Post Traumatic Epilepsy
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The incidence of post-traumatic epilepsy varies widely among clients with traumatic brain injury depending upon the type and severity of the injury as well as on other related factors, including the patient's age. A number of different types of post-traumatic epilepsy exist, some of which involve frequent seizures with acute symptoms, while other types involve only rare seizure occurrence and/or symptoms of a mild nature. Employability of clients with post-traumatic epilepsy is often possible but vocational rehabilitation issues need to be considered on a case-by-case basis.

The cause, severity, and type of post-traumatic epilepsy, as well as the probability of remission, tend to vary widely from case to case. For this reason, it is critically important to categorize the specific components involved. Early epilepsy (within the first week after injury) should be distinguished from late epilepsy. The total number of seizures, which have occurred, should be ascertained, as well as whether the seizures are of the generalized or partial type.

Post-traumatic epilepsy appears to be the result of brain lesions secondary to traumatic brain injury. Laidlaw and Richins (1982) suggest that focal residual lesions resulting from the trauma are the primary cause. It is also possible that microscopic lesions caused by the head injury may induce the post-traumatic epilepsy on a subcortical basis. Iudice and Murray (2000) still emphasize that lack of definitive knowledge regarding the development of post-traumatic epilepsy affects the ability to provide appropriate prophylactic anticonvulsant strategies. It is important to note that many other types of epilepsy exist other than the post-traumatic type. Epilepsy can also be caused by brain tumors, cerebrovascular disease and other neurological difficulties.

This reading assignment presents an overview of the etiology, risk factors, evaluation methods, and treatment of post-traumatic epilepsy, as well as a review of specific seizure types and appropriate anticonvulsant usage. It should be noted that, in many cases of post-traumatic seizure, the accompanying neuropsychological sequelae of the head injury, and not the seizure incidents, will be the primary area of concern to the rehabilitation counselor.

Also reviewed in the reading assignments are counseling considerations, life care plan issues, and job-development strategies for use with the post-traumatic epilepsy population. For many clients, a situational assessment program will be mandatory to establish accommodations related to both cognitive concerns and seizure-related safety issues.

Incidence
The incidence of seizures needs to be considered by the time interval following the trauma. Iudice and Murray (2000) classify seizures as:

1) "impact seizures occurring within 24 hours;"
2) early seizures occurring within one week; and
3) late seizures observed from day eight through two years or later."

They emphasize that concussive convulsions occurring in sports are not epileptic in nature and require no specific therapy or antiepileptic medication. Perron et al. (2001) underscore that these concussive convulsions evidence no structural brain abnormalities or long-term abnormalities, and do not carry an increased risk for or late epilepsy.

Murray and Iudice (2000, p 1092) indicate an impact frequency rate of 1.26% within 24 hours and an early seizure (within one week) frequency rate of 3.6%. There tends to be a higher proportion of generalized seizures at the early stage and aggressively increasing partial seizure occurrences later. Iudice and Murray, in their review, indicate that on an overview, late post-traumatic epilepsy occurs in 7% of all cases, but it occurs in 34% of those experiencing brain injury in combat, and the frequency is approximately 50% for those experiencing severe trauma.

Jennett (1979) indicates that in about 80% of late epilepsy cases (cases in which the onset of epilepsy occurs after the first week post-injury), seizures persisted for a period of two to five years. In a number of such cases, however, seizure remission of two years or more was reported prior to recurrence. Two factors seem to be causative in the onset of seizures: (1) the nature of the specific type of brain impairment involved; and (2) a constitutional tendency toward seizures.

Most patients, according to Salazar et al. (1995) can be 95% certain of avoiding PTE if they are seizure free for three years, regardless of the risk factors present.

Regarding age distribution, a comprehensive study by Kollevold (1976) suggests that the preponderance of cases of post-traumatic epilepsy involve males between the ages of 10 and 50, with the highest incidence occurring between the ages of 15 and 19. Some studies have related the latter higher incidence occurring between the ages of 15 and 19. Some studies have related the latter higher incidence to the prevalence of alcohol abuse in younger males (Grudzinska et al., 1974). In the Kollevold study, it was also suggested that children tend to be twice as susceptible to post-traumatic epilepsy as adults.

