Posttraumatic Epilepsy

by

A. EARL WALKER, M.D.

Formerly, Professor of Neurological Surgery
University of Chicago, Chicago, Illinois
Now, Professor of Neurological Surgery
The Johns Hopkins University, Baltimore, Maryland

Today modern medical and surgical therapy offer patients suffering from posttraumatic epilepsy a more hopeful future. Here is a practical approach to the problem in the light of the latest, accepted advances.

90 pages **28 illustrations**

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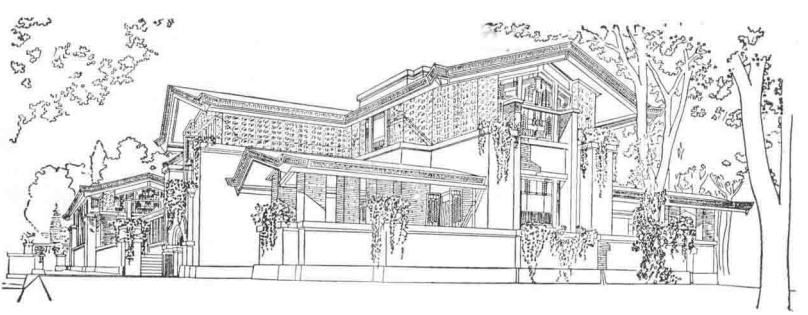

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This Book Is Dedicated

to

STEPHEN POLYAK

My First Preceptor

in

Neurology

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POSTTRAUMATIC EPILEPSY

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Introduction

Since Hippocrates (1) who observed convulsions following cranial wounds, cerebal trauma has been considered a cause of epilepsy. To the neurologist, posttraumatic epilepsy is not a satisfying concept for the neural and chemical mechanisms inducing a fit as the result of cerebral trauma are poorly understood. But to the laity, a diagnosis of posttraumatic epilepsy is both adequate and encouraging, for in their minds it removes the case from the awful and stigmatized category of idiopathic epilepsy. Accordingly, relatives often emphasize minor head injuries, which they subconsciously hope will be considered etiologically related to the convulsions of their kin. And physicians, too, frequently assume that the occurrence of a fit after a head injury justifies a post hoc, propter hoc conclusion.

Because a convulsion is evidence of cerebral dysfunction, trauma etiologically related to epilepsy must affect the brain. A blow on the head causing only a scalp bruise is not sufficient evidence to establish a cerebral injury. Neurological abnormalities such as loss of consciousness, hemiparesis, aphasia etc., are reliable criteria of brain

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damage. Excitatory focal manifestations of cerebral damage at the point of the blow, such as Jacksonian seizures, localized electroencephalographic abnormalities, are corroborative evidence, but their absence does not rule out a posttraumatic etiology. A pneumoencephalogram may indicate an area of brain atrophy secondary to cerebral trauma. Although all of these evidences of cerebral trauma may not be present in every case, their complete absence makes the diagnosis of posttraumatic epilepsy difficult to substantiate.

The convulsions in posttraumatic epilepsy usually begin within two years of the cerebral injury; if a longer latent period exists, the possibility of some other etiological factor is enhanced. The traumatic etiology in such late developing cases of epilepsy must be established from the clinical history and findings, the electroencephalographic and pneumoencephalographic alterations and by the elimination of other possible causes.

II

Incidence Of Posttraumatic Epilepsy

The real frequency of convulsive seizures following brain trauma is difficult to assess. The inadequate follow-up of head injury cases, the difficulty in determining the extent of the wound and the differences in treatment are probably the reasons for the wide variations in incidence reported by different authors.

The frequency of posttraumatic epilepsy is directly related to the severity of the cerebral injury. In closed head injuries, which tend to be less severe than open cranial wounds, and to affect the brain-stem and basal ganglia rather more than the cerebral cortex, the incidence of posttraumatic epilepsy is approximately 2.5% (58) to 3.5% (60).

Open wounds of the head are followed by convulsions in from 4.5% (61) to 49% (9) of cases. As the result of wounds incurred in World War I, statistics are available from three different countries: Germany, England, and Canada. In 1926 Steinthal and Nagel (67) reported a 39.8% incidence of posttraumatic epilepsy in German patients who had had penetrating dural wounds; four years later Credner (9) found that 49.5% of such patients had epilepsy. In England Wagstaffe (71), in a series of 176 penetrating wounds, found only

18.7% with epilepsy. In 1941 Ascroft (2) reported an incidence of 45%. Two years later Gliddon (23) found only 18.9% of Canadian pensioners from World War I with penetrating dural wounds suffering from epilepsy.

