READINGS ON TRAUMATIC EPILEPSY

for the Fulbright Trial

Prosecution Clinic

Michael D. Stevens, The Case for Extended Recovery in Posttraumatic Epilepsy Medial Trial Technique Quarterly 201 (1981 annual); Law Library: RA 1001 .M4

Causes of Epilepsy

In 80 to 90 percent of all cases of epilepsy, no organic cause can be found; these are classified as being idiopathic (cause unknown), Lennox 1960.[6] There are many forms of epilepsy that are undramatic and may pass unnoticed by the average observer yet they definitely becloud judgment, alter awareness, paralyze initiative and self-control and leave the victim with genuine amnesia lasting from seconds to hours. Many seizures also occur during sleep and are therefore unknown even to the patient. Still more convulsions occur only during infancy and seem to burn themselves out in that they do not recur.

Just as any person may develop a fever, so too may any person have seizures. This means that even a normal healthy brain can be overstimulated by such factors as alcohol, drugs, concussion (a shaking up of the brain with resultant pinpoint to pinhead size hemorrhages), infection, fright or shock so as to explode into a series of convulsive neuromuscular (nerve and muscle) discharges of electrical energy leaving the person confused, disoriented, unconscious or amnesic for a variable period of time.[7]

The *convulsive threshold* represents a natural or artificial barrier in terms of physiological resistance in the same way that a fuse resists amperage overloading in an electrical circuit. Various drugs can be employed in an effort to increase this resistance in the epileptic.[8] However, warnings or aurae, when they do occur, may be so fleeting that no protection is possible.

[5] Houts, M., Presenting the Medical Research: Post-Traumatic Epilepsy, Trauma, vol 21, No. 1, p 70 (1979).

[6] Lennox, W. G., Epilepsy and Related Disorders, Little, Brown, Boston (1960).

[7] Stein, C., Mental Competency and the Law, 1964 MTTQ 155, at 169.

[8] Id.

Consequently, self-injuries such as fractures, cerebral concussion, a lacerated mouth or tongue, dislocation of the jaw or arm and multiple bruises are common.

There are many causes of epilepsy. The following list is fairly complete.

1. Genetically induced familial defects.
2. Genetic accidents.
3. Poisons, including drugs and alcohol.
4. Cerebral vascular accidents (strokes).
5. Disorders of the blood.
6. Meningitis.
7. High fevers due to infectious diseases.
8. Brain tumors.
9. Lack of oxygen to the brain because of drowning or other accidental causes.
10. Hysteria.
11. Trauma.[9]

It is the last cause, and the 17,000 to 95,000 individuals who develop seizures annually as a result of trauma, to which we now focus our inquiry.

Development of Traumatic Epilepsy

Traumatic epilepsy may develop within a few hours after a head injury, but this is rare. A convulsion developing so soon after the accident is probably due to a direct cerebral injury, an acute toxic reaction or a hysterical (condition simulating physical disease with mental manifestations) seizure, rather than the beginning of chronic posttraumatic epilepsy. Most cases of traumatic epilepsy develop within one year, but up to four years after the head injury is a possibility.[10]

When convulsions due to traumatic epilepsy are alleged, it is legally and medically desirable that they be witnessed and described by a physician or other professionally trained observer. Sometimes, the epileptic fit causes the head injury rather than

[9] Schwartz, R., The Epilepsies: New Dimensions for the Trial Lawyer, Trauma, vol 20, No. 6, p 49 (1979).

[10] Davidson, H. A., Psychiatric Aspects of Head Injury, 1959 MTTQ 201, at 205.

vice versa. In any case involving an apparently traumatic epilepsy it is advisable to make an exhaustive study of the patient/client's pretraumatic medical history. An epileptic may have only brief nighttime fainting spells without convulsions up to a certain point, and then may develop daytime convulsive seizures. If during one of these minor attacks, he falls and hurts his head, and if later he exhibits major seizures with convulsions, he will honestly ascribe the convulsive spells to the initial head injury.

The occufrence of mysterious, severe, disabling and long-delayed aftereffects of head injury are common gossip among laymen, and frequently a source of fear and great concern to the patient, his family and his lawyer. For the most part these fears are unwarranted. It is perfectly true that not uncommonly there can be a formation of a blood clot between the inner surface of the skull and the outer surface of the brain due to an injured or ruptured blood vessel resulting in a continuing leakage of blood and, therefore, accumulation of a blood clot. Depending on whether these lie to the outside or inside of the flexible layer of meninges called the dura (brain and spinal cord coverings), these are then termed epidural (between the inner portion of the skull and the dura) or subdural (between the dura and the arachnoid, the middle layer of the meninges) hematomas. [See Subdural Hematomas: From Diagnosis to Prognosis, 1975 MTTQ 349.] The important point is not on which side of the dura they lie but the fact that the epidural hematoma arises from a torn artery, meaning that the higher pressure blood available in an artery results in a more rapid collection of a large clot and a more immediate and dramatic danger to the person's life. The subdural hematoma is usually due to the rupture of veins, therefore leaking blood under low pressure, with far slower accumulation of clot and less dramatic effects. The epidural (arterial) hematoma generally makes its presence known within hours after the injury. The accumulation of a subdural (venous) hematoma may be much more delayed and in some cases may not be suspected or diagnosed until a matter of several weeks after injury. Far more commonly, however, it would become apparent within a matter of days following injury. In general, aside from these two special instances, if a patient makes a good and clinically complete re-

covery from a head injury, he is in the clear. However, there is a single exception and under the heading of permanent residuals, the prospect of posttraumatic epilepsy lurks behind every head injury.

Injury resulting in laceration (tearing) of the brain, whether due to penetration by a fragment of bone or by an environmental object inevitably leaves a scar, just as does a laceration elsewhere in the body. With aging this scar undergoes contraction or shrinkage and other changes closely parallel to scars in other tissue. In doing so, this scar may create an area of irritation in the brain leading to seizures, and this is what we know as posttraumatic epilepsy. This is one of the few aspects of head injury in which immediate recovery may seem excellent only to be followed by a delayed disability.[11] Following compound depressed skull fractures in which the dura is torn, the incidence of seizures is about 50 percent.[12] If the dura is not torn, though the underlying brain is compressed and contused, the frequency is about 23 percent. In closed head injuries the incidence is about 5 percent. Ninety percent of posttraumatic epilepsy occurs within the first year; the greatest percentage within the first three months. Convulsions can be due to cranial defect (the pressure of the overlying depressed skin irritating the brain), though this is very infrequent.[13] Actually, it's extremely difficult, if not impossible, to ascertain whether the seizures are due to the irritated brain from the injury itself or to the depressed skin over a cranial defect. This is where the diagnostic tests previously mentioned can play an important role. When it does develop after an injury, epilepsy is far more apt to develop from contusion than from concussion because it is nature's repair with scar tissue which, after months or years, becomes the irritative focus.[14]

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[11] Stein, note 7, at 198.
[12] Id.
[13] Id.
[14] Davidson, note 10, at 206.

Section 295

Epilepsy: Relationship to Trauma, Proof of

Trauma to the head may cause epilepsy, as in the instance of a skull fracture or penetrating wound of the skull. Head injury may result in three basic patterns of convulsive seizures. The first and most common is the grand mal seizure, characterized by a loss of consciousness and a generalized fit in which the patient stiffens into a so-called tonic phase of seizure, with a rhythmic jerking of his extremities known as the clonic phase of the attack. An interval of relaxation follows, extending into the muscles of the bladder and bowel, with loss of control.

The second type of epileptic fit is known as the focal seizure, usually beginning in a specific part of the body, as for example, convulsive movements of the arm, leg or mouth, or an isolated part thereof. The seizure may begin in the thumb or corner of the mouth, indicating a basic disturbance in the brain, and spreading to other areas.

There is also a so-called psychomotor type of seizure, characterized by spells of psychotic behaviour or irrational conduct, as for example, the performance of a physical act at a time and place not normally associated with such act. Other seizures

Q. What was the diagnosis that you finally made, doctor?

A. Post-traumatic epilepsy.

Q. Did you take a history from this patient before your examination?

A. I did.

Q. Did the patient disclose a history of prior epileptic seizures?

Q. Or convulsions of any form whatever?

A. None at all.

Q. Did you specifically inquire upon this point?

A. I did.

Q. Upon what symptoms or signs did you base your diagnosis?

A. On results of an electroencephalogram showing a focal abnormality, and the following neurological signs, etc.

Q. What is an electroencephalogram, doctor?

Q. Describe the manner in which it is taken.

assume characteristics of all three basic types, without readily lending themselves to precise classification.[1]

Authorities are not entirely agreed on the frequency or degree of relationship between trauma and epilepsy, some believing that head injuries rarely cause epilepsy, but merely precipitate an already existing predisposition to convulsive seizures. Other observers believe the incidence of epilepsy as a result of head injuries is relatively high.

From a legal point of view, however, the difference in degree of causation may have slight significance. A tortfeasor is liable for the full consequences of his wrongful act, regardless of whether the resulting convulsive seizures are due to an aggravation of a latent condition or represent an original injury.[2]

Where a mild trauma to the head is followed by a seizure, the injury is usually a precipitating factor, the predisposition being already present.

Q. Please explain the significance of the findings this test disclosed.

Q. Now, doctor, will you explain to this jury the nature of the neurological tests that you conducted?

(Explain fully)

Q. What did you learn as to the time of the first seizure?

A. It took place two days after the accident.

Q. What is the significance of this time interval?

Q. What did the history of the patient disclose with respect to an accident or physical injury during July of last year?

A. He stated he was thrown from a car during a collision and

1. The history of a few isolated dizzy spells or blackouts, following a head injury, together with abnormal electroencephalographic findings, do not in themselves necessarily establish a diagnosis of post-traumatic epilepsy. It is necessary to carefully evaluate the entire clinical pattern over a sufficient period of time before such a diagnosis can properly be made.

