Recognition of Pseudoseizures

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Failure to recognize pseudoseizures is a common problem, affecting both epileptic patients who additionally have pseudoseizures and nonepileptic patients inappropriately called epileptic. Pseudoseizures most commonly mimic generalized tonic-clonic (GTC, "grand mal") seizures or complex-partial seizures. Several patients whose pseudoseizures were not recognized are described. Adhering to logical principles of diagnosis for epilepsy, as for other medical problems, and remaining aware of basic behavioral dynamics should eliminate the confusion between pseudoseizures and epileptic seizures.

Many patients diagnosed and treated for long periods for "epilepsy" do not have epileptic seizures at all. The causes of pseudoseizures are myriad, arising at times from classical hysterical conversion reactions, at times from frank malingering, or at times as "acting out" behavior generated by various internal conflicts. It may be argued whether such episodes in fact should be called "seizures" at all. The term "pseudoseizure" is used here to designate such spells, implying only that they are not epileptic in origin. The widely used term "hysterical seizures" implies a specific etiology or dynamics which may not be accurate for most nonepileptic seizures.

The reason that an apparent epileptic seizure should be the specific symptom of a conversion or other psychological reaction is a complicated and fascinating question, for which there are as yet no definitive answers. However, once a diagnosis of epilepsy is made, particularly by the highly valued family physician, it is difficult to later convince a patient or family that certain episodic behavioral traits are not indeed epileptic.

The physician who treats patients with seizures should be aware of the international classification of seizures established by the World Health Organization. This current classification does more than merely provide a uniform terminology; it establishes the guidelines for a physiologic framework in understanding seizures (Table 1). Pseudoseizures most often resemble either generalized tonic-clonic seizures (GTC, "grand mal"), or complex-partial ("psychomotor," often assumed to be from the temporal lobe) seizures.

Case Illustrations

Four patients who suffer because of the misdiagnosis of epilepsy demonstrate the importance of recognizing pseudoseizures.

Case 1

B.W. is a 22-year-old right-handed seaman. He was sent to the hospital for evaluation because he

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Table 1. International Classification of Epileptic Seizures[1]

I. Partial Seizures (Seizures Beginning Locally)
  A. Partial Seizures with Elementary Symptomatology (Generally without Impairment of Consciousness)
    1. With Motor Symptoms (Includes Jacksonian Seizures)
    2. With Special Sensory or Somatosensory Symptoms
    3. With Autonomic Symptoms
    4. Compound Forms
  B. Partial Seizures with Complex Symptomatology (Generally with Impairment of Consciousness)
    1. With Impairment of Consciousness Only
    2. With Cognitive Symptomatology
    3. With Affective Symptomatology
    4. With "Psychosensory" Symptomatology
    5. With "Psychomotor" Symptomatology (Automatisms)
    6. Compound Forms
  C. Partial Seizures Secondarily Generalized
II. Generalized Seizures (Bilaterally Symmetrical and Without Local Onset)
    1. Absences (Petit Mal)
    2. Bilateral Massive Epileptic Myoclonus
    3. Infantile Spasms
    4. Clonic Seizures
    5. Tonic Seizures
    6. Tonic-Clonic Seizures (Grand Mal)
    7. Atonic Seizures
    8. Akinetic Seizures
III. Unilateral Seizures (Or Predominantly)

had several seizures on his ship. The product of a normal birth and childhood, he had no previous neurologic or medical illness. One brother was said to have had generalized tonic-clonic convulsions since age six, and was treated with medications. With each of B.W.'s spells, he would say to those around him, "I'm going to have a seizure," fall rapidly to the floor, flexed at the hips, knees, and neck, and then have violent, shaking movements. His arms would flail out to the side in dramatic movements. Often he would grab a bystander, frequently shouted, and invariably would strike some observer with his feet or hands. On one occasion he urinated during a spell.

