

Nonepileptic Behavioral Disorders: Diagnosis and Treatment

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ABSTRACT

Purpose of Review: This article will review the important steps in making an accurate diagnosis of psychogenic nonepileptic events or episodes (PNEE), and recent developments in diagnosis and treatment.

Recent Findings: Several clues can be obtained from the history to help the clinician suspect the diagnosis of PNEE. While none of these clues are diagnostic on their own, each is valuable, and there are often multiple clues in a given patient. Clinical clues have limitations, and once PNEE is suspected, video-EEG monitoring remains the gold standard and the only way to make a definite diagnosis of PNEE. Like most tests, video EEG has its limitations, but in most cases the diagnosis can be made and is not difficult. Regarding treatment, growing evidence exists that psychotherapy, especially cognitive behavior therapy, is effective, and a recent finding is that pharmacotherapy may have a role.

Summary: The diagnosis of PNEE can be made reliably, but the management of PNEE remains problematic, in large part because of the insufficient involvement of mental health professionals.

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INTRODUCTION

Seizures are common, and so are nonepileptic seizure mimics that are mistaken for seizures.¹ In fact, about a quarter of patients previously diagnosed with epilepsy who are not responding to drugs are found to be misdiagnosed.^{1,2} While the differential diagnosis of seizures can theoretically be broad,¹ the reality is that most patients misdiagnosed as having epilepsy who are seen at specialized epilepsy centers are eventually shown to have psychologically induced, psychogenic, or “behavioral” episodes. The terminology used can at times be confusing. Strictly speaking, terms such as pseudoseizures and nonepileptic seizures include both psychogenic and nonpsychogenic (ie, organic) episodes

that mimic epileptic seizures. Examples of nonpsychogenic episodes include syncope (the most common), paroxysmal movement disorders (eg, dystonia), cataplexy, complicated migraines, and (in children) breath-holding spells and shuddering attacks. On the other hand, terms such as psychogenic or behavioral events refer to the subset of nonepileptic seizures, adding the very important connotation of a psychological origin. In other words, *nonepileptic* is not synonymous with *psychogenic*. “Psychogenic nonepileptic seizures” has become the most often used term, but because the word *seizure* in this context creates confusion among patients and families, the author prefers (and recommends) to omit it and use terms

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KEY POINTS

- Psychogenic nonepileptic events or episodes are very commonly seen at epilepsy centers, where patients with psychogenic nonepileptic events or episodes represent about 30% of those referred for refractory seizures. In addition to being common, psychogenic nonepileptic events or episodes may represent a challenge in diagnosis and management, and many health care professionals are uncomfortable dealing with them.
- Despite the ability to make a diagnosis of psychogenic nonepileptic events or episodes with near certainty, the average delay in diagnosis remains long at about 7 to 10 years.

such as psychogenic nonepileptic events or episodes (PNEE), which will be used here.^{3,4} Psychogenic simply means “generated by the psyche” or “of psychological origin” and does not imply a specific type of psychological disturbance or diagnosis (eg, somatoform, conversion, dissociative, factitious, malingering, or anxiety disorder).

PNEE are very commonly seen at epilepsy centers, where patients with PNEE represent about 30% of those referred for refractory seizures.¹ In addition to being common, PNEE may represent a challenge in diagnosis and management, and many health care professionals are uncomfortable dealing with them. In addition, regardless of the condition that was misdiagnosed as seizures (psychogenic or not), the misdiagnosis of epilepsy has serious consequences. Unfortunately, a diagnosis of seizures is easily perpetuated without being questioned and is difficult to undo, which explains the usual diagnostic delay^{5,6} and its cost.⁷ Despite the ability to make a diagnosis of PNEE with near certainty, the average delay in diagnosis remains long at about 7 to 10 years,^{5,6} which suggests that treating neurologists do not have a

high enough index of suspicion. This article will first review the steps involved in making the diagnosis and then turn to management considerations.