**Risk of Occurrence**

Iudice and Murray (2000, p 1093) summarize risk factors for developing PTE in over 30% of patients with penetrating head injuries, intracerebral or subdural hematoma, depressed skull, fracture, or early seizures after injury. PTE seems
associated with the penetrating nature of the trauma, extent of tissue loss, and brain trauma severity - penetration of the dura increasing occurrence up to 57%.

Salazar et al. (1995) developed a model, which predicts PTE in the range of 71% - 77% using CT findings alone of lesions in five brain areas:

1. right vertex gray matter;
2. left convexity cortex;
3. left temporal white matter;
4. right frontal white matter; and
5. right corona radiata.

Weiss and colleagues (1983, 1986) have generated a relatively accurate model for predicting post-traumatic epilepsy using both clinical and anatomic correlates-the analysis resulting in predictive tables associated within months post-injury. Again, these factors become less meaningful three years or more post-injury. According to Jennett's earlier study, two-thirds of those with first-time seizures did not have recurring seizures. It is important to remember that epilepsy is defined as "two or more seizures."

Seizure remission, according to Caveness (1963), tends to correlate with the total number of seizures a patient has experienced: patients with only one to three seizures have an 85% remission rate, those having four to 30 seizures have a 55% remission rate, and patients having more than 30 seizures have a 21% remission rate.

In the study by Annegers et al. (1980), injuries were classified as severe (brain contusion, intracerebral, or intracranial hematoma, or either unconsciousness or amnesia lasting 24 hours or more), moderate (skull fracture or 30 minutes to 24 hours of unconsciousness or amnesia), and mild (briefer unconsciousness or amnesia). The risk of post-traumatic seizures following severe injury was 7.1% within one year, and 11.6% within five years; after moderate injury, the risk was .7% within one year and 1.6% within five years; after mild injury, the risk was .1% within one year and .6% within five years. The incidence of seizures after mild head injuries was only slightly greater than in the general population, and after five years post-injury it was the same as within the general population. Epilepsy occurs with a higher incidence rate (DeSantis et al. 1979) in cases requiring surgery (11.6%) than in non-operative cases (1.2%). For purposes of life care planning, a consulting neurologist or neurosurgeon can estimate the probability of epilepsy or recurring seizures (general partial) occurring, the costs of diagnosis and treatment, probability of remission, etc. The risk factor data that is available will enable this to be considered as related to the life care plan. In a minority of cases, costs related epilepsy surgery or use of a vagal nerve stimulator may need to be considered to optimize seizure control or promote a seizure free status.

Seizure types

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There are two major categories of seizure activity:

1. generalized seizures; and
2. partial seizures.

Generalized seizures involve the whole brain and include generalized tonic clonic or "grand mal" type of seizure (old terminology) and the absence or "petit mal" type of seizure (old terminology). The "grand mal" seizure can be divided into two component stages: (1) a tonic stage in which, for a short period, the body is rigid and (2) a clonic stage in which there is muscle jerking and contraction. The average seizure of this type lasts from two to three minutes. Early post-traumatic seizures tend to be generalized. It should be noted that some seizures begin as focal partial and then generalize into "grand mal" seizures. The absence or "petit mal" type of seizure lasts for only a few seconds, during which there is a disruption of consciousness, a fluttering of the eyelids, or blank staring. If a "grand mal" seizure lasts more than ten minutes, it is considered a medical emergency termed status epilepticus, requiring immediate transfer to a hospital emergency room. There are a number of other generalized seizure types (simple tonic or clonic, atonic, myoclonic, etc.) which occur considerably less frequently.

Partial seizures begin locally in a part of one cerebral hemisphere. They can be categorized as either partial elementary or partial complex. Partial elementary seizures involve only one system, such as the motor or sensory system, and are associated with some type of jerking movement of the legs or hands with no loss of consciousness. Partial complex, or psychomotor seizures, are among the common encountered by the neurologist or rehabilitation counselor. They are times associated with psychical changes (deja vu experiences), always involve a loss of consciousness, and can induce repetitive motor movements such as lip smacking or fumbling with one's hands, as well as wandering or nonsensical verbalization. Approximately 60% of those with seizures have partial seizures (Pedley & Hauser, 1988). They tend to occur in late post-traumatic epilepsy, after the first week, and can secondarily generalize. It should be noted that some events occur that mimic seizures that are really non-epileptic and a function of conversion hysteria, post-traumatic stress, etc.

