

Psychogenic Nonepileptic Seizures

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OVERVIEW

Psychogenic nonepileptic seizures (PNES) are routinely seen at epilepsy centers, where they represent 15% to 30% of patients referred for refractory seizures (1,2). They occur fairly often in the general population, with an estimated prevalence of 2 to 33 per 100,000 persons, making this condition nearly as common as multiple sclerosis (MS) or trigeminal neuralgia. In addition to being common, PNES represent a challenge, both in diagnosis and in management.

Terminology

The terminology used to describe PNES is variable and at times confusing. A number of terms have been used, including pseudoseizures, nonepileptic seizures, nonepileptic events, psychogenic seizures, and hysterical seizures. Strictly speaking, terms such as pseudoseizures, nonepileptic seizures, and nonepileptic events include both psychogenic and nonpsychogenic (i.e., organic) episodes that mimic epileptic seizures. Examples of nonpsychogenic episodes include syncope (the most common); paroxysmal movement disorders (e.g., dystonia); cataplexy; complicated migraines; and, in children, breath-holding spells and shuddering attacks. Terms such as psychogenic or hysterical seizures, on the other hand, refer to a subset of nonepileptic seizures with the connotation of a psychological origin. Use of the term hysteria has long since fallen into disfavor. The term psychogenic seizures could possibly be interpreted as epileptic seizures triggered or exacerbated by a psychological factor. For these reasons, PNES is the preferred term (3) and is used throughout this chapter.

The Misdiagnosis of Epilepsy

The erroneous diagnosis of epilepsy is relatively common. Approximately 25% of patients previously diagnosed with epilepsy and who are not responding to antiepileptic drug (AED) therapy are found to be misdiagnosed, both in epilepsy referral clinics (4,5) and in epilepsy monitoring units (1). Most patients misdiagnosed with epilepsy are eventually shown to have PNES (1,2) or, more rarely, syncope (6,7). Occasionally, other paroxysmal conditions can be misdiagnosed as epilepsy, but PNES are by far the most common condition, followed by syncope. Often, electroencephalograms (EEGs) that are interpreted as providing evidence for epilepsy contribute to this misdiagnosis (4,6,8). As is true with other chronic conditions (e.g., MS), whenever a wrong diagnosis of epilepsy has been given, it can be very difficult to “undo.” Unfortunately, once the diagnosis of “seizures” has been made, it becomes easily perpetuated without being questioned, which explains the usual diagnostic delay (9,10) and associated cost (11,12). It is disconcerting that despite the ability to render a diagnosis of PNES with near-certainty, the delay in diagnosis remains long, at about 7 to 10 years (9,10), indicating that neurologists may not have a high enough index of suspicion when AED treatment fails. This chapter begins by reviewing the steps involved in making that diagnosis and then turns to management considerations.

MAKING THE DIAGNOSIS

Suspecting the Diagnosis

PNES are initially suspected in the clinic on the basis of history and examination. A number of “red flags” are useful

in clinical practice and should raise the suspicion that seizures may be psychogenic rather than epileptic. Of course, resistance to AEDs can be the first clue and is usually the reason for referral to an epilepsy center. Most (approximately 80%) of the patients with PNES have been treated with AEDs for some time before the correct diagnosis is made (13). This is because a diagnosis of epilepsy is usually based solely on history and may be difficult, especially for nonneurologists (e.g., emergency department physicians and primary care physicians). A very high frequency of episodes that are completely unaffected by AEDs (i.e., no difference whether on or off medication) should also suggest the possibility of a psychogenic etiology. The presence of specific triggers that are unusual for epilepsy can be very suggestive of PNES, and this should be asked specifically when obtaining the history. For example, emotional triggers ("stress" or "getting upset") are commonly reported in patients with PNES. Other triggers that are suggestive of PNES include pain, certain movements, sounds, and lights, especially if they are alleged to *consistently* precipitate a "seizure." The circumstances under which attacks occur can be very helpful. Like other psychogenic symptoms, PNES tend to occur in the presence of an "audience," and, for example, occurrence in a physician's office or waiting room may be predictive of a psychogenic etiology (14). Similarly, PNES tend not to occur in sleep, although they may seem to and may be reported as such (15,16).

If the historian and witnesses are astute enough, the detailed description of the spells often includes characteristics that are inconsistent with epileptic seizures. In particular, some characteristics of the motor ("convulsive") phenomena are associated with PNES (see "Electroencephalogram-Video Monitoring"). However, witnesses' accounts are rarely detailed enough to describe the episodes accurately; in fact, even seizures witnessed by physicians and thought to be epileptic often turn out to be PNES. The patient's medical history can be useful as well. Although it has not been documented, coexisting poorly defined and "fashionable" (probably psychogenic) conditions, such as fibromyalgia, chronic pain, irritable bowel, or chronic fatigue, are associated with psychogenic symptoms. In a population referred for refractory seizures, a history of fibromyalgia or chronic pain has a strong association with a diagnosis of PNES (14). Similarly, a florid review of systems suggests somatization. A psychosocial history with evidence of maladaptive behaviors or associated psychiatric diagnoses should raise the level of suspicion of PNES. The examination, paying particular attention to mental status evaluation, including general demeanor and appropriate level of concern, overdramatization, and hysterical features, can be very telling, often uncovering such histrionic behavior as "give-way" weakness or "tight-roping." Performing the examination can, in itself, act as an "induction" in suggestible patients, making a spell more likely to occur during the history taking or examination.

