Düşünen Adam The Journal of Psychiatry and Neurological Sciences 2012;25:170-178
DOI: 10.5350/DAJPN2012250210

Approach to Patients with 'Psychogenic Non-Epileptic Seizures': A Review

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ABSTRACT

Approach to patients with 'psychogenic non-epileptic seizures': a review

Several terms have been used for pseudo-seizures, including psychogenic non-epileptic seizures (PNES), psychogenic seizures, hysterical seizures and hysteroepilepsy. Among them, the term 'non-epileptic seizures' is preffered. PNES outwardly may appear similar to epilepsy, but are caused purely by emotion. PNES are defined by modern psychiatry as conversion and dissociative disorders, but these disorders may coexist with many other psychiatric disorders including depression, posttraumatic stress disorder and personality disorders. The aim of this article is to describe and to compare similarities and differences in epidemiological, psychiatric and semiologic variables between patients with psychogenic non-epileptic seizures and other patients with epilepsy.

Understanding the nature of psychogenic non-epileptic seizures is necessary in order to reach the right diagnosis based on clinical symptoms and signs. Once a firm diagnosis has been reached, presenting that diagnosis to the individual patient is the first, and one of the most important part of the treatment. Non-epileptic seizures, also reffered to as pseudoseizures, are paroxysmal behaviors with psychological comorbidities and are unresponsive to treatment with antiepileptic drugs. Systematic comparisons between patients with recent onset psychogenic seizures and patients with recent onset non-psychogenic seizures may lead to a better understanding of psychological concomitants and perhaps the aetiology of psychogenic seizures.

A traumatic experience may be a potential risk factor for patients with PNES. The evidence suggests that a history of childhood abuse might be involved in the aetiology of at least some cases of psychogenic seizures. Such studies are important considering the early differential diagnosis of true epileptic versus psychogenic fits is notoriously difficult.

Key words: Conversion disorders, non-epileptic seizures, epilepsy

ÖZET

Psikojenik epileptik olmayan nöbetleri olan hastalara yaklaşım: Bir derleme

Epileptik olmayan nöbetler için histerik nöbet, histeroepilepsi veya psikojenik nöbet kavramlarını da içeren pek çok tanımlama kullanılmaktadır. Bu tanımlamalar arasında 'epileptik olmayan psikojenik nöbet' terimi daha çok tercih edilir. Epileptik olmayan psikojenik bu nöbetler, görünüşte epileptik nöbetlere tamamen benzese de, ortaya çıkış nedenleri tamamen psikojeniktir. Epileptik olmayan psikojenik nöbetler, modern psikiyatride konversiyon ve dissosiyatif bozukluk olarak tanımlanır ve bu hastalıklar, aynı zamanda depresyon, posttravmatik stres bozuklukları ve kişilik bozuklukları gibi diğer psikiyatrik problemlerle bir arada bulunur. Bu makalenin amacı psikojenik epileptik olmayan nöbetleri olan hastalarla epilepsisi olan hastalar arasında epidemiyolojik, psikiyatrik ve semiyolojik değişkenlerdeki benzerlik ve farklılıkları karşılaştırmak ve tanımlamaktır.

Klinik belirti ve bulgulara dayanarak, doğru teşhise ulaşmak için epileptik olmayan psikojenik nöbetlerin doğasını iyi anlamak, doğru tedaviyi uygulayabilmenin en önemli parçasıdır. Psikojenik nöbetler paroksismal davranışlarla seyreder ve antiepileptik tedaviye yanıt vermezler. Yeni başlangıçlı psikojenik nöbetlerle psikojenik olmayan nöbetler arasındaki sistematik kıyaslamalar, eşlik eden psikolojik semptomları ve etiyolojiyi anlamak açısından önemlidir.

Travmatik deneyimler, psikojenik nöbet geçiren hastalar için aynı zamanda potansiyel bir risk faktörüdür. Kanıtlar, bazı psikojenik epilepsilerin etiyolojisinde çocukluk çağı travmalarının rolü olabileceğini düşündürtmektedir. Buna benzer çalışmalar, gerçek epileptik vakalarla psikojenik epilepsilerin erken ayırıcı tanısının oldukça zor olduğuna dikkat çekmesi açısından incelemeye değerdir.

