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Irwin N. Perr

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Post-Traumatic Epilepsy and the Law

Irwin N. Perr, M.D.*

Post-traumatic epilepsy is a condition of great importance to the lawyer in personal injury work in that it can result from injury. In any specific case, it must be differentiated from epilepsy resulting from other causes. In addition, it is an unusual entity in that it may develop long after the injury. Therefore the probability or improbability of such a complication may be an important medicolegal consideration. This paper analyzes many of these factors and illustrates how present knowledge can be better used in the legal handling of such problems.

"It is a capital mistake to theorize before one has data. Insensibly one begins to twist facts to suit theories, instead of theories to suit facts."—Sherlock Holmes.

COURTS TODAY are more and more called upon to render decisions based on the relation of legal liability to injury or disease. Dean Pound¹ has stated of this, that: "The conditions of today call for planned and orderly cooperation of the lawyer and man of science in doing systematically for types of questions what has been done unsystematically and often blunderingly for each case as it arose."

Previous articles² have commented on various aspects of the relation between epilepsy and the law. From the standpoint of the lawyer, the most important problem in this area is the

* B.S., Franklin and Marshall College; M.D., Jefferson Medical College; Diplomate in Psychiatry, American Board of Neurology and Psychiatry; Clinical Director, Cleveland Regional Treatment Center; Second year law student at Cleveland-Marshall Law School.

[*Editor's Note:* This is a sequel to an article entitled "Epilepsy and the Law" which appeared in 7 Clev.-Mar. L. R. (2) 280 (May 1958).]

¹ Pound, R., Forward, Symposium on Scientific Proof and Relations of Law and Medicine. 1 Clinics 1350 (April, 1943).

² (a) Barrow, R. L. and Fabing, H. D., Epilepsy and The Law, Paul B. Hoeber, Inc., N. Y. (1956).

(b) Fabing, H. D. and Barrow, R. L., Medical Discovery as a Legal Catalyst: Modernization of Epilepsy Laws to Reflect Medical Progress. 50 NW. U. L. R. 42 (1955-6).

(c) Fabing, H. D. and Barrow, R. L., Medical Progress in Treating Epilepsy and the Need for Reform of Laws Affecting Epileptics. 3 Epilepsia 92 (Nov., 1954).

(d) Friedman, G. A., Epilepsy and The Law. 84 Med. Times 1359 (Dec., 1956).

(e) Perr, I. N., Epilepsy and The Law. 7 Clev.-Mar. L. R. 280 (May, 1958).

(f) Smith, H. W., Medico-Legal Facets of Epilepsy. 31 Tex. L. R. 765 (1953).

correlation of legal liability, injury and epilepsy. This paper covers the following questions—(1) What is the relation between injury and epilepsy, (2) How is post-traumatic epilepsy recognized and differentiated, (3) What are some of the features of the course of post-traumatic epilepsy, and (4) How does one estimate the likelihood of post-traumatic epilepsy developing following a head injury? It will be seen from the data in this article that there is no clear-cut answer to any of these questions. Nonetheless a vast amount of information has been accumulated, and it is possible to reach many general conclusions. Although this information is of vital importance to lawyers in personal injury work, this article by its nature necessitates an almost purely medical exposition. It is hoped that this will make it more useful to the lawyer than a mere rehashing of case decisions.

Before delving into the medical aspects of the problem, a few comments regarding injury and disease are in order. Following this, examples will be given of some legal cases in which the problems discussed have been a principal topic of consideration.

Our primary concern here is how to utilize existing knowledge, within present limitations, in order to achieve the highest standard of justice. Hamby,³ in discussing this problem, said: "In cases of injury to the head, obviously the physician's primary duty is medical and not legal. In a culture less socialized than our own, this duty is more easily discharged than here. Now the law so jealously encloses us that there are few opportunities for man to be injured 'on his own,' so to speak. After awakening in the morning, a person injured in his own house may legally claim compensation, for trauma even of his own making, from the owner of the house or from the carrier of his personal liability insurance. Spared this possibility, he departs for work in the car owned and insured by himself or jointly with a finance company. . . .

"When the mishap finally occurs, the patient often finds himself obliged to seek legal aid and to obtain the financial coverage he had so fondly imagined earlier was his by right of purchase, of employment, or by virtue of citizenship in the modern state. He now suddenly plunges into a bewildering pool, the currents of which he only vaguely may have suspected earlier. To stay

³ Hamby, W. B., *Medicolegal Aspects of Head Injuries*. 56 *New York State J. M.* 1253.

in business, his insurers attempt to minimize his complaints and thereby his compensation. To counteract this tendency, his lawyers inflate the estimate of his damage. Members of the jury bend toward fat or lean settlements according to their generosity with other people's money or to their cynicism. The doctor's dilemma lies in giving testimony based on medical fact in the midst of frank and frantic partisanship."

An example of the attitude of a prominent plaintiff's attorney is in this statement by Belli⁴: "Traumatic epilepsy may not show itself for as much as 18 years after the damage to the brain. One time the author (Belli) was presented with a case of a 3 month old child, that had been placed in a hospital for surgery on a cleft palate. The day after the operation, unaccountably, the child was seen in its bed with a stellate (star) skull fracture. The recovery was uneventful. A year later the most complete examination revealed not a sign of brain damage. Should settlement be postponed for some 21 years until majority is reached, when there would probably be no chance of sequela (although there have been cases that manifested themselves years later)?"

Of course, the inferences raised concerning the cause of the injury are not relevant to this article. The question of epilepsy developing at a late date from a head injury is relevant, and some answers will be given later in this paper.

A different aspect of this problem, which is most important, was reported by Hyslop⁵, who made a specific study of 750 head injury cases involving litigation. Of these 65 (8.6%) raised the possibility of post-traumatic epilepsy. In 13 (20%) he found focal brain damage and verified seizures. In two of the cases, the seizures occurred in the first six days following injury, with no further attacks (these then should not be considered as "epilepsy"). The other 11 cases all developed within 26 months following the injury. The remaining 52 cases were studied quite carefully; all claimed a head injury at least of a concussion nature. In 12 cases, investigation revealed no injury at all; in three cases there had been merely a laceration of the scalp, with "careful coaching by the claimant's lawyers" (Hyslop's words)

⁴ Belli, M. M., 1 *Modern Trials* 569, Bobbs-Merrill Co., Inc., Indianapolis, 1954.