2. One authority, Dr. Desmond S. O'Doherty, Professor of Neurology at Georgetown University School of Medicine, pointed out that in an analysis of 2500 cases the incidence of epilepsy as a result of head injury ranged up to 50%. Where the scalp alone is involved, the incidence of seizures was 17%; involvement of the dura raised the figure to 22%. An actual penetration of the dura (the membrane comprising the outermost covering of the brain) indicated an incidence of seizures in 33% of the cases. Proceedings of Medico-Legal Seminar, Washington, D.C. Jan. 17, 1957.

The question whether the patient suffered convulsive seizures prior to such trauma would then assume paramount importance.

A severe blow to the head may cause a hemorrhage inside the brain, or a skull fracture can result in bony fragments being driven into the brain matter, with seizures following as a direct result thereof. It is important for counsel to note that the scar tissue forming after such injuries may, unlike scar tissue of the skin, grow slowly and not fully mature until many years after the trauma, with a resulting delay in appearance of the seizures or the characteristic symptoms of epilepsy.[3]

Counsel should, in preparing proof of relationship between the trauma of a specific accident and epilepsy, carefully evaluate the following factors with his medical witness:

a. How soon after the trauma did the actual convulsive seizures begin? The time interval may prove decisive, although a period of several years does not necessarily preclude a finding of causal relationship.

b. What symptoms preceded the first convulsive seizure, and with what frequency did they occur? Where these symptoms form a consistent or

struck his head on the ground, becoming unconscious.

Q. How long was he unconscious?

A. He stated the period was six hours.

Q. Did you form an opinion, based upon the history received from the patient and your examination, as to a relationship between the physical injury and the epileptic seizures?

A. I did.

Q. Are you able to state, with reasonable medical certainty, whether this accident was a competent producing cause of the epileptic seizures?

A. I can.

Q. Will you so state, doctor?

(Examination of plaintiff)

Q. Describe your general health prior to the accident on July 7th, last.

3. The precise mechanics by which a traumatic scar in the brain causes epilepsy is not known. A lesion accompanied by destruction of the cerebral cortex may, for example, cause speech disorders or impairment of vision in one person, and post-traumatic convulsive seizures in another. There is no restriction as to site of the lesion: it can occur anywhere in the brain and still result in such seizures.

classical pattern for a post-traumatic epilepsy, the diagnosis is accordingly made more credible.

c. How severe was the trauma to the head? A slight blow with no more than dizziness at isolated periods may be more difficult to establish as a causative factor than a severe form of trauma, as a skull fracture, with loss of consciousness, major reflex changes or paralysis.[4]

The extent to which epilepsy has rendered a person disabled from normal physical activity or the duties of his occupation must be determined from the frequency of his seizures, their severity and general characteristics, and the demands of his particular job or craft.

Q. Did you ever suffer from any type of convulsive seizure?

Q. When was the last time you consulted a doctor prior to that accident?

Q. For what condition did you visit the doctor?

Q. Did you ever suffer blackouts, or loss of memory?

Q. Were you rendered unconscious following a blow, prior to this accident?

4. The results of all neurological or other tests should be prepared for use at the trial. The indication of a focal abnormality in an electroencephalogram, for example, may be highly important in establishing the head injury as the cause of the epileptic seizures.

The time interval between the physical injury and the onset of the convulsions is not necessarily a dependable guide in evaluating the causal relation between the two. There have been instances of convulsions taking place as late as fifteen years after a brain injury, although it has been noted that most authorities believe that where convulsions or significant symptoms do not appear within two years after the trauma they are not related thereto. McBride, Earl D., Disability Evaluation, Fifth edition, J. B. Lippincott Co., p. 643.

§ 18.06 Abnormal EEG

The types of electrical abnormalities have been traditionally described as falling into four rather broad groups. The EEG is abnormal because:

1) it contains too much "slow" activity, or
2) too much "fast" activity, or
3) there are transient or "paroxysmal" abnormalities in the record, or
4) there is an electrical event over a single part of the brain—a "focal" abnormality.

(1) Abnormally Slow

"Abnormally slow" inplies that the frequencies obtained are much slower than those usually seen for the patient's chronological age, and that the slowing is not part of the slower activity seen normally in drowsiness or sleep.

So, what does "abnormally slow" mean that the patient has? Like many other laboratory results, these are *nonspecific findings*. The slow record reflects an alteration in brain physiology, but a host of pathological processes can reproduce the same electrical picture. For example, it is quite difficult to identify by an electrical sign alone the abnormally slow activity produced by a recent trauma to the head, infections of the central nervous system, increased intracranial pressure, disease of the cerebral blood vessels, or brain tumors.

In other words, a 3 cps wave looks the same regardless of the mechanisms responsible for its production.

This is not to say, however, that there are not additional clues in a recording besides the slow activity. Other considerations must be given to the presence or absence of focal abnormalities, the character of the background rhythm, and changes observed in serial (separate examinations made in series, usually on different days, and spread over a period of several days or weeks) recordings. These factors may aid the electroencephalographer in incriminating the particular abnormally slow activity observed to a particular cause.

(2) Abnormally Fast

"Abnormally fast" usually is even less specific regarding cause than is the slow activity. However, it is generally a more benign abnormality; and in many cases, the clinical correlations that can be made with an unusual amount of fast EEG activity are quite poor.

The most common cause of an unusual amount of fast activity in the EEG is probably related to recent drug ingestion or administration.

(3) Paroxysmal Abnormality

A paroxysmal abnormality implies that the electrical changes appear in a transient or intermittent fashion. The connotation has been that paroxysmal abnormalities imply the possibility of a convulsive disorder—epilepsy.

The electrical paroxysms may consist of intermittent slow activity, or they may consist of sudden changes in the amplitude of the waves. Spikes and sharp waves are brief duration signals, often high in amplitude, that always are a rather important indication of central nervous system abnormality. These are often seen in the convulsive disorders, and appear as paroxysmal patterns.

(4) Focal Abnormalities

Focal abnormalities indicate that a specific area of the brain is not producing normal electrical activity. The abnormality may be an area of slow waves, or fast frequencies, or a region of increased or decreased amplitude, or the site of transient, paroxysmal type of abnormalities.

Detection of a focal abnormality may be an important diagnostic clue and is an important consideration in the evaluation of any recording.

§ 18.07 EEG in Diseased States

The following is a brief description of the electrical abnormalities observed in specific diagnostic categories. Equally important, however, is the problem of electrical *normality* in the known presence of disease.

(1) Convulsive Disorders

The greatest contribution of electroencephalography to clinical medicine has been in relation to the convulsive disorders.

Though it is not necessary to rely on the EEG in making a clinical diagnosis of a convulsive disorder, the EEG is abnormal in approximately 75 to 85 percent of all cases of epilepsy. In addition, the percentage of abnormality may be increased by securing serial records, and by using such "activiation" techniques as sleep, metrazol, and photic (by light) stimulation. The EEG serves as a valuable supplementary diagnostic tool in this disorder.

Though the percentage of electrical abnormalities observed is quite high, the type of electrical abnormality may not be specific for either the type of convulsive phenomenon or its cause.

(2) Petit Mal Epilepsy

Of all the types of electrical abnormalities, the now classical *rhythmical spike* and *3 cps paroxysmal* abnormalities observed in association with petit mal epilepsy are the most specific.

If this EEG abnormality is observed in a tracing, the probabilities approach 95 percent that the patient has had, or will have, brief seizures characterized by short lapses of consciousness, blinking of the eyes, staring, and perhaps mild abnormal motor activity.

This does not mean, however, that this is the only type of seizure the patient may have; for, in addition, the patient may suffer from an occasional generalized, or more rarely, a focal type of seizure. The high potential spike and 3 cps activity (*Fig. 18-2*) is often not present in the "resting" part of the tracing, but only appears in response to hyperventilation (overbreathing) or during drowsiness and light sleep. In many instances, this abnormal spike and 3 cps wave pattern

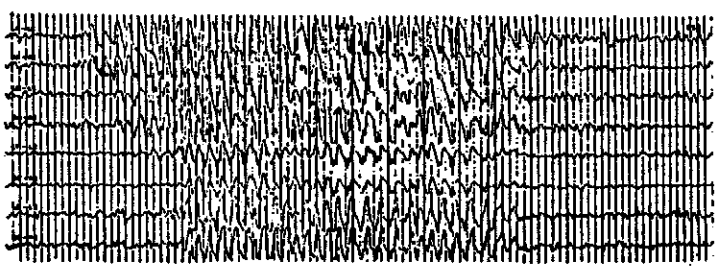

FIG. 18-2. An example of the spike and 3 cps wave activity associated with petit mal epilepsy. The sample was recorded over a twenty-second interval.

is extremely sensitive to changes in the level of circulating carbon dioxide; hence, most laboratories include a period of overbreathing for its detection.

Petit mal spells are characteristically a type of convulsive disorder found in children between the ages of two and fifteen years, with a tendency to subside after this time. It is also a condition of unknown cause, with factors such as birth injury, infections and injuries in later life playing a less definite role than in some other types of convulsive disorders.

(3) Petit Mal Variant

A variation of the spike and 3 cps activity has been called the *petit mal variant*. This abnormality is characterized by a less clearly rhythmical relationship between the spikes and the 3 cps waves, with the spiking often of a multiple or polyphasic type, and the slow waves ranging from 1-to-4 per second.