Anahtar kelimeler: Konversiyon bozukluğu, epileptik olmayan nöbet, epilepsi

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Date of receipt:
May 23, 2011

Date of acceptance:
July 31, 2011

INTRODUCTION

The aetiology of non-epileptic non-organic seizures, also referred to as psychogenic seizures, hysterical seizures, dissociative seizures, non-epileptic attack disorder or pseudoseizures, is largely unknown. Psychogenic nonepileptic seizures (PNES) are characterized by sudden and time-limited alterations of consciousness and are associated with a disturbance in controlling motor, sensory, autonomic, cognitive,

emotional and/or behavioural functions (1). These seizures can mimic any kind of epileptic seizures and thus may be mistaken for generalized tonic-clonic seizures, absence seizures, and simple or complex partial seizures. But these seizures do not occur as a consequence of abnormal cortical discharges; they are thought to be mediated by a dysfunction in the processing of psychological or social distress (2) Also the clinical and EEG features characterizing epileptic seizures are lacking and there is no organic explanations in pseudoseizures. Recent estimates suggest that between 9% and 50% of patient referrals to epilepsy services are of a non-epileptic nature (3). In some instances, patients with reliably established epilepsy exhibit additional seizures of apparently unknown origin (up to 33%), although many patients may present exclusively with these so called non-epileptic attacks. Hence, these seizures are nearly as common as multiple sclerosis and trigeminal neuralgia (3,4). Such patients are notoriously difficult to work with, and the cost to the health service can be high.

There is no completely reliable set of procedures for identifying PNES and that's why distinguishing them from epileptic events can be an extremely difficult task. As a result, many patients with PNES have received an incorrect diagnosis of epilepsy, a misjudgement that can have far reaching implications. Mostly, these group of patients are treated for epilepsy for several years and by the time the correct diagnosis is made, they will commonly have taken more antiepileptic drugs (AEDs) at higher doses and experience more side effects than an equivalent cohort of patients with epilepsy (5). One in 10 patients will present in apparent status epilepticus. Astonishingly, about one quarter of referrals to any specialty epilepsy center with refractory status were found to have "pseudostatus'' and among these patients physiologic nonepileptic seizures are less common than psychogenic nonepileptic seizures (4,6).

What this disorder should be called has been the subject of considerable debate. Some terms (hysterical seizures, pseudoseizures) are pejorative, unacceptable to patients (7) and have largely been abandoned (8). Others (nonepileptic seizures [NES], nonepileptic attack disorder) merely describe what the condition is not, rather than conveying what it is. Furthermore, these terms used with different meanings: the term NES, for example, is sometimes used to refer to the group of neurological, cardiological, and other medical conditions, in addition to psychiatric disorders, which constitute the differential diagnosis for epilepsy, while on other occasions, the term is used as a form of loose shorthand to refer to the psychological attacks alone (3,9). The terms psychogenic nonepileptic seizures (PNES) and functional seizures overcome some of these objections but formal psychiatric classification systems provide clearly defined labels. Unfortunately, though, there are stil inconsistencies: thus, within DSM-IV-TR such attacks are classified under somatoform disorder as ''conversion disorder'' and in ICD-10 the diagnostic label "dissociative convulsions'', is classified within the group of conversion disorders. It is the current terminology that will be adopted here.

As we have seen, PNES are common, the diagnosis is often missed, and patients, not only fail to receive appropriate treatment, but are subject to unnecessary, costly, and potentially harmful medical interventions (10,11). In considering the management of the disease, we will therefore focus on assessment and diagnosis before considering contemporary approaches to treatment.