⁵ Hyslop, G. H., *Seizures, Head Injuries, and Litigants*. 31 *J. Indust. Hyg. and Toxicol.* 336 (1949).

to bring in the question of epilepsy. In 18 cases the injury occurred as the result of a seizure; of these seven admitted previous attacks and five others were later shown to have had previous epilepsy (which had been diagnosed prior to the alleged injury). Six cases were those in which the individual had developed idiopathic epilepsy without an antecedent injury. Four cases had had previous head injuries which at first had been denied. In not one of the cases of focal brain injury was there an attempted fraud; of the other 52, 28 (54%) showed malingering or fraud with respect to the character of the injury or its effects. Thus, in his study, the number of frankly fraudulent cases outnumbered the true cases of post-traumatic epilepsy by more than two to one. No other similar studies of the subject have been found (making evaluation of this report difficult), but this information does point out some of the difficulties inherent in the evaluation of this type of case.

Turning to cases, some of the features of several will be presented in order to illustrate how this problem becomes manifest in the courts. From the medical standpoint, there is little opportunity for comment, inasmuch as insufficient medical data are given. The cases are those in which post-traumatic epilepsy developed; those in which there has been a severe brain injury with other neurological defects demonstrated, but with no proof of epilepsy; and those in which there is no defect from the injury but in which there is a possibility of later epilepsy. These are extremely important matters for two main reasons. One, epilepsy is a very unpleasant disease and a great handicap, as has been indicated previously.⁶ Two, epilepsy in general is considered by laymen, to be a most revolting disease, and thus compensatory awards tend to be high.

In *Kuemmel v. Vradenburg*,⁷ the claimant had a depressed skull fracture requiring surgery in order to remove bone and dirt from the brain, with resultant neurologic damage. The medical expert testified that the patient was likely to have a spastic paralysis and that such injuries are "likely to cause people to have convulsions or epileptic fits." In a New York case,⁸ the plaintiff was hit by a falling rock at a state park, sustaining a deep penetrating compound fracture. One of the results was

⁶ See n. 2(e) above.

⁷ *Kuemmel v. Vradenburg*, 239 S. W. 2d 869 (Tex., 1951).

⁸ *Dakin v. State*, 130 N. Y. S. 2d 39, 288 (1954).

post-traumatic epilepsy. The higher court ruled that an award of \$72,867.28 was not excessive. In *Nagala v. Warsing*,⁹ a boy, three years ten months old suffered a fractured skull, and was unconscious 13 days. There was severe damage to the brain, with brain tissue extruding from the wound. Among the elements considered in reaching an award were the possibilities of epilepsy and of personality problems developing. In this case no specialist in neurology testified. However, there was no question of damage, with both eye difficulties and poor coordination on the right side of the body already present.

A common problem was that present in *Cochran v. Wimmer*¹⁰, where there was no external evidence of a physical injury but where the claimant developed epilepsy four days after the alleged injury. The court ruled that the problem was to decide whether this was traumatic or idiopathic epilepsy, and that this was a question of fact and thus one for the jury. In *Thompson v. Anderman*,¹¹ the court ruled that an award of \$54,000 was not excessive where there was a fracture through the base of the skull with concussion and contusion in the brain stem, hemorrhage from the left ear and profuse bleeding, and in view of the disability, pain and suffering and the possibility of developing epileptic seizures, as well as his age and life expectancy. This was a boy of 13 with a mentality of 10. All three doctors agreed on a possibility of epilepsy, but did not feel that it was likely in view of the period of time since the injury; one stating "we are always leery about a seizure developing three years after the injury." In *Bartholomew v. Impastato*,¹² the court ruled that opinions voiced by two physicians, as to mental injuries which might result from cerebral injuries sustained by a three year old child due to fault, were too speculative to warrant an award of damages based on the conclusion that the injuries were permanent.

In an Indiana case¹³ there was an award of \$15,000 for a skull fracture and lacerations sustained by a six year old child "who would probably suffer in later years from convulsive disorder." Testimony of a doctor as to the probability of focal epilepsy resulting from injury was admissible. In this case there

⁹ *Nagala v. Warsing*, 219 P. 2d 603 (Wash., 1950).

¹⁰ *Cochran v. Wimmer*, 81 N. E. 2d 790 (Ind., 1948).

¹¹ *Thompson v. Anderman*, 285 P. 2d 507 (N. M., 1955).

¹² *Bartholomew v. Impastato*, 12 S. 2d 700 (La., 1943).

¹³ *Fort Wayne Transit v. Shomo*, 143 N. E. 2d 431 (1957).

was intracranial bleeding, and surgical intervention was needed in order to remove bony fragments from the brain. A small metal disc was placed in the skull and the boy was hospitalized a total of eight days. The EEG suggested a focal convulsive disorder in the left temporal area "probably with some grand mal component." The two year time interval indicated permanent brain damage, and the bony fragments must have bruised the underlying brain and resulted in scar tissue. Testimony stated that "during the war 50-75% of those receiving such wounds developed convulsive disorder" and that "75% of those with an abnormality like that of the appellee, resulting from injury, will sometime develop focal epilepsy." These figures can be compared with others later in the paper.

An interesting case was that of *Melendez v. N. Y. C. Omnibus Corp.*¹⁴ where a 46 year old cook, alighting from a bus, suffered a skull fracture and direct brain damage, with resultant traumatic epilepsy, post-traumatic psychosis, right sided paralysis, and aphasia. Here the court reduced the award when it was shown that the plaintiff had not worked for 23 months prior to the injury and that he had a history of psychoneurosis for which he had previously been discharged from the army.

The preceding paragraphs bring into focus how important the problem of post-traumatic epilepsy is in the law. The remainder of this paper will deal with the medical aspects which must be the basis of evaluation in any such case.

Frequency of Post-traumatic Epilepsy as Compared with Other Types of Epilepsy

As indicated in a previous case,¹⁵ when epilepsy develops, the type and cause must be ascertained. Post-traumatic epilepsy is compensable; idiopathic epilepsy and other types are not. From the standpoint of statistical incidence, post-traumatic epilepsy is a relatively insignificant type of epilepsy. In general, idiopathic epilepsy constitutes about 78% of epilepsy while post-traumatic epilepsy is found in about four to five percent of cases; which means that idiopathic epilepsy is found fifteen to twenty times more commonly.