In addition, the abnormality is more apt to be focal in type as well as paroxysmal—in marked contrast to the classical spike and 3 cps wave pattern in petit mal. The type of clinical convulsive symptoms associated with the petit mal variant are less likely to be limited to seizures of brief duration, and are also more likely to be related to birth injury, anoxia, infections or injuries in later life.

(4) Grand Mal

Grand mal, or generalized tonic-clonic types of seizures, are not as well correlated with a specific electrical abnormality as are petit mal seizures. The types of electrical abnormalities may range from generalized, paroxysmal slow, 1-to-3 or 5-to-7 cps waves of a higher potential than the background rhythm, to intermittent generalized runs of spiking and/or slow wave activity. In addition, focal abnormalities may be present that consist of either focal slow waves and spikes in varying combinations.

Grand mal epilepsy is not a disease but a symptom of many possible causes; and similarly, the EEG pattern may be quite diverse. For example, a patient may experience generalized seizures resulting from a small area of scarring on the cortex secondary to trauma, or the seizures in other individuals may

be of a less clearly defined origin. Hence, they are classed as "idiopathic" or "cryptogenic." However, both spells may appear the same clinically.

(5) Generalized Body Disturbance

Generalized seizures may also be a manifestation of a more generalized bodily disturbance, such as pathologically low blood sugar levels, or in uremia secondary to kidney disease. Toxic agents—lead, alcohol, barbiturate withdrawal—also may be manifest by generalized seizures.

Again, there is no single specific pattern for any of these clinical situations, though clues may be obtained regarding possible cause by considering the character of the background rhythm or the presence or absence of focal abnormalities.

It should be pointed out that the electrical paroxysm may not be associated with any clinical change in the patient while the EEG is being obtained and that such electrical abnormalities appear between clinical seizures.

(6) Psychomotor Seizures

Psychomotor or temporal lobe type of seizures have become clarified more precisely both clinically and electroencephalographically in the last ten years.

"Psychomotor" is perhaps an unfortunate word in that there is some connotation of a possible psychiatric or functional origin for this disorder. Such is not the case. Clinically, these spells are manifest by a cluster of signs and symptoms such as brief periods of unawareness, with smacking of lips, flushing or blanching of the face, the performance of fairly elaborate motor acts such as fumbling with clothing or walking aimlessly for short distances—all without awareness or recall. These acts have been termed "automatisms"—examples of automatic behavior.

Superficially, the automatisms resemble the *fugue states* observed in some psychiatric diseases, but a differentiation can be made by the presence of the changes in the autonomic nervous system—the flushing or blanching, and the time relationships involved.

Psychomotor seizures are mainly events occupying seconds

or minutes, while fugue states of psychic origin are hours or even days in duration.

It is now generally recognized that the EEG is abnormal in 75 to 90 percent of these cases, and that a fairly specific abnormality is present in these recordings. This consists of a spiking discharge appearing over one or both temporal regions, most easily demonstrated during drowsiness or light sleep (*Fig. 18–3*). The spiking may be the only abnormality present, or it might appear in association with generalized or focal slow waves.

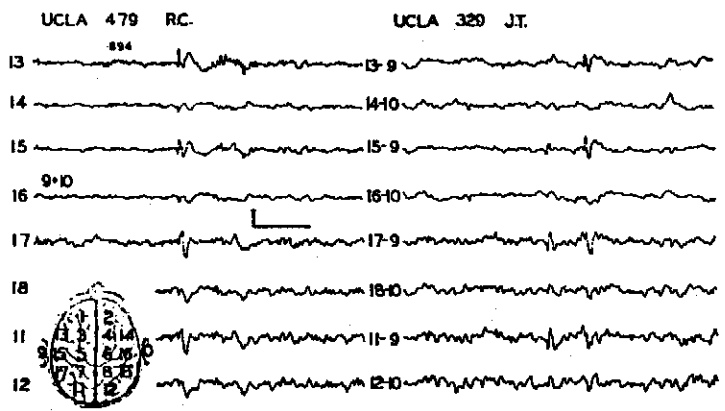

FIG. 18-3. Spiking activity is apparent over the left side of the head, as recorded from two individuals, both of whom had temporal lobe types of seizures.

This type of seizure strongly suggests a pathological involvement of one or both temporal lobes, more specifically structures deep within this region. The pathology may be secondary to trauma to this area, either at birth or later in life; with tumors in this vicinity; or as a secondary result of many previous generalized seizures with associated periods of oxygen deprivation.

(7) Focal Seizures

Another clinical category of convulsive phenomena is the *focal* types of seizures. The clinical manifestations are dependent upon the *area* of the brain that is involved. Psychomotor spells, for example, might be considered as a focal type of seizure in that the temporal lobes are involved—with both motor and behavioral changes resulting. Other types of focal seizures may be largely motor in type with the electrical abnormality appearing at or close to the part of the cortex controlling voluntary movement—the so-called "motor strip."

The clinical symptoms may also be largely sensory in type, in which case the focal electrical abnormality is close to or on the sensory receiving area of the cortex.

This classification of seizures is based on location only, and may result from trauma, old infections, brain abscesses, changes in blood supply to that region, or brain tumors.

(8) Myoclonic Jerks

There are other clinical types of seizures that are less commonly encountered. Myoclonic jerks are brief, fairly generalized, contractions of large groups of muscles associated with generalized, high-potential spike discharges in the EEG. The pathological basis for this type of seizure activity is quite diverse, though infections of the central nervous system and degenerative disease have been most frequently incriminated.

(9) Fourteen and Six Positive Spikes

Fourteen and six per second positive spikes, probably incorrectly called *hypothalamic epilepsy,* constitutes a relatively new electrical and clinical entity. The electrical abnormality consists of positive spikes at a repetitive rate of 14 and/or 6-per-second over the temporal-occipital region. (*Fig. 18–4*).

This is almost always observed in the stage of drowsiness and light sleep, and is much more commonly seen in children.

The clinical correlations of this abnormality are quite vague, ranging from disturbances in behavior, dizziness, headache, and a variety of definite seizure activity. It has already appeared as a medico-legal problem in its possible relationship to brain trauma.

(10) Correlation of Abnormal Activity to Convulsive Disorders

Several comments are necessary to classify the relationship of abnormal electrical activity to convulsive disorders.

Traumatic Epilepsy, 24 Am Jur, Proof of Facts 2d 1 (1980)

II. PROOF THAT PLAINTIFF'S EPILEPTIC SEIZURES WERE RESULT OF TRAUMA RESULTING FROM AUTOMOBILE ACCIDENT

A. Elements of Proof

§ 26. Guide and checklist

The following facts and circumstances, among others, tend to establish that the plaintiff is suffering from posttraumatic epilepsy and will probably continue to suffer from this condition in the future for an indefinite period of time.

- ☐ Circumstances of accident [§ 27]
- ☐ Nature of plaintiff's injuries [§§ 28, 36]
- ☐ Plaintiff's first seizures [§§ 29, 31]
- ☐ Electroencephalogram (EEG) test [§§ 30, 37]
- ☐ Other neurological tests [§§ 30, 38]
- ☐ Frequency and duration of plaintiff's seizures [§§ 32, 41]
- ☐ Absence of prior accidents involving head injury to plaintiff [§ 33]
- ☐ Plaintiff's freedom from seizures prior to accident [§ 33]
- ☐ Absence of epilepsy among plaintiff's ancestors and blood relatives [§§ 33, 39]
- ☐ Nature of treatment of plaintiff's seizures [§§ 34, 42–44]
  —Degree of control achieved by medication [§§ 34, 42]
  —Side effects of medication [§ 44]
- ☐ Effect of seizures on plaintiff's activities [§ 35]
- ☐ Neurological examination of plaintiff [§ 36]
- ☐ Nature of plaintiff's seizures [§§ 36, 40]
- ☐ Trauma as probable cause of plaintiff's seizures [§§ 36, 38–41]
- ☐ Causes and effects of seizures of type suffered by plaintiff [§§ 40, 41, 45]
- ☐ Prognosis for plaintiff's condition [§ 45]

[It is assumed in the following proof that an eight-year-old boy was involved in an automobile accident during which he received a blow to the head, and that since that time, or commencing shortly after the accident, he has suffered from periodic disabling seizures which are controlled with only partial success by medication.]

B. Testimony of Plaintiff's Mother

§ 27. Circumstances surrounding accident

[After introduction and identification of witness]

Q. What relation are you to the plaintiff in this case?

A. I am his mother.

Q. How old is the plaintiff now?

A. He is ten years old.

Q. How old was he when the accident in which he was injured occurred?

A. He was eight years old.

Q. Where were you when the accident happened?

A. I was in the same car with my son, and my husband was driving.

Q. Would you tell us briefly what happened, please.

A. We were traveling along the highway near town when my husband's car was hit broadside by the defendant's truck; I was knocked out for a few minutes, and my son was thrown right out of the car.

Q. What happened to your son?

A. He was thrown onto the pavement and knocked out.

Q. For how long was he unconscious?

A. For about 10 or 15 minutes, as far as I know.

> ☐ **Note: Duration of unconsciousness.** Evidence as to how long a trauma victim was unconscious should be introduced, if available, since studies have shown that there is a direct correlation between the length of unconsciousness occurring after a blow to the head and the frequency of occurrence of posttraumatic epileptic seizures. **Cross-reference:** As to factors that tend to increase the likelihood of development of posttraumatic epilepsy, see § 17, supra.

Q. Were you yourself conscious at the time?

A. Yes, I was.

Q. After your son regained consciousness, what did he do?

A. He just lay on the ground, very white in color, and started to cry. He said he could hardly breathe and that his chest hurt.