By contrast, the presence of certain symptoms argues in favor of epileptic seizures and should warrant caution.

These include significant postictal confusion, incontinence, and, most important, significant injury (17–21). Although some injuries have been reported in PNES, data that describe injuries in patients with PNES are based largely on patients' self-reports (22). In particular, tongue biting is highly specific to generalized tonic-clonic seizures (18) and thus is a very helpful sign when present.

Confirming the Diagnosis

EEG and Ambulatory EEG

Because of its low sensitivity, routine EEG is not very helpful in diagnosing PNES. However, the presence of repeated normal EEGs, especially in light of frequent attacks and resistance to AEDs, certainly can be viewed as a red flag (23). Ambulatory EEG is increasingly used, is cost-effective, and can contribute to the diagnosis of PNES by recording the habitual episode and documenting the absence of EEG changes. However, because of the difficulties involved in conveying this diagnosis (see "Management"), it should always be confirmed by video-EEG monitoring.

Video-Electroencephalogram Monitoring

This is the gold standard for diagnosis of PNES (2,3,9,15–19,21), and, in fact, is indicated in all patients who continue to experience frequent seizures despite the use of AEDs (24). In the hands of experienced epileptologists, the combined electroclinical analysis of both the clinical semiology of the ictus and the ictal EEG findings allows a definitive diagnosis in nearly all cases. If an attack is recorded, the diagnosis is usually easy, and it is unusual that this question (i.e., PNES versus epilepsy) cannot be answered.

The principle of video-EEG monitoring is to record an episode and demonstrate that (a) there is no change in the EEG during the clinical event, and (b) the clinical spell is not consistent with seizure types that can be unaccompanied by EEG changes. Ictal EEG has limitations because it may be negative in simple partial seizures (25,26) and in some complex partial seizures, especially frontal ones (21). Ictal EEG may also be uninterpretable or difficult if movements generate excessive artifact.

Analysis of the ictal semiology (i.e., video) is at least as important as the ictal EEG, as it often shows behaviors that are obviously nonorganic and incompatible with epileptic seizures. Certain characteristics of the motor phenomena are strongly associated with PNES, including a very gradual onset or termination; pseudosleep; discontinuous (stop-and-go) activity; and irregular or asynchronous (out-of-phase) activity side-to-side head movement, pelvic thrusting, opisthotonic posturing, stuttering, and weeping (15–17,19,21,27–30). A particularly useful sign is preserved awareness during bilateral motor activity, which is relatively specific for PNES. This is because unresponsiveness is almost always present during bilateral motor activity, with the notable exception being supplementary motor area seizures (31,32).

Inductions

Provocative techniques, also known as activation procedures, or "inductions," can be extremely useful for the diagnosis of PNES, particularly when the diagnosis remains uncertain and no spontaneous attacks occur during monitoring. Many epilepsy centers use some sort of provocative technique to aid in the diagnosis of PNES (33,34). Some variability exists among the methods used. Although intravenous (IV) saline injection has traditionally been the most common (35–38), a number of other techniques have been described (39–42), which may be preferable (see below).

The principle behind provocative techniques is suggestibility, which is a feature of somatoform disorders in general. For example, in psychogenic movement disorders, where the diagnosis rests solely on phenomenology (i.e., there is no equivalent of the EEG), response to placebo or suggestion is considered a diagnostic criterion for *definite* psychogenic mechanism (43).

There are many advantages to the use of provocative techniques. First, when carefully studied and used simultaneously with EEG, their specificity approaches 100% (44). Second, difficult situations exist in which the combination of semiology (video) and the EEG does not allow one to conclude that an episode is psychogenic in origin. As mentioned earlier, two relatively common scenarios are (a) the ictal EEG is uninterpretable because of movement-related artifacts, and (b) the ictal EEG is normal, but the symptoms are consistent with a "simple partial" seizure. In these situations, the very presence of suggestibility (i.e., suggestion triggers the episode in question) is the strongest argument to support a psychogenic etiology. Third, at least theoretically, nonepileptic is not quite synonymous with psychogenic. The combination of a recorded attack and a normal ictal EEG qualifies as a nonepileptic spell but cannot in itself be categorized as psychogenic. On the other hand, a positive induction does stamp the episode as psychogenic, and even difficult-to-convince laypersons and attorneys understand this concept. Fourth, there is a strong economic argument for the use of these techniques, especially with the constraints imposed by third-party payers. When spontaneous attacks do not occur in the allotted time for monitoring, the evaluation may be inconclusive. In such situations, provocative techniques often turn an inconclusive evaluation into a diagnostic one.