The following Table illustrates this incidence in two studies.

¹⁴ *Melendez v. N. Y. C. Omnibus Corp.*, 139 N. Y. S. 2d 788 (1955).

¹⁵ See n. 10 above.

TABLE

Recurrent Convulsive Seizures in 2000 non-institutional cases of epilepsy at all ages:

Presumed causes of seizures	(after Lennox ¹⁶)
Idiopathic epilepsy	77.6%
Cerebral trauma	5.7
Birth injury or congenital defect	5.6
Brain infection	4.2
Brain tumor	2.6
Cerebral circulatory defect	1.9
Extracerebral causes	0.9

Analysis of 689 Patients whose attacks began after 20 years of age:

	(after Livingston ¹⁷)
	Number
Idiopathic epilepsy (78%)	527
Hypertension or cerebral arteriosclerotic	83
Alcohol	28
Post-traumatic (2.5%)	17
Neurosyphilis	11
Pregnancy	8
Cerebral birth trauma	4
Brain abscess	2
Brain tumor	2
Cysticercosis	1

Another large report from the Montreal Neurological Institute¹⁸ showed that in 2000 cases of epilepsy, only 86 (4.3%) were post-traumatic in origin. Thus, when the physician is presented with a case of epilepsy, he must make a careful study in order to rule out all other factors, especially since clarification may be life-saving, as in the case of a brain tumor.

Some Pathologic Factors

In order to clarify the reports that follow, some basic concepts may be useful. Epilepsy is a disease of the brain; in order for post-traumatic epilepsy to develop there must be an injury to the brain. "Laceration of the brain is an essential factor—

¹⁶ Lennox, W. G., *Epilepsy and The Epileptic*. 162 J. A. M. A. 118 (Sept. 8, 1956).

¹⁷ Livingston, S., *Etiologic Factors in Adult Convulsions*. 254 New Engl. J. Med. 1211 (June 28, 1956).

¹⁸ Jasper, H. and Penfield, W., *Electroencephalograms in Post-traumatic Epilepsy; Preoperative and Post-operative Studies*. 100 Am. J. Psychiat. 365 (Nov., 1943).

whether or not there is injury to the skull.”¹⁹ Injuries to the scalp and even to the skull may be totally irrelevant to the development of epilepsy. The brain is protected from injury by the scalp, the skull (which is a spherical container well designed to dissipate force), and the dura mater (a hard fibrous layer beneath the skull). Inside this layer, the brain more or less floats in a sea of cerebrospinal fluid. Injury to the brain can occur in one of two ways—the force of the blow can jar the brain (for example, the tips of the temporal poles may be traumatized as in *contre coup* injury) or by some direct injury in the area of the blow. This can occur, for example, when a piece of bone or a foreign body such as a bullet, penetrates directly into the skull. It will be seen that the type of injury is most important, as this will help to determine the likelihood of epilepsy. In general, head injuries can be categorized into two main types. In the first, there is a blow to the head with laceration, concussion, or even fracture with no depression of the broken bone. In the second, there is fracture with direct injury to the brain beneath, which means penetration of the dura mater into the brain substance. Factors such as bleeding may not be relevant if the hematoma forms a mass outside the brain tissue itself.

The basis of epilepsy is destruction of brain tissue itself, with formation of a scar surrounded by irritable brain tissue from which the abnormal electrical discharge spreads.

Incidence of Post-traumatic Epilepsy Following Head Injury

Analyzing this problem requires many distinctions to be made (1) between war and civilian studies and (2) between closed and penetrating injuries, as well as careful analysis in order to see what each study is reporting, since few are comparable. This is most important, as otherwise one can indiscriminately pick statistics to support any given point of view, and thus make any scientific attempt at evaluation ludicrous. For example, one might say that the incidence of epilepsy following head injury is 0.1% to 50%; such a statement, however, conveys absolutely nothing. Defendant's attorneys can show that the likelihood of epilepsy following head injury is less likely than in the absence of any injury, while plaintiff's attorneys can quote

¹⁹ Denny-Brown, D., *Clinical Aspects of Traumatic Epilepsy*. 100 *Am. J. Psychiat.* 585 (1944).

statistics indicating almost the opposite. Both sets of statistics are true, except that they are reporting different things. Most of the balance of this paper will be a discussion of these differences, in order to indicate how, in a given situation, the most reasonable probabilities can be obtained.

Among the most commonly quoted articles on the subject are a series on head injuries from World War I and World War II. Different studies here report an incidence of from 1.5% to 49%. In 18,000 gunshot wounds of the head, the British Ministry of Pensions reported an incidence of 800 epileptics (4.5%). Some French figures were 12.1%, German figures, 44% (of 562 cases). Other studies report 27%, 49.5% (of 1234 cases), 45%, and 43% (of 820 cases). Others report two, seven, six percent, etc. The percentage of epilepsy in the population at large is estimated at 0.5%. On the other hand, in the largest single study ever reported, by Feinberg in Switzerland—of the civilian population in a 14 year period of 1919 to 1933—there were only 50 cases of traumatic epilepsy in 47,130 head injuries, an incidence of 0.12%, or less than that of the general population.

Why is it then, that the war studies report very high figures in comparison to studies of civil injuries? Let us look at a well-known study—that of Ascroft.²⁰ In 1939 he reported on 317 cases from World War I. Of these, 34% had seizures. In cases where there was penetration of the dura mater, the incidence was 45%; when there was no penetration, the incidence was 23%. These figures are about the highest of any reported, and there are many factors which are responsible. First, a large number of persons were included who had seizures immediately following the injury (even if only one seizure) without recurrence (today these would not be considered as “epilepsy”). Secondly, many cases were excluded because of insufficient data. Many minor injuries were not included, and neither were cases where the damage was to the cerebellar area of the brain. The group in general was one in which the members were severely injured, and these statistics are probably maximal. Another very important factor was that these injuries were caused by high velocity missiles, which caused great brain damage in contrast to the type of injury received in civil life by the usual type of injury, from a blunt instrument.