CLINICAL ASSESSMENT

The clinical diagnosis of PNES requires the exclusion of epilepsy and other physical disorders; indeed, DSM-IV-TR places nonepileptic convulsions in the somatoform rather than the dissociative disorders category to emphasis the importance of excluding physical illness in the differential diagnosis of these episodes. Several factors should be considered in the diagnostic process and judgements should always be based on converging lines of evidence. Great care must be taken to establish the precise sequence of events during an attack and history taking is not complete until an eyewitness account has been obtained. The duration of each phase of symptoms, including recovery from the attack should be determined. Any habitual pattern in the circumstances that trigger attacks should be

sought (6). Patients and eyewitnesses should be prompted for specific symptoms because significant features may not be mentioned spontaneously (e.g., psychic and cognitive symptoms, auras, automatisms, occurrence during sleep).

Medical Differential Diagnosis

Physiologic nonepileptic seizures have multiple causes. Of medical disorders mistaken for epilepsy, syncope is the most common and in non-specialist settings is the condition most likely to be misdiagnosed as epileptic. It is important to note that tonic or clonic movements may be seen during syncope. At the same time raised postictal serum prolactin concentrations can occur false-positively. However, characteristic prodromal symptoms (lightheadedness, clammy sweatiness, a sense of receding sound and vision, nausea), associated cardiac symptoms, and a relation in some cases to postural changes or valsalva usually make identifying cases of syncope straightforward (12). In patients with cerebrovascular disease, the differentiation of transient iscemic attacks from partial seizures may sometimes be difficult. Iscemic epizodes may last for seconds to minutes but usually occur with preserved consciousness, are associated only with a loss of function, and are not followed by more typical epileptic features. A comparatively long duration of symptoms is useful in recognizing migraine and vertigo. Abnormal startle phenomenon, including hyperekplexia, is rare but often mistaken for epilepsy and needs to be distinguished from startle induced seizures (13). Paroxysmal movement disorders may be mistaken for epilepsy. Sudden loss of muscle tone that may produce falls in response to an emotional trigger suggest cataplexy, which is usually found in association with other features of the narcolepsy syndrome (narcolepsy, hypnopompic or hypnogogic hallucinations, and sleep paralysis). Other parasomnias giving rise to complex behavioural episodes, arising from sleep may be confused with epileptic automatisms, although the former lack any preceding ictus and are usually of comparatively long duration (14). Space occupying lesions in the 3rd ventricle may produce intermittent CSF obstruction associated with visual symptoms and are a rare cause of sudden episodes of collapse with loss of cosciousness. Metabolic and endocrine disorders associated with loss of cosciousness usually have a protracted time course and are suggested by other features in the history (3). Psychogenic nonepileptic seizures also may be due to alcohol and drug intoxication or withdrawal.

Psychiatric Differential Diagnosis

Psychogenic nonepileptic seizures are a physical manifestation of psychologic distress. Once epilepsy and other organic causes of seizures have been excluded, there are three categories of psychiatric diagnoses that must be considered: 1. dissociative seizures, 2. factitious disorder, 3. other psychiatric disorders that have been mistaken for epilepsy.

Dealing with the last category first, paroxysmal symptoms of psychiatric disorders may sometimes raise the question of epilepsy. The most common example of this is panic disorder (6,15). Patients may report detachment type dissociations (depersonalization, derealization) and tremulousness during panic attacks while partial epileptic seizures may include both emotional and somatic symptoms of anxiety. Features that are useful in distinguishing the two conditions include a longer duration, cognitive symptoms, and the presence of specific environmental triggers in panic disorder and in partial seizures, the unique quality of the emotional symptoms ("ictal fear") together with associated more characteristic epileptic features in partial seizures (16). Paroxysmal symptoms in psychosis may sometimes raise the question of epilepsy, but such symptoms (e.g., hallucinations) lack the highly stereotyped quality of epileptic phenomena and epizodes are usually of long and variable duration. Other psychiatric disorders sometimes confused with epilepsy include depersonalization disorder and attention deficit hyperactivity disorder in which failing school performance and poor concentration may sometimes raise the possibility of juvenile absence epilepsy (17).

In most cases, however, the seizures will be the

principal symptoms and cannot be accounted for by another psychiatric condition. The two diagnostic possibilities are dissociative seizures and factitious disorders distinguished from one another by whether the seizures are thought to arise through unconscious processes like in dissociative seizures (DS) or are delibaretely enacted. In factitious disorder, the patient is held to be deliberately simulating epilepsy for reasons understandable in terms of their psychological background. It is distinguished from malingering (not a medical diagnosis) in which people are simulating illness for some obvious practical gain (e.g., compensation, avoidance of criminal responsibility).