The main limitation of provocative techniques is that they introduce ethical concerns. Several valid ethical arguments against placebo induction have been raised and acknowledged, making these techniques controversial (33,34,45,46). Of primary concern is the fact that physicians cannot honestly disclose the content of the syringe (for IV saline) or cannot say that the maneuver (e.g., tuning fork or patch) induces seizures. Even if the term "seizures" is then used in a broader sense, encompassing PNES, a degree of disingenuousness persists. The problem is particularly acute when a placebo is used, which results in

deceptive "beating around the bush." Thus, techniques that do not use placebo may be preferable, which circumvents these ethical problems while retaining similar diagnostic value (42,45). The best-documented technique uses a combination of hyperventilation, photic stimulation, and strong verbal suggestion (42,47). If hyperventilation is contraindicated or ill advised, counting aloud with arms raised will work equally well. The sensitivity is comparable to that with other methods, ranging from 60% to 90% (35–39,42,44,47). One major advantage of this technique is that hyperventilation and photic stimulation truly induce seizures, so that deception is not inherent to the procedure. Indeed, these maneuvers are performed during most EEGs, so that most patients will have undergone them previously. For this reason, patients or their families are not intrigued by the induction technique and do not ask about it (42). In fact, a comparable provocative technique using "psychiatric interview" was found not to be harmful and even useful by patients (39). Provocative techniques should only be performed along with video-EEG monitoring. Without the use of a placebo, provocative techniques are similar to other clinical maneuvers performed during the neurologic examination when nonorganic symptoms are suspected.

Short-Term Outpatient Video-EEG with Activation

An extension of the use of inductions is that when patients are strongly suspected, on clinical grounds, of having PNES, they can undergo outpatient "video-EEG with activation." This can be very cost-effective, while retaining the same specificity and a reasonably high level of sensitivity. In one published series, 10 of 15 patients had their habitual nonepileptic seizures with hyperventilation plus photic stimulation plus suggestion (47). In another study, short-term outpatient video-EEG with saline induction yielded a diagnosis in 60% of patients (48). At our center this is routinely used, and in two-thirds of cases the typical episode is obtained, thus obviating the need for "long-term" video-EEG monitoring (49).

DIFFICULT AND SPECIAL ISSUES IN DIAGNOSIS

Previous Abnormal Electroencephalogram

This is a very common problem. Many patients with PNES who are seen at epilepsy centers have had previous EEGs interpreted as epileptiform activity. When carefully reviewed, the vast majority turn out to be normal variants that were overinterpreted (8). In this situation, it is essential to obtain and review the actual tracing previously read as epileptiform activity, because no amount of normal subsequent EEGs will "cancel" the previous abnormal one. Unfortunately, obtaining prior EEGs can be difficult. First, records are not always available or accessible, and second,

digital electroencephalograph systems are incompatible with each other. In this regard, software that allows one to read *any* digital EEG format is very valuable and may become a necessity at referral epilepsy centers.

In children, coexisting benign focal epileptiform discharges of childhood (BFEDC) on the EEG are a common “red herring.” Such discharges are frequently seen in asymptomatic children and do not necessarily confirm that the reported episodes are epileptic. When epileptic seizures do occur in patients with perirolandic BFEDC on interictal EEG, they are usually facial sensorimotor or nocturnal generalized tonic clonic in nature. When the clinical presentation is mismatched with the expected manifestations of BFEDC—for example, in children with medically refractory “convulsions” or staring spells—video-EEG is appropriate to allow examination of the EEG during clinical events. In children with nonepileptic events, the “ictal” EEG will remain normal despite the BFEDC during interictal recording.

Coexisting Epilepsy

There is a widely held belief that many or most patients with PNES also have epilepsy. A careful review of the literature shows that this belief is inaccurate. Reports that have found high percentages of patients with PNES who also have epilepsy are based on loose criteria, such as an “abnormal EEG,” whereas those that required definite evidence for coexisting epilepsy found percentages between 9% and 15% (50,51).

Coexisting Organic Disease

A related phenomenon is that seizures are especially likely to be overdiagnosed as epileptic in patients with other organic neurologic diseases, such as MS, stroke, or antecedent brain surgery (52), or a history of head injury. For example, among patients in one study with traumatic brain injury diagnosed as posttraumatic epilepsy, 30% had psychogenic seizures instead (53). Thus, as is the general rule, if seizures do not respond to AEDs, a diagnosis of PNES should be considered despite the coexistence of organic disease. A diagnosis of PNES following some types of head injury may be particularly problematic if the injury involves litigation.

Psychogenic Nonepileptic Seizures After Epilepsy Surgery

PNES can occur following epilepsy surgery (54–56) and should always be considered if seizures recur and are somewhat different than they were preoperatively. In general, PNES tend to occur within 1 month after surgery (55). Risk factors include neurologic dysfunction in the right hemisphere, seizure onset after adolescence, low intelligence quotient (IQ), serious preoperative psychopathologic conditions, and major surgical complications (55,56).