Q. Did he attempt to move at all?

A. No, hardly at all. He just moved his arms a little bit and kept crying.

Q. What injuries did your son receive?

A. I couldn't tell; there was no blood that I could see.

Q. How long did you remain at the scene of the accident?

A. It was about 20 minutes before the ambulance came.

Q. And then you were taken to the hospital?

A. Yes.

§ 28. Plaintiff's hospitalization after accident

Q. After the ambulance took you to the hospital, what was done for you and your son?

A. A doctor examined both of us, and took X-rays of my son's head and body.

Q. Did you stay with your son while these examinations were going on?

A. Yes.

Q. Were you with him when the X-rays were taken?

A. Yes, I was allowed to stay with him most of the time he was in the hospital.

Q. Were there any other diagnostic procedures made at that time?

A. No.

Q. Was there any positive indication that your son had received a blow to the head?

A. He had a big bruise on the side of his head.

Q. Was he bleeding?

A. No, there was no blood.

Q. After you had been checked over at the hospital did you and your son go home?

A. They said I could go home right away but that I should leave my son there for observation.

Q. After they took the X-rays, what did they do with your son?

A. They took him up to the intensive care unit and kept him there for three days.

Q. Then what happened to him?

A. Then he was put in the pediatric ward and finally they said he could go home.

Q. How long was that after he had been admitted to the hospital?

A. Just a week.

§ 29. Plaintiff's first seizures

Q. How did your son seem after he got home?

A. He was tired and he slept a lot, and we had to bandage his ribs with a sort of strap that they gave us to wrap him in.

Q. Was he able to go out and play?

A. No, he had to stay in and we were told to watch him carefully.

Q. For what reason were you supposed to watch him?

A. The doctor at the hospital just before we left said to watch him carefully for signs of strange or different behavior.

Q. And did you notice any different behavior?

A. Not particularly for the first few weeks, but later on he began to sort of tune out, right in the middle of a sentence or in the middle of doing something. All of a sudden he just seemed to not be mentally there anymore. Sometimes he would fall down when this happened.

Q. Were there any other signs of peculiar behavior?

A. One morning I went in to his bedroom to wake him and he looked very peculiar, even while he was asleep; suddenly he gave a strange sort of moan and he went into a very deep sleep and slept for two hours, which was unusual. Then when he woke up I found that he had wet the bed, something he had not done since he was a baby.

Q. After this happened did you attempt to seek medical advice?

A. No, not right away because when he woke up he seemed to be fine. However, he wouldn't eat anything for breakfast, and almost nothing for lunch. This was very unusual too.

Q. When was the next time that anything strange occurred with regard to your son?

A. About three or four weeks after the bed-wetting incident, he suddenly turned very pale one afternoon and his eyes turned to the right side of his head, his right hand curled up and his right foot went sideways.

Q. Did he speak to you?

A. I spoke to him and he make some kind of an answer but he really wasn't quite there, if you know what I mean.

§ 30. Plaintiff's second hospitalization

Q. After your son had the strange right-side attack, what did you do?

A. We decided, my husband and I, that it was time he went to the hospital, so we called our doctor and said we were taking him down to the hospital right away.

Q. What did your doctor say?

A. He said to go ahead and that he would phone the hospital and have him admitted.

Q. Did you have any indication at that time that your son might develop epilepsy?

A. Yes, the doctor at the hospital mentioned that it was a possibility and that was one reason we should watch him very carefully —to see if he had any seizures of any kind.

Q. In addition to the X-rays which you mentioned were taken when your son first entered the hospital after the accident, were other diagnostic procedures performed when he reentered the hospital?

A. Yes, he had several.

Q. How do you know?

A. I was present when they performed two of them, and then all of the tests they did were itemized on the hospital bill.

Q. Were any of these tests painful?

A. Yes, they did a spinal tap which caused him a great deal of pain afterwards.

Q. Did they do any other tests?

A. Yes, they also did a brain scan and an electroencephalogram.

Q. Were these tests painful to your son?

A. No, he seemed not to mind them so much.

Q. How long was your son in the hospital this time?

A. About six or seven days.

§ 31. Continuation of seizures

Q. Did you take him home after that?

A. Yes, they said he could go home but that I was to watch him very carefully and keep track of any seizures he might have.

Q. Did you find it fairly easy to recognize these seizures when they occurred?

A. Oh, yes, certainly; there was no question about it.

Q. How were you able to distinguish between normal behavior and the type of seizure he had?

A. The doctor explained to me the various kinds of behavior that my son might exhibit—rapid eye blinking, sudden brief periods of falling asleep, and that sort of thing.

Q. How soon after his discharge from the hospital did he have his first seizure?

A. During the first week he was home.

Q. How did this seizure manifest itself in his behavior?

A. He would be walking or sitting or doing something and all of a sudden he would freeze; everything would just stop for him.

Q. Did he ever fall or run into things?

A. Yes, if he was walking and his balance was not right at the time the seizure occurred, he would fall. At other times, depending on his balance, he would just stop.

Q. How would you describe your son's appearance at such times?

A. He looked like a statue.

Q. Did you observe this on many occasions?

A. Yes, very often.

Q. Did you make any kind of written record of your observations?

A. Yes, the doctor suggested that I should keep a record of events of that nature, so I did.

Q. You kept a sort of a diary, is that it?

A. Yes.

Q. Did you make an entry in the diary each time your son exhibited strange behavior?

A. Yes, I carried the diary with me everywhere, and I made up a kind of shorthand to help me record what kind of action he was performing at a particular time.

Q. And were you actually with your son for much of that first period of time he was home?

A. I practically never left his side, not even for meals.

Q. How were you able to manage this?

A. Luckily my mother and my two older daughters helped with

the meals and the housework—did it all, really—and I was able to spend most of my time with my son.

§ 32. Frequency and duration of seizures

Q. Approximately how many times during the course of a day did your son have seizures, before he started taking medication for his condition?

A. About 25 or 30 times a day; some days even more. It would depend on what he was doing.

Q. What do you mean?

A. Well, when he was sitting quietly, reading or watching television, he seemed to have more short seizures than when he was engaged in some sort of physical activity.

Q. Did you keep track of them as requested?

A. Yes, I would write down the time and the duration of every seizure that I noticed. Of course, I probably did not see all of them, no matter how carefully I tried to keep track. But I kept the diary pretty faithfully.

Q. For how long did you keep this diary?

A. I kept it intensively for about ten days.

Q. And after that time?

A. After that I only made note of the longer seizures my son was having; by then he was on medication and the seizures were fewer.

Q. When you were with him do you think that you were able to observe all of the seizures he had?

A. Not always. One time when he was sitting watching television I went over to him and spoke to him and tickled him and he was obviously having a seizure. I wouldn't have known about it if I hadn't gone over to him.

Q. How long did these seizures last usually?

A. Some of them were only a few seconds.

Q. Were most of the seizures of fairly short duration?

A. Yes.

Q. About how long, would you say, on the average?

A. I would say that about ¾ of them would last for maybe 10 to 30 seconds each. Some were even less than that.

Q. And the others?

A. Some of the others went on for a minute or two, but seldom more than that.

Q. Were these seizures of the same type you mentioned a while ago?

A. Yes; rapid eye movements and blinking, short periods of unconsciousness, losing his balance while walking, sudden freezing in the middle of doing something, and things like that.

§ 33. Family history; prior injuries

Q. Has your son ever had any seizures such as you describe happen to him prior to the accident?

A. No.

Q. Had anything like this accident ever happened to him before?

A. No, never.

Q. Has your son ever had any other head injury?

A. No, never.

Q. Could he have had a head injury that you would not have known about?

A. No, he always tells me if he has been hurt in any way.

Q. Are either you or your husband subject to epileptic seizures?

A. No.

Q. Is there any epilepsy in your family or in your husband's family that you know of?

A. No.

Q. Had anything like this ever happened to anyone in your family before?

A. No, not that I know of.

§ 34. Effect of medication

Q. When you found he was having up to 25 and 30 seizures a day, did you take him back to the hospital or see your doctor?

A. Yes. They said to bring him back in and they would give him medication.

Q. What sort of medication did he get?

A. They gave him Dilantin and phenobarbital.

Q. How did you know what drugs he was taking?

A. The doctor told me when he wrote out the prescriptions. And

the pharmacist and I discussed the two drugs when I had the prescriptions filled.

Q. After your son started taking the medication prescribed for him, did the seizures become less frequent?

A. Yes, they did.

Q. Did they stop altogether?

A. No.

Q. About how often does he have seizures now?

A. About every two or three weeks; of course, I would have to be looking at him at the time of the seizure in order to know that he was having one. It is possible that he has them more often than I know about.

Q. How long has he been taking this medication?

A. About two years.

Q. Now that he is taking medication, is your son able to do everything that the other children in the neighborhood do?

A. No. He cannot climb trees or go swimming or ride a bicycle alone, and we don't allow him to play football or other rough sports.

Q. Does he play with other children?

A. Yes, he has playmates both at home and at school.

Q. Do they understand about his condition?

A. Most of them do; his teacher and I have tried to explain a bit about it and they have accepted it very well. He has had two or three nasty experiences with ignorant schoolmates, however.

> ☐ **Practice Note: Emotional impact of epilepsy.** At this point the attorney may wish to continue with this line of questioning, eliciting from the epileptic child's mother some of the more unpleasant experiences the child has undergone since becoming epileptic. Ignorance and cruelty on the part of people, especially children, who are unaware of the nature of epilepsy is a fact of life even in our own supposedly enlightened time. The jury could, at this point, be made aware of this additional type of suffering undergone by a victim of epilepsy.