**Early Management**

Prophylaxis and diagnostics are two key components in the early management of post-traumatic epilepsy. Management will often begin prior to confirmation of the diagnosis. Studies have shown that early diagnostics have proven valuable in identifying cases, which later develop into serious epilepsy disorders.

Before a client can begin to come to terms with epilepsy, it is necessary to fully evaluate the type and severity of the condition and to perceive it clearly in relation to the primary brain injury, which preceded it. Anticonvulsant medication must be prescribed with extreme care to avoid unnecessary side effects. It is
especially important that the rehabilitation counselor, the treating neurologist, and the patient/client maintain open lines of communication to discuss any problems or developments.

**Prophylactic Use of Anticonvulsants**

The prescribing of anticonvulsants following head injury to prevent seizure occurrence should not be done indiscriminately. The risk factors identified in the studies cited previously should be carefully weighed, as anticonvulsants often have adverse side effects and their effectiveness in preventing post-traumatic epilepsy varies from case to case.

The present state of the art (Temkin, 2001) suggests that standard prophylactic use of anticonvulsants in order to prevent early post-traumatic epilepsy is the standard of care with carbamazepine (Tegretol) or phenytoin (Dilantin) being recommended for a week or so after the injury. Conversely, no acceptable effective prophylactic strategies have proven effective in managing late post-traumatic epilepsy (Temkin, 2001; Iudice & Murray, 2000) while some have negative side effects related to long-term use including cognitive loss, peripheral neuropathies, and even slightly increased morbidity as in the case of sodium valproate (Depakote), reviewed by Temkin (2001).

A recent survey reviewed by Iudice and Murray (2000, p. 1096) indicated that across 127 neuropsychological departments, 36% of neurosurgeons do not prophylactically treat any of their brain injured patients, 12% prescribe anticonvulsants prophylactically in any trauma, with 52% making the decision within the context of existing risk factors (e.g., a penetrating injury, intracranial hemorrhage, etc.). Iudice and Murray (2000, p. 1096) suggest prophylactic use of anticonvulsants past the acute period only within the context of salient risk factors and positive benefit/cost evaluation.

**Diagnostic Procedures in Evaluating Post-Traumatic Epilepsy**

The EEG (electroencephalogram) is a procedure that provides a written record of the brain's spontaneous electrical activity. The brain wave patterns are electronically traced on a moving display paper, usually in a simultaneous 16-channel format. These tracings are then reviewed by a neurologist specializing in reading of the EEG to investigate the existence of any aberrant electrical discharges and determine if there is a focus for the seizure activity within the brain. Jennett (1975, p 156) indicates that the EEG cannot be used to predict epilepsy following injury. The abnormalities it records often relate only to the brain impairment caused by the trauma. On the one hand, a normal EEG is only partially reassuring on the other hand, the presence of EEG abnormalities does not necessarily foreshadow the later occurrence of epilepsy. Scherzer and Wessley (1978) corroborate the difficulty of generating valid criteria for the prognosis of post-traumatic epilepsy with the electroencephalogram. It should be
noted that in using awake and asleep EEGs, 25% of the partial seizures can still be missed and specialized neuroradiological, noninvasive techniques can be needed to scan the brain and identify the small focal lesions causing the seizure.

The CT (computed tomography) scan, which is a technological approach in radiology that permits scanning of the brain for abnormal structures without hospitalization or complicated invasive techniques, has proven valuable in the location of post-traumatic epilepsy. Magnetic resonance imaging (MRI), however, is usually superior to CT scanning and is generally the gold standard due to its definitiveness and sensitivity in clarifying small lesions or cerebral cortex abnormalities (International League Against Epilepsy, 1997). The MRI, CT and single photon emission computed tomography (SPECT) or positive emission tomography (PET) can truly clarify issues relating to structural lesions. Multichannel magnetoencephalography (MEG) is one of the newer techniques for measuring magnetic fields and appears more definitive in circumscribing causes of epileptic discharge.

Medical Evaluation Concerns

Ideally, the person with a seizure disorder should be evaluated by a neurologist, particularly if seizure control has not been established in three months (National Association of Epilepsy Centers, 1990). It is also crucial for those patients taking anticonvulsants to have their blood serum levels assessed to determine whether the medication is within their therapeutic range.