Epilepsy Surgery in Patients with Psychogenic Nonepileptic Seizures

Occasionally, patients evaluated for epilepsy surgery also have PNES, triggered especially by activation procedures. Under the right circumstances, this is not a contraindication to surgery (57). If the epilepsy is refractory and the epileptic seizures are the most disabling ones, it may be appropriate to perform surgery to provide relief from the burden of seizures and high-dose AEDs, while approaching the PNES with psychiatric intervention.

PSYCHOPATHOLOGY

PNES are, by definition, a psychiatric disorder. According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) classification (58,59), physical symptoms caused by psychological causes can fall under three categories: somatoform disorders, factitious disorders, and malingering. Somatoform disorders are, by definition, the *unconscious* production of physical symptoms caused by psychological factors, which means that the symptoms are not under voluntary control—that is, the patient is not faking and not intentionally trying to deceive. Somatoform disorders are subdivided into several disorders, depending on the characteristics of the physical symptoms and their time course. The two somatoform disorders relevant to PNES are conversion disorder and somatization disorder. In fact, the DSM-IV added a new subcategory of conversion disorder (from the DSM-III-R), specifically termed conversion disorder with seizures. In contrast to the unconscious (unintentional) production of symptoms of the somatoform disorders (including conversion), factitious disorders and malingering imply that the patient is purposely deceiving the physician—that is, faking the symptoms. The difference between the two (i.e., factitious disorder and malingering) is that in malingering, the reason for doing so is tangible and rationally understandable (albeit possibly reprehensible), whereas in factitious disorder, the motivation is a pathologic need. An important corollary, therefore, is that malingering is not considered a mental illness, whereas factitious disorder is (58,59).

It is generally accepted that most patients with PNES fall under the somatoform category (unconscious production of symptoms) rather than the intentional faking type (malingering and factitious). However, although the DSM classification is simple in theory, it is nearly impossible to know if a given patient is faking. Intentional faking can only be diagnosed in some circumstances by catching a person in the act of doing so (e.g., self-inflicting injuries, administering medications or eye drops to cause signs, putting blood in the urine to simulate hematuria). Malingering may be underdiagnosed (60), partly because the “diagnosis” of malingering is essentially an accusation.

From a practical point of view, the role of the neurologist and other medical specialists is to determine whether organic disease exists. Once the symptoms are shown to be psychogenic in nature, the exact psychiatric diagnosis and its treatment are best handled by a psychiatrist.

The role of antecedent sexual trauma or abuse is thought to be important in the psychopathology of psychogenic seizures and psychogenic symptoms in general. A history of abuse may be more common in the convulsive, rather than the limp, type of PNES (61).

PROGNOSIS

Overall, the outcome in adults is tenuous. After 10 years of symptoms, more than half of patients continue to have seizures and remain dependent on social security benefits (62,63). The outcome is better in patients with greater educational attainments, younger age at onset and diagnosis, attacks with less dramatic features, fewer additional somatoform complaints, lower dissociation scores, and lower scores on the higher-order personality dimensions "inhibitedness," "emotional dysregulation," and "compulsivity" (63). The limp or catatonic type may have a better prognosis than the convulsive or thrashing type (64). Quality of life is severely affected in patients with PNES (65).

Duration of illness is probably the single most important prognostic factor in PNES—that is, the longer patients have been treated for epilepsy, the worse the prognosis (10,64,66). Thus, obtaining a definite diagnosis of PNES early in the course is critical. Currently, the average delay in the diagnosis of PNES remains long at 7 to 10 years (9,10), indicating that the index of suspicion for psychogenic symptoms may not be high enough. In addition, an accurate diagnosis of PNES also significantly reduces subsequent health care costs (12).

Overall, the outcome in patients with PNES is better in children and adolescents (67), probably because the duration of illness is shorter and the psychopathology or stressors are different from those in adults (66,68). School refusal and family discord may be significant factors. Serious mood disorders and ongoing sexual or physical abuse are common in children with PNES and should be investigated in every case.

MANAGEMENT

Role of the Neurologist or Epileptologist

The role of the neurologist or epileptologist does not end when the diagnosis of PNES is made. In fact, perhaps the most important step in initiating treatment is in the *delivery of the diagnosis* to patients and families (10,69–71). Most patients with psychogenic symptoms have received an initial diagnosis of organic disease (e.g.,

epilepsy), so that patients' reactions typically include disbelief and denial, as well as anger and hostility ("Are you accusing me of faking?" or "Are you saying that I am crazy?"). Written information can be useful in supplementing verbal explanations, but, unfortunately, patient information on psychogenic symptoms is rather scarce. Remarkably, the American Psychiatric Association (APA) has abundant patient education materials available on diverse topics, but none on somatoform disorders (72). Patient education materials on PNES are scarce but available (73). Patient education is particularly important in psychogenic symptoms. Unless patients and families understand and accept the diagnosis, they will not comply with recommendations. Therefore, communicating the diagnosis is critical. In fact, patients' understanding and reactions to the diagnosis have an impact on outcome (10).