When seen in neurologic examination, he was told that the vibrating tuning fork touched to his forehead might cause a seizure, but that the examining neurologist could "control" the seizure. When the tuning fork was applied to the forehead, the patient had sudden truncal flexion, he shouted a monosyllable, and began to flail about with all four extremities in random, but violent, groping movements. He grabbed the examining neurologist by the necktie and pulled him toward the examining table. When the patient's nose and mouth were held closed so that there was no airway, the patient opened his eyes quickly, and forcibly pushed the examiner's hands away from his face.

Several electroencephalograms were all normal, including one performed during a seizure.

Comment

It is important to note that this patient had a family history of epileptic seizures. The physician who made the initial diagnosis of epilepsy had interpreted this history to suggest a familial type of epilepsy. Many patients with pseudoseizures either have true epileptic seizures themselves in addition to their pseudoseizures, or have a family member with seizures.[2,3]

Although many seizures appear to occur in response to specific sensory inputs, the so-called "reflex" epilepsies, the suggestible onset of this patient's seizure should be noted. The maneuver of suggesting the onset of a seizure and using a vibrating tuning fork on the forehead only works in certain, very suggestible patients, but it is a useful way to expose pseudoseizures. Another important fact is that the patient having a GTC is invariably apneic, certainly not consciously aware of obstruction of the airway. The physiologic substrate of the epileptic seizure, an uncontrolled excessive electrical discharge from central nervous system (CNS) neurons, if generalized, must obliterate consciousness or conscious-purposeful behavior.

Therefore, it is impossible for a person having a generalized tonic-clonic convulsion to respond to such stimulus as closing the airway; a person who does respond is not having a GTC.

The most important observation of this and most other pseudoseizures interpreted as GTCs or "grand mal seizures" is the dramatic and violent flailing about in all directions. It is important to be aware of the usual sequence of events in the clinical propagation of a GTC. The patient becomes very rigid, and usually assumes a very extended posture, approaching a classical decerebrate posture. At this time, virtually all muscle groups, including the diaphragm and intercostal muscles are violently tonically contracted, and the EEG characteristically is dominated by generalized, high voltage, rapid spikes. Within a very short interval (2 to 30 seconds), there are repetitive, generalized jerks of flexor, then extensor, muscles in the trunk and extremities. These occur first slowly, often at one per second, becoming more rapid as the patient has a generalized clonic convulsion. The clonic movements then become less rapid and less rhythmical, the patient often emits a very deep terminal sigh, and remains postictally confused or somnolent for a variable period of time.

Although it is well known that a person may have real epileptic seizures despite normal interictal electroencephalograms (EEGs), when several records using appropriate provocative maneuvers and sleep are completed, fewer than ten percent of epileptic patients will have exclusively normal EEGs.[4] By contrast, it is exceedingly rare for the EEG to be normal while a true epileptic seizure is occurring. A normal EEG during an apparent GTC effectively eliminates an epileptic origin of that particular spell.[4]

Case 2

S.G. is a 34-year-old right-handed homemaker, mother of four. She came for neurologic evaluation because of momentary lapses of consciousness, particularly triggered by bright lights. She had majored in education in college, and had special academic interests in Special Education, which led her to become aware of features of epilepsy.

She had a negative neurological and medical history, and her neurologic examination was normal. During an electroencephalogram, at flicker rates above 8 Hz, she had sudden flexion at trunk, elbows, and wrists followed by generalized shaking of the two upper extremities and the head. These always ceased immediately after photic stimulation. Despite the patient's apparently convulsive movements during photic stimulation, the EEG was completely normal. On one occasion, videotape was performed while a flickering light precipitated one of her attacks. With the more careful review afforded by videotape, it was noted that the first movements occurring with her spells were rapid but deliberate lifting of the hands from the lap, supination of the hands and forearms, and folding of the arms across the chest. This lasted less than a second, and then was followed by the repetitive flexion movements of the trunk and arms. The legs were never involved in the otherwise widespread, bilateral, shaking movements.

On careful discussion with the patient afterward, it became apparent that she was currently depressed and anxious about recent difficulties in her marriage. She was also worried about her romantic and sexual role with her husband and her changing role as a mother while her children were approaching adolescence.

