

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,100

Open access books available

167,000

International authors and editors

185M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.  
For more information visit [www.intechopen.com](http://www.intechopen.com)



# Psychogenic Non-Epileptic Seizures (PNES)

*Nirmeen A. Kishk and Mai B. Nassar*

## Abstract

Psychogenic non-epileptic seizures (PNES) are a common presentation to the emergency rooms and neurology departments, and they are difficult to discriminate from epileptic seizures (ES). PNES present as paroxysmal time-limited, alterations in motor, sensory, autonomic, and/or cognitive signs and symptoms, but unlike epilepsy, PNES are not caused by ictal epileptiform activity. There is no exact known etiology or mechanism for PNES so far. The most recognized factors discussed in the literature include trauma and child adversity, dissociation, somatization, emotional processing, psychiatric comorbidities, coping styles, and family dysfunction. The use of a comprehensive assessment model may ease the transition of patient care from the diagnosing team to the outpatient treatment provider. Recognition of the characteristic clinical features of PNES and utilization of video-EEG to confirm the diagnosis are critical. Communicating the diagnosis, discontinuation of treatment for epilepsy (unless comorbid PNES and epilepsy are present), and implementing proper liaison with a multidisciplinary team with clinical psychologists, neurologists, and psychiatrists improve patient and healthcare outcome.

**Keywords:** PNES, DES, definition, etiopathology, management

## 1. Introduction

According to Hingray et al. [1], between 12 and 20% of adults presenting in epilepsy clinics have dissociative seizures.

Psychogenic non-epileptic seizures present as paroxysmal time-limited, alterations in motor, sensory, autonomic, and/or cognitive signs and symptoms that are not caused by ictal epileptiform activity, and positive evidence for psychogenic factors that may have caused the seizure is present [2].

PNES were formerly given different names including the name hystero-epilepsy, pseudo-seizures, and behavioral spells. However, most of these terms became abandoned in the literature because of being either vague or pejorative, implying that the seizures are unreal or fake. So, the accepted terminology in the medical community became psychogenic non-epileptic seizures (PNES), non-epileptic attack disorder (NEAD) [3], or dissociative non-epileptic seizures (DES) [4].

## **2. Epidemiology**

The prevalence of PNES remains somewhat uncertain but has been estimated at up to 50/100000<sup>3</sup>; the incidence of video electroencephalography (vEEG)-confirmed PNES has been determined as 4/100000 per year [5]. However, data from epilepsy centers estimate a much higher incidence rate.

## **3. Etio-pathology**

Up to now, there is no exact known etiology or mechanism for PNES. Some of the most commonly presumed factors include trauma and childhood adversity, dissociation, somatization, alexithymia and defective emotional processing, illness perception, family dysfunction, psychiatric comorbidities and personality factors, age, gender, and organicity (including comorbid epilepsy and anti-seizure medication use) [3, 6, 7].

## **4. Psychogenesis**

Considering the previously mentioned factors, multiple theories for the psychogenesis or the mechanism by which PNES operate were hypothesized. All of these have agreed about the multifactorial nature of PNES that can be explained by different models.

One of the convenient proposed models for the psychogenesis of PNES is the one proposed by Bodde et al. [7]. This model shows five different layers or levels that highlight how each of these factors represents a heterogeneous group and may have a differential impact on the causation, development, and prolongation of PNES, emphasizing that not all factors have a similar impact. The proposed model is as follows:

### *Level 1. Psychological etiology*

This includes the factors involved in the causation of PNES, such as sexual adversity or other traumatic experiences.