²⁰ Ascroft, P. B., Traumatic Epilepsy after Gunshot Wounds of the Head. 1 (1941) *Brit. Med. J.* 739.

The differences between war and civil injuries have been commented on by many. Siris²¹ reports: "Among the ways (in) which head injuries of war differ from those of civil life is in the incidence of subsequent epilepsy. . . . The development of this condition following all types of civil head wounds is considerably lower, running in general between 0.5 and 2 per cent, not much higher than the incidence of epilepsy in the population at large." Other such comments are quoted below.²²

In another war study²³ of 200 cases of severe brain injury, caused by penetration of the dura mater by artillery shell fragments and rifle bullets, the incidence of epilepsy was 16.5% (33 cases). A European study²⁴ showed a less than two percent incidence where the dura was not penetrated, compared with a 27% incidence where there was penetration with brain damage. Another English study²⁵ of 820 cases of penetrating brain wounds reported an incidence of 43%. However, here again one runs into that fact that one seizure meant classification as an epileptic—a concept which will be discussed below. Others²⁶ were 44%

²¹ Siris, J. H., *The Management of Post-Traumatic Epilepsy*. 42 U. S. Nav. M. Bull. 144 (Jan., 1944).

²² (a) Sachs, E., *Two Important Postwar Problems in Neurologic Surgery*. 101 J. Nerv. and Ment. Dis. 460 (May, 1945).

"It is very striking that the incidence of epilepsy following fractures of the skull in civil life is far less frequent than in war wounds, and, of course, one obvious difference is that in the war cases, compound fractures are much more common." He also stresses the difference between missiles and blunt injuries in causation.

(b) Garland, H. G. and Walter, W. G., *Traumatic Epilepsy: discussion*. 35 Proc. Roy. Soc. Med. 773 (Oct., 1942).

"Suffice it to say that the highest incidence claimed is 20% and the lowest a good deal less than that in the general population; and that it is at least very probable that the first figure relates to a selected group of severe head injuries and the second is diluted with many trivial cases."

In civilian head injury, epilepsy occurs in about 3% (excluding simple concussion which would lower the number).

²³ Maltby, G. L., *Penetrating Craniocerebral Injuries; evaluation of late results in a group of 200 consecutive penetrating cranial war wounds*. 3 J. Neurosurg. 239 (1946).

²⁴ Wagstaffe, W. W., *The Incidence of Traumatic Epilepsy after Gunshot Wounds of the Head*. 2 Lancet 861 (1928).

²⁵ Russell, W. R. and Whitty, C. W. M., *Studies in Traumatic Epilepsy. 1. Factors influencing the incidence of epilepsy after brain wounds*. 15 J. Neurol. Neurosurg. and Psychiat. 93 (1952).

²⁶ (a) Baumm, H., *Erfahrungen uber Epilepsie bei Hirnverletzten*. 130 Ztschr. f. d. ges. Neurol. u. Psychiat. 279 (1927).

(b) Credner, L., *Klinische und sociale Auswirkungen von Hirnschadigungen*, 126 Ztschr. f. d. ges. Neurol. u. Psychiat. 721 (1930).

and 49.5%. Another military figure²⁷ in 279 American cases was 36.2% within two years.

Seizures following lobotomy (which is a direct injury to the brain) occur in 25.6%; 60% of which are controlled by medication.²⁸ Bickers²⁹ reports the incidence of open head injuries as 5.5 to 20%, and closed head injuries as 2.5% with seizures after simple concussion almost unknown.

Most reports concerning closed head injuries give an incidence of two to six percent.³⁰ Military reports indicate a higher incidence, but here again one sees the results of high velocity missiles causing injury.

In general, reports from civilian injury run at less than one-third the rate reported from military wounds.

One of the most important reports is that of Penfield and Shaver³¹, who found the following:

<u>Type</u>	<u>Total Cases</u>	<u>No.</u>	<u>Epilepsy %</u>
Scalp Wounds without fracture	193	1	0.5%
Concussion, contusion, or compression above	40	0	0
Fracture without proven dural tear (including subarachnoid hemorrhage)	136	7	5.1
Fracture with dural tear	38	3	7.9

Most important was the finding that in 126 brain concussions there were no cases of post-traumatic epilepsy—a finding which has been supported elsewhere. Here the total incidence was less than 2.5% (eleven in 407 injuries).

The larger the series of cases, the lower the incidence reported; the largest series ever reported being that of Feinberg³², where the incidence was 0.12% in over 47,000 cases. Of these,

²⁷ Watson, C. W., The Incidence of Epilepsy following Craniocerebral Injury. 26 *A. Res. Nerv. and Ment. Dis.* 516 (1947).

²⁸ Freeman, W., Lobotomy and Epilepsy; a Study of 1000 Patients. 3 *Neurology* 479 (1953).

²⁹ Bickers, D. S., The Medicolegal Aspects of Head Injury. 44 *J. M. Assn. Georgia* 431 (Sept., 1955).

³⁰ Phillips, G., Traumatic Epilepsy after Closed Head Injury. 17 *J. Neurol. Neurosurg.* 1 (Feb., 1954).

³¹ Penfield, W. P. and Shaver, M. S., The Incidence of Traumatic Epilepsy and Headache after Head Injury in Civil Practice. 24 *Res. Publ. Ass. Nerv. Ment. Dis.* 620 (1945).

³² Feinberg, P., *Epilepsie und Trauma*. Inaugural Dissertation. U. Zurich. Uznach, 1934, publ. by K. Oberholzer.

Denny-Brown³³ says: "The series of Feinberg is therefore by far the largest unselected group of civil head injury, and the best figure we have at present for such a group." He reports a rate of five per 1000 with fractures (0.5% or the same as that in the general population).

Question of Fracture, Hemorrhage, and Other Related Injuries

One pertinent question is "what is the likelihood of epilepsy following a fracture that does not involve the pushing of bone into the brain?" Denny-Brown³⁴ states: "It may be noted. . . how clearly the figures show that fracture of the skull is without importance in the question of epilepsy." Penfield³⁵ comments: "Closed injury to the skull, regardless of its severity, rarely results in post-traumatic epilepsy. . . . The likelihood of epilepsy is greatly increased in case the dura has been penetrated and the brain lacerated by fragments of depressed bone or missile. This is apparently quite independent of the severity of the cerebral concussion and intracranial hemorrhage which may have attended the injury." Epilepsy is rarely found after subdural hematoma, meningitis, thrombophlebitis, thrombosis, etc. He³⁶ further states: "Brain laceration more often causes seizures than cerebral contusion or closure of a cerebral vessel. Subdural hematoma and internal hydrocephalus never do unless some other local complication is present."