The psychiatric consultant who noticed a preoccupation with physical appearance, insecurity about sexual adequacy, suggestibility, and slightly seductive characteristics felt that the patient had many of the traits of the hysterical personality, and would probably relinquish her current symptoms as her recent adjustment reaction was successfully negotiated.

In the following seven months, without antiepileptic drugs, the patient's spells have disappeared. Two repeated EEGs have been normal.

Comment

Several important points can be made from this case. The patient was familiar with some of the characteristics of photic sensitive epilepsy. As with many patients with pseudoseizures, this was a person who had firsthand and rather thorough knowledge of the features of epilepsy. However, flicker-sensitive seizures almost never have focal or anatomically restricted clinical signs.[5,6] Furthermore, the EEGs of persons with flicker-sensitive seizures during a seizure are always abnormal. Although on rare occasions seizures due to focal cerebral disease can be triggered by flicker-

ing lights, flicker-sensitive seizures are usually caused by primary generalized epilepsy, a condition which usually begins in childhood between ages four and nine. To have photic-sensitive seizures develop at age 34 would be exceedingly unusual. If antiepileptic medication had been given to this patient during the first month of her complaint, the eventual disappearance of her pseudoseizures might easily have been attributed erroneously to the medication. Under those circumstances, it would be difficult to persuade the patient, or indeed many physicians, that the diagnosis of epilepsy was in error and that she should discontinue the antiepileptic drug. As the nature of her spells was recognized, inappropriate medication was avoided.

Case 3

B.G., a 35-year-old right-handed nulliparous woman was told at age 18 that she had "seizures," but not "epilepsy." At that time she was on active duty in the Navy, was depressed, and began to have episodes of nervousness at work manifested by tremulousness and anxiety, often followed by periods of unresponsiveness for 10 to 30 minutes. She was told these episodes were "petit mal" or "psychomotor" seizures. Neurologic evaluation at that time, accompanied by two electroencephalograms, was normal. She was treated with phenobarbital and trimethadione, but the spells persisted. Eventually, she was separated from the military service because of "epilepsy," for which she was treated for the ensuing 17 years.

Numerous medications were tried, including phenytoin, primidone, acetazolamide, mephobarbital, and carbamazepine. Her spells continued, however, and were particularly notable at times in her life when she was under emotional stress. She was referred for neurologic evaluation because her seizures began to interfere with her current job as a secretary/typist. She was taking primidone (phenobarbital concentration 25 μg/ml, primidone concentration 8 μg/ml), phenytoin (serum concentration 12 μg/ml), and carbamazepine (serum concentration 5.5 μg/ml). She was aware of mild acne, mild hirsutism of her face, and some gingival hypertrophy since initiating phenytoin eight years before, but she had tolerated these side effects in order to control her seizures. Despite these medications, at usually effective serum concentrations, the patient continued to have her spells.

By the time she was referred, she was having five to ten spells per day, usually lasting from 10 to 30 minutes. Her spells were characterized by unresponsiveness or prolonged lapses of alertness. She said that during her spells she could hear people speaking to her but the words often sounded "funny." However, during her spells, she would often carry on what appeared to observers to be normal conversations. On a few occasions, she would continue typing a letter, but at the end of the episode note that the words were all in error. The spells often lasted 30 to 60 minutes, never occurred while the patient was driving, and could always be associated within ten minutes of a stressful or angry discussion with another person. She never performed purposeless activities nor appeared confused to observers during her spells.

Eight electroencephalograms were normal over the previous 17 years. The only abnormal electroencephalogram was reported shortly before the patient was separated from military service. Review of that electroencephalogram later demonstrated that the discharges concluded to be epileptic by the initial interpreter were normal K-complexes during physiologic sleep.

In the hospital, her medications were discontinued gradually one at a time and several EEGs, including sleep, nasopharyngeal electrode, hyperventilation, and photic stimulation were all normal after the medications were discontinued. No detectable levels of anti-epileptic drugs could be noted in the blood. During one of the EEG recordings, the patient reported having one of her characteristic spells while the EEG was completely normal.