§ 35. Plaintiff's schooling

Q. Does he go to a regular public school?

A. No.

Q. Why not?

A. Because he is too far behind in his work.

Q. Does he go to any school at all?

A. Yes, we decided that he needed special training and he goes to a special school now.

Q. Who decided that he needed special training?

A. My husband and I, and the school authorities.

Q. Did your doctor agree?

A. Yes, he thought it would be best for him.

Q. How does he get along at the school?

A. It has been all right so far. He still suffers seizures every few weeks, however, and it is very tiring for him so that it is almost a whole day gone out of his life each time it happens.

Q. Would you say that he has caught up in his work with other children of his own age?

A. Oh, no. He is at least two years behind most of them.

Q. Do his teachers think that he will be able to catch up, or at least keep up with his work, year by year?

A. No, they say he will never catch up and he will be lucky to complete a year's schooling within the year.

Q. Does this special schooling cost you any money?

A. Yes, we pay ..... dollars per month tuition.

Q. Do you anticipate that it will cost you money for his education in the future?

A. Yes, certainly.

☐ **Practice Note: Collateral source rule applicable in damage award.** Where there was strong evidence that an epileptic child would need special schooling for a long time and the actual cost of such education was established by testimony, an award to the boy's father of damages for future expenses during the boy's minority was upheld against the contention that evidence was speculative as to whether the public school program which offered the child special education would be discontinued. The collateral source rule was applicable so as to preclude and render irrelevant evidence as to benefits provided by a third party, that is, the state or municipality, that were independent of the tortfeasor; damages otherwise recoverable could not therefore be diminished. Healy v White, 173 Conn 438, 378 A2d 540.

C. Testimony of Pediatric Neurologist (Expert Witness)

§ 36. Examination of plaintiff by neurologist

[After introduction and identification of witness]

Cross-Reference: For proofs regarding the qualifications of a neurological surgeon and of a pediatrician, see Physicians and Surgeons, 9 POF 241.

Q. Doctor, would you explain the term neurology for us, please?

A. Neurology deals with disorders of the nervous system, which includes the brain, the spinal cord, and the peripheral nerves and muscles.

Q. How is it that there is a separate specialty, pediatric neurology, within the general specialty of neurology?

A. The bodies of children are constantly growing and changing so that when their nervous systems are injured or diseased, a different set of reactions and events may occur than will occur in adults who have the same diseases or injuries.

Q. When was the first time you examined the plaintiff in this case, doctor?

A. He was brought to the emergency room of the ..... Hospital following an automobile accident. He remained there for about a week and then was sent home. About three weeks later he came to the pediatric department of the hospital where I examined him.

Q. Did you see the boy when he was first brought to the hospital following the accident?

A. No.

Q. Why was he sent to the pediatric department?

A. He had been having seizures at home, his mother said.

Q. What was the purpose of your examination?

A. To ascertain the cause of the seizures the boy had been having.

Q. Did you reach a conclusion as to what the cause was?

A. Yes, I did.

Q. What was your conclusion?

A. My diagnosis was that the boy was suffering from posttraumatic epilepsy as a result of the concussion he suffered in the accident.

Q. What was the basis for your conclusion that he had suffered a concussion?

A. It was based primarily on the fact that the boy had struck his head in the automobile accident, which was confirmed by the large bruise on his head, and by the fact that the boy was unconscious for several minutes after the accident.

Q. Did you take an X-ray of the boy's head?

A. No, he had already had an X-ray when he was first admitted to the hospital after the accident, and it revealed that there was no fracture of the skull.

Q. Would a skull X-ray reveal that a person has suffered a concussion?

A. No.

Q. Was the plaintiff's concussion sufficiently severe to cause epilepsy?

A. Yes, definitely.

Q. Did you yourself actually see the boy have a seizure?

A. Yes, a small one; it was only a brief fluttering of the eyelids, but it was typical of the sort of seizures children of his age might have.

Q. What particular criteria or factors would have to exist before one could determine that a person's epilepsy resulted from a head injury as opposed to some other cause?

A. There are three basic requirements that must exist for a diagnosis of posttraumatic epilepsy. First, the patient has never had epileptic seizures before; second, he has sustained a head injury of sufficient severity to cause epileptic seizures; and third, there is no other disease or anomaly present in the patient's body that would cause epileptic seizures.

Q. On the basis of your examination of this boy, do you believe that all of these factors exist here?

A. Yes, I do.

§ 37. Electroencephalogram (EEG) test

Q. Did you perform any diagnostic tests on the boy before diagnosing his condition as epilepsy?

A. Yes, there were several diagnostic tests.

Q. What diagnostic tests did you perform on the plaintiff?

A. First we did electroencephalograms, or EEG, as it is known.

Cross-Reference: For a proof regarding the authentication of an electroencephalogram, see Electroencephalograms, 4 POF 641.

Q. Could you please tell us what an EEG is and what it measures?

A. An EEG is basically a recording of the electrical activity of the brain. Electrodes are placed over various areas on the head. They are sensitive enough to detect changes in electrical activity in the brain. These changes are recorded and printed out in the form of a graph.

Q. Is the EEG similar to an electrocardiogram?

A. Yes, in a way. The chief difference is that the brain wave voltage of an EEG is much less strong than the heartbeat voltage of an electrocardiogram, so that the amplification of the EEG has to be much greater in order that the brain waves can be recorded and read.

Q. Does a child's EEG differ from that of an adult?

A. In a normal adult there is a predominance of what is called alpha rhythm, wherein the waves occur in a certain frequency, anywhere from 8 to 13 waves per minute. The frequency of children's waves is somewhat slower, and a normal range has been established for children.

Q. What was your purpose in making the EEG?

A. People who are suffering from epilepsy often give evidence of their condition on their EEG. Instead of the normal brain wave, you see what is called a spike and slow wave. This type of wave is always abnormal.

Q. When this type of wave appears, does this support or confirm a diagnosis of epilepsy?

A. Yes, this type of wave definitely supports the diagnosis, although it does not itself make the diagnosis.

Q. Does such an EEG show that epileptic seizures will occur in the future?

A. No, it shows that there has been cerebral damage, an injury to the brain, but it cannot be used as a reliable indication that seizures will occur in the future.

Q. If the EEG was normal, that is negative as to indicating any seizures, would this tend to cast doubt on your diagnosis of posttraumatic epilepsy?

A. If several EEG readings in a row were negative and showed no

spike and slow wave, we would certainly look for some other cause of the patient's seizures.

[At this point counsel should have the witness identify and interpret the plaintiff's EEG tests.]

☐ **Case Illustration: Admissibility of electroencephalographic tests.** Testimony and doctor's office reports supplemented by three electroencephalographic tests taken in three different years was admissible to show that plaintiff's epileptic condition had worsened from "a mild abnormality" in 1950, through "positive spike seizures" in one hemisphere of the brain in the EEG tracings in 1955, to "spike seizures" in both hemispheres of the brain in 1956. Testimony of plaintiff's expert in neurology to the effect that the injury was permanent was admissible (as against the contention that it was in answer to a hypothetical question based on the opinion of another expert) where his opinion was based on evidence of plaintiff's physical condition after the fall which caused his injury, and on testimony of the electroencephalographs showing progressive abnormality. Melford v Gaus & Brown Constr. Co., 17 Ill App 2d 497, 151 NE2d 128, 66 ALR2d 528.

§ 38. Other tests

Q. Besides the EEG test, what other diagnostic tests did you perform?

A. We made a thorough physical examination of the boy, of course. Then we performed a spinal tap.

Q. For what reason did you do the spinal tap?

A. To rule out the possibility that he might have been suffering from some kind of destructive brain lesion.

Q. What did the results of this test indicate?

A. It showed a normal protein content, which tends to rule out the possibility of a brain lesion and confirm the diagnosis of epilepsy.

Q. Did you perform any blood tests on the plaintiff?

A. Yes.

Q. Are you able to tell whether or not a person has epilepsy by performing blood tests?

A. No.

Q. Then why are they performed?

A. They are useful for subsequent monitoring of chronic effects of drug treatment in people with epilepsy.

Q. Did any other tests you did tend to confirm the posttraumatic epilepsy diagnosis?

A. Yes, we performed a brain scan, also known as computerized axial tomography, which enabled us to find out whether or not the boy had a tumor or blood clot in his brain as a result of his head injury, or whether there was some evidence of cerebral hemorrhage or infarct that may have affected the blood supply to the brain, or whether there were any abnormalities of the boy's cerebral vascular system.

Q. What were the results of the brain scan?

A. The results were negative, that is, the boy showed no evidence of blood clots or any other such abnormalities I have mentioned.

Q. If the results were negative, doctor, how this does support your finding of brain concussion and epilepsy?

A. The brain scan results merely ruled out other possibilities of brain injury, and reinforces the presumption that the boy was suffering the effects of concussion, which is what we had tentatively diagnosed all along.

Q. Did the brain scan and other test results confirm your tentative diagnosis?

A. Yes, though we still had not determined from the boy's parents whether or not there was any familial predisposition to epilepsy, that is, whether there was any history of epilepsy in the family, or whether the boy had shown any sign of it in the past.

Q. How is this information obtained?

A. By questioning friends and members of the family of a suspected epileptic one can often determine if there is a hereditary predisposition to the disease.

§ 39. Family history; predisposition

Q. Did the plaintiff have any predisposition or tendency toward epilepsy?

A. Not that we know of. It is quite possible that he did, but nothing in the family would indicate that he has a predisposition to epilepsy.

Q. What were you able to find out concerning the boy's family history as regards the possibility of epilepsy occurring?

A. I questioned both the father and the mother closely, and separately, but was unable to elicit anything but negative replies to my questions concerning the possibility of epilepsy in the family.

Q. Are you sure of the family history?

A. It is rare that a family history is completely accurate. It is quite possible that the boy has a relative or someone in his genetic background with a tendency toward epilepsy; it is very difficult to be sure.

