Can Emotions Precipitate Seizures—A Review of the Question

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Controversy over the emotional precipitation of seizures has existed for centuries. Large clinical studies have generally shown a significant proportion of seizures to be triggered by emotions. Laboratory studies have been reported which describe seizure activity on the electroencephalogram during periods of experimentally induced stress. However, very few of these studies have utilized control groups and blind evaluators. The numerous reports of the emotional precipitation of seizures indicate that the phenomenon may be genuine and that further research is warranted. Research on emotional interventions such as biofeedback techniques and psychotherapy is the next step, and early work on these interventions is promising.

Currently, physicians disagree over whether or not emotional stimuli can precipitate seizures. When a patient reports that he/she thinks a stressful situation caused a seizure, some physicians will accept it while others discount it. In spite of the controversy, current neurology textbooks either discount such reports or else simply state that seizures can be provoked by emotions and stress without indicating the experimental work upon which this is based. ¹⁻⁶ Betts⁷ recently reviewed the psychosomatic aspects of epilepsy, but his summary of the literature on emotional stimuli as precipitants was incomplete. This paper is a comprehensive review of the literature and the issues involved in this controversy.

Historical Perspective

The controversy is actually an old one. The history of seizures parallels that of hysteria, which is

well described by Veith.8 Seizures were included as symptoms of hysteria by Hippocrates and also by Galen in the second century AD. From the 2nd to 18th centuries, seizures were thought to occur in people who were in communication with the devil, and sexual taboos were involved in the explanations of seizures. In the early 19th century, hysterical symptoms were considered to be emotional disorders in idle or dissatisfied people. In the mid 19th century, gynecological explanations were in vogue, and ovariectomies were performed to control seizures. Jules Falret, a psychiatrist at Salpetriere in Paris (circa 1866), stated that his female patients with seizures were actresses. Twenty years later at the same institution, Charcot coined the word "hysteroepilepsy" to describe a disorder which he felt only imitated genuine epilepsy. John Hughlings Jackson regarded epilepsy as distinct from hysteria.9 Freud also felt that hysteroepilepsy was to be distinguished from organic epilepsy.10 Freud provided a psychodynamic explanation for Dostoyevsky's seizures, which he felt were hysterical in nature. Later psychoanalysts have kept less to this distinction and have extended the psychodynamic explanation to genuine seizures as well. 11 Thus,

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seizures in general and hysterical seizures in particular have been described by psychoanalysts as a wish to escape from a difficult situation or from sexual conflicts, as a discharge of frustration and aggression, as a suicidal wish, and as expression of frustrated dependency and of separation anxiety. Mittelmann¹¹ has reviewed the literature on these explanations.

That emotions are involved in hysterical seizures is accepted. But can strong emotions precipitate a seizure in someone with a documented organic lesion or with a positive electroencephalogram (EEG)? To answer this question, one must distinguish between hysterical and organic seizures. This, of course, is easier said than done, and it may be one reason for the disagreement over stress-induced seizures. In 1901, Gowers12 listed criteria which he felt distinguished epileptic from hysterical fits: tonic-clonic contractions, cyanosis, complete unconsciousness, tonguebiting, and incontinence were typical of epilepsy, while wild, uncoordinated movements, partial consciousness, talking, and opisthotonic posturing were typical of hysteria. More recent literature¹³ adds to this the postictal abnormal muscle tone and the abnormal reflexes (Babinski, pupillary, and corneal) as signs useful in distinguishing organic from hysterical seizures. However, others feel that such distinctions cannot be made. For example, Aggernaes14 concluded that purely hysterical and purely organic seizures can scarcely ever be found. He thought it more reasonable to talk of predominantly hysterical or predominantly organic states of clouded consciousness. Even a definition of epilepsy is difficult to provide, and many authorities do not attempt one. In spite of these problems of definition and distinction, it is still possible to study the effect of stress and emotions in persons with predominantly organic seizures—those with known organic lesions, such as tumors, or those with positive EEGs. Most of the studies which follow have been based on cases with positive EEGs.

Clinical Observations

Much of the published work consists of clinical impressions that seizures can be provoked by emotional stress. John Hughlings Jackson⁹ wrote that fright, excitement, and anxiety can be causes of a seizure discharge, but he emphasized that this occurred only if there were already an unstable

focus of nerve tissue. Since Jackson's time, the emotional triggering mechanisms for seizures have included fright, anxiety, anger, embarrassment, crying, and worry¹⁵; these are usually obtained from reports by the patient. Other reports are based on the clinical impressions of physicians; these reports describe the emotional precipitation of seizures in considerably more detail. ¹⁶⁻²⁷ However, not all physicians shared the impression that emotions could cause seizures. Gastaut and Tassinari²⁸ described numerous triggering mechanisms for epilepsy, but they stated that, in their long careers, they had seen only ten cases of patients with seizures precipitated by emotions.