Subsequently, long-term EEG recordings were performed: one with 24-hour recording of eight EEG channels during waking and sleep while the patient was free to move about the hospital ward, and another eight-hour recording of simultaneous videotape and eight channels of EEG. During both of these recording sessions, the patient reported having several of her spells, which were not noted by observers. The EEG remained unchanged as the spells occurred.

As all of these results were discussed with the patient, she became angry that the physicians doubted her spells were epileptic in origin, and said that her doctors were implying that she "was crazy."

A psychiatric consultant noted that the patient had generally very good adaptive skills, despite some deep-seated sense of inadequacy and some hysterical personality traits. After two interviews with the psychiatrist, the patient had no more of her seizures in the ensuing five days, a longer episode-free interval than she had noted in the previous several years. In seven months of follow-up without medications, the patient has had no more spells.

Comment

This case illustrates several very important points. Although EEG is an invaluable tool in the diagnosis of epilepsy, injudicious reliance upon any laboratory report may interfere with sound clinical judgment. Unfortunately, in many laboratories in this country, EEGs are interpreted by persons with insufficient training in interpretation. Many normal patterns, such as those seen in adolescents or during sleep in all ages, are erroneously interpreted as epileptic by the unwary observer. Such a misinterpretation of normal sleep features influenced the management of this patient. It should be noted that the initial symptoms which were interpreted as epileptic were very probably anxiety attacks triggered by the patient's adolescent adjustment reaction at the time. After the diagnosis of epilepsy was made, and features of epilepsy were discussed with the patient, the nature of her spells flourished and grew more complicated, and she clung ever more firmly to the diagnosis of epilepsy. As they became more difficult to control, rather than questioning the diagnosis of epilepsy, her physicians added more and more medications.

Although complicated behaviors during complex-partial seizures are well recorded, there is usually a quality of aimlessness or confusion and randomness about the behaviors.[4,7-9] Complicated tasks have been documented, but these are distinctly the exceptions rather than the rule.[10] Therefore, any spell involving a complicated task such as playing music, traveling to a particular place, or typing a letter, should be called epileptic only with caution. In the case of this patient, the ability to strike all typewriter keys and return the carriage to original points at proper times did not really exclude the possibility of epilepsy, but should have raised some degree of hesitation. Likewise, although there may be only an alteration or blurring of consciousness during a complex-partial seizure, involvement of the limbic system is usually sufficient to provide some degree of amnesia for the seizure. The fact that this patient often could recall that people were speaking to her implies that memory was preserved during the spells. Also, her ability to carry on apparently normal conversations during the spell strongly argues against an epileptic seizure, particularly a complex-partial seizure in which consciousness is altered.

Case 4

B.F. was a 24-year-old right-handed male with frequent complex-partial seizures and a well-documented right anterior temporal seizure focus. His typical seizures began with interruption of activity, a look of exasperation or discomfort upon his face, and confused asking of single word questions such as "what?" or "who?" He then would wander confusedly around the room, lifting cushions from chairs or sofas, or looking under items of furniture. Sitting quietly in a chair he would fumble at the buttons on his shirt or trousers. These confused episodes lasted two to three minutes, and were followed by periods of confusion, lethargy, and depression, which would last 30 minutes to 2 hours.

In addition, when under stress or when angered, he had frequent episodes of staring blankly, not responding to speech. On most occasions, at the termination of such spells of unresponsiveness, he would grab the garments of persons near him and threaten violence or shake the person angrily. Afterward, he would shake his head and say, "where am I, what am I doing?" On several long-term, simultaneous EEG and videotape recordings, clear-cut seizure discharges emanating from the right temporal lobe could be seen preceding and occurring during the first type of seizure. However, during his staring spells, or violent outbursts, the EEG was completely normal.

Comment

The normal EEG during the staring spells and violent outbursts excludes an epileptic origin for these particular spells. Furthermore, there are strong theoretical, clinical, and physiological grounds to dismiss violent behavior as epileptic in origin,[11-13] despite concepts of the lay press to the contrary.