Diagnosis of PNES is suspected by anamnesis, physical examination, ictal semiology and personal and psychiatric history. The presence or absence of self injury and urine or fecal incontinence, the ability to induce seizures by suggestion, psychologic tests, historical factors and ambulatory EEG have been found to be insufficient for the diagnosis of PNES (18,19). Postictal prolactin levels greater than two times the upper limit of normal once were thought to differentiate generalized and complex partial seizures from PNES, but recently have been shown to be unreliable. Inpatient video-electroencephalography (v-EEG) monitoring is the gold standard for the diagnosis of PNES. This tecnique results in a definitive diagnosis in almost 90% of patients (15,18). Definitive diagnosis is achieved when a patient is observed having typical seizures without accompanying EEG abnormalities. Family members or witnesses who are familiar with the patient's seizures must agree that the recorded epizodes are typical events.

The Semiology of PNES

Typical clinical features of these events include gradual onset, long duration, a waxing and waning course, and disorganized asymmetric motor activity. The events lack the stereotypy of epileptic seizures because the pattern of symptoms and sequence of events vary between seizures. Not all seizures with these features are PNES, however. Frontal lobe seizures often are mistaken for PNES because of the associated dramatic motor and vocal outbursts, possible retained consciousness, and short postictal period. Frontal lobe seizures may be distinguished by their brief duration, stereotypical nature, and tendency to begin during sleep (19,20). Gelastic seizures (in which the primary automatism is laughter), reflex epilepsies, and myoclonic jerks also have been mistaken for PNES. Avoidance behavior during seizures, closed eyes during seizures and on recovery, evidence that the patient is able to recall events for a period of unresponsiveness, dystonic posturing, emotional or situational trigger for the seizures, ictal crying, weeping, pelvic movements (especially forward thrusting), resisted eyelid opening, side-to-side head movements and ability of the observer to modify the patient's motor activity are the features suggesting a diagnosis of PNES (8,21).

Historical features strongly suggesting the diagnosis of PNES include being associated (often multiple) with psychiatric disorders, onset of seizures after age of 10, flurries of seizures or recurrent pseudo-status epilepticus that lead to multiple emergency department visits or hospitalizations, high seizure frequency, history of sexual or physical abuse, lack of concern or an excessive or exaggerated emotional response, multiple unexplained physical symptoms, no history of injury from seizures, no response to antiepileptic drugs or a paradoxical increase in seizures with antiepileptic drug treatment, personal, family, or professional experience with epilepsy or presence of the learning difficulties and seizures that occur only in the presence of others or only when the patient is alone (22-25).

PSYCHIATRIC FORMULATION

Epidemiological Consideration, Comorbidity and Risk Factors for PNES

The prevalence of nonepileptic seizures ranges from 2-33 cases per 100.000 persons in the general population. About three quarters of patients are women (15,18,21). Seizures usually begin in the late teens or early 20's but there is a wide range. Patients in lower socioeconomic groups and with less educational achievement are probably overrepresented. Histories of previous

medically unexplained symptoms are present in up to 80% of patients. The prevalence of PNES is increased in patients with head injuries, learning disabilities, or isolated neuropsychologic deficits and patients with PNES have higher than average rates of abnormal results on magnetic resonance imaging (MRI) and EEG. These factors suggest that physical brain disease may play a role in the development of the events. The events also occur in patients with central nervous system lesions that are associated with an increased risk of developing epilepsy, such as stroke, trauma, infection, and malformation, as well as in patients with hippocampal sclerosis, which often is identified as a cause of temporal lobe epilepsy. Thus, the presence of MRI or EEG abnormalities may delay diagnosis and treatment of PNES (2). Estimates of the coexistence of epilepsy and PNES vary from 5% to more than 60%, depending on the study setting and diagnostic criteria. In some cases, symptoms will have attracted a medical diagnosis although objective evidence of pathology is lacking. The recently reported association of a diagnosis of asthma in patients with PNES is an example of this.