These factors can be summed up in the following statement:³⁷

"Depression of an area of bone in the cranial vault is not necessarily a severe or dangerous happening. . . . The important feature is whether or not the dural lining of the skull is torn by a sharp edge of bone jutting inwards. . . a simple fissure in the vault of the skull is not of itself harmful. . . . The cases of war injury demonstrate that fracture per se is not of any real moment in this question. . . . It must be remembered that the cause of epilepsy is damage to the brain."

³³ See n. 19 above.

³⁴ *Ibid.*

³⁵ Penfield, W., *Post-traumatic Epilepsy*. 100 *Am. J. Psychiat.* 750 (1944).

³⁶ Penfield, W., *Epileptogenic Lesions*. 56 *Acta Neurol. et Psychiat. Belg.* 75 (Feb., 1956).

³⁷ Denny-Brown, D., *Symposium on Scientific Proof and Relation of Law and Medicine—Factors of Importance in Head Injury—A General Survey*. 1 *Clinics* 1405 (April, 1943).

What Is the Meaning of a Convulsion Soon After Injury?

As pointed out in an earlier paper³⁸, an essential element of epilepsy is its recurrent or periodic nature. Since there are many other causes of convulsions, this element must be found in addition to its other characteristics. An epileptiform attack immediately following an injury does not necessarily denote epilepsy. Denny-Brown³⁹ states: "It should be at least considered whether early convulsions deserve the name traumatic epilepsy or should the term 'immediate traumatic epilepsy' be given some special annotation. There are cases where the diagnosis of epilepsy was made on a single convulsion in the early stage of severe head injury, without subsequent disability, and where diagnosis interfered with subsequent employment. . . . Because a drug, or electric shock, or anoxia, will provoke a convulsion, it cannot be maintained that 'epilepsy' is thereby produced." Thus convulsions immediately following the injury may indicate only a temporary response to an injury. As such, they usually disappear. In contrast, the basic pathology behind post-traumatic epilepsy is scar formation which usually takes months to develop.

Other comments are given below.⁴⁰

³⁸ See n. 2(e) above.

³⁹ See n. 19 above.

⁴⁰ (a) Ascroft, P. B., see n. 20 above.

If seizures develop in the first week, they probably will be transient.

(b) Marsh, C., *Post-traumatic Epilepsy; Pathogenesis and Treatment*. 9 Bull. Los Angeles Neurol. Soc. 79 (March, 1944).

Not every case of convulsive disorder which follows an injury to the head is necessarily a bona fide case of post-traumatic epilepsy.

(c) Cavins, H., *Head Injuries in War, with Especial Reference to Gun-shot Wounds*. 2 War Med. 772 (Sept., 1942).

Ascroft's investigation appears to show that the fits which occur in the first two weeks after injury and operation do not predispose to epilepsy at a later date. This is in agreement with the experiences of patients who have fits in the first days after subarachnoid hemorrhage or after the removal of brain tumors.

(d) Watson, C. W., see n. 27 above.

Penfield, in discussing the article, states: "In Ascroft's figures of 45 per cent incidence of epilepsy after injury, he included the patients who had seizures during the first two or three weeks after brain injury. But only 20% of those patients will become chronic epileptics who have recurring seizures. The percentage is thus too high."

Very pertinent are some comments by Walker,⁴¹ who has written a great deal on the subject of post-traumatic epilepsy. He states⁴²: "Paroxysmal alterations in the state of consciousness very commonly follow a head injury. Even shortly after a blow producing only a momentary loss of consciousness, the victim is likely to feel dizzy and lightheaded and to black out when he assumes an erect position. These minor lapses are generally considered as due to nervous instability producing a temporary cerebral ischemia." He further comments⁴³: "Some members of the legal profession . . . imply that a few dizzy spells or momentary blackouts after a head injury and an abnormal electroencephalographic finding are sufficient to establish the diagnosis of epilepsy, with all the stigmas attached to the 'falling sickness,' and who, on this basis, ask a large award to compensate their 'epileptic' clients for the recurrent seizures that will mar his or her future. Such a contention is obviously false since neither these clinical manifestations nor abnormal brain waves are adequate for the diagnosis of a convulsive disorder per se."

Importance of Heredity in the Development of Post-traumatic Epilepsy

Comments have been made that predisposed individuals develop post-traumatic epilepsy, and that constitutional factors exclusive of the injury should be considered in discussing causation. Defense attorneys may try to magnify this point. However, the bulk of present evidence indicates that such a position is not tenable. There is little agreement amongst medical authorities on this point. Many specific studies indicate that this is not a

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- 41 (a) Walker, A. E., *Post-traumatic Epilepsy*. C. C. Thomas, Publ. Springfield, Ill. (1949).
 - (b) Walker, A. E., *Medicolegal Aspects of Head Injuries*. 24 *Postgrad. Med. A-34* (1958).
 - (c) Walker, A. E., *Prognosis in Post-traumatic Epilepsy*. 164 *J. A. M. A.* 1636 (Aug. 10, 1957).
 - (d) Walker, A. E., Caveness, W. F., Barrow, R. L., and Allen, W. S., *Medico-legal Aspects of Head Injuries*. 2 *Clinic. Neurosurg.* 55 (1955).
 - (e) Walker, A. E., *The Natural History of Post-traumatic Epilepsy*. 81 *Tr. Am. Neurol. Assn.* 37 (1956).
 - (f) Walker, A. E., and Quadfasel, F. A., *Follow-up Report on Series of Post-traumatic Epileptics*. 104 *Am. J. Psychiat.* 781 (1948).
 - (g) Quadfasel, F. A. and Walker, A. E., *Problems in an Army General Hospital*. 26 *A. Res. Nerv. and Ment. Dis., Proc.* 461 (1947).

⁴² See n. 41(b) above.