### *Level 2. Vulnerability*

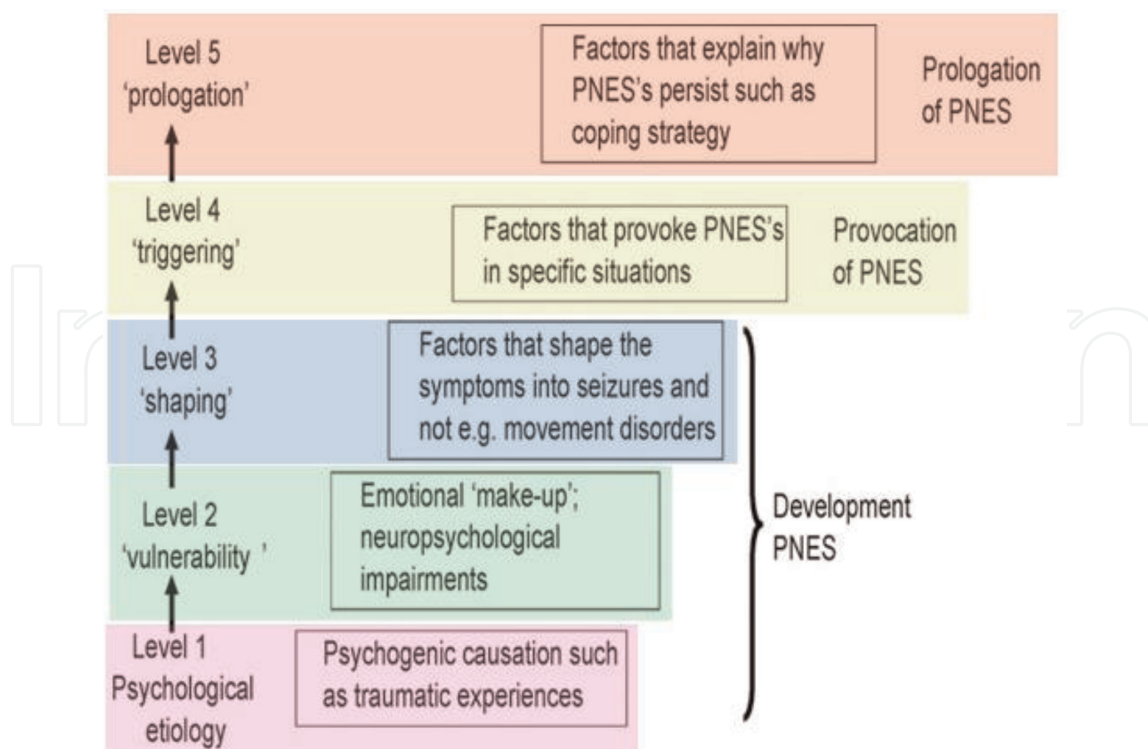
It refers to factors that act as predisposing elements for a person to develop psychosomatic symptoms like PNES, for example, personality factors, gender, neuropsychological impairments, organicity, and age. Many authors have pointed to the specific vulnerability of patients with PNES in terms of both their emotional “make-up” and their neuropsychological functioning.

### *Level 3. Shaping factors*

Some factors can specifically shape the symptoms in the direction or form of “seizures” rather than other forms, for example, movement disorders or headache-like symptoms. A shaping factor may be a relative with epileptic seizures (symptom modeling) or the person himself having past history of epilepsy.

### *Level 4. Triggering factors*

These are factors that create circumstances or situations that provoke and precipitate PNES, such as factors that refer to primary gain. Psychological mechanisms that transfer an emotional state into a seizure can be part of these triggering factors, such as dissociation and somatization. These factors explain why seizures occur on a



**Figure 1.**  
 Model of psychological factors involved in PNES [7].

specific day or in a cluster or why there is a period of remission. This differentiates PNES from conversion states that have a more predictable presentation.

*Level 5. Prolongation factors*

The previous factors are specifically important in the development of PNES, whereas prolongation factors are important in explaining why the seizures persist and PNES may become a chronic disorder. These factors tailor PNES frequency and resistance against therapy. Such modulating factors include the coping strategy of the patient and secondary gain aspects (**Figure 1**).

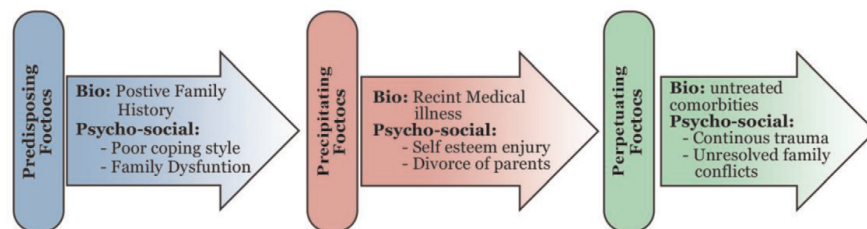
## 5. Predicting PNES: a multivariate approach

All the current research and studies aim to make that leap of “predicting” PNES, to change PNES from being a “diagnosis of exclusion” to being a “predictable,” early detected diagnosis.