SUSPECTING THE DIAGNOSIS

The diagnosis is initially suspected in the clinic on the basis of the history and examination. A number of “red flags” are useful and should raise the suspicion that so-called seizures may be psychogenic rather than epileptic (Table 8-1). Of course, resistance to antiepileptic drugs (AEDs) is usually the reason for referral to the epilepsy center, but the features shown in Table 8-1 should raise the suspicion that the episodes in question may be psychogenic rather than indicative of intractable epilepsy. Knowledge of the circumstances in which attacks occur can be very helpful. Like other psychogenic symptoms, PNEE tend to occur in the presence of an audience, and occurrence in the physician’s office or the waiting room or during the examination is suggestive of PNEE.⁸ A detailed description of the events often includes characteristics that are inconsistent with epileptic seizures. However, description by witnesses may

TABLE 8-1 Historical Features That May Suggest a Psychogenic Origin

- ▶ A dramatically high frequency of episodes that is completely unaffected by antiepileptic drugs
- ▶ Specific triggers that are unusual for epilepsy—including emotional triggers (stress or becoming upset), pain, movements, sounds, or lights—especially if they are alleged to *consistently* trigger a seizurelike episode
- ▶ Episode in the doctor’s office or waiting room
- ▶ History of “fibromyalgia” or unexplained chronic pain
- ▶ Florid review of systems
- ▶ A psychosocial history with evidence for maladaptive behaviors or associated psychiatric diagnoses

be inaccurate and have obvious limitations,^{9–11} so that the characteristics of the motor (convulsive) phenomena are best assessed during video EEG (see below under Video-EEG Monitoring). Increasingly, patients and family can provide video recordings (on mobile phones or cameras), which can be almost as helpful as video-EEG monitoring. Chronic pain or “fibromyalgia” diagnoses are often associated with psychogenic symptoms, and the presence of these diagnoses can have a high predictive value of 70% to 80%.⁸ Some other (often poorly documented) diagnoses, such as chronic fatigue, multiple allergies, or Lyme disease, often have the same value. The examination—especially mental status evaluation including the general demeanor, appropriate level of concern, overdramatization, or histrionic features—can be very telling. Lastly, the examination often uncovers inorganic signs or behaviors such as give-way weakness or atasia-abasia. 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, including significant postictal confusion, incontinence, occurrence in sleep, and significant injury. In particular, tongue biting is highly specific to generalized tonic-clonic seizures¹² and is therefore a very helpful sign when present. PNEE tend to not occur in sleep, although they may seem to and may be reported as doing so.¹³ Postictal stertorous breathing is quite specific for convulsive epileptic seizures.^{14,15} Some of the signs associated with seizures, such as tongue biting, injuries, and incontinence, can be reported by patients with PNEE. Obviously these are much more specific if they are documented rather than reported.

CONFIRMING THE DIAGNOSIS

Routine EEG and Ambulatory EEG

Because of low sensitivity, routine EEG is not very helpful in making a diagnosis of PNEE. The presence of repeated normal EEGs should raise suspicion, however, especially in light of frequent attacks and resistance to medications.¹⁶ Ambulatory EEG is increasingly used, is cost effective, and can contribute to the diagnosis by recording the habitual episode and documenting the absence of EEG changes. However, because ictal EEG can only be interpreted in the context of the video, and because of the difficulties in conveying this diagnosis (see MANAGEMENT, below), a diagnosis of PNEE should always be confirmed by video-EEG monitoring. If technically adequate, home or ambulatory EEG with video may become as good as inpatient video EEG in the future.

Video-EEG Monitoring

Video-EEG monitoring is without question the gold standard for diagnosis,^{1,17} and is indicated in all patients who continue to have frequent seizures despite medications. 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 episode is recorded, the diagnosis is usually easy, providing a clear answer concerning the question of PNEE versus epilepsy. Furthermore, the procedure has a high diagnostic yield, and most patients have their first event in the first 2 days.¹⁸

The principle of video-EEG monitoring is to record an episode and demonstrate that (1) there is no change in the EEG during the clinical event, and (2) the clinical episode is not consistent with a frontal lobe partial seizure or other epileptic seizure types

KEY POINTS

- The presence of certain symptoms argues in favor of epileptic seizures, including significant postictal confusion, incontinence, occurrence in sleep, and significant injury. In particular, tongue biting is highly specific to generalized tonic-clonic seizures.
- A diagnosis of psychogenic nonepileptic events or episodes should always be confirmed by video-EEG monitoring.
- Video-EEG monitoring is the gold standard for diagnosis of psychogenic nonepileptic events or episodes.

that may lack characteristic EEG changes. Ictal EEG has limitations because it may be negative in some partial seizures, especially those without alteration of awareness.^{19,20} Ictal EEG may also be uninterpretable or difficult if movements generate excessive artifact (see below under Pitfalls of Video EEG).