If seizure control is not achieved by a general neurologist within nine months, referral should be made to a tertiary or fourth level epilepsy center (National Association of Epilepsy Centers, 1990). These centers will have neurologists specializing in epilepsy (epileptologists) and allied health staff specializing solely in epilepsy (look for membership in the American Epilepsy Society). These specialists can address specific pharmacological issues, non-epileptic seizures, the need for invasive, intracranial video/EEG recording, the potential for epilepsy surgery, and specific neuropsychological or other expertise.

In order for the rehabilitation counselor to develop an optimal rehabilitation plan, a considerable amount of specific information is needed. This information includes the following:
~ the identification of any precipitants (e.g., fatigue or alcohol) related to the seizure activity;
~ the identification of any temporal or situational pattern to the seizure;
~ a clear description of seizure behavior, and;
~ frequency and identification of any aura or other consistent warning signs related to the seizures, as well as precise information about the duration of the seizure aura or any other preceding warning events.

It is also important to note the client's prescription or nonprescription medications
and to indicate his or her compliance with medical regimens, as well as any suggestion of a change of anticonvulsant regimen or any potential side effects of medication. The amount of time required for individual recovery from a seizure should also be noted. A final concern is the individual's ability to maintain a driver's license. In some states, an individual requires several years of freedom from seizures in order to maintain a license while in other states the matter is left to the discretion of the treating physician.

**Anticonvulsants and Other Treatments**

It is common for an individual to come to the rehabilitation counselor on either the wrong or too many medications. The treatment goal has generally been the use of primary anticonvulsant at a maximum tolerable dosage for the patient without undue negative side effects. Following earlier use of phenytoin (Dilantin) and phenobarbital, in the 1970s we had the utilization of carbamazepine (Tegretol) and valproic acid (Depakote, Depakene). In the 1990s, new medications such as topiramate, lamotrigine, gabapentin, vigabatrin, etc. were developed as add on or adjunctive medications - some of which are now being used as primary treatment agents with continued study of some of these compounds. Side effects of the new wave of medications have been chiefly subtle and all have shown effectiveness against complex partial and secondarily generalized seizures. Side effects of the cautions may be additive and the blood serum level of each medication needs to be evaluated carefully and frequently.

The names of anticonvulsants used in cases of generalized tonic-clonic or “grand mal” seizures include phenytoin (Dilantin), carbamazepine (Tegretol), and lamotrigine (Lamictal). Ethosuxamide (Zaronrin) has been used in the treatment of “petit mal” or absence seizures. Valproic acid (Depakene, Depakote) has also been used as a primarily anticonvulsant for generalized tonic-clonic and absence seizures. Dilantin and Tegretol are also used to treat psychomotor or partial-complex seizures. Simple partial seizures are often treated by Dilantin. A number of new wave anticonvulsants can be used as a secondary medication to insure better seizure control. Some, such as lamotrigine or topirimate, are now being used as primary treatment resources for both partial and generalized seizures.

It is very important that the rehabilitation counselor maintain an open dialogue with the patient and the treating physician in the management of an anticonvulsant side effects. This dialogue will prove particularly valuable when identifying and remedying anticonvulsant side effects. Barbiturate anticonvulsants, such as phenobarbital, can induce fatigue, increase irritability, and affect motor speed and dexterity. An individual in a toxic range on an anticonvulsant is going to have difficulties with life functioning as a direct result of the medication side effects. Other anticonvulsant side effects can include double vision, difficulties concentrating, nausea, dizziness, weight gain, gum and facial hair growth. More severe side effects, such as liver function disturbances or suppression of bone marrow, have been observed in some cases. It is to be
noted that for many individuals taking anticonvulsants, particularly with progress during the 1990s, effects, including the cognitive, are minimal. The anticonvulsant regimen for those with post-traumatic epilepsy, as discussed, will vary according to the known factors of each case in relation to a persistent seizure condition.

Works Cited:


National Association of Epilepsy Centers: Recommended Guidelines for Diagnosis and Treatment in Specialized Epilepsy Centers, Epilepsia 21 (Supp. 1), 1-12, 1990.