Communicating the diagnosis is where the failure and breakdown often occur, and this is the main obstacle to effective treatment. Typically, physicians are uncomfortable with this diagnosis and tend to be uneasy formulating a conclusion. Reports frequently remain vague and fail to give clear interpretations, leaving the clinician hanging (e.g., "there was no EEG change during the episode" or "there is no evidence for epilepsy" or "seizures were nonepileptic"), with no explanations given to patients and families. In these situations, patients often continue to be treated for epilepsy, possibly with the understanding that the test was inconclusive. The diagnosis should be explained clearly, using unambiguous terms that patients can understand, such as "psychological," "stress-induced," or "emotional." The physician communicating the diagnosis must be compassionate (remembering that most patients are not faking), but firm and confident (avoiding "wishy-washy" and confusing terms).

The neurologist should also continue to be involved and not "abandon" the patient. The neurologist can assist in weaning patients off AED therapy, and may be helpful in addressing such issues as driving and disability. With regard to driving, few data are available, and there is no evidence that patients with PNES have an increased risk for motor vehicle accidents (74), probably for the same reason that they do not usually sustain serious injuries. Nevertheless, caution is advised, and each case should be evaluated individually and jointly by the neurologist and the mental health professional. Another sensitive issue is that of disability. PNES can be truly disabling, and this should be made clear. However, logic dictates that in these cases, a disability claim should be filed and justified on the basis of a *psychiatric diagnosis*, not a neurologic one. Another reason for the neurologist to continue following these patients is that one should keep an open mind about the possibility of coexisting epilepsy.

Role of the Mental Health Professional

Psychogenic symptoms are, by definition, a psychiatric disease, and mental health professionals should treat these patients. Treatment includes psychotherapy and adjunctive medications for coexisting anxiety or depression. Unfortunately, mental health services are not always easily available, especially for the uninsured. Another obstacle is that psychiatrists tend to be skeptical about the diagnosis of psychogenic symptoms, and even in patients with PNES in whom video-EEG monitoring allows a near-certain conclusion, they tend not to believe the diagnosis (75). A useful approach to combating this skepticism is to provide the treating psychiatrist with the actual video recordings of the PNES, as these can be more convincing than written reports.

PSYCHOGENIC NONEPILEPTIC SEIZURES IN CHILDREN

Although PNES are more common in adolescence, they may occur in children as young as 5 or 6 years of age. Most of what has been emphasized here applies to children as well as to adults. However, there are certain features specific to children. First, the differential diagnosis of seizures is broader in children, with many nonepileptic, nonpsychogenic conditions to be considered (76), including tics, breath-holding spells, and shuddering attacks. In addition, children experience nonepileptic staring spells (77), which are actually episodes of behavioral inattention that are misinterpreted by adults. The gender difference of female predominance is not observed until adolescence (78), and PNES are as common in preadolescent boys as in preadolescent girls. As described above, BFEDC are a common confounding feature on the interictal EEG, and the outcome in children and adolescents with PNES is generally better than that in adults (67).

PSYCHOGENIC NONEPILEPTIC SEIZURES IN PERSPECTIVE

The literature on PNES (at least the neurology and epilepsy literature) often gives the impression that PNES represents a unique disorder. In reality, PNES are but one type of somatoform disorder. How the psychopathology is expressed (PNES, paralysis, diarrhea, or pain) is only different in the diagnostic aspects. Fundamentally, the underlying psychopathology, its prognosis, and its management are no different with PNES than with other psychogenic symptoms. Whatever the manifestations, psychogenic symptoms represent a challenge both in the diagnosis and management.

Psychogenic (nonorganic, "functional") symptoms are common in medicine. Conservative estimates are that approximately 10% of all medical services are provided for

psychogenic symptoms (60). They are also common in neurology, representing approximately 9% of inpatient neurology admissions (79) and probably an even higher percentage of outpatient visits. Common neurologic symptoms that are found to be psychogenic include paralysis, mutism, visual symptoms, sensory symptoms, movement disorders, gait or balance problems, and pain (79–81). For several neurologic symptoms, signs or maneuvers have been described to help differentiate organic from nonorganic symptoms. For example, limb weakness is often evaluated by eliciting the Hoover sign, for which a quantitative version has been proposed (82). Other examples include looking for give-way weakness and alleged blindness with preserved optokinetic nystagmus. More generally, the neurologic examination often attempts to elicit signs or symptoms that do not make neuroanatomic sense (e.g., facial numbness affecting the angle of the jaw, gait with astasia-abasia, or tight-roping).

Every medical specialty has its share of symptoms that can be psychogenic. In gastroenterology, these include vomiting, dysphagia, abdominal pain, and diarrhea. In cardiology, chest pain that is noncardiac is traditionally referred to as "musculoskeletal" chest pain but is probably psychogenic. Symptoms that can be psychogenic in other medical specialties include shortness of breath and cough in pulmonary medicine, psychogenic globus or dysphonia in otolaryngology, excoriations in dermatology, erectile dysfunction in urology, and blindness or convergence spasms in ophthalmology. Pain syndromes for which a psychogenic component is likely include tension headaches, chronic back pain, limb pain, rectal pain, and pain in sexual organs. Of course, because pain is, by definition, entirely subjective, it is extremely difficult, and perhaps impossible, to ever confidently say that pain is psychogenic. It could even be argued that all pain is psychogenic, and thus psychogenic pain is one of the most "uncomfortable" diagnoses to make. In addition to isolated symptoms, some syndromes are considered to be at least partly psychogenic by some and possibly entirely psychogenic (i.e., without any organic basis) by others. These controversial but "fashionable" diagnoses include fibromyalgia, fibrositis, myofascial pain, chronic fatigue syndrome, irritable bowel syndrome, and multiple chemical sensitivity. As mentioned previously, there seems to be a relationship between fibromyalgia and PNES (14).