A multivariate approach may predict the development of PNES and provide useful markers for early identification of patients with potential PNES [6, 8, 9]. The multivariate approach proposed comprises the following:

### 5.1 The biopsychosocial/3P (BPS/PPP) psychiatric assessment

Multiple studies suggest that the biopsychosocial/3P (predisposing, precipitating, and perpetuating) model for approaching the diagnosis of PNES is one of the most comprehensive integrative models for screening and early identification of variables that can be readily and cost-effectively obtained in patients with non-diagnostic V-EEG evaluations, or eventually in an outpatient setting, and may prompt more rapid diagnosis and treatment [10–13]. See **Figure 2** [13].



**Figure 2.**  
*Biopsychosocial conceptualization of PNES [13].*

This involves a thorough psychiatric clinical interview to obtain precise history including demographic characteristics, present and past psychiatric history, medical history, family history, personal history, current living circumstances, and family dynamics to identify the possible biological, psychological, and social etiological factors that may interact as predisposing, precipitating (triggering), or perpetuating factors for PNES and present them in a BPS/3P (biopsychosocial/predisposing, precipitating, and perpetuating) formulation to establish a proper individualized treatment plan [10].

## 5.2 Clinical and neurophysiological assessment

Although none of the clinical signs by themselves carry a strong enough diagnostic value unless the psychiatric, neurologic, and neurophysiologic backgrounds are taken into account, the following clinical signs and serological and EEG findings were claimed to have a predictive value for PNES when integrated with the other previously mentioned psychological factors and semiological features [6, 8, 9, 11].

- *Self-reported length of the attacks:* Patients with PNES have longer (>2 minutes) events compared to patients with epileptic seizures, where the length of the attacks is usually less than or equal to 2 minutes [8].
- *Age of onset of seizures:* Patients with PNES have older age of onset compared to patients with epileptic seizures, with “30 yrs. old” as the average age of onset 12.
- *Frequency of seizures:* PNES have more frequent attacks than epilepsy; diagnosis entitles a frequency of at least 2 seizures per week [14].
- *Duration of illness (years since the first seizure):* Shorter duration/less years since the first seizures is considered a good predictor for PNES with an average of 8 yrs. since the first seizure [6, 8, 9].
- *Occurrence of an episode during clinic visits:* In a patient with “refractory epilepsy,” the occurrence of an episode during clinic visits is a predictor of the episode being PNES with a high specificity (99%) and positive predictive value (PPV 77%) but a low sensitivity (3%) [12].



- *Prolonged PNES or NEPS:* Recurrent hospital admissions with prolonged PNES or NEPS (episode longer than 30 minutes) suggest PNES [6].
- *Response to medications:* Resistance to at least two anti-seizure medications is a predictor for PNES [14].
- *EEG findings:* At least 2 normal EEG studies are required to assume PNES15.

Added to that, the ILAE reported that predictors of PNES include the “rule of 2s” with an 85% PPV for PNES15. The rule of 2 s suggests that the diagnosis of PNES requires the following: at least two normal electroencephalography (EEG) studies with at least two seizures per week and resistance to two anti-seizure medications [6].

## 6. Patients’ characteristics

In an attempt to discriminate factors underlying this heterogeneity and detect important predictors of dissociative seizures, Hingray et al. [1] identified dissociative seizure patients into three profiles; each had some factors in common, but from a statistical point of view, participants’ trauma history pattern emerged as the strongest discriminating feature between these three profiles. Accordingly, Hingray et al. [1] named the identified patient subtypes according to their trauma history: Group 1, “No/Single Trauma”; Group 2, “Cumulative Lifetime Traumas”; and Group 3, “Childhood Traumas” (see **Table 1**).

## 7. Clinical presentation

### i. *Semiology: Behavioral seizure manifestation:*

According to Hingray et al. [1], cluster analysis data collected on patients with PNES were categorized using the proposed classification distinguishing five different semiological profiles, which were simplified to establish three groups based on categories most frequently used in the previous literature into hyperkinetic seizures (commonest semiology involves excessive movement of limbs, trunk, and head), paucikinetic seizures (seizures with stiffening and tremor), and syncope-like events or seizures (with atonia and loss of consciousness). The latter is less frequent [15].