Analysis of the ictal semiology (ie, video) is at least as important as the ictal EEG because the video often shows behaviors that are obviously nonorganic and incompatible with epileptic seizures. The most important behavioral features of PNEE are shown in **Table 8-2**. None of these features has 100% specificity or is completely diagnostic, and all should be interpreted with caution. For example, preserved awareness during bilateral motor activity is very useful because unresponsiveness is almost always present during epileptic bilateral motor activity, although a notable exception is supplementary motor area seizures. Similarly, the value of eye closure has been ques-

tioned²¹ but remains high in predicting PNEE,²² especially when prolonged and with complete unresponsiveness. Most patients with PNEE show more than one of these behavioral characteristics, often making the diagnosis relatively easy. A simple and practical semiologic classification divides PNEE into six types: rhythmic motor, hypermotor, complex motor, dialeptic, subjective, and mixed.²³ The presentation as limp immobile unresponsiveness with eyes closed (ie, pseudosyncope) presents unusual challenges, because such patients often see cardiologists rather than neurologists and are rarely sent for video-EEG monitoring. Many cases of “syncope of unknown origin” could possibly be undiagnosed psychogenic episodes.²⁴ When recorded in the epilepsy monitoring unit (EMU), the diagnosis of psychogenic syncope is not difficult, because these episodes can be induced by suggestion whereas true syncope shows a reliable series of ictal EEG changes.²⁴

TABLE 8-2 The Most Useful/Specific Semiologic (Ictal) Features Suggestive of Psychogenic Nonepileptic Events

- ▶ Pseudosleep
- ▶ Discontinuous (stop-and-go) activity
- ▶ Irregular or asynchronous (out-of-phase) activity including side-to-side head movement
- ▶ Nonclonic shaking with variable rhythm and direction
- ▶ Pelvic thrusting
- ▶ Opisthotonic posturing
- ▶ Stuttering
- ▶ Weeping
- ▶ Preserved awareness during bilateral motor activity
- ▶ Ictal eye closure
- ▶ Prolonged immobile unresponsiveness with eyes closed (pseudosyncope)
- ▶ Postictal whispering or other partial motor responses

Inductions

Provocative techniques, or inductions, can be particularly helpful in cases when the monitoring period has failed to record any spontaneous attacks and the diagnosis remains uncertain. Many epilepsy centers use these techniques as an additional method for diagnosing PNEE. IV saline was commonly used for some time, but other modalities are now preferred as a result of ethical concerns associated with IV placebo. The principle of suggestibility (a feature of all somatoform disorders) is crucial to all provocative techniques. In psychogenic movement disorders, for example, where the diagnosis is based entirely on phenomenology, response to induction is considered a strong diagnostic criterion for a psychogenic etiology.²⁵

The chief advantage of provocative techniques is a very high specificity, especially when combined with ictal video-EEG monitoring.²⁶ In addition, there are situations where the combination of semiology (on video) and ictal EEG is inconclusive regarding whether an episode is psychogenic in origin (eg, movement-related artifacts may render the ictal EEG uninterpretable). Symptoms may also be consistent with a simple partial seizure, which can be accompanied by a normal ictal EEG. The presence of suggestibility (ie, suggestion triggers the episode in question) in such situations strongly supports a psychogenic etiology. Lastly, the ability of these techniques to turn an inconclusive evaluation (with no episode recorded) into a diagnostic one may provide an economic argument in favor of utilizing them.

Potential ethical concerns form the only disadvantage of provocative techniques, including several valid ethical arguments that have been raised against placebo induction.^{27,28}

Techniques that avoid the use of placebo are preferable because, while they retain similar diagnostic value, they generally circumvent ethical problems.²⁸⁻³⁰ The technique that is most thoroughly documented combines hyperventilation, photic stimulation, and verbal suggestion;^{29,30} counting aloud with arms raised can be used in cases where hyperventilation is contraindicated. The sensitivity (ranging from 60% to 90%) is comparable to other induction techniques. One major advantage of this method is that hyperventilation and photic stimulation are used during routine EEGs so that patients will not be intrigued by these procedures.^{28,29} In fact, patients found a similar provocative technique using psychiatric interview to be not harmful and even useful.³¹ Video-EEG monitoring should always be performed in conjunction with provocative techniques. Without the use of a placebo, most of the objections against inductions are theoretical and become far outweighed by the practical consequences of perpetuating a wrong diagnosis of epilepsy.