How are Psychogenic Nonepileptic Seizures Unique Among Psychogenic Symptoms?

Among psychogenic symptoms, PNES are unique in one main characteristic: with video-EEG monitoring, they can be diagnosed with near-certainty. This is in sharp contrast to other psychogenic symptoms, which almost always involve a diagnosis of exclusion. This feature allows a clarity and confidence of diagnosis that may assist in the criti-

cal step of convincing the patient and family of the nonorganic nature of the PNES.

REFERENCES

- Benbadis SR, Hauser WA. An estimate of the prevalence of psychogenic non-epileptic seizures. *Seizure* 2000;9:280-281.
- Benbadis SR, Heriaud L, O'Neill E et al. Outcome of prolonged EEG-video monitoring at a typical referral epilepsy center. *Epilepsia* 2004;45:1150-1153.
- Gates JR. Nonepileptic seizures: time for progress. *Epilepsy Behav* 2000;1:2-6.
- Smith D, Defalla BA, Chadwick DW. The misdiagnosis of epilepsy and the management of refractory epilepsy in a specialist clinic. *QJM* 1999;92:15-23.
- Scheepers B, Clough P, Pickles C. The misdiagnosis of epilepsy: findings of a population study. *Seizure* 1998;7:403-406.
- Eiris-Punal J, Rodriguez-Nunez A, Fernandez-Martinez N, et al. Usefulness of the head-upright tilt test for distinguishing syncope and epilepsy in children. *Epilepsia* 2001;42:709-713.
- Zaidi A, Clough P, Cooper P, et al. Misdiagnosis of epilepsy: many seizure-like attacks have a cardiovascular cause. *J Am Coll Cardiol* 2000;36:181-184.
- Benbadis SR, Tatum WO. Overinterpretation of EEGs and misdiagnosis of epilepsy. *J Clin Neurophysiol* 2003;20:42-44.
- Reuber M, Fernandez G, Bauer J, et al. Diagnostic delay in psychogenic nonepileptic seizures. *Neurology* 2002;58:493-495.
- Carton S, Thompson PJ, Duncan JS. Non-epileptic seizures: patients' understanding and reaction to the diagnosis and impact on outcome. *Seizure* 2003;12:287-294.
- Nowack WJ. Epilepsy: a costly misdiagnosis. *Clin Electroencephalogr* 1997;28:225-228.
- Martin RC, Gilliam FG, Kilgore M, et al. Improved health care resource utilization following video-EEG-confirmed diagnosis of nonepileptic psychogenic seizures. *Seizure* 1998;7:385-390.
- Benbadis SR. How many patients with pseudoseizures receive antiepileptic drugs prior to diagnosis? *Eur Neurol* 1999;41:114-115.
- Benbadis SR. A spell in the epilepsy clinic and a history of "chronic pain" or "fibromyalgia" independently predict a diagnosis of psychogenic seizures. *Epilepsy Behav* 2005;6:264-265.
- Benbadis SR, Lanctman ME, King LM, et al. Preictal pseudosleep: a new finding in psychogenic seizures. *Neurology* 1996;47:63-67.
- Thacker K, Devinsky O, Perrine K, et al. Nonepileptic seizures during apparent sleep. *Ann Neurol* 1993;33:414-418.
- Desai BT, Porter RJ, Penry JK. Psychogenic seizures. A study of 42 attacks in six patients, with intensive monitoring. *Arch Neurol* 1982;39:202-209.
- Benbadis SR, Wolgamuth BR, Goren H, et al. Value of tongue biting in the diagnosis of seizures. *Arch Intern Med* 1995;155:2346-2349.
- Guberman A. Psychogenic pseudoseizures in non-epileptic patients. *Can J Psychiatry* 1982;27:401-404.
- Hoefnagels WA, Padberg GW, Overweg J, et al. Transient loss of consciousness: the value of the history for distinguishing seizure from syncope. *J Neurol* 1991;238:39-43.
- Meierkord H, Will B, Fish D, et al. The clinical features and prognosis of pseudoseizures diagnosed using video-EEG telemetry. *Neurology* 1991;41:1643-1646.
- Peguero E, Abou-Khalil B, Fakhoury T, et al. Self-injury and incontinence in psychogenic seizures. *Epilepsia* 1995;36:586-591.
- Davis B. Predicting nonepileptic seizures utilizing seizure frequency, EEG, and response to medication. *Eur Neurol* 2004;51:153-156.
- Benbadis SR, Tatum WO, Vale FL. When drugs don't work: an algorithmic approach to medically intractable epilepsy. *Neurology* 2000;55:1780-1784.
- Devinsky O, Sato S, Kufta CV, et al. EEG studies of simple partial seizures with subdural electrode recordings. *Neurology* 1989;39:527-533.