⁴³ See n. 41(c) above.

factor. For instance, one study⁴⁴ reports that families of post-traumatic epilepsy show a 4.5% incidence of seizures compared to 3.4% in normals and 17% in families of all epileptics. That there is a familial disposition in idiopathic epileptics is well verified, as is shown above. Slater⁴⁵ states: "Some degree of inherent susceptibility may be present in persons who suffer 'traumatic' epilepsy." Walker⁴⁶ and Siris⁴⁷ discuss this theory. Expressing the contrary view, Phillips⁴⁸ states: "There is no reason to suppose that the subject of cranial trauma is more likely to suffer a fit if he has a . . . family history of epilepsy. . . ." Others agree with this viewpoint.⁴⁹ Thus at present not enough is known regarding this subject, and for the present this factor would not be relevant in a legal proceeding.

Time Interval Between the Injury and the Development of Post-traumatic Epilepsy

This subject is extremely important to the lawyer, as the incidence of epilepsy is related directly to the time interval following the injury. In a case where there has been an injury, a lapse of time, and no development of epilepsy, the lawyer is most concerned over the reasonable or probable likelihood of such a complication developing. The claimant's attorney will try to include this as an element of damages, if possible (which well he should). One way of doing so is to quote a few cases in which the development following the injury occurred fifteen or twenty years later, and to point out the necessity for settlement based on this possibility (see the earlier statement by Belli). Fortunately this factor can be reasonably well evaluated.

Cases where epilepsy develops many years after an injury are relatively rare. Mann⁵⁰ reports a case where there was a 24 year interval between injury and onset of seizures. He published

⁴⁴ See n. 41(g) above.

⁴⁵ Slater, J. K., *Trauma and the Nervous System*. 53 *Edinburgh Med. J.* 623 (1946).

⁴⁶ See n. 41(a) above.

⁴⁷ See n. 21 above.

He states: "Apart from the extracerebral factor, it appears probable that an underlying epileptic predisposition or diathesis is necessary for the development of the disease."

⁴⁸ See n. 30 above.

⁴⁹ See n. 27, 40(b) above.

⁵⁰ Mann, L. B., Jr., *Post-traumatic Epilepsy; report of a case with 24 year interval between injury and onset of seizures*. 14 *Bull. Los Angeles Neurol. Soc.* 187 (1949).

this paper in 1949, and found after a review of the literature only five cases where the epilepsy developed subsequent to ten years. His case was unusual in that a three year old girl had been kicked in the head by a horse, and had a depressed skull fracture which was never relieved surgically, so that one could feel the hole in her head. In this case, surgical removal many years later cured the epilepsy.

Such cases are so rare as to be meaningless statistically, and are useful only for their theatrical value.

Let us then turn to various studies which analyze this specific problem. Phillips⁵¹ in a study of 190 cases developing after closed head injuries showed that the epilepsy developed in the first three months in 104 (55%), by one year in 156 (82%), by two years in 162 (85%), that 23 or 12% developed in two to four years, and five (or less than 3%) developed in four to 11 years. In another series⁵² where epilepsy developed in 53 of 630 head injuries, epilepsy developed in one month in 22 (42%), in one to six months in 16 (30%), in six to 12 months in eight (15%), one to two years in no cases, and more than two years in seven (13%). In an Army series⁵³, 27% developed within three months and 58% by six months. The vast majority are reported, in most studies, to develop within 18 months. Walker⁵⁴ also states that 50% develop within nine months and, of those in whom epilepsy develops within five years, 80% have the initial seizure within two years. Jasper and Penfield⁵⁵ report an incidence of 46% in the first year, 63% in three years, 80% in five years.

Thus, between 55% and 85% of cases develop in the first two years. Accordingly they may be brought into the trial proceedings as an existing complication, rather than as a potential one.

Other Factors Concerning Type of Injury

In addition to the force of the injury and the type of injury, other factors play a role. If the brain is divided, going from front to back, into (1) Frontal (2) Temporal (3) Parietal, and (4) Occipital areas, one finds a difference in incidence of epilepsy. In-

⁵¹ See n. 30 above.

⁵² See n. 19 above.

⁵³ See n. 41(g) above.

⁵⁴ See n. 41(b) above.

⁵⁵ Jasper, H. and Penfield, W., *Electroencephalograms in Post-Traumatic Epilepsy; preoperative and post-operative studies*. 100 *Am. J. Psychiat.* 365 (Nov., 1943).

juries to the motor area (parietal) will give the highest incidence of epilepsy. However, these do not differ so greatly from the injuries to the frontal and temporal area as to have great statistical significance, and so various reports on this will not be described. Injuries to the occipital area or the midbrain, however, are not characterized by epilepsy. Russell and Whitty⁵⁶ comment on this distribution, as do many others such as Ascroft.⁵⁷ Whether or not the presence of pieces of bone or metal embedded in the brain play a role is another pertinent question. Statistics here indicate that this factor is not especially relevant to the incidence of epilepsy; the reason being that large foreign bodies are usually removed surgically, and that the ones left do not seem to play a very important role. Where there is infection of the brain, the incidence seems to be higher. Early surgery does not seem to lower the incidence. Some report that the incidence is higher where there is a prolonged period of post-traumatic amnesia (PTA). For instance, in one series⁵⁸ of 38 cases, there was a PTA of more than three hours in 28, under three hours in eight, and under one-half hour in two. No definite statement can be made at this point as to the importance of unconsciousness (it has been reported that 23 to 36% showed no unconsciousness at the time of injury).

As mentioned, the incidence is directly related to damage in the brain, and one would expect on neurologic examination to find evidence of brain damage. In a well studied series of Army cases⁵⁹, 94.3% showed neurological damage. Only 14 of 246 cases showed no abnormality on neurological examination. On the other hand, the presence of severe head injury does not mean that epilepsy will develop. One study⁶⁰ mentions a head injury group, with no convulsions, that was characterized by greater injuries than the cases which developed post-traumatic epilepsy.

⁵⁶ Russell, W. R. and Whitty, C. W. M., Studies in Traumatic Epilepsy.

1. Factors influencing the incidence of epilepsy after brain wounds. 15 *J. Neurol. Neurosurg. and Psychiat.* 93 (1952).
2. Focal Motor and Somatic Sensory Fits. 16 *J. Neurol. Neurosurg. and Psychiat.* 73 (1953).
3. Visual Fits. 18 *J. Neurol. Neurosurg. and Psychiat.* 79 (1955).