Short-Term Outpatient Video EEG with Activation

When patients are strongly suspected to have PNEE on clinical grounds, outpatient video EEG with activation is a very useful and cost-effective extension of induction techniques that retains high specificity and sensitivity,^{30,32} including in the veteran population.³³

Pitfalls of Video EEG

The most obvious limitation of ictal EEG is that it may be negative in some partial seizure types. Knowing which types of clinical seizures may be unaccompanied by ictal EEG changes is therefore critical in avoiding errors. The most common type of seizures that are unaccompanied by ictal EEG

KEY POINTS

- Many patients with psychogenic nonepileptic events or episodes seen at epilepsy centers have had previous EEGs, and often at least one of these was interpreted as epileptiform.
- When reviewed, the vast majority of EEGs interpreted as epileptiform in patients with psychogenic nonepileptic events or episodes will turn out to show overinterpreted normal variants.

changes are those without impairment of awareness—that is, “simple partial” seizures having purely subjective phenomena (ie, auras). Motor simple partial seizures may include focal clonic seizures and brief tonic seizures, typically of frontal lobe origin; they are usually brief (5 to 30 seconds) and tonic, or may be hypermotor, but not usually as dramatically flailing or thrashing as PNEE. If multiple episodes are recorded, stereotypy (ie, highly similar behavioral features between seizures) is a feature that strongly suggests epileptic seizures rather than PNEE. Ictal EEG may be uninterpretable if movements generate excessive artifact. In those situations, it can be impossible to “prove” that such episodes are psychogenic. For example, brief episodes of déjà vu or fear or tonic stiffening with no EEG changes can never be proven to be psychogenic. Arguments in favor of PNEE include suggestibility (triggered by placebo maneuvers), or events that never progress to clear seizures. Lastly, PNEE episodes do not occur during sleep, so attacks that arise out of EEG-verified sleep may reliably be diagnosed as organic (ie, epileptic seizures or parasomnias). Epileptic seizures with altered awareness and no EEG changes are very rare but exist, and if the clinical events are strongly suggestive of seizures, it is best to err on the side of treating them as epileptic.

Of course, *nonepileptic* does not always mean *psychogenic*, and other diagnoses must be considered before making a diagnosis of PNEE.¹ Common nonepileptic organic causes to consider are syncope and paroxysmal movement disorders for episodes that occur while awake and parasomnias for episodes that occur in sleep.

A common misconception is that a recorded episode with a negative EEG is all it takes to make a diagnosis of PNEE. This is of course grossly inac-

curate. A negative EEG can only be interpreted in the context of the semiology of the attack in question. Therefore, both the video and EEG must be available—in fact the diagnosis would probably be more accurate with video alone than with EEG alone. When used properly, video EEG allows the diagnosis of paroxysmal seizurelike events, and in particular the diagnosis of PNEE, with a high degree of confidence. A study of the inter-rater reliability of the diagnosis by video EEG, sampling a group of epileptologists, found a good inter-rater agreement,¹⁷ indicating that there is a certain component of subjective artful judgment. Results also confirmed that there was very good agreement on the vast majority of cases, which indicates that the merely “good” agreement was accounted for by a small handful of difficult cases.

DIFFICULT AND SPECIAL ISSUES IN DIAGNOSIS**Previous Abnormal EEG**

A very common problem, illustrated in **Case 8-1**, is previous abnormal EEG results. Many patients with PNEE seen at epilepsy centers have had previous EEGs, and often at least one was interpreted as epileptiform. In this situation, illustrated by **Case 8-2**, it is essential to obtain and review the actual tracing previously read as epileptiform, since no amount of normal subsequent EEGs will invalidate the supposedly abnormal one. When reviewed, the vast majority will turn out to show overinterpreted normal variants.³⁴ By far the most common errors in EEG interpretation, and the main source of over-reading, are benign temporal sharp transients or wicket rhythms that are read as temporal spikes. The same errors in diagnosis occur for benign, nonspecific episodic symptoms not even suggestive of seizures (eg, lightheadedness,

Case 8-1

A 30-year-old woman was referred for a 7-year history of seizures that continued despite several antiepileptic drugs. She had a diagnosis of chronic pain and “fibromyalgia” and was on multiple pain medications. Outside records documented several seizurelike events in the waiting room of her primary care physician and neurologist. Video EEG confirmed the suspected diagnosis of psychogenic nonepileptic events/episodes (**Supplemental Digital Content 8-1**, links.lww.com/CONT/A44). The episode was induced by activation (photic stimulation and verbal suggestion). Note the multiple features suggestive of the diagnosis: whole body side-to-side low-amplitude nonclonic trembling, which is initially horizontal but later changes to vertical (variability in rhythm and direction); the eyes continuously closed; the side-to-side head shaking; and the asynchronous leg movements (bicycling).