Q. What about the possibility of birth trauma, that he sustained some injury at birth that might have damaged his brain?

A. I inquired specifically about that but the mother denied any knowledge of birth trauma.

Q. Could there have been some injury to the boy at birth that the mother did not know about?

A. It is possible; some physicians believe that there are many more seizure-causing birth traumas than we know about. However, this is a matter of conjecture and we have as yet no way of proving such injuries occurred.

Q. Is epilepsy an inherited disease?

A. At one time it was thought to be, but few believe this nowadays. It is believed that there is a genetically acquired threshold of propensity to epileptic seizures.

Q. Would you explain what that means, doctor?

A. It means that the propensity of certain people to having epileptic seizures is stronger than it is in other people, because it takes less of a trauma, for some reason, for them to begin having epileptic seizures. It is sometimes termed the "constitutional" factor which inclines these people towards susceptibility to epilepsy.

Q. Is this predisposition to epilepsy always inherited?

A. Not necessarily, though it is difficult to say for sure; it may occur in families where it has never occurred before. However, if a child has one or more parents or close relatives with epilepsy in his or her background, the child's risk of being born with a tendency to epilepsy is far greater.

Q. Why is it difficult to determine the inherited factor in suspected epileptics?

A. Family histories are often inaccurate.

Q. Why is that?

A. There is or was a stigma attached to a person having epilepsy, and many people, on being interviewed in the hospital, will deny that they have any parents or close relatives who are epileptic. In some cases, of course, they may be completely ignorant of the fact that they have epileptic relatives. In any case, a completely accurate picture of a patient's background with regard to possible epilepsy is very difficult to acquire.

Q. What effect, if any, would trauma or an injury have upon an individual who is predisposed toward epilepsy?

A. Such an injury would usually increase the possibility of the person having epileptic seizures.

Q. What type of injury would be necessary to produce this?

A. No particular type of injury is required; any injury to the head or its contents could produce epileptic seizures.

Q. Can you make a fairly certain assumption that there is no history of epilepsy in the family?

A. No, not a certain one, but we must make that assumption since there is no other information we have to go on.

Q. Did you find anything else that might indicate that he had, or was susceptible to having, epileptic seizures?

A. No, nothing beyond the head injury.

Q. Did you inquire concerning other possible illnesses or injuries that might have caused a predisposition to epilepsy?

A. Yes, I did.

Q. What were you able to find out?

A. That the plaintiff had no known history of previous illness or injury that would have predisposed him to seizures of the type he was having.

☐ **Practice Note: Inquiry into prior accidents.** As part of the pretrial investigation, plaintiff's counsel should inquire carefully into prior accidents involving head trauma. Where there is a history of a prior injury to the head, hospital records and physicians' notes should be examined, in order that a determination can be made as to whether the last accident is the sole producing cause of the alleged

disabilities. Failure to explore fully the possibility of a prior accident contributing in some degree to the present complaints may invite the embarrassing situation where counsel learns for the first time at the trial that the head injury forming the basis of his action is actually an aggravation of a prior injury. Masland, Epileptics—Their Medicolegal Problems & Confrontations, 7 Law Med J 2d 339 (Feb 1979).

§ 40. Plaintiff's seizures—Effect of psychomotor seizures

Q. Could you give us a brief explanation concerning the mechanism by which trauma of the kind suffered by the plaintiff causes epileptic seizures?

A. It is generally assumed that seizures are due to localized damage to the brain which causes the formation of a scar made up largely of dead brain tissue, which then shrinks, causing disturbance to the surrounding live brain tissue, or neurons. It is this disturbance of the surrounding brain tissue that causes various types of seizures that may occur.

Q. Why do the types of seizure vary so much?

A. Because different parts of the brain control different movements, emotions, feelings, and so on. If the damage occurs in the area of the brain where memory is stored, there may be disturbances in the patient's memories. If the damage occurs in the area of the brain controlling vision, the patient may have various kinds of visual disturbances. And so on.

Q. What type of seizures does the plaintiff have?

A. He suffers from psychomotor seizures, which are the most common type of attack, after the convulsion or grand mal seizure; they account for about 35 to 40 percent of all seizures.

Q. How is the patient with psychomotor seizures affected?

A. The seizures affect and usually begin in the temporal lobe of the brain; they may spread without causing a convulsion, and may disturb sensations of smell and hearing, as well as memory. The patient does not always lose consciousness but often behaves like an automaton. Following the attack the patient usually sleeps heavily, has a headache, and may have a period of confused thinking, or perhaps all three.

Q. Does the patient remember such attacks?

A. No.

Q. Does the patient fall down when he has psychomotor seizures?

A. No, not usually, since there is seldom complete loss of consciousness. However, if he or she is off balance at the moment the seizure occurs, there is a possibility of falling.

Q. Do the plaintiff's seizures indicate to you any particular cause and effect?

A. Yes, the plaintiff's symptoms and seizure pattern form a consistent or classical pattern for a posttraumatic epileptic condition.

Q. When you say posttraumatic epileptic condition, does this differ from merely an epileptic condition?

A. Only in the fact that it is more likely to have been caused by a specific trauma rather than another cause whose origin is unknown, or epilepsy that is not related to a particular trauma.

§ 41. —Time interval between accident and seizures

Q. What was the approximate time interval between the time of the accident and the plaintiff's first seizures?

A. According to the patient history it was approximately one month.

Q. Could you describe what there was in the patient history that indicated the boy had had seizures, in your opinion?

A. When the mother brought the boy in to the hospital for diagnosis and evaluation, she reported that about a month after the accident the boy began to exhibit symptoms that I interpreted as probable seizures.

Q. What were these symptoms?

A. She reported that the boy would suddenly appear to tune out from time to time, very briefly, sometimes falling down when this occurred for no apparent reason. In view of the subsequent history and diagnosis in this case, I believe that these sudden attacks were probably epileptic in nature.

Q. It is significant that the first seizures did not occur immediately after the accident?

A. No, epileptic seizures do not usually occur immediately after trauma. There may be a time lag of many months or even years. For this reason it is sometimes difficult to ascribe seizures that commence after trauma with absolute certainty to that trauma.

Q. Was the time interval which occurred in this case consistent with posttraumatic seizure?

A. Yes, the onset of seizures about one month after trauma is seen fairly frequently in posttraumatic seizures.

§ 42. Treatment; drug therapy

Q. What treatment did you prescribe for the plaintiff's condition?

A. Initially I prescribed 30 milligrams of phenobarbital twice daily.

Q. What is the action of phenobarbital in connection with epileptic seizures?

A. Phenobarbital is a central nervous system depressant which decreases the excitability of the brain and thereby diminishes the tendency to seizures.

Q. Did this dosage work for the plaintiff to eliminate his seizures?

A. It helped for a while, but later we had to put him on Dilantin as well as phenobarbital. After he began having 15 or 20 seizures per day it was necessary to increase medication.

Q. What is the function of Dilantin?

A. The effects of Dilantin are fairly similar to those of phenobarbital in that Dilantin decreases the excitability of the brain, thereby diminishing the threshold for seizures.

Q. How much Dilantin and phenobarbital is the plaintiff now taking daily?

A. He is taking 150 milligrams of Dilantin and 60 milligrams of phenobarbital twice a day.

Q. Are the plaintiff's seizures completely controlled by the medication he is taking?

A. No, not completely. He has seizures about once or twice every month. They are in fairly good control, however, and should remain so.

Q. Does this dosage appear to control his seizures adequately?

A. Yes, so far it has proved fairly workable for him. It may be necessary to reduce or increase the dosage as time goes by.

Q. Would these seizures be completely controlled if the plaintiff took more of the medication prescribed?

A. No, drug therapy controls the type of seizures the plaintiff suffers from, that is, psychomotor seizures, in about 25 or 30 percent of the cases and reduces the frequency of seizures in about 50 percent of the cases. The plaintiff falls into the latter category, it would seem.

Q. Have you examined the plaintiff recently?

A. Yes, about a month ago.

Q. Based upon your recent examination and upon his history, can you tell the court to what degree the course of drug treatment with phenobarbital and Dilantin has been effective in controlling his seizures?

A. After commencement with Dilantin, in addition to the phenobarbital, his seizure control was good, but not complete.

Q. And he is now experiencing one or two seizures per month?

A. Yes. It is possible that he is having other small seizures that go unobserved, but from what his mother and teacher and he himself tell us, he has a seizure every two or three weeks now.

§ 43. —Necessity for continuation of therapy

Q. For how long do you anticipate the plaintiff will require medication?

A. Possibly for the rest of his life; possibly less than that. It will depend upon whether or not he continues to have seizures.

Q. Why is it important to know if the seizures have stopped?

A. We can decrease the dosage when no more seizures occur. If after decreasing the dosage there are still no more seizures, we continue to decrease or eliminate the medication and watch the patient closely to see if he has any more seizures.

Q. How will you know if he stops having seizures?

A. It is sometimes difficult to know this; he would have to be watched carefully, since some seizures can occur at night during sleep. The boy's mother and teacher and others who see him frequently should necessarily keep on the alert for signs of further seizures.

Q. Can you determine if an epileptic patient is completely free of seizures?

A. I would rarely, if ever, state that a patient with a history of seizures has reached a point where he will never have one again.

Q. Have you tried reducing the plaintiff's dosage lately?

A. No, since he is still showing signs of seizures and having one or two every month or so it would not be advantageous to cut down on his medication.

Q. When would you think of cutting down on his medication?

A. If he goes for a period of about five years without a seizure, then medication can be cut down or discontinued.

Q. Is this period of time the same for all types of epileptic seizures?

A. No, for the most common kind, which are grand mal seizures, a two-year period without seizures is usually a sufficient length of time before one begins to cut down or discontinue medication.