In addition to a history suggestive of somatization, there is a high rate of psychiatric comorbidity (22). Maladaptive personality features of borderline type and histrionic type are common, often in the form of trait accentuations rather than personality disorder in itself. However, all of psychogenic seizures in association serves as a coping mechanism.

Patients with these events are more likely to use maladaptive coping strategies to handle stress. In PNES, psychologic conflicts are translated into a physical symptom –seizure. In this way, intolerable distress is dissociated from the painful conscious experience of the trauma or forbidden emotions that are causing the distress. Thus, genuine PNES (as opposed to factitious disorder or malingering) are not intentional: they are created as a psychologic defense mechanism to keep internal stressors out of conscious awareness.

PNES do not have a single aetiology; rather, they are the product of several different causal pathways. The seizures may be the result of psychopathologic processes, a response to acute stres in patients without evidence of psychopathology, or a reinforced behavior pattern in cognitively impaired patients. Rarely, malingering or factitious disorder presents as PNES. Approximately, 73% of patients with PNES have concurrent psychiatric disorders which tend to be related to trauma, and include post traumatic stres disorder (PNES) and other anxiety disorders; depressive disorders; and conversion, somatization, and dissociation disorders. Patients with PNES frequently have a history of (or current) physical or sexual abuse or significant psychosocial stressors for which there is no perceived resolution (15,23). Although there have been negative findings, a number of large studies in which abuse has been carefully defined have shown higher rates of reported abuse in patients with PNES compared with epileptic controls and unselected psychiatric patients. Traumatic, abusive experiences in adulthood have also been implicated. These ''unspeakable dilemmas'' often involve dysfunctional family interaction and communication. Other traumatic experiences or situations that foster low self esteem, for example bullying at school or unrecognized learning difficulties, may be overrepresented but have not been studied in adults with PNES (1,2,23).

There is evidence that adverse life events are more common in the year preceding the onset of PNES, but triggers for initial seizures are often not apparent. Adverse family interactions may serve both as predisposing and maintaining factors for PNES. A pattern of avoidant behaviour, often exacerbated by caretakers overprotective reactions, is a comparatively underrecognised maintaining factor but readily apparent on history taking in the form of an agoraphobic pattern of avoidance ostensibly for fear of having a seizure in an embarrassing or vulnerable setting. Finally, for some patients the psychological and social advantages inherent to a medical sick role are undoubtedly important mantaining factors. Such advantages include both an avoidance of responsibility and positive benefits such as the caring response elicited in others (26).

It should be noted that PNES share many possible aetiological factors with other somatoform disorders. The paroxysmal nature of the symptoms, however, gives this order a unique quality that creates special difficulties for diagnosis but also raises specific treatment

approaches. To determine why a patient is having psychogenic nonepileptic seizures, the physician must identify the psychologic function of the seizure. A detailed, systematic psychiatric evaluation and an assessment of family, social, financial, and employment problems should provide insight (5,7).

TREATMENT

Explaining the PNES Diagnosis to Patients

Treatment begins with an explanation of diagnosis. This must be handled openly but sensitively: if it is not, the patient is likely to reject the diagnosis, decline treatment, and go elsewhere for more investigations: a disaster in terms of time and expense, both for the patient and medical services. Following points should be covered during discussing the diagnosis with the patient:

1. Reasons for concluding they do not have epilepsy
2. What they do have (describe dissociation)
3. Emphasize they are not suspected of ''putting on'' the attacks
4. They are not ''mad''
5. Triggering ''stresses'' may not be immediately apparent
6. Relevance of etiological factors in their case
7. Maintaining factors
8. May improve after correct diagnosis
9. Caution that AED withdrawal should be gradual
10. Describe psychological treatment

It is important to involve patients' carers in this explanation. Firstly, a clear explanation must be given of the reasons for concluding that the patient does not have epilepsy. This should cover any aspects of the patient's seizure semiology that are inconsistent with epilepsy and features in their history that make epilepsy less likely (for example, a failed response to AEDs, and lack of risk factors for epilepsy). A through explanation of investigation results should follow that, if relevant, must address any non-specific ''abnormalities'' that the patient may previously have been told about and the way in which these can be put in context (3,7,11,27).