It is too early to know the incidence of posttraumatic epilepsy in World War II. Maltby (35) found 34 patients who had had convulsions in his series of 250 penetrating wounds of the head, but his cases were followed for only a short period. In a series of 279 cases Watson (74) reported a 36% incidence of posttraumatic epilepsy within two years of injury. Both Maltby's and Watson's series were of patients in Army General Hospitals, where the severely wounded cases tend to gravitate.

At the request of the author, Dr. George C. Culbreth reviewed the Veterans Administration's file of head injury cases from World War II in the Chicago area. From a list of 830 cases of cerebral trauma incurred 2-5 years previously 454 "C" files were obtained at random and examined in June and July of 1947. Since discharge from the service, all of these cases had been seen, and examined one or more times, in the majority of instances within the 6 months period prior to review of the file.

In this entire group 48 patients (10.8%) had had seizures. In the 188 penetrating head injuries due to bullet, shell fragment and compound fractures, 28 or 14.9% of the patients had experienced convulsions. However all but one of these posttraumatic epileptics were well controlled by anti-

convulsant medication. The low incidence of seizures and their response to medical management is probably related to the careful debridements and relative freedom of the wounds from infection.

It has been stated that a late developing epilepsy has a poorer prognosis than one which comes on shortly after the head injury (2). In the Cushing Hospital series* (56) the seizures came on within three months in 27.0% of the cases and between three to six months of the injury in 30.7%. Within these limits, the delayed onset of the attacks did not seem to have a prognostic import in this series.

* In the latter part of November, 1945, the Surgeon General of the United States Army designated Cushing General Hospital, Framingham, Massachusetts, as a center for the treatment of patients suffering from posttraumatic epilepsy. Approximately 250 patients — the majority transferred from other Army General Hospitals — were examined and treated for this condition in Cushing General Hospital. Much of the information in this discussion was acquired as the result of the author's experience in this center for posttraumatic epilepsy.

III

Some Factors in the Pathogenesis of Posttraumatic Epilepsy

In symptomatic epilepsy the factors precipitating seizures are probably similar irrespective of the basic neuropathological condition, be it tumor, abscess, or scar. While the precise anatomical, physiological, and biochemical mechanisms are not yet known, some information is available concerning the nature of a few factors.

Susceptibility to Convulsions

The capricious occurrence of epilepsy after cerebral injuries has been ascribed to an individual susceptibility to convulsions or "a relatively low degree of stability of the nervous system" (61). This theoretical conception has not received physiological confirmation. When cerebral dysrhythmia was propounded as the basic factor in idiopathic epilepsy, it was suggested as the convulsive diathesis. Dysrhythmia is, however, no more frequent in cases of posttraumatic epilepsy, than it is in the general population (22, 30). Moreover, if dysrhythmia, a hereditary factor (32), were associated with the convulsive diathesis in brain disease, the incidence of epilepsy in relatives of posttraumatic

epileptics should be high. Such is not the experience of Penfield and Erickson (50). Nor did Ziskind and Ziskind (80) find evidence in favor of this hypothesis. In the group of posttraumatic epileptic patients studied at Cushing General Hospital (56) only 4.5% of the patient's relatives had convulsions compared to an incidence of 3.4% in normal families and 17% in epileptic families (33).

The posttraumatic epileptic is more susceptible to certain convulsive agents, such as metrazol, than normal individuals, but not to other convulsants such as electric shock (29, 69). Dandy and Elman (10, 11) showed that dogs having cerebral lesions had convulsive seizures with smaller doses of absinthe than normal dogs. Whether the posttraumatic epileptic is more susceptible to metrazol than the non-epileptic brain injured patient is a crucial point which has not yet been demonstrated. If that could be proved, it is understandable how fluctuations in cerebral metabolites such as glucose, oxygen, etc., within the physiological range for a normal person, might precipitate a seizure in a highly susceptible individual.

Cerebral Vascular Instability

That spasm of the pial vessels due to irritation by the scar initiates the convulsive seizure, often has been suggested as the proximate cause of a fit. Vascular constriction and pallor of the cerebral cortex has been reported just before an attack. However, most neurosurgeons (43, 50) have been unable to confirm this phenomenon either by direct