Comment. This case illustrates typical features: delayed diagnosis, chronic pain, and an episode in the waiting room (as summarized in **Table 8-1**). The recorded episode also shows several typical features of the episode itself (as summarized in **Table 8-2**).

Case 8-2

A 46-year-old woman was diagnosed (with video EEG) with clear psychogenic nonepileptic events or episodes (PNEE) but had a prior EEG that reportedly “showed epilepsy.” The report indicated “rare right temporal spikes,” so a doubt about coexisting epilepsy persisted. With some difficulties (eg, software compatibility), the author was able to obtain and view the EEG in question, and the “spikes” are shown here (**Figure 8-1**). These benign fluctuations on background activity in the temporal region are the most common over-read patterns (see reference 34 for further discussion).

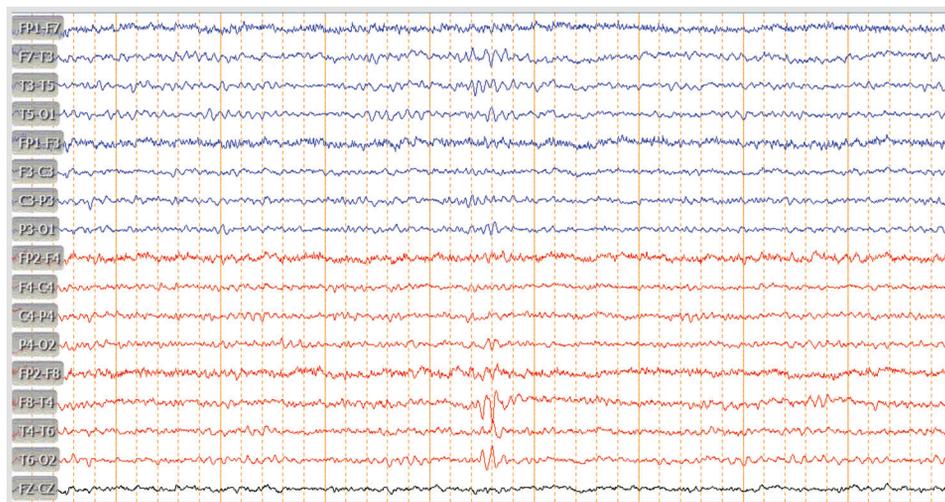


FIGURE 8-1 Routine bipolar “double banana” montage. This sample shows the benign sharp transient with phase reversal at T4, which was over-read as a “spike.”

Comment. As discussed in the text, it is common for a prior EEG to be overinterpreted as abnormal, making the diagnosis of seizures more difficult to “undo.”

dizziness, weakness, and numbness), resulting in the diagnosis or misdiagnosis of seizures being entirely based on the over-read EEG.

Psychogenic Nonepileptic Events or Episodes in an Unexpected Setting

There are several situations in which the degree of suspicion for PNEE may be a priori lower, especially when there is a previous diagnosis of epilepsy or comorbid organic disease involving head injury. A widely held yet erroneous belief persists that many or most patients with PNEE also have epilepsy. Reports that have found high percentages of patients with PNEE to also have epilepsy are based on loose criteria (such as “abnormal EEG”), whereas those that require definite evidence for coexisting epilepsy have found percentages between 9% and 15%.³⁵ These patients present obvious management difficulties. The assumption that patients with PNEE also have epilepsy is a common “cop-out” strategy to justify the use of AEDs.

Seizures are also especially likely to be overdiagnosed as epileptic in patients with other known organic neurologic diseases, such as multiple sclerosis, stroke, or antecedent brain surgery.³⁶ For example, in patients with moderate to severe traumatic brain injury diagnosed with post-traumatic epilepsy, 30% are found to have PNEE instead.³⁷ PNEE after head injury are particularly thorny because they often involve litigation.

Several specific patient populations may be likely to be overdiagnosed with epileptic seizures, including veteran populations, the elderly, and those having previous epilepsy surgery. Since many veterans have had previous traumatic brain injury, many are assumed to have post-traumatic

epilepsy, which probably explains the prolonged diagnostic delay in this population.^{33,38} PNEE, like other psychogenic symptoms, tend to begin in younger patients but may occur and even begin in older patients.³⁹ PNEE may also occur after epilepsy surgery⁴⁰ and should always be considered if seizures recur and are somewhat different than preoperatively.