### ii. *Phenomenology: Subjective seizure experience*

Many patients describe physical symptoms of panic or hyperventilation during their seizures without feeling anxious; it has been suggested that panic symptoms are more common in adolescents with PNES than in adults. Even in the absence of panic symptoms, most patients experience their seizures as confusing and beyond their control. At the same time, patients with epilepsy are more likely to conceptualize their seizure as a hostile agent acting of its own volition [1].

iii. *Autonomic seizure manifestations:*

More than one-quarter of patients with PNES give a history of ictal incontinence of urine; fecal incontinence is also reported. Sinus tachycardia is common but is more gradual in onset, less marked, and less persistent postictally than in epileptic seizures [15].

## 8. Confirming the diagnosis: A staged approach

Conversation analysis of history taking (**Table 2**) [16] and characteristic semiological and clinical features (**Table 1** [1]) and **Table 2** [16]) may help discriminate PNES from ES, but individually, they cannot not be a reliable diagnostic discriminator [17] (**Table 3**). To provide greater clarity about the process and certainty of the diagnosis of PNES and improve the care for the patients, the ILAE proposed a staged approach to confirm the diagnosis of PNES in which levels of diagnostic certainty were developed (see **Table 4**).

Key: + means history characteristics consistent with PNES, \*PNES = psychogenic non-epileptic seizures, EEG = electroencephalogram.

## 9. Delivery of the diagnosis: communication protocol

The process of communicating the diagnosis is one of the most important and potentially effective therapeutic steps in the management pathway of patients with PNES with both immediate (within 24 hours of diagnosis presentation) and long-term

Criteria	Group 1 No/Single Trauma	Group 2 Cumulative Lifetime Traumas	Group 3 Childhood Traumas
Predominant gender	Male	Female	Female
Educational level	Low	High	Intermediate
Triggers	<ul style="list-style-type: none"> <li>• Non-identifiable</li> <li>• Frustration more than anxiety</li> </ul>	<ul style="list-style-type: none"> <li>• Identifiable</li> <li>• Anxiety (80%) more than frustration (50%)</li> </ul>	<ul style="list-style-type: none"> <li>• Identifiable</li> <li>• Anxiety (84.1%) more than frustration (31.8%)</li> </ul>
Trauma history	Non-significant	Significant Multiple emotional trauma (most common type)	<ul style="list-style-type: none"> <li>• Significant</li> <li>• Childhood onset</li> <li>• Child sexual abuse and emotional trauma</li> </ul>
PTSD Prevalence	Non-significant	PTSD in 33.3% of cases	PTSD in 63.6%
Comorbid epilepsy	43.4% (common)	16.7% (rare)	52.4% (commonest)
Seizures semiology	Non-hyperkinetic Seizures (paucikinetic 42.2%)	Hyperkinetic most common	Hyperkinetic: Non-hyperkinetic 1:1

**Table 1.**  
*Patients' characteristics in groups [1].*

Diagnostic, linguistic, and interactional features yielded by conversation analysis	
PNES	ES
Patients tend to focus on the situations in which seizures have occurred or the consequences of their seizures rather than subjective seizure symptoms.	**Patients readily focus on the subjective seizure symptoms.
Subjective seizure symptoms may be listed but are not described in detail.	**Subjective seizure symptoms are given in detailed accounts with extensive formulation efforts (including reformulations, re-starts, neologisms, and pauses).
When the doctor tries to direct the patient's attention to particularly memorable seizures (e.g., the first, last, or worst seizure), patients commonly show focusing resistance by not providing further information or by generalizing rapidly to the description of their events in general.	**When the doctor tries to direct the patient's attention to particularly memorable seizures (e.g., the first, last, or worst seizure), patients readily provide more information about their subjective seizure symptoms in these particular seizures.
Patients tend to catastrophize their seizure experiences.	Patients tend to normalize their seizure experiences when talking to a doctor.
Patients prefer metaphors depicting their seizures as a place or space they traveled through or to which they were confined.	Patients tend to describe their seizures as acting independently (and often as doing something to the patient).