Q. Would it not be safer for the patient just to continue medication for the rest of his life, without cutting down or eliminating it?

A. No, the medication should be decreased or stopped if it is possible to do so.

Q. Why is that?

A. It is always better not to take drugs than to take them, if possible, but where there are unpleasant side effects of the drugs, which is the case here, it is all the more reason to discontinue taking them.

§ 44. —Side effects of drugs

Q. Why is it deemed necessary to cut down or eliminate an epileptic's medication when possible to do so?

A. Because there are undesirable side effects from taking anticonvulsive drugs.

Q. Do the medications that plaintiff is taking, phenobarbital and Dilantin, have any known side effects?

A. The side effects of these drugs are primarily those of sedation. Dilantin has other side effects, including dizziness, insomnia, nervousness, twitching of the muscles, headache, and other central nervous system effects. It also can cause nausea, vomiting, and constipation. The most frequent side effect is gingival hyperplasia.

Q. Has the plaintiff manifested any of these side effects?

A. Yes, he is subject to rashes, and he has developed gingival hyperplasia, which is uncomfortable and must be watched constantly.

Q. Would you define that term for us please, doctor.

A. Gingival hyperplasia is an abnormal increase in the number of normal cells in the gums; a patient who has this condition may reduce its effects by massaging the gums and brushing teeth frequently.

Q. Has this condition had any deleterious effects upon the plaintiff?

A. Yes, some of his upper teeth have shown a tendency toward displacement so that he may need the services of an orthodontist to check his teeth and to keep them in line.

Q. Is this gum condition fairly common among patients who are taking Dilantin?

A. Yes, it occurs in approximately 15 percent of people who take the drug.

Q. Is this condition likely to be permanent?

A. Quite possibly. At least as long as he continues to take Dilantin; if he discontinues the Dilantin therapy his gum condition would probably disappear in a year or so.

Q. Why do you continue to prescribe Dilantin for the plaintiff, in that case?

A. Because other drugs that control his seizures have not been found effective, or their side effects have been even worse than those of Dilantin.

Q. What would happen if the plaintiff took too much of either one of the drugs he is taking?

A. A child of his age can take fairly large doses of phenobarbital without too much bad effect because it is rapidly excreted in children. He would be subject to drowsiness, probably, and perhaps dysarthria.

Q. Would you define that term please, doctor?

A. Dysarthria is a thick, slurred kind of inarticulate speech that people who have taken overdoses of phenobarbital, and also Dilantin, manifest.

Q. Are there any other side effects from overdosing on Dilantin?

A. Yes, a patient can develop ataxia, or unsteadiness of gait, and sometimes the patient will have diplopia, which means double vision.

Q. Has the plaintiff shown any indication of these side effects?

A. Not so far as I know.

Q. If he did, what would you do about it?

A. Probably cut down the dosage of Dilantin, at least temporarily. These conditions are sometimes caused by a temporary buildup of Dilantin, or sometimes a temporary increase in dosage, whether deliberate or accidental. Sometimes these conditions

occur with no apparent reason and then disappear almost as suddenly.

Q. Are there many other side effects of Dilantin?

A. Yes, there are quite a few, some of them even fatal.

Q. Would you describe some of the other, nonfatal side effects?

A. Well, some patients develop hirsutism, which is excessive body hair, and which is particularly disconcerting in females; then there are various gastrointestinal disturbances, such as nausea, vomiting, constipation, and abdominal pains. Some people who are particularly sensitive to the drug develop rashes.

Q. Has the plaintiff shown signs of any of these other side effects?

A. Yes, occasionally he is nauseous and has stomach pains, but they are not bad enough to warrant discontinuance of Dilantin therapy.

Q. Why is Dilantin prescribed so frequently?

A. Because it is so effective in the treatment of epilepsy. In most cases, the effect of doing without Dilantin is far worse than some of the side effects that can and do occur. If the side effects are too dangerous, of course, the drug would be discontinued and some other drug will be tried.

§ 45. Prognosis

Q. Do you have an opinion based on reasonable medical certainty as to whether or not the condition you diagnosed, that is psychomotor seizures, is permanent in his case?

A. Psychomotor seizures usually do not disappear as the patient grows older. Therefore there is a better than 50 percent chance that this child will be left with seizure problems throughout his life.

☐ **Practice Note: Statistical probability of permanency of epileptic condition.** In an action based on injury to a seven-year-old boy in an automobile who suffered "permanent minimal brain dysfunction syndrome with associated multiple psychmotor seizures," two pediatric neurologists were permitted to express themselves statistically in arriving at the conclusion that plaintiff's epilepsy was a permanent condition. One neurologist stated that "psychomotor seizures usually do not disappear as the patient grows older" and that "there is a better than 50 percent chance that this child will be left with the seizure problem throughout his life"; when cross-examined as to how much better than 50 percent statistically, he replied that "it is probably about 80 to 90 percent." This opinion was corroborated by the other neurologist who stated there was certainly a better than 60 percent probability that plaintiff would continue to have seizures, and that the "odds are very much against" plaintiff ever being seizure-free. Healy v White, 173 Conn 438, 378 A2d 540. See **Annotation:** Admissibility of expert medical testimony as to future consequences of injury as affected by expression in terms of probability or possibility. 75 ALR3d 9.

Q. Do you think that the plaintiff will suffer permanent brain damage from his epileptic seizures?

A. There is a possibility of that.

Q. Would you say that it is probable that he may suffer permanent brain damage?

A. No, I could say that it is possible but I could not say that it is probable.

Q. If the patient stopped having seizures for a period of time, would that necessarily indicate that he would continue not having them, indefinitely, perhaps?

A. No, not necessarily.

Q. How long a period of time would he have to be seizure-free in order for a physician to say that he was cured?

A. If an epileptic who is subject to psychomotor seizures remains seizure-free for a period of five years, we might go so far as to say that he was in remission. However, it is doubtful that any reputable physician would ever say that a patient of this kind is cured and will never again be subject to seizures.

Q. You mean that seizures may begin again at any time?

A. Yes; sometimes it will take a particular accident or event to set them off, but it is certainly possible that they may start again. Just how it happens medical science has no answer.

Q. Do you have an opinion based on reasonable medical certainty as to whether or not future treatment will be required in the plaintiff's case?

A. There is no question but that he will need further treatment in the future.

Q. What sort of treatment will that probably be?

A. The prescription of anticonvulsant medicine, and supervision of its administration, should be sufficient.

Presenting Plaintiff's Medical Proof, Traumatic Epilepsy, 6 Am Jur Trials

F. Traumatic Epilepsy

§ 20. In General

When epilepsy occurs without apparent cause, it is medically classified as idiopathic epilepsy. This type of epilepsy, frequently observed, has a hereditary or congenital basis. However, epilepsy may also occur as a sequel or complication of head trauma. It is then called traumatic epilepsy.

When epilepsy is alleged as a consequence of an injury, the defense may contend that the plaintiff's epilepsy was caused not by his brain injury but by unknown congenital or hereditary factors. Plaintiff's counsel must prove that the plaintiff's condition is traumatic epilepsy, a sequel of head trauma.[13]

13. Clinical Neurology (9th ed), by Israel S. Wechsler. Saunders (Philadelphia, 1963), p 489.

14. Acute Injuries of the Head (4th ed), by G. F. Rowbotham (late Hunterian Professor of Surgery and Dickinson Scholar). Livingstone (Edinburgh, 1964) pp 365–366.

15. Proof of epilepsy resulting from brain injury—testimony of attending physician. 4 Am Jur Proof of Facts, Epilepsy p 703.

ALR Annotations:

Admission of opinion evidence as to cause of epilepsy. 66 ALR2d 1089, 1094.

Excessiveness of damages for epilepsy resulting from injury. 16 ALR2d 129, 294.

§ 21. Incidence or Possibility of Occurrence

The incidence or chance of developing epilepsy in a head trauma case is apparently dependent on two factors: (1) the area of the brain that receives the trauma, and (2) the degree of trauma or penetration into this area.

The brain has three meninges or membranes (tissue coverings): the dura mater, the pia mater, and the arachnoid. The outermost of these three membranes is the dura, a tough, fibrous tissue. A fracture of the skull will frequently occur without any penetration of the dura; to penetrate this layer the fracture must be depressed so that the fragments will press down.

Nevertheless, according to Dr. D. Denny-Brown, Professor of Neurology at the Harvard Medical School, even when the dura is not penetrated, the incidence of traumatic epilepsy is still about 20 per cent in severe head injury cases.

> "Severe head injury, *without penetration of the dura,* carries about 20 per cent liability to epilepsy . . . and this liability is *not changed by the presence or absence of fracture of the skull.*"[16]

If the dura has actually been penetrated there is almost a probability of developing epilepsy. Dr. Denny-Brown states:

> "If the dura has been lacerated the subsequent liability to epilepsy is almost 50% (45% Ascroft, 49% Credner, in follow-ups over a long period)."[17]

The incidence of epilepsy may increase if certain areas of the brain are involved in the injury. For example, the possibility of developing convulsions or fits is reported to be as high as 67.7 per cent if the central parietal region is involved in the injury. Two

Anxiety as to future epilepsy as element of damages in personal injury action. 71 ALR2d 345, 347.

Accident insurance as affected by pre-existing epilepsy condition. 84 ALR2d 176.

16. Factors of Importance to Head Injury, by D. Denny-Brown, (Professor of Neurology, Harvard University Medical School). Clinics, vol 1 p 1416.

