropriate
therapeutic agents have been administered alone and in combination without
beneficial results. Surgery should be considered only when these measures
have failed. Obviously, if the seizures are symptomatic evidence of a
progressive cerebral lesion such as tumor, abscess, or hematoma,
surgery is the method of treatment.

SURGICAL TREATMENT

A patient who presents focal clinical seizures or focal electro-
encephalographic abnormalities should always be considered as a potential
candidate for surgical treatment. Surgical therapy is performed only
when the neurosurgeon determines that the site of the cerebral abnormality
is in an accessible area of the brain, the removal of which would be
compatible with life and would not cause the patient more of a handicap
than the seizures themselves.

The operations most frequently used for the treatment of epilepsy
are:

1) Excision of localized cortical areas exhibiting constant abnormal
electrical discharge. In such cases, the excision is based on the
premise that a single, localized cortical area is responsible for the
seizures.

2) Temporal lobectomy for patients with clinical psychomotor seizures
associated with focal, temporal lobe electroencephalographic abnormalities.
This operation has been used mainly in adults.

3) Hemispherectomy, more accurately termed hemicorticectomy, is a newer development in the surgical treatment of certain types of epilepsy associated with unilateral cerebral atrophy.
CONCLUSION

"Attitude is a framework of reference that predisposes persons to act in a certain way . . . . What is greatly needed in epilepsy is more research on attitude and less authoritative statements that imply we have the answers.\(^5^8\)." The ultimate, most challenging obstacle with which any epileptic is faced is the attitude of people whose reactions to seizures are based on ignorance. "Epilepsy has been attributed to everything from lizards in the brain to radioactive fallout, and one in every seven Americans still believes that it is a form of insanity.\(^4^9\)."

To the uninformed observer, a seizure is still an extremely mysterious event. After all, something is "going on" within the brain, something over which the individual has no control. That loss of control, occurring unpredictably and for reasons which are not well-understood, touches a deep dread. We fear loss of control in anyone, in any form, whether temporary or permanent. Most of all, we fear the possibility within ourselves—and each reminder in others renews that fear. Harry Sands, Ph.D., a New York City psychotherapist and consultant to the Epilepsy Foundation of America, feels that a lot must be done to improve the welfare of the epileptic. "Certainly science should continue efforts to develop better medications that will effect greater control . . . but that is by no means the answer to the attitude problem. Physicians must not merely see that their patients are properly diagnosed and medicated; they must develop far more understanding of what the epileptic patient
feels and thinks. One epileptic states, "I think many people see epilepsy as a disease that somehow makes you less of a person. Not that the epileptic is 'looked down on' exactly, but that he is seen as 'different,' as someone who has something 'wrong' with him. It's not viewed the same way as other chronic problems like heart disease or arthritis. Epilepsy is seen as a mental disorder, simply because the problem is located in the brain. I think that's mainly due to ignorance. It may be hard for some laymen to understand, especially if they have seen an epileptic have a major seizure. They conclude that he has 'something wrong up there.' They avoid him. And he becomes a second-class person."

In reference to driving, he says, "I resent it, sure. But I have to admit it's logical. I could kill people if I had a seizure behind the wheel. Of course, I want to drive, but I'll just have to wait and see how it goes. When I've been seizure-free for 365 days instead of only 45, then I'll make plans. Epilepsy is a phenomenon, not a disease. "Every person will have a fit if suitably provoked." So why the stigma? We have always been somewhat fearful of the unknown, but our fear often prevents us from seeing the person rather than the condition with which he suffers.

Efforts to help epileptic in the future will have to be a team approach. Centers for the comprehensive evaluation of difficult epileptic patients are gradually being developed at various teaching centers and should be utilized by physicians when confronted by the care of such problem cases. Such centers offer comprehensive diagnostic services as well as follow-up care of patients with questionable, complicated, or uncontrolled epilepsy. A team of specialists provide comprehensive services to the medical profession and all referred patients. As a rule,
a neurologist, a social worker, and a public health nurse constitute the basic team. Additional out-patient consultants, such as psychiatrists, psychologists, vocational rehabilitation workers, and others are utilized depending upon the needs of each patient. Numerous and varied programs have been developed for the vocational rehabilitation of epileptics. The first task of rehabilitative programs should be to establish realistic goals that take into consideration the types of disabilities to be included and their philosophy of operation. In general, the limited and protective programs of the past are giving way to comprehensive approaches utilizing aggressive techniques of recruitment and work programs aimed at making their clients as self-supporting and independent as possible. The difference in these approaches is considerable, both for the individual and for society.
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