⁵⁷ See n. 20 above.

⁵⁸ See n. 22(b) above.

⁵⁹ See n. 41(g) above.

⁶⁰ Gibbs, F. A., Wagner, W. R., and Gibbs, E. L., Electroencephalogram of Traumatic Epilepsy. 100 *Am. J. Psychiat.* 738 (May, 1944).

Some Features of Post-traumatic Epilepsy

Two features of post-traumatic epilepsy should be mentioned—(1) often the course is quite mild, and (2) often the condition disappears completely. In 207 cases,⁶¹ less than one-half had more than two attacks of any type per year. In major attacks, only 30% had more than two seizures a year, and of the group studied 47% had had no attacks for two years, 35.6% had no attacks in the period from the fifth to the tenth year after the injury (this study was a 10 year follow-up), and 14.6% only one or two attacks a year in the last five years of the period. If in the first five years, seizures cease for a year, the chances are four out of five that there will be no seizures in the next five to eight years. If there is a cessation of attacks for two years, the chance of recurrence is only two in 100. Probably 40% of those with seizures in the first few weeks will have no further attacks.

The greater the neurologic deficit, the greater the disability from such other factors as post-traumatic psychosis or neurosis, and the lower the basic intelligence—the more likely is the individual to be handicapped in his future adjustment. These factors seem to play a greater role than the epilepsy or even paralysis alone.

Post-traumatic Epilepsy and the EEG

Electroencephalography is a science in itself. Suffice it to say that it is obviously of importance in attempting to support a diagnosis of post-traumatic epilepsy and in estimating the likelihood of its developing. The other question of importance is the potential use of the EEG in helping to differentiate essential from post-traumatic epilepsy. This section is concerned only with these factors, but in view of the intricacies of the EEG, comments will be most generalized.

As was mentioned in a previous article⁶², the essence of epilepsy is in its recurrent seizures. The EEG does not relate well with this problem, depending on the types of findings. It must be kept in mind that there are many kinds of findings on the EEG, that so-called abnormal patterns are found in many

⁶¹ See n. 41(b), 41(c) above.

⁶² See n. 2(e).

conditions, and that there is considerable deviation on normal subjects. Therefore, Penfield⁶³ says: "We should agree immediately that dysrhythmia is not epilepsy and that, particularly in cases of compensation, we should be very loath to let dysrhythmia or alteration in the EEG record influence us very much. The patient who is an epileptic should be defined only as a patient who has recurring seizures."

An essential point to remember is that the EEG will show various findings after head injury. These findings are so common as to be of basically no prognostic significance. In judging the statistics to follow, one must keep in mind the difference between generalized and focal abnormalities, and between slow irregular focal discharges and "spiky" focal discharges. Of the consistent slow wave focus on the EEG, Marsh⁶⁴ states that this does not prove that the patient has or will have post-traumatic epilepsy. "It signifies a focus of abnormal cellular activity which, in the majority of cases of craniocerebral injury, even of the penetrating type, does not result in convulsive seizures." Williams⁶⁵ states: "An abnormal EEG persisting after a head injury does not necessarily increase the likelihood of traumatic epilepsy, but the presence of episodic outbursts of abnormal waves does. . . . Immediately after a head injury, it is usual to find some gross abnormality characteristic of severe cerebral damage during the period of resolution (which) may mimic the picture of epilepsy, but which in a few weeks subsides with gradual reappearance of normal rhythms." Therefore EEG's are of not much help immediately following injury. As mentioned, there are many kinds of non-specific abnormalities which may be picked up on EEG. Of these, Williams states that "the presence of this kind of abnormality in patients with head injury does not seem to be closely related to the likelihood of traumatic epilepsy." In his series, he found larval epileptic outbursts in only nine percent, but that these were helpful in diagnosis as such findings occur three times more frequently than in idiopathic epilepsy. Paroxysmal outbursts are not very helpful, with similar rates of incidence in other head injuries.

⁶³ Penfield, W. P., in *Discussion of the Natural History of Post-traumatic Epilepsy*. 81 *Tr. Am. Neurol. Assoc.* 37 (1956).

⁶⁴ Marsh, C., *Post-traumatic Epilepsy; Pathogenesis and Treatment*. 9 *Bull. Los Angeles Neurol. Soc.* 79 (Mar., 1944).

⁶⁵ Williams, D., *Electroencephalogram in Traumatic Epilepsy*. 7 *J. Neurol. Neurosurg. and Psychiat.* 103 (July, 1944).

Walker⁶⁶ states: "Some years ago it was hoped that the EEG would be of diagnostic and prognostic importance in epilepsy. Experience has shown, however, that the brain waves may denote cerebral damage but do not reliably indicate or forecast convulsive complications."

In one prominent study⁶⁷, only eight percent of post-traumatic epilepsy had normal records, as compared with 53% in severe head injury, 84% in normals, and 15% in unselected epileptics. Another investigation⁶⁸ indicated localized findings in 90% of verified cases (either random spikes or sharp waves) and stated that it is "questionable whether the diffuse or bisynchronous disorders are truly of post-traumatic etiology One may assume the probabilities are greatest that they are essential (idiopathic) rather than post-traumatic epilepsy."

Since the article by Gibbs, Wagner and Gibbs⁶⁹ is such an important and classic study, some of the findings will be given in detail. In this study, comparisons were made of the EEG's of 125 cases of post-traumatic epilepsy, 215 cases of head injury without convulsions, 1161 other epileptics, and 1000 normal patients. After cautioning about the dangers inherent in generalization, the authors presented their findings based on this study.

⁶⁶ See n. 41(b) above.

Two useful articles on groups of patients with post-traumatic epilepsy are the following: Kaufman, I. C., Marshall, C. and Walker, A. E.

- (a) Activated Electroencephalography. 58 Arch. Neurol. and Psychiat. 533 (1949).
- (b) Metrazol Activated Electroencephalography. 26 A. Res. Nerv. and Ment. Dis., Proc. 476 (1947).

Here at least 88% showed some EEG abnormality; 78% showed focal abnormality.

⁶⁷ See n. 60 above.