17. Factors of Importance to Head Injury, by D. Denny-Brown. Clinics, vol 1 p 1416.

English physcians, Dr. W. Ritchie Russell and Dr. C. W. M. Whitty, have stated:

> "Previous writers have pointed out that the site of the brain wound affects the incidence of fits. Ascroft (1941) considered that fits frequently followed wounds around the Rolandic area and diminished in incidence as the poles of the brain are approached, though he utters a caveat about the accuracy of localization in his material. Baumm's figures also support this: 67.7% fits in the 'central parietal region' compared with 15.8% in the frontal and 6.9% in the occipital areas, though Credner (1930) finds a smaller difference with 47.3% parietal and 45% frontal."[18]

Dr. Denny-Brown similarly observes:

> "The liability [*for development of epilepsy*] is greatest when the damage is to the frontal or parietal region of the brain, less in the temporal lobe and least in the occipital region. The question of life expectancy is one on which reliable statistics are few . . . ."[19]

§ 22. Time of Occurrence

The time interval between an accident and an ensuing epileptic seizure is, clearly, important. Indeed, it might well be supposed that the task that faces plaintiff's counsel who seeks to establish that the accident caused plaintiff's epilepsy ordinarily grows increasingly onerous as this time interval becomes greater.

Medical authorities, however, indicate that a traumatically caused epileptic seizure may occur at almost any time following head trauma. Thus, it has been said:

> *"An epileptic seizure may occur at any time interval after a head injury."*[20]

18. Studies in Traumatic Epilepsy—I. Factors Influencing the Incidence of Epilepsy After Brain Wounds, by W. Ritchie Russell and C.W.M. Whitty (Department of Neurology, Radcliffe Infirmary, Oxford, England). Journal of Neurology, Neurosurgery and Psychiatry, vol 15 p 96.

19. Factors of Importance in Head Injury—A General Survey, by D. Denny-Brown (Professor in Neurology, Harvard University Medical School). Clinics, vol 1 p 1417.

20. The Post-Traumatic Convulsive and Allied States (4th ed), by A. R. Elvidge (Assistant Professor of Neurosurgery, McGill University; Associate Neurosurgeon, Montreal Neurological Institute; Consulting Neurosurgeon, Montreal General Hospital), in Injuries of the Brain and Spinal Cord and Their Coverings, Samuel Brock, ed. Williams & Wilkins (Baltimore, 1960) p 287.

> *"Convulsive seizures are not uncommon after cranio-cerebral injuries. They may be of any type and occur at any time after the injury."*[1]

Dr. Walter E. Dandy, late Professor of Neurosurgery at the Johns Hopkins Medical School, states that *convulsions are not expected to occur until eighteen months to two years after the accident,* in cases where the fracture of the skull is not depressed:

> "Moreover, there is a very definite and important time relationship between the accident and the onset of convulsions. Again excluding depressed fractures from which the convulsions may appear immediately after the accident, convulsions due to cerebral trauma rarely begin within a year and usually not until 18 months or 2 years after the accident."[2]

Dr. D. Denny-Brown, Professor of Neurology at the Harvard Medical School, declares that traumatic epilepsy will not appear until the scar tissue in the brain eventually begins to contract and exert traction on the neighboring brain substance. He notes that cases have been reported in which traumatic epilepsy did not occur until twenty years after the head injury:

> "The attacks of epilepsy, due to brain injury, begin to appear at an interval of time after the injury, probably only when the scar shortens, as all scars do, with age, and exerts traction on the neighboring brain substance. *Cases have been recorded in which the attacks did not commence until 15 (Feinberg),*

1. Craniocerebral Injuries, by Donald Munro (Surgeon in Chief for Neurological Surgery at Boston City Hospital; Assistant Professor of Neurological Surgery, Harvard University Medical School; Associate Professor of Neurological Surgery, Boston University Medical School), Oxford University Press (New York, 1938) p 234.

Other Aids:

Late Complications of Brain Injury in the Presence of a Normal Neurological Examination, by Samuel A. Stornelli, M.D. Lawyer's Medical Journal, vol 2, no 1 (May 1966) p 49.

2. The Brain—Epilepsy, by Walter E. Dandy. Lewis' Practice of Surgery, Dean DeWitt Lewis, ed. W. F. Prior Co. (Hagerstown, Md., 1932) p 329.

16 (Symonds), or 20 years (Ascroft) after the injury, yet owing to their focal or Jacksonian character were clearly related to the damage of the brain."[3]

The following excerpts present similar opinions:

". . . Convulsions may set in months or years after an injury to the head."[4]

"While the convulsions may become manifest soon after the injury, they generally set in a few months or years later."[5]

"The convulsions may set in six months to two years or longer after the accident, particularly if there is scar formation."[6]

"Both grand and petit mal [forms of epilepsy] *may appear anywhere from one to ten years after the injury."*[7]

"Traumatic epilepsy may develop as late as ten years or more after severe injury of the head, but usually there has been in the interval vertigo, paroxysmal headache, or other premonitory symptoms."[8]

§ 23. Seriousness of Occurrence at Time Remote From Trauma

As has just been noted,[9] traumatic epilepsy may not appear until many years after the infliction of a brain injury. This fact is important to plaintiff's counsel since his case may be set for trial

3. Factors of Importance in Head Injury—A General Survey, by D. Denny-Brown. Clinics, vol 1 p 1418.

4. Clinical Neurology (9th ed), by Israel S. Wechsler. Saunders (Philadelphia, 1963) p 610.

5. Clinical Neurology (9th ed), by Israel S. Wechsler. Saunders (Philadelphia, 1963) p 491.

6. Clinical Neurology (9th ed), by Israel S. Wechsler. Saunders (Philadelphia, 1963) p 612.

7. Sequelae of Head Injury, by Samuel Brock (Professor of Neurology, College of Medicine, New York University; editor of "Injuries of the Brain and Spinal Cord and Their Coverings"). Journal of Medical Society of New Jersey, vol 49 p 319.

8. Epilepsy and Gunshot Wounds of the Head, by W. E. Stevenson. Brain, vol 54 p 223.

9. § 22, supra.

or settled long before it is clear that his client will or will not develop epilepsy as a consequence of his trauma.

Epilepsy that develops long after the trauma has a far graver prognosis than that which appears at an earlier date. This important fact has definite support in medical literature. Dr. D. Denny-Brown states:

"Attacks which begin in the first month after injury respond well to treatment and tend to disappear, whereas those with later onset respond less well to treatment and often lead to mental deterioration."[10]

Dr. A. Earl Walker, Professor of Neurological Surgery at The Johns Hopkins Medical School, makes the same observation:

"It has been stated that a late developing epilepsy has a poorer prognosis than one which comes on *shortly after the head injury*."[11]

10. Factors of Importance in Head Injury—A General Survey, by D. Denny-Brown (Professor of Neurology, Harvard University Medical School). Clinics, vol 1 p 1418.

11. Posttraumatic Epilepsy, by A. Earl Walker (Professor of Neurological Surgery, Johns Hopkins University). Charles C. Thomas (Springfield, Ill., 1949) p 7.

Epilepsy Foundation of America
Frequently Asked Questions

This page is intended to provide the basic information about epilepsy and seizure disorders to the general public. It is not intended to, nor does it, constitute medical advice, and readers are warned against changing medical schedules without first consulting a physician.

What causes epilepsy?

In about seven out of ten people with epilepsy, no cause can be found. Among the rest, the cause may be any one of a number of things that can make a difference in the way the brain works. For example, head injuries or lack of oxygen during birth may damage the delicate electrical system in the brain. Other causes include brain tumors, genetic conditions (such as tuberous sclerosis) , lead poisoning, problems in development of the brain before birth, and infections like meningitis or encephalitis. Epilepsy is often thought of as a condition of childhood, but it can develop at any time of life. About 30 percent of the 125,000 new cases every year begin in childhood, particularly in early childhood and around the time of adolescence. Another period of relatively high incidence is in people over the age of 65.

Can epilepsy be outgrown?

In some forms of childhood epilepsy the chances of outgrowing the disorder seem to be quite good. This is true especially in absence (petit mal) epilepsy and a condition known as benign childhood (Rolandic) epilepsy. However, there is no way to tell in advance whether a child's seizures will disappear in later life. Many children (and some adults) can be successfully weaned off the medicine (by a physician) after a couple of years of freedom from seizures, and in many of those cases, the seizures will not return.

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Does epilepsy get worse with time?

Epilepsy generally does not get worse unless the seizures are being caused by an underlying brain disorder that gets worse with time, such as a brain tumor. If anything, seizures seem to happen less often as people grow older. However, it is also possible to develop epilepsy for the first time as an older person.

Does physical activity increase seizures?

Research suggests that seizures may be less likely to occur when someone with epilepsy is engaged in physical activities than when bored or inactive. Living a normal, active life inevitably involves some risks. This is true for all people, not just those with epilepsy. Exercise is a necessary part of a healthy life style and, indeed, may improve seizure control. If there might be any special concerns or individual precautions, these can be discussed with the physician.

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Can head injury cause epilepsy?

Yes, epilepsy may result from serious head injury, and is known as post-traumatic epilepsy. It is most likely to occur after an open or penetrating wound, though on sometimes it develops after a closed head injury. The seizures generally do not begin until at least three months after the injury, but the time interval is variable. Evaluation of seizures resulting from head trauma includes a thorough history, physical examination, EEG, and CT scan or MRI. Injury caused epilepsy is treated with antiepileptic medications; later on, if seizures cannot be controlled, surgery to remove the scarred area may be an option.

What's the connection between stroke and epilepsy?

Cerebrovascular disease (stroke) is the most common cause of epilepsy in mid to late adulthood. Seizures resulting from effects of a stroke may occur from six months to two years after the incident in 20% of stroke patients. While strokes are more frequent in older adults, they may occur at any age.

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