A potential problem in accurate diagnosis in each of these scenarios is that the patient's episodes are assumed to be epileptic. Assuming a diagnosis of epilepsy without proof is also exemplified by PNEE patients receiving seizure dogs,⁴¹ and patients receiving vagus nerve stimulators,⁴² both of which can also be found, once monitored, to have PNEE.

PSYCHOPATHOLOGY

PNEE are by definition a psychiatric disorder. According to the *Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition) (DSM-IV)* classification,⁴³ 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 due to psychological factors, which means that symptoms are not under voluntary control (ie, 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. Thus far, the two somatoform disorders relevant to PNEE have been conversion disorder and somatization disorder. In fact, the *DSM-IV* has a subcategory of conversion disorder specifically termed conversion disorder with seizures. By contrast to the unconscious, unintentional production of symptoms of

the somatoform disorders (including conversion), factitious disorder and malingering imply that the patient is purposely deceiving the physician—that is, faking the symptoms. The difference between factitious disorder and malingering is that in malingering the reason for doing so is tangible and rationally understandable, while in factitious disorder the motivation is a pathologic need for the sick role. An important corollary, therefore, is that factitious disorder is considered to be a mental illness, while malingering is not.⁴³ The *DSM* classification is evolving, and the fifth edition (*DSM-V*) is currently under development^{44,45} with release planned for May 2013. The *DSM-V* will have a more inclusive category named somatic symptom disorders, which includes not only somatoform disorders but also psychological factors affecting medical conditions (psychosomatic), and factitious disorders. In addition, because somatization disorder, hypochondriasis, undifferentiated somatoform disorder, and pain disorder share certain common features (namely, somatic symptoms and cognitive distortions), they may be grouped under the common rubric of complex somatic symptom disorders. Most often in clinical practice PNEE will fit best under conversion or somatization disorders (unconscious production of symptoms), with only a small minority under factitious disorder or malingering (intentional feigning). In the *International Classification of Diseases, 10th Revision (ICD-10)*,⁴⁶ somatoform disorders are found in chapter V, titled “Mental and behavioural disorders”—specifically sections F40–F48, titled “Neurotic, stress-related and somatoform disorders.” Interestingly, section F44 combines dissociative and conversion disorders and specifically includes dissociative convulsions (F44.5). From a practical point of view, the role of the

neurologist is to determine whether there is organic disease. Once the symptoms are shown to be psychogenic, the subtleties of the psychiatric diagnosis and its treatment are (or should be) best handled by mental health professionals.

The role of a clear antecedent traumatic experience is thought to be important in the psychopathology of PNEE and psychogenic symptoms in general. About three-quarters of patients report antecedent traumatic factors, such as abuse, bereavement, health-related trauma, and accident or assault. Sexual abuse in particular is associated with more severe situations, including self-harm, other medically unexplained symptoms, more features suggestive of epilepsy (eg, convulsive and more severe attacks, nocturnal attacks, injuries, incontinence), more emotional triggers and flashbacks, and more disability.^{47,48}

Patients with PNEE perform similarly on measures of effort compared to intractable epilepsy patients,⁴⁹ supporting the notion that the vast majority are not in the consciously faking category. In addition, Minnesota Multiphasic Personality Inventory (MMPI) findings are also poor in discriminating between patients with PNEE and those with intractable epilepsy.⁵⁰ Therefore, while psychological profiles may be useful for treatment strategies, they are not particularly helpful for diagnosis.

PROGNOSIS

In adults, the outcome of PNEE is mediocre.^{51,52} Over half of patients continue to have seizurelike events and remain disabled after 10 years of symptoms. Patients with higher education, younger age of onset and diagnosis, events with less dramatic features, fewer additional psychogenic symptoms, lower dissociation scores, and lower scores on the personality

dimensions *inhibitedness*, *emotional dysregulation*, and *compulsivity* may have better outcome.^{40,50} Quality of life is severely affected in patients with PNEE.⁵³ Importantly, improvement in the seizurelike events does not necessarily translate into overall improvement or productivity, as the underlying psychopathology may not be improved.⁵⁴ The single most important prognostic factor is likely to be the duration of illness—that is, the prognosis grows worse the longer the patient has been treated for epilepsy.^{6,55} Therefore, making a definite diagnosis of PNEE early after onset is crucial.