- Sperling MR, O'Connor MJ. Auras and subclinical seizures: characteristics and prognostic significance. *Ann Neurol* 1990;28:320-328.
- Gates JR, Ramani V, Whalen S, et al. Ictal characteristics of pseudoseizures. *Arch Neurol* 1985;42:1183-1187.
- Gulick TA, Spinks IP, King DW. Pseudoseizures: ictal phenomena. *Neurology* 1982;32:24-30.
- Bergen D, Ristanovic R. Weeping as a common element of pseudoseizures. *Arch Neurol* 1993;50:1059-1060.
- Vossler DG, Haltiner AM, Schepp SK, et al. Ictal stuttering: a sign suggestive of psychogenic non-epileptic seizures. *Neurology* 2004;63:516-519.
- Kanner AM, Morris HH, Lüders H, et al. Supplementary motor seizures mimicking pseudoseizures: some clinical differences. *Neurology* 1990;40:1404-1407.
- Morris HH 3rd, Dinner DS, Lüders H, et al. Supplementary motor seizures: clinical and EEG findings. *Neurology* 1988;38:1075-1082.
- Schachter SC, Brown F, Rowan AJ. Provocative testing for nonepileptic seizures: attitudes and practices in the United States among American Epilepsy Society members. *J Epilepsy* 1996;9:249-252.
- Stagno SJ, Smith ML. Use of induction procedures in diagnosing psychogenic seizures. *J Epilepsy* 1996;9:153-158.
- Walczak TS, Williams DT, Berten W. Utility and reliability of placebo infusion in the evaluation of patients with seizures. *Neurology* 1994;44:394-399.
- Cohen RJ, Suter C. Hysterical seizures: suggestion as a provocative EEG test. *Ann Neurol* 1982;11:391-395.
- Bazil CW, Kothari M, Luciano D, et al. Provocation of nonepileptic seizures by suggestion in a general seizure population. *Epilepsia* 1994;35:768-770.
- Slater JD, Brown MC, Jacobs W, et al. Induction of pseudoseizures with intravenous saline placebo. *Epilepsia* 1995;36:580-585.
- Cohen LM, Howard GF 3rd, Bongar B. Provocation of pseudoseizures by psychiatric interview during EEG and video monitoring. *Int J Psychiatry Med* 1992;22:131-140.
- Luther JS, McNamara JO, Carwile S, et al. Pseudoepileptic seizures: methods and video analysis to aid diagnosis. *Ann Neurol* 1982;12:458-462.
- Riley TL, Berndt T. The role of the EEG technologist in delineating pseudoseizures. *Am J EEG Technol* 1980;20:89-96.
- Benbadis SR, Johnson K, Anthony K, et al. Induction of psychogenic nonepileptic seizures without placebo. *Neurology* 2000;55:1904-1905.
- Fahn S, Williams DT. Psychogenic dystonia. *Adv Neurol* 1988;50:431-455.
- Lanctman ME, Asconape JJ, Craven WJ, et al. Predictive value of induction of psychogenic seizures by suggestion. *Ann Neurol* 1994;35:359-361.
- Benbadis SR. Provocative techniques should be used for the diagnosis of psychogenic nonepileptic seizures. *Arch Neurol* 2001;58:2063-2065.
- Gates JR. Provocative testing should not be used for nonepileptic seizures. *Arch Neurol* 2001;58:2065-2066.
- McGonigal A, Oto M, Russell AJ, et al. Outpatient video EEG recording in the diagnosis of non-epileptic seizures: a randomised controlled trial of simple suggestion techniques. *J Neurol Neurosurg Psychiatry* 2002;72:549-551.
- Bhatia M, Sinha PK, Jain S, et al. Usefulness of short-term video EEG recording with saline induction in pseudoseizures. *Acta Neurol Scand* 1997;95:363-366.
- Benbadis SR, Siegrist K, Tatum WO, Heriaud L, Anthony K. Short-term outpatient EEG video with induction in the diagnosis of psychogenic seizures. *Neurology* 2004;63:1728-30.
- Benbadis SR, Agrawal V, Tatum WO 4th. How many patients with psychogenic nonepileptic seizures also have epilepsy? *Neurology* 2001;57:915-917.
- Lesser RP, Lüders H, Dinner DS. Evidence for epilepsy is rare in patients with psychogenic seizures. *Neurology* 1983;33:502-504.
- Reuber M, Kral T, Kurthen M, et al. New-onset psychogenic seizures after intracranial neurosurgery. *Acta Neurochir (Wien)* 2002;144:901-907.
- Hudak A, Agostini MA, Van Ness P, et al. Use of video-EEG monitoring in the differential diagnosis of posttraumatic seizure disorders. *Epilepsia* 2003;44(Suppl 9):5.