Approach to the patient should be as follows. Many patients will react unfavourably to the news that no medical explanation has been found and great care should be taken to emphasize that the doctor understands the attacks are real, disabling, and that the patient is not suspected of ''putting them on''. A useful approach can be to tell the patient that they have attacks in which their mind or brain ''switches off'', and they lose control. It is often helpful to describe the concept of dissociation, explaining that the attacks represent an extreme form of something that is part of everyday experience using examples illustrating selective and divided attention (for example, reading a book and not hearing your name called, travelling from work and remembering nothing of the journey). It should be explained that the symptoms are stress related but that it is usual for the stresses to be difficult to identify. Patients commonly object that the seizures cannot be caused by stress as they occur at times when they are relaxed. In this situation, it may be helpful to explain that attacks may be triggered by stressful or unpleasant thoughts that the patient is barely aware of (or cannot remember) and may have little to do with their immediate circumstances. The concept that thinking may occur on a number of different levels at any one time can be described. Examples of the link between physical symptoms and emotional state (e.g., crying, autonomic symptoms of arousal) and complex involuntary behavioral accompaniments to emotions (e.g., as seen with sudden grief or with rage) may help illustrate some of the physical attributes of seizures. If the patient experiences somatic symptoms of arousal during their seizures, the relation of these features to anxiety can be described and the seizures likened to a ''panic attack without the panic'' in which dissociation (''switching off'') protects the patient from unplesant or frightening emotions (28).

Patients often express a fear that they are being told they are ''mad''. They should be reassured that the condition they have is common and the profile of a typical patient should be described, emphasizing the points that apply to them. A link between traumatic experiences in childhood may be made meaningful by explaining that children exposed to abuse, especially if it is repeated, learn to ''switch off'' as a way of coping: PNES may represent a re-emergence of this once

adaptive response in the face of challenges, stress, or perhaps something that reminds the patient of painful memories. Some have recommended raising the subject of abuse as a causal factor even if a history of it has not emerged. However, such an approach risks encouraging ''false memories'' and may best be avoided (1,26,29).

A description of maintaining factors is important and is often welcomed by patients who are sceptical about supposed psychological origins of their symptoms. It can be explained that whatever caused the seizures in the first place may remain unknown, but that worry about seizures, including what they are attributable to, and worry about the consequences of having a seizure may actually make the seizures worse and more frequent. Patients will often relate to the confusion and anxiety engendered by receiving contradictory advise from a succession of doctors and the role this may have in perpetuating attacks. The concept of how avoidant behaviour, often exacerbated by a well meaning family's protective reactions acts to reinforce anxiety about attacks, may give the patient and their family a rationale for change (3,16).

Finally, the patient should be given the hope that their problems can be treated. Most patients are delighted to hear they may discontinue AEDs, but they should be warned that this must be done gradually for fearing of triggering a withdrawal seizure. It is worth emphasizing that once confusion about diagnosis is resolved, a significant proportion of patients find that this explanation often alone leads to a resolution of the attacks over time.

Approaches to Treatment

There have been no randomized controlled trials of treatment in PNES. The evidence comes from case reports and small, uncontrolled treament studies. In the small proportion of patients who have significant comorbid depression or anxiety, appropriate pharmacotherapy (for example, SSRIs) is indicated (3). For most, however, some form of psychotherapy will be the mainstay of treatment. In patients with learning difficulties, operant behavioral programmes using simple reward systems are often helpful. The early literature includes a number of compelling descriptions of insight oriented, dynamic psychotherapeutic approaches in patients with a history of PNES and sexual abuse. More recent reports have described psychoeducational group therapy and eye movement desensitization (EMDR) in similar patient groups. Variations of therapy based on psychodynamic, insight oriented, and educational principles are undoubtedly widely practiced and believed to be effective but further studies of such iterventions are needed.