MANAGEMENT

The Role of the Neurologist

The role of the neurologist should continue after the diagnosis is made. In fact, the initial delivery of the diagnosis to patients and families is probably the most important step in initiating treatment;⁶ patients and families are not likely to comply with recommendations unless they understand and accept the diagnosis. Because patients' understanding of and reactions to the diagnosis can even affect outcome, the neurologist's communication of the diagnosis is vital.⁶ Most patients have carried a diagnosis of epilepsy, so the reactions can be negative (eg, disbelief, denial, anger). Written information can be useful in supplementing verbal explanations.⁵⁶

In practice, delivering and explaining the diagnosis can frequently be the main obstacle to treatment. Physicians are often uncomfortable with the diagnosis and tend to hesitate when formulating a conclusion, thus giving reports that may remain euphemistically vague and offer no clear conclusion (eg, "There was no EEG change," or "There is no evidence for epilepsy"); as a result, patients and their families can be left without clear

explanations. In these situations, patients typically continue to be treated for epilepsy, often under the mistaken impression that their tests were inconclusive or that they have a rare disease. The physician should explain the diagnosis clearly using unambiguous terms (eg, "psychological," "stress-induced") that laypeople can understand. In delivering the diagnosis, the physician must be compassionate (keeping in mind that most patients are not faking) but firm and confident (avoiding ambiguous and perplexing terms).

The neurologist should continue to be involved and can assist in weaning AEDs and addressing issues such as driving and disability. Few data are available regarding PNEE and driving, and no evidence indicates that patients with PNEE have an increased risk of car accidents⁵⁷ (most likely for the same reason that they do not usually sustain serious injuries). Nevertheless, caution is warranted, and each patient merits consideration in this regard on an individual basis with input from the neurologist (and ideally also the mental health professional). Disability is another difficult issue; PNEE can be disabling, but disability should ideally be determined and filed on the basis of a psychiatric rather than a neurologic diagnosis. The ever-present possibility of coexisting epilepsy provides another reason for the neurologist to continue following these patients.

The Role of the Mental Health Professional

Psychogenic symptoms are by definition a psychiatric disease, which mental health professionals should treat. Treatment includes psychotherapy and psychotropic medications.^{58,59} Unfortunately, mental health services are not always easily available, especially for the noninsured. A significant obstacle is that psychiatrists tend to be

skeptical about the diagnosis of psychogenic symptoms, and even for PNEE where video-EEG monitoring allows a near certain diagnosis, they tend to not believe the diagnosis.^{60,61} Providing the treating psychiatrist with the video recordings of the PNEE can be a useful approach to combat this skepticism, as these recordings can be more convincing than written reports. In treatment of PNEE, the evidence for the efficacy of cognitive behavior therapy is growing,^{62,63} and pharmacologic treatment with a selective serotonin reuptake inhibitor antidepressant may be similarly helpful.^{58,59}

The author pointed out previously that the American Psychiatric Association had no patient information on this category of conditions, despite an elaborate website emphasizing patient education.⁶⁰ Seven years later, the site still has no information on the entire category of Somatoform or “Somatic Symptom” Disorders.⁶⁴ In keeping with this “omission,” it is very difficult to find psychiatrists interested in the treatment of somatoform or somatic symptom disorders, and even large epilepsy centers do not have consistent access to good psychiatric care for patients with PNEE. Psychology organizations do not fare any better.⁶⁴ Articles on somatoform disorders are also rare in the psychiatric literature. For example, a review of 589 research abstracts presented at the 2012 American Psychiatric Association annual meeting, using title words, yielded none for *conversion*, none for *somatization*, one for *somatic*, and one for *somatization*. It is illustrative that the recent advances in PNEE treatment are in neurology journals^{58,62,63} and not the psychiatry or psychology literature. Clearly, there remains a significant unmet need for both clinical care and research of patients with somatoform or somatic symptom disorders.^{60,64}

PSYCHOGENIC NONEPILEPTIC EVENTS OR EPISODES IN CHILDREN

Although PNEE are more common in adolescence, they may occur in children as young as 5 or 6 years of age. Most of what has been presented regarding adults with PNEE also applies to children as well. 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.¹ In particular, children also have nonepileptic staring spells,⁶⁵ which are behavioral inattention that is misinterpreted by family or physicians. These are easily clarified with video-EEG recordings. The gender difference of female predominance is not seen until adolescence, and PNEE are as common in preadolescent boys as in girls. Benign focal epileptiform discharges of childhood are a common confounding feature on interictal EEG. Another aspect specific to children is that serious psychosocial stressors, such as abuse, may be ongoing at the time of diagnosis and require acute intervention. Outcome of PNEE is overall better in children and adolescents,⁶⁶ probably because the duration of illness is shorter, and the psychopathology or stressors are different from those in adults. School refusal and family discord may be significant factors. Serious mood disorders and ongoing sexual or physical abuse are common in children with PNEE and should be sought in every case.