Only a few statistical clinical studies could be found. Gowers¹² studied 1,665 persons with seizures; a cause could be found in only 696 (42 percent) and of these, one third reported emotion as a cause (fright, excitement, and anxiety). Fremont-Smith²⁹ studied 42 unselected persons with seizures with loss of consciousness; in 31 of 42 a direct relationship was found between major convulsions and emotions (usually fear, guilt, or frustration). Many of his patients also had evidence of organic brain disease as a predisposing factor for the seizure. Allen30 reported that 42 of 182 cases had an emotional factor related to the occurrence of a seizure. Berlin and Yeager³¹ studied four cases retrospectively and found a correlation between the person's emotional state and the frequency and severity of seizures and abnormalities on the EEG. Mignone et al³² found that 53 percent of their patients reported stress precipitation of seizures in their sample of 151 patients who had "unequivocally positive" EEGs.

Emotions Induced During EEG Recordings

Some workers have induced emotions in a laboratory setting while recording EEG activity to see what direct correlations they could make. Zegans et al³³ and Kemph et al³⁴ described such a case report. They reported a highly significant increase in galvanic skin response and heart rate (presumptive evidence of emotional stress during a stressful interview) and a decrease of 3 cps burst activity. Bureau et al³⁵ used EEG telemetry and reported that petit mal absences occurred most often during boredom. Barker and co-workers³⁶⁻³⁹ reported that EEG abnormalities were induced during interviews at such times when there was

recall of stressful events, during stress-induced disruptions of integrative activity, during the use of stimuli which were designed to startle, disturb, irritate, or annoy, and during periods of inactivity. Vidart and Geier40 used EEG telemetry to show that mood increased or decreased spike and wave abnormalities, that interest shown in the patient reduced the abnormalities, and that traumatic events raised the number of abnormalities. Gottschalk⁴¹ described a few patients in whom doing silent mental arithmetic and talking reduced the frequency of abnormal, high-amplitude, slow-wave paroxysms on the EEG, and one patient in whom the expression of emotionally charged ideas was regularly followed by slowwave, high-voltage activity. Stevens^{42,43} studied 30 patients, using the EEG during a stressful interview. She reported that one third had epileptiform abnormalities on the EEG precipitated by emotional stress and one third reduplicated or exaggerated previously abnormal EEG changes during emotional stress; no such EEG changes were observed in nine normal persons during the stressful interview. Small et al44,45 similarly stressed 44 persons with known seizure disorder. Of 37 who showed an emotional response, 12 had activation of the EEG.

Discussion

The above studies have not settled the controversy. Numerous clinical observers have reported that emotions precipitate seizures and have felt that this could be proved if only the proper techniques could be found. The simultaneous use of EEG recordings and stressful interviews provided the believers of stress-induced seizures with some experimental data on which to base their claims. Yet others have remained skeptical.

There are a number of reasons why such disagreements arise. In the studies of simultaneous use of EEG and interviews, it is probable that the person making the correlation knew if there was epileptiform activity on the EEG in advance of making his evaluation of the emotional state. Very few studies had control groups and few mentioned the use of blind evaluators to control for observer bias. Another reason for controversy is that many studies and case reports relied upon reports by the patient concerning emotions. It could be argued that the patient is also a biased observer or that stressful life events occur all the time but

may especially be remembered when they coincidentally occur prior to a seizure. Physicians differ in the extent to which they will take the word of the patient that emotions precipitated a seizure. Sometimes disorders of association are taken as evidence for emotional stress when they also are evidence of seizure activity.

An important question is whether the emotions reported prior to a seizure are the result rather than the cause of seizure activity. It has long been known that emotions can be part of the aura or ictal state. It is possible that a changing physiological state which precedes a seizure could make a person more emotionally labile; he would, thus, have more emotional outbursts and would experience a stressful situation as being more stressful than he would at other times. Descriptions of seizures prior to the era of anticonvulsant medications are interesting in this regard because they provide an account of the natural course of a seizure. Spratling worked for many years in a colony of epileptics and in 1904 published a book which provides numerous such clinical descriptions.46 He described transitory periodic irritability prior to seizures:46

Among the scores of cases I have been privileged to see almost daily for years. I have learned to detect with almost unfailing certainty—through noting temperamental changes alone the moment the patient enters the room and begins to speak—the approach of a convulsion a few hours or even days in advance of the convulsive period. An almost imperceptible change in personality has been wrought. The patient is querulous, fussy, fault-finding, nothing goes right; trifles that ordinarily produce no effect on him now completely engage his attention. His friends ignore him, his family is indifferent to his needs and his condition, his fellow patients are no longer congenial, their attacks disturb him, he cannot endure their jocose remarks, distorting them into expressions of ridicule. Finally, these ideas may persist in their growth, looming up larger and larger on the horizon of a morbidly heated mind, until they pass into qualified delusions, all being dependent upon the subtle, pernicious, autocratic influence of the approaching attack, and all completely disappearing, as if by magic, after the attack is over.