*\*\*features that revealed statistically significant differences between PNES and ES patients.*

**Table 2.**  
 Conversation analysis diagnostic features in PNES and ES [16].

Signs that favor PNES	Evidence from primary studies
Long duration	Good
Fluctuating course	Good
Asynchronous movements	Good*
Pelvic thrusting	Good*
Side-to-side head or body movement	Good**
Closed eyes	Good
Ictal crying	Good
Memory recall	Good
Signs that favor ES	Evidence from primary studies
Occurrence from EEG-confirmed sleep	Good
Postictal confusion	Good
Stertorous breathing	Good
Other signs	Evidence from primary studies
Gradual onset	Insufficient
Nonstereotyped events	Insufficient
Flailing or thrashing movements	Insufficient
Opisthotonus "Arc de cercle"	Insufficient



Signs that favor PNES	Evidence from primary studies
Tongue biting	Insufficient
Urinary incontinence	Insufficient

*\*Frontal lobe partial seizures excluded. \*\*Convulsive events only.*

**Table 3.**  
Summary of evidence that supports the signs used to distinguish PNES from ES [6].

Diagnostic level	History	Witnessed event	EEG
Possible	+	By witness or self-report or self-description	No epileptiform activity in routine or sleep-deprived interictal EEG
Probable	+	By clinicians who reviewed recording or in person, showing semiology typical of PNES	No epileptiform activity in routine or sleep-deprived interictal EEG
Clinically established	+	By clinician experienced in diagnosis of seizure disorders (on video or in person), showing semiology typical of PNES while not on EEG	No epileptiform activity in routine or ambulatory ictal EEG during a typical ictus/event in which the semiology would make ictal epileptiform EEG activity expectable during equivalent epileptic seizures
Documented	+	By clinician experienced in diagnosis of seizure showing semiology typical of PNES while on video EEG	No epileptiform activity immediately before, during, or after ictus captured on ictal video EEG with typical PNES semiology

**Table 4.**  
Proposed diagnostic levels of certainty for PNES [6].

reduction of PNES [18]. The summary of four reasonably detailed communication strategies that have been published [19–22] is shown in **Table 5** [23].

## 10. Treatment of PNES

### I. Treatment of the underlying etiological factors and comorbidities:

Recognition and treatment of the “3Ps” (predisposing, precipitating, and perpetuating factors) are almost always necessary for symptom resolution. It may even be sufficient to treat the comorbid condition in conjunction with proper presentation of the diagnosis [12, 13].

### II. Patient engagement:

Brief *psychoeducation* of the patient and *motivational interviewing* after presenting diagnosis can reduce ambivalence about treatment and facilitate behavioral change in favor of the patient’s health and give the patient a sense of control (internal locus of control) [20]. *Motivational interviewing* can be particularly useful in patients who find it difficult to trust their claimed diagnosis and thus recurrently seek new healthcare providers despite previous findings documenting PNES [24].

Covered topic	Communication points delivered to patient
Negative diagnosis	What you do not have (i.e., epilepsy) What you do not need (i.e., treatment with AEDs*) – unless needed for other indications
Diagnostic method	How diagnosis was made (i.e., video-EEG* captured typical event) “It is common!,” frequently seen in long-term monitoring units
Genuine symptoms	Symptoms are real, not fabricated
Explanatory model (positive diagnosis)	Role of accumulating risk factors over time and automatic functional brain patterns
Suggestion	Some patients improve with reassurance that their events are not epileptic and once diagnosis is explained
Treatment and expectations	There are effective treatments Psychotherapy works through skills learning, “brain re-training” There is no sudden cure; treatment requires time and training

\*AEDs = antiepileptic drugs, EEG = electroencephalogram.

**Table 5.**  
*Diagnosis delivery: Summary of communication protocol [23].*

### III. Psychotherapeutic interventions:

#### A. Cognitive behavioral therapy

In a randomized controlled trial that compared cognitive behavioral therapy (CBT) to standard medical care, individual CBT was evaluated with a significant reduction in monthly event frequency after 12 sessions [25]. The following concepts were addressed in the CBT sessions: (1) treatment engagement; (2) reinforcement of independence; (3) distraction, relaxation, and refocusing techniques when episode is imminent; (4) graded exposure to avoided situations; (5) cognitive restructuring; and (6) relapse prevention.

#### B. Psychodynamic psychotherapy

Psychodynamic psychotherapy has not been examined as frequently as CBT, but favorable results have been demonstrated in uncontrolled studies using individual and group formats [26, 27].

#### C. Family therapy

Family therapy may be indicated when family system dysfunction is present since it is a contributor to symptoms of depression and to a poorer quality of life in PNES [28].

#### D. Mindfulness techniques

Mindfulness techniques promote the challenging of experiential avoidance while delineating personal values. In a case series that utilized a mindfulness-based treatment protocol, event reduction was attained using mindfulness techniques [29].