BORDER ZONE OF PSYCHOGENIC NONEPILEPTIC EVENTS OR EPISODES

The border zones of PNEE are psychogenic, broadly speaking, but in different categories from the typical somatoform disorders discussed above, and may be

KEY POINT

- In treatment of psychogenic nonepileptic events or episodes, the evidence for the efficacy of cognitive behavior therapy is growing, and pharmacologic treatment with a selective serotonin reuptake inhibitor antidepressant may be similarly helpful.

better characterized as behavioral. Panic attacks are paroxysmal manifestations of anxiety or panic disorder, typically include intense autonomic symptoms (especially cardiovascular and respiratory in nature), and may be mistaken for seizures.⁶⁷ In these attacks, abrupt, intense fear is accompanied by at least four of the following symptoms: palpitations, diaphoresis, tremulousness or shaking, shortness of breath or sensation of choking, chest discomfort, nausea or abdominal discomfort, dizziness or lightheadedness, derealization or depersonalization, fear of losing control, fear of dying, paresthesias, and chills or hot flashes. The symptoms typically peak within 10 minutes. Other manifestations of anxiety—such as agoraphobia, social phobia, and depressive disorder—often coexist with panic disorder. Similarly, if the symptoms of post-traumatic stress disorder resemble seizures, they can be viewed as a variant of PNEE and lead to a misdiagnosis.⁶⁸ Unusual repetitive and purposeless behaviors or mannerisms are common in neurologically impaired patients,^{65,69} and abnormal motor behaviors are often observed in the intensive care unit.⁷⁰ Rather than truly psychogenic, these manifestations are often simply misinterpreted by families and physicians and are easily diagnosable with video-EEG recordings.

CONCLUSION: A MORE GENERAL PERSPECTIVE ON PSYCHOGENIC SYMPTOMS

The literature on PNEE often implies that they represent a unique disorder. In reality, PNEE are but one type of somatoform disorder. How the psychopathology is expressed (seizurelike episodes, paralysis, diarrhea, or pain) is only different in the diagnostic aspects. Fundamentally, the underlying psychopathology and its prognosis and management are no different for

PNEE than they are for other psychogenic symptoms.⁷¹

Psychogenic (ie, nonorganic, “functional”) symptoms are common in all of medicine. Conservative estimates consider that at least 10% of all medical services are provided for psychogenic symptoms.²⁵ Common neurologic symptoms that are found to be psychogenic include paralysis, mutism, visual symptoms, sensory symptoms, movement disorders, gait or balance problems, and pain. Several neurologic symptoms, signs, or maneuvers have been described to help differentiate organic from nonorganic symptoms. Among psychogenic symptoms, PNEE are unique in one principal characteristic: with video-EEG monitoring, they can be diagnosed with near certainty. This is in sharp contrast to other psychogenic symptoms, which are almost always a diagnosis of exclusion. This feature allows a clarity and confidence of diagnosis that may assist in the critical step of convincing the patient and his or her family of the nonorganic nature of the PNEE. Once the diagnosis of PNEE has been established by video EEG, the role of the neurologist is to convey the diagnosis clearly and compassionately and to mediate referral for mental health management. Unfortunately, difficulty in access to appropriate mental health management of somatoform or somatic symptom disorders remains a vexing and frustrating limitation for clinicians and patients with PNEE alike.

USEFUL WEBSITES

PNEE Patient Information brochure.
health.usf.edu/NR/rdonlyres/C4AD7955-E93A-4702-BB3A-C5F5A5381B44/0/PNESbrochure.pdf

American Psychiatric Association.
www.psych.org.

American Psychological Association.
www.apa.org.

Treatment for Psychogenic Nonepileptic Seizures.
www.webspawner.com/users/cbarmagick/index.html.

There are several “nonepileptic seizures” groups that are searchable on social media sites.

VIDEO LEGEND

Supplemental Digital Content 8-1

Psychogenic nonepileptic episode. Video demonstrates a psychogenic nonepileptic episode induced by activation (photic stimulation and verbal suggestion). Note the multiple features suggestive of the diagnosis: whole body side-to-side low-amplitude nonclonic trembling, which is initially horizontal but later changes to vertical (variability in rhythm and direction); the eyes continuously closed; the side-to-side head shaking; and the asynchronous leg movements (bicycling).

links.lww.com/CONT/A44

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