Table 2. Social Effects of Epilepsy

Popular Misconceptions About the Condition
Restrictions in Driving
Exclusions from Employment
Exclusion from Military Duty
Prohibition of Consumption of Alcohol
Difficulty (or High Costs) of Obtaining Life Insurance, Auto Insurance, and Health Insurance
Constant Expense for Medication
Potential Facial Effects from Medications (Hirsutism, Gingival Hypertrophy, Acne, Eczema, Loss of Hair)
Potential Sedative Effects of Medications (Barbiturates, Phenytoin, Carbamazepine, Clonazepam)
Potential Irritant-Hyperactive Effects of Medications (Barbiturates)

In managing this person's seizures, it would be important to recognize which of the spells were epileptic and which were not, in order to plan management and medication appropriately. If the epileptic seizures were adequately controlled with medication, and pseudoseizures were assumed erroneously to be epileptic, extra or additional medications might be added inappropriately. Niedermeyer et al demonstrated that toxicity from antiepileptic drugs may in fact *promote* pseudoseizures.[3]

It often appears curious that a person who suffers from the various physical and social repercussions of having epilepsy should "produce" apparent seizures from a psychological mechanism. However, it is possible for the epileptic person to view seizures as a type of punishment. For the person with guilt or remorse, a seizure may serve a punishing or redeeming function. Also, there are cases of persons with epilepsy who suspect their family or physician is not sensitive enough to their plight, and who feel that they must have more seizures in order that they might be understood or the severity of their illness appreciated. In fact, such motivations may cause a patient to avoid medications in order to have more seizures.[14]

Discussion

Other than seizures first revealed by status epilepticus or as part of an acute neurologic disease, there is very seldom a pressing need to initiate treatment for epilepsy until a diagnosis can be clearly established. Once a diagnosis of epilepsy is applied, for several reasons the patient is unlikely to relinquish the diagnosis easily. This is unfortunate because there are many social, as well as physical, repercussions of having epilepsy in current society (Table 2). Such repercussions are unfortunate and often unfair for the person who really has epileptic seizures, but especially should be spared the person who does not.

There are several fundamental principles which can distinguish pseudoseizures from epileptic seizures (Table 3). A spell characterized by violent movements of all four extremities but preserving consciousness can rarely be epileptic in origin. Likewise, when the violent movements of the extremities constitute a random flailing with asynchronous movements of the right and left side, an epileptic origin must be questioned. If the patient has violent movements of all four extremities, or appears unconscious, responsiveness to somatic stimulation may be pivotal. A person with a GTC cannot respond to obstruction of the airway. Therefore, a very important diagnostic test in the person with doubtable "grand mal" seizures is to pinch the nose and close the mouth firmly during an attack. If the person either awakens or responds by pushing the hands of the examiner away, the spell is not epileptic.

Often, a complex-partial seizure may be more difficult to distinguish from a pseudoseizure. A few important characteristics should be remembered, however. Goal directed behavior is not part of an epileptic disturbance. Therefore, the patient who in a fugue state was able to drive long distances in a car, follow traffic rules appropriately, and arrive at a destination (even a wrong one) without incident probably did not have an epileptic seizure. Certainly, such cases have been reported, but they are reportable by virtue of their bizarre deviation from the norm. The lay press, and even neuroscientists, have expounded and expanded upon affective features of complex-partial seizures. Indeed, the complex-partial seizure may cause foreign, unrecognized CNS discharges which, of course, elicit perplexed, unpleasant, and fearful subjective or emotional experiences for the