The paroxysmal nature of the attacks, the occurence of somatic symptoms of arousal in many patients, and the association with agoraphobic avoidant behaviour suggest that techniques developed in cognitive behavioural therapy (CBT) for the treatment of panic disorder might readily be adapted for PNES. This CBT model also provides a useful rationale for treatment in patients who report no history of past traumatic experiences or who have received psychotherapy for this but continue to have seizures. A study involving 20 patients treated with CBT reported a significant reduction in seizures six months after treatment ended and perhaps more importantly, found improvements in work and social outcome. CBT techniques developed for personality disorder may be helpful but these and other techniques require evaluation (28).

A significant proportion of patients continue to have seizures despite intensive and varied treatment. A realistic approach in such cases is to offer long term follow up to provide support for the patient and their family, social interventions to improve quality of life despite seizures, and also to limit the cost and morbidity associated with further unnecessary investigations and medical interventions (6). Antiepileptic drugs should be tapered in patients with exclusively nonepileptic seizures, and appropriate psychotropic drugs are titrated for the treatment of psychiatric comorbidities.

Disease Course and Outcome

Because psychogenic nonepileptic seizures are not a single entity or disorder, the course is variable and depends on the underlying cause. Prognostic factors also vary. Some factors for favorable outcome are

acceptance of nonepileptic nature of episodes, family structure that supports autonomy, female gender, having friends currently, having good relationships with friends as a child, higher ability to express emotions, higher intelligence and education, independent lifestyle, less dramatic PNES, less exreme scores on traits defining emotionel dysregulation, less tendency to dissociate, shorter duration of PNES, younger age at diagnosis. Poor prognostic factors in patients with PNES are coexisting epilepsy, disbelief of diagnosis, presence of psychiatric comorbidity including personality disorder, long delay in diagnosis, family structure that supports dependency and illness, longer duration of PNES, male gender, ongoing physical or sexual abuse, ongoing psychosocial stressors, pending litigation, persistent somatization, reluctant self-disclosure, restricted expression of anger and positive feelings, unemployment or disability (26,28).

A review of outcome studies found that after a mean follow up period of three years, about two thirds of patients continued to have PNES and more than half remained dependent on social security. Receiving psychiatric treatment has been associated with a positive outcome in some studies, but not in others.