⁶⁸ See n. 55 above.

⁶⁹ See n. 60 above.

To illustrate how severe brain injuries can be without epilepsy developing, all of the severe head injury cases *without convulsions* were unconscious at least an hour, 23% had brain laceration, 55% bloody spinal fluid, 5% depressed fracture, 21% compound fracture, 8% subdural hemorrhage, and 2% extradural hemorrhage. The authors point out that EEG's done immediately after head injury are of little use as at this time practically all patients demonstrate some findings—so EEG's were done 3 or more months subsequent to the injury. Here the incidence of abnormality and especially focal abnormality was much greater in the post-traumatic series. Children were more likely to show an EEG abnormality. While in the post-traumatic series, the incidence of abnormalities remained almost constant, in the head injury group they gradually declined over a two year period. Focal findings were four times as frequent in post-traumatic cases as in unselected epileptics, and focal naroxysmal findings were twenty-one times as common.

(1) Focal EEG abnormality is strongly suggestive of brain damage.

(2) Other things being equal, if generalized EEG abnormality is present three or more months after a mild head injury, the chances are 16 to one that the abnormality antedated the injury.

(3) In post-traumatic cases, even though the EEG is normal, the brain may be damaged (found in three cases of 160, or less than one in 50).

(4) If a paroxysmal abnormality is found three or more months after the injury, the chances are at least 27 to two that the patient has epilepsy.

(5) If a patient has seizures and shows focal paroxysmal abnormality three or more months after head injury, the chances are 21 to seven that he has the seizures as a result of the injury rather than as a result of the other known or unknown factors that produce seizures in an unselected group of epileptics.

(6) If a normal EEG is found three or more months after head injury, the chances are at least 53 to eight that the patient is not a post-traumatic epileptic.

Utilization of Medical Evaluation and Statistics in Legal Proceedings

If seizures exist, the problem is to determine if it is post-traumatic, and if possible to evaluate the severity. This is a purely medical problem based on some of the principles given above.

The perplexing problem, to physician, lawyer and plaintiff alike, is how to establish a reasonable probability that a given complication will develop. Utilizing the information here presented, one can make some quick mathematical estimates.

Let us take an example. In a civilian head injury caused by a blunt instrument, not a missile (with clear-cut penetration of the skull and dura mater), the incidence of epilepsy will probably not reach 20% (this is a liberal estimate for the purpose of this example). Since at least two-thirds of cases of post-traumatic epilepsy will develop within a two year period, if by the end of two years the patient has not developed epilepsy, he now has only a $6\frac{2}{3}\%$ chance of doing so. Thus there is an im-

mediate presumption that the odds are 16 to one against such a complication developing.⁷⁰

As another example, one might return to the semi-hypothetical case of Belli mentioned earlier in this article. To review the facts briefly, the key features are these—(1) a non-depressed skull fracture, (2) no evidence of penetration of dura or local brain injury, (3) no evidence of abnormal EEG, (4) negative neurologic examination, (5) the passage of a year, (6) no history of injury (and if one was present, apparently not an injury by a missile, nor a severe head blow at high speeds by a blunt instrument).

It is not necessary to comment on all these features. As to the first, the incidence of post-traumatic epilepsy in such cases may be hypothecated as two percent (although as stated previously, many neurologists feel that, without local brain injury, such an injury is almost totally irrelevant to the development of epilepsy). As to number (5) above, since, in one year, more than a majority of cases will develop epilepsy if in fact it will develop at all—then the chances here become less than one percent. Thus immediately there is a presumption statistically that the odds are more than 100 to one against the development of epilepsy. The odds here are now so low that without localizing brain injury, EEG, etc.—if epilepsy did develop, it would statistically be most likely a case of idiopathic epilepsy rather than post-traumatic epilepsy. Without laboring the point, it may be summed up by saying that this case becomes a statistical nullity.

To quote Walker⁷¹, an expert in this subject (admittedly out of context): “May we not say, then, with reasonable medical certainty, that if a patient without neurological symptoms or deficit and having a normal EEG has gone two years after his injury without seizures, he will not develop post-traumatic epilepsy?”

Smith⁷², in his excellent article, states: “The risk of epilepsy following head injury is of the following order after simple concussion of the brain—0.02%; after linear fracture of the cranial vault—0.521%; after severe head injury with depressed fracture of the skull, fragments of which have lacerated the dura

⁷⁰ Here we have not taken into account any of the many other factors previously discussed, so that the example will be clear in meaning.

⁷¹ See n. 41(d) above.

⁷² See n. 2(f) above.

mater and brain—20 to 45%. It follows that in no case can the plaintiff prove probable future occurrence of traumatic epilepsy which has failed to materialize by the time of trial without adducing strong corroborative evidence of impending epilepsy such as significant changes in serial electroencephalograms interpreted and supported by competent neurological opinion.”

Thus the claimant's attorney faces an uphill statistical battle in any effort to indicate probability of occurrence where in fact it has not yet occurred. Utilization of the many varied aspects discussed here will be of help in the giving of some mathematical estimate by the physician, and its subsequent use by the court.

An Interesting Suggestion as an Alternative Method of Handling These Claims

Walker⁷³, in a very imaginative suggestion, states: “If there is a reasonable suspicion that attacks have occurred and it is feared that recurrent epileptic attacks may develop, I believe that it would be fair to both the patient and the compensating agency to award not compensation for traumatic epilepsy but an insurance policy against the possibility of an epileptic attack developing within a reasonable time, say 5 or 10 years. . . . If seizures develop, the patient will be compensated; if they do not, he will not have been stigmatized and the primary compensating agent will not pay a penalty.

“Even if post-traumatic seizures do develop, it should be clearly understood that they do not have the same prognosis and implications as does so-called idiopathic epilepsy. In fact, if a patient has had only one or two attacks within the first year or two after a head injury, I would certainly hesitate to suggest compensation on that basis. Probably some type of epilepsy insurance would be the most equitable means of handling these cases. There is excellent evidence that such patients have a good chance of living lives which will not be punctuated by convulsions.”

He feels that actuaries could work out some system. A key problem in any system would be a high standard of evaluation in order to screen out fraudulent claims, which would fit in so nicely with such a system, and which would still protect the rights of the injured.

⁷³ See n. 41(b) above.