Table 3. Differential Features of Seizures and Pseudoseizures

	Generalized Tonic-Clonic (Grand Mal) Seizures	Complex Partial (Psychomotor) Seizures	Absence (Petit Mal) Seizures	Pseudoseizures
Mode of Onset	Very rapid or abrupt May be focal	May be slow or abrupt, may begin with confusion or other "aura" of vague premonition. Usually unpleasant	Instantaneous	Dramatic, may be in response to stressful situation
Duration	20 seconds-3 minutes	Few seconds-30 minutes	1-20 seconds	Usually at least a minute, highly variable, up to hours
Purposeful Behavior	Never	Only in a confused, incoordinated manner	Never	Very common, often violent or angry
Speech	Occasionally a grunt or shout at onset	Uncommon; confused, repetitive, or irrelevant when present	Never	Frequent. Often dramatic, tragic, or mystical
"Convulsive" Movements	First, tonic posture, then, generalized, symmetric clonic movements	May occur, usually focal. Clearly non-purposeful	Extremely rare	Often uncoordinated, flailing of all four extremities
Incontinence	Urine common, almost never feces	Urine sometimes, although not common. Never feces	Urine uncommon. Never feces	Urine frequent, feces not uncommon
Amnesia for Seizure	100%	May be aware of onset. Details rarely recalled	100%	Variable. Events of "seizure" sometimes recalled
Age of Onset	Any time after infancy	Any time after infancy	Childhood (4-9 years)	Usually adolescence to mid-twenties

patient. However, anger, rage, or violence are quite unusual manifestations of seizures, and some authorities doubt whether rage outbursts or violence are possible results of an epileptic seizure at all.[10-13] (Irritability often seen in a *post*ictal person may result in violent behavior, however.)

Despite the many debates about Dostoevsky's or Prince Mishkin's ecstasy or pleasure from seizures, Henri Gastaut has shown that pleasure to any degree has been only questionably documented in a few patients.[15] Therefore, experiences reported as pleasurable or ecstatic, however bizarre they might appear, are not likely to be epileptic.

The more common manifestations of epileptic seizures should not be obscured by exceptional possibilities. Therefore, very flamboyant or bizarre behavior, particularly when it involves criminal acts, violence, or profound secondary gain for the individual, should not too quickly be attributed to epilepsy. In fact, the behavior occurring in complex-partial seizures with psychomotor

activity is usually aimless, confused, and uncomplicated.[4] Such spells may last for several minutes, but rarely a half-hour or longer.

Role of Electroencephalogram

Certainly, the physician should not be restricted to the laboratory for making any diagnosis. This is especially true in the use of the EEG for evaluating seizures. However, a questionable seizure occurring in a patient with a normal EEG should be concluded as epileptic only with some trepidation. Even the interictal EEG, when bolstered appropriately with flashing lights, hyperventilation, and periods of sleep, will show an epileptiform abnormality in 60 to 90 percent of persons with epileptic seizures, particularly if more than one record is performed.[4,10] Especially, an EEG recorded during a seizure is important. Although there are cases reported in which implanted electrodes have demonstrated epileptic seizure activity which could not be recorded with conventional scalp EEG,[16] such cases are exceedingly rare, and have not been seen by most experienced electroencephalographers.[11,16,17] Therefore, when the EEG is normal while one of the characteristic spells occurs, the diagnosis of epilepsy should be challenged.

An inappropriate diagnosis of epilepsy may deprive a patient of necessary psychotherapy. In fact, an inappropriate diagnosis may fuel those pathologic mechanisms or internal conflicts responsible for the "spells." Accordingly, the diagnosis of epilepsy tends to be self-propagating, and the patient and family may be dissuaded from an inappropriate diagnosis later only with great difficulty. It should not be forgotten that antiepileptic medications, and indeed the diagnosis of epilepsy itself, may be harmful to the patient. As Niedermeyer reported, high doses of antiepileptic drugs may facilitate occurrence of hysterical seizures.[3] Many of the antiepileptic drugs have either sedative or other psychoactive properties. If pseudoseizures are a manifestation of anxiety or depression, to pharmacologically alleviate the underlying psychologic disturbance with a sedative drug may result in the erroneous conclusion that the drug worked because of antiepileptic properties.

Although the underlying physiology of epilepsy is not yet fully understood, and although every patient is of course a unique individual, there are logical and comprehensible principles that should be observed and careful diagnostic criteria and points of history that should be noted before diagnosis and treatment are hastily applied.

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