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54. Davies KG, Blumer DP, Lobo S, et al. De novo nonepileptic seizures after cranial surgery for epilepsy: incidence and risk factors. *Epilepsy Behav* 2000;1:436-443.
55. Glosser G, Roberts D, Glosser DS. Nonepileptic seizures after resective epilepsy surgery. *Epilepsia* 1999;40:1750-1754.
56. Ney GC, Barr WB, Napolitano C, et al. New-onset psychogenic seizures after surgery for epilepsy. *Arch Neurol* 1998;55:726-730.
57. Reuber M, Kurthen M, Fernandez G, et al. Epilepsy surgery in patients with additional psychogenic seizures. *Arch Neurol* 2002;59:82-86.
58. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders: DSM-III-R*, 3rd ed., rev. Washington, DC: Author, 1987.
59. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders: DSM-IV*, 4th ed. Washington, DC: Author, 1994.
60. Ford CV. The somatizing disorders. *Psychosomatics* 1986;27:327-337.
61. Abubakr A, Kablinger A, Caldito G. Psychogenic seizures: clinical features and psychological analysis. *Epilepsy Behav* 2003;4:241-245.
62. Lancman ME, Brotherton TA, Asconape JJ, et al. Psychogenic seizures in adults: a longitudinal analysis. *Seizure* 1993;2:281-286.
63. Reuber M, Pukrop R, Bauer J, et al. Outcome in psychogenic nonepileptic seizures: 1 to 10-year follow-up in 164 patients. *Ann Neurol* 2003;53:305-311.
64. Selwa LM, Geyer J, Nikakhtar N, et al. Nonepileptic seizure outcome varies by type of spell and duration of illness. *Epilepsia* 2000;41:1330-1334.
65. Szaflarski JP, Hughes C, Szaflarski M, et al. Quality of life in psychogenic nonepileptic seizures. *Epilepsia* 2003;44:236-242.
66. Gudmundsson O, Prendergast M, Foreman D, et al. Outcome of pseudoseizures in children and adolescents: a 6-year symptom survival analysis. *Dev Med Child Neurol* 2001;43:547-551.
67. Wyllie E, Friedman D, Lüders H, et al. Outcome of psychogenic seizures in children and adolescents compared with adults. *Neurology* 1991;41:742-744.
68. Wyllie E, Glazer JP, Benbadis S, et al. Psychiatric features of children and adolescents with pseudoseizures. *Arch Pediatr Adolesc Med* 1999;153:244-248.
69. Benbadis SR, Stagno SJ, Kosalko J, et al. Psychogenic seizures: a guide for patients and families. *J Neurosci Nurs* 1994;26:306-308.
70. McCahill ME. Somatoform and related disorders: delivery of diagnosis as first step. *Am Fam Physician* 1995;52:193-204.
71. Shen W, Bowman ES, Markand ON. Presenting the diagnosis of pseudoseizure. *Neurology* 1990;40:756-759.
72. American Psychiatric Association. 2001. Available at: http://www.psych.org/public_info/index.cfm, and http://psych.org/public_info/fact_sheets/dpa_fact.cfm. Last accessed July 11, 2005.
73. Benbadis SR, Heriaud L. Psychogenic (non-epileptic) seizures: a guide for patients & families. Available at: <http://hsc.usf.edu/com/epilepsy/PNESbrochure.pdf>. Last accessed July 11, 2005.
74. Benbadis SR, Blustein JN, Sunstad L. Should patients with psychogenic nonepileptic seizures be allowed to drive? *Epilepsia* 2000;41:895-897.
75. Harden CL, Burgut FT, Kanner AM. The diagnostic significance of video-EEG monitoring findings on pseudoseizure patients differs between neurologists and psychiatrists. *Epilepsia* 2003;44:453-456.
76. Wyllie E, Benbadis S, Kotagal P. Psychogenic seizures and other nonepileptic paroxysmal events in children. *Epilepsy Behav* 2002;3:46-50.
77. Rosenow F, Wyllie E, Kotagal P, et al. Staring spells in children: descriptive features distinguishing epileptic and nonepileptic events. *J Pediatr* 1998;133:660-663.
78. Kotagal P, Costa M, Wyllie E, et al. Paroxysmal nonepileptic events in children and adolescents. *Pediatrics* 2002;110:e46.
79. Lempert T, Dieterich M, Huppert D, et al. Psychogenic disorders in neurology: frequency and clinical spectrum. *Acta Neurol Scand* 1990;82:335-340.
80. Keane JR. Hysterical gait disorder: 60 cases. *Neurology* 1989;39:586-589.
81. Kapfhammer HP, Dobmeier P, Mayer C, et al. Conversion syndromes in neurology. A psychopathological and psychodynamic differentiation of conversion disorder, somatization disorder and factitious disorder [German]. *Psychother Psychosom Med Psychol* 1998;48:463-474.
82. Ziv I, Djaldetti R, Zoldan Y, et al. Diagnosis of "non-organic" limb paresis by a novel objective motor assessment: the quantitative Hoover's test. *J Neurol* 1998;245:797-802.