REFERENCES

1. Bakvis P, Roelofs K, Kuyk J, Edelbroek PM, Swinkels WA, Spinhoven P. Trauma, stress, and preconscious threat processing in patients with psychogenic nonepileptic seizures. Epilepsia 2009; 50:1001-1011.
2. Dikel TN, Fennell EB, Gilmore RL. Posttraumatic stres disorder, dissociation, and sexual abuse history in epileptic and nonepileptic seizure patients. Epilepsy Behav 2003; 4:644-650.
3. Benbadis SR, Blustein JN, Sunstad L. Should patients with psychogenic nonepileptic seizures be allowed to drive. Epilepsia 2000; 41:895-897.
4. Alsaadi TM, Marquez AV. Psychogenic nonepileptic seizures. Am Fam Physician 2005; 72:849-856.
5. Reuber M, Elger CE. Psychogenic nonepileptic seizures: review and update. Epilepsy Behav 2003; 4:205-216.
6. Mellers JDC, Shaw P, Henderson M, Polkey C, David AS, Toone BK. Schizophrenia-like psychosis arising de-novo following a temporal lobectomy: timing and risk factors. J Neurol Neurosurg Psychiatry 2004; 75:1003-1008.
7. Stone J, Campbell K, Sharma N, Carson A, Charles P, Sharpe WM. What should we call pseudoseizures? The patient's perspectives. Seizure 2003; 12:568-572.
8. Volow Micheal R. Pseudoseizures: an overview. South Med J 1986; 79:600-607.
9. Scull DA. Pseudoseizures or non-epileptic seizures; 15 synonyms. J Neurol Neurosurg Psychiatry 1997; 62:200.
10. Martin RC, Gilliam FG, Kilgore M, Faught E, Kuzniecky R. Improved health care resource utilization following video-EEG-confirmed diagnosis of nonepileptic psychogenic seizures. Seizure 1998; 7:385-390.
11. Işıkay CT, Yiğit A, Öncü B, Şarman C. Anamnezin epileptik nöbet ile psikojenik nöbeti ayırmadaki güvenilirliği / The reliability of anamnesis in distinguishing between epileptic seizures and psychogenic seizures. Epilepsi 1999; 5:70-74.
12. Sheldon R, Rose S, Ritchie D, Connolly SJ, Koshman ML, Lee MA, Frenneaux M, Fisher M, Murphy W. Historical criteria that distinguish syncope from seizures. J Am Coll Cardiol 2002; 40:142-148.
13. Manford MR, Fish DR, Shorvon SD. Startle provoked epileptic seizures: features in 19 patients. J Neurol Neurosurg Psychiatry 1996; 61:151-156.
14. Schenck CH, Mahowald MW. Parasomnias. Managing bizarre sleep-related behavior disorders. Postgrad Med 2000; 107:145-156.
15. Alper K, Devinsky O, Perrine K, Vasquez B, Luciano D. Psychiatric classification of nonconversion nonepileptic seizures. Arch Neurol 1995; 52:199-201.
16. Iriarte J, Parra J, Urrestarazu E, Kuyk J. Controversies in the diagnosis and the management of psychogenic pseudoseizures. Epilepsy Behav 2003; 4:354-359.
17. Bowman ES. The differential diagnosis of epilepsy, pseudoseizures, dissociative identity disorder, and dissociative disorder not otherwise specified. Bull Menninger Clin 2000; 64:164-180.
18. Cragar DE, Berry DTR, Fakhoury TA, Cibula JE, Schmitt FA. A review of diagnostic techniques in the differential diagnosis of epileptic and nonepileptic seizures. Neuropsychol Rev 2002; 12:31-64.
19. Rowan AJ, Gates JR (editors). An introduction to current practice in the diagnosis of non-epileptic seizures. In: Non-Epileptic Seizures. Newton, Mass: Butterworth-Heinemann, 1993, 1-7.

20. Barry JJ. Nonepileptic seizures: an overview. CNS Spectr 2001; 6:956-962.

21. Shahid A, Shagufta J, Amir A, Wassef T, Ibrahim A. How to use your clinical judgement to screen for and diagnose psychogenic nonepileptic seizures without video electroencephalogram. Clin Neurosci 2011; 8:29-35.

22. Goldstein LH, Mellers JD. Ictal symptoms of anxiety, avoidance behaviour, and dissociation in patients with dissociative seizures. J Neurol Neurosurg Psychiatry 2006; 77:616-621.

23. Harden C. Pseudoseizures and dissociative disorders: a common mechanism involving traumatic experiences. Seizure 1997; 6:151-155.

24. Ercan ES, Dizdarer G, Tütüncüoğlu S. Epileptik ve psikojenik nöbetleri olan çocuk ve ergenlerin, psikiyatrik, ailesel ve sosyodemografik özellikler bakımından karşılaştırılması. Ege Pediatri Bulteni 2001; 8:21-26 (Article in Turkish).

25. Koçer A, İnce N, Gözke E, Koçer E. Epileptik nöbetlerin tanısında öykünün duyarlılık ve seçiciliğinin değerlendirilmesi. Epilepsi 2002; 8:9-15 (Article in Turkish).

26. Fiszman A, Alves-Leon SV, Nunes RG, D'Andrea I, Figueira I. Traumatic events and posttraumatic stres disorder in patients with psychogenic nonepileptic seizures: a critical review. Epilepsy Behav 2004; 5:818-825.

27. Lesser RP. Treatment and outcome of psychogenic nonepileptic seizures. Epilepsy Curr 2003; 3:198-200.

28. Prigatano GP, Stonnington CM, Fisher RS. Psychological factors in the genesis and management of nonepileptic seizures: clinical observations. Epilepsy Behav 2002; 3:343-349.

29. Tezer Fİ, Uluşahin A, Saygı S. Epilepsi cerrahisi sonrası ortaya çıkan epileptik olmayan psikojenik nöbetler: olgu sunumu. J Neurol Sci Turk 2007; 24:170-177 (Article in Turkish).