Spratling said that these "ill-humor periods" may begin days or weeks prior to a seizure and he estimated such temperamental changes to occur in up to 80 percent of persons with seizures.

Another reason that disagreements arise is the

difficulty in distinguishing real from hysterical or mixed types of seizures based on clinical observation alone. It might be argued that some clinical reports are based on hysterical seizures because of this difficulty. Even simultaneous scalp EEG monitoring during apparent seizure activity may not be entirely reliable. Goldensohn⁴⁷ described studies in which the EEG did not reliably indicate when a seizure began or ended. This weakens the usefulness of the studies which utilized the EEG and stressful interviews simultaneously.

One might wonder why we should even try to settle this controversy. There are several reasons why the question of emotionally precipitated seizures is important. One is that if it exists, then understanding it may provide further understanding of the biological mechanisms of emotion. Another reason is that if emotions or stress can induce seizures, then intervention can be designed to prevent this. Behavior modification techniques could be used to treat seizures. A recent review of these techniques as applied to the control of seizures suggested there is promise in their clinical use and offered guidelines for future work. 48 Sterman⁴⁹ reported that use of biofeedback of 12 to 14 Hz rhythm recorded over the sensorimotor cortex to increase its occurrence resulted in clinical improvement in four of five cases of seizures, as shown by EEG changes and by decreased seizure frequency. While there is a question whether the reduction in seizure frequency was due to the production of a suppressive premotor rhythm or to a change in psychological state as a result of biofeedback practice, either possibility holds promise.

Intervention in emotionally precipitated seizures would include psychotherapy for crisis intervention and benzodiazepines during stressful periods, whether the seizures are of the hysterical or mixed type. Most of the studies above that were case reports described such crisis intervention and psychotherapy. There were surprisingly few statistical studies on the effectiveness of psychotherapy with epileptics. Cobb⁵⁰ reported results of psychotherapy of 45 epileptics who were referred to him after the neurologist felt he could do no more for the patient; 67 percent had marked improvement and ten percent had slight improvement after psychotherapy. Gottschalk⁵¹ reported no seizure recurrences for two years in two of three cases given intensive psychotherapy.

Future research might take several directions to help resolve the controversy. There could be more use of blind evaluators to control for observer bias. The reports of stressful life events need to be controlled by comparison with other persons and with periods in the same person when he does not have seizures. Scalp EEG recordings may not be reliable, but depth electrode studies may be useful. This could ethically be done best during surgical procedures such as temporal lobectomy for control of seizures. Groethuysen et al52 used depth electrodes in studying an epileptic patient who had not had a seizure for 40 years. They observed a seizure when psychologically traumatic material was presented to the patient and the seizure activity was recorded near the amygdala. Papez⁵³ and McLean⁵⁴ have shown that the limbic system is associated with emotional experiences. Groethuysen's findings suggest the possibility that seizure activity may originate in the limbic system during emotional stress by activation of that system. This would be similar to activation of sensory areas in reflex epilepsy, in which sensory stimuli precipitate seizures. Future research might also investigate other suggested mechanisms. For example, Revitch⁵⁵ suggested that emotional precipitation of seizures is due to secretion of epinephrine, and Liberson⁵⁶ suggested acetylcholine release and hypoglycemia secondary to insulin release. Mattson et al⁵⁷ described three patients who unconsciously hyperventilated when anxious; EEG abnormalities and seizure activity increased at such times and decreased with administration of five percent carbon dioxide. Green⁵⁸ reported two patients who self-induced their seizures by hyperventilation. While animal studies of seizures have been done, such studies have not focused upon the induction of seizures by psychological stress. For example, a recent study showed that noxious stimuli such as pinching and electrical shock increased seizure expression-muscular jerkingwithout altering its electrocortical aspect.⁵⁹ Similar studies could be designed to see what effects psychological stress have on seizure expression. Thus, there are a number of directions which future research might take to help clarify the present controversy over the emotional precipitation of seizures.

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