IV. Pharmacotherapy:

- A. The pharmacologic treatment of patients should begin with early tapering and discontinuation of the anti-epileptic drugs (AEDs).
- B. In people with mixed epileptic seizures (ES) and PNES, reduce high doses of AEDs or polytherapy if possible.
- C. Use psychopharmacologic agents to treat comorbidities.

Protocol of personalized psychological interventions in PNES

---

Triage:

- Patient's thoughts on diagnosis and potential treatment (locus of control, attributions, and perceived responsibility for recovery)
- Seizures occurrence and response to seizures (seizure description, frequency, hospital contact, and medications)
- Onset factors (home, work, and life events in the months prior to onset)
- Current circumstances (home, family, work, pastimes, and social support)
- Past history (other illness, traumatic events, and long-term life history)

Treatment:

- Treatment approach was based on a psychological formulation developed with the patient.
- The broad outline of the treatment covered the following: psychoeducation to patients and their families to develop an understanding of PNES\* and awareness of triggers, both external and internal; considering the context that may both prevent and perpetuate attacks; and identifying the attack prodromal phase and how to take remedial action.
- While the models used were integrative and varied according to the formulation, intervention was predominantly delivered in a CBT\* framework; other approaches were used on a case-by-case basis (see below).
- Session 1: assessment and formulation
- Sessions 2–10: interventions are used according to treatment targets that emerge from formulation:
  1. When social factors predominate in cause and maintenance:
    - Family therapy
    - Interpersonal therapy
    - Social interventions
  2. When internal thought processes/personal conscious behavior predominate in cause and maintenance:
    - Cognitive behavioral therapy
    - Behavioral management advice
  3. When internal conflicts such as grief or reaction to past trauma predominate in cause and maintenance:
    - Mindfulness and compassionate mind
    - Acceptance and commitment therapy
    - Counseling
    - Focused analytic therapy
    - Dialectical behavioral therapy
  4. When physiological states, current health problems, or habitual reactions to these problems predominate in cause and maintenance:
    - Psychological treatment for sleep dysregulation
    - Cognitive assessment remediation
    - Behavioral management advice

---

\*CBT = cognitive behavioral therapy, PNES = psychogenic non-epileptic seizures.

**Table 6.**  
*Protocol for psychological interventions in PNES.*

In an attempt to reach a consensus on a specific protocol of psychological interventions when dealing with PNES, Duncan et al. [30] proposed a protocol (see **Table 6**).

## 11. Evidence-based guide for management of PNES

The ILAE proposed the following management algorithm shown in **Table 6** [6] in an attempt to provide an evidence-based protocol for the management of PNES (**Table 7**).

Treatment steps	Direct evidence	Indirect evidence
Diagnosis	X	
Consider early Investigate (vEEG)	X	
Assessment	X	
Characterize: Neurologic comorbidity	X	
Psychiatric comorbidity Social/family conflict	X	
Communication of diagnosis	X	X
Explain: What PNES are not What PNES are		X
Psychiatric/psychological treatment	X	X
Patient engagement	X	X
Psychotherapy: CBT for PNES Family therapy	X	X
Antidepressants	X	X
Case management		X
Rehabilitation		X

*Note: vEEG = video electroencephalogram, CBT = cognitive behavioral therapy, PNES = psychogenic non-epileptic seizures.*

**Table 7.**  
 Management of psychogenic non-epileptic seizure and evidence basis [6] (updated from [31]).

IntechOpen

### **Author details**

Nirmeen A. Kishk<sup>1\*</sup> and Mai B. Nassar<sup>2</sup>


1 Department of Neurology, School of Medicine, Cairo University, Giza, Egypt

2 Department of Psychiatry, School of Medicine, Cairo University, Giza, Egypt

\*Address all correspondence to: [nirmeenkishk@kasralainy.edu.eg](mailto:nirmeenkishk@kasralainy.edu.eg)

### **IntechOpen**

---

© 2022 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 



## References

- [1] Hingray C et al. Heterogeneity of patients with functional/dissociative seizures: Three multidimensional profiles. *Epilepsia*. 2022;**00**:1-16
- [2] LaFrance WC, Devinsky O. The treatment of nonepileptic seizures: Historical perspectives and future directions. *Epilepsia*. 2004; **45**(s2):15-21
- [3] Brown RJ, Reuber M. Towards an integrative theory of psychogenic non-epileptic seizures (PNES). *Clinical Psychology Review*. 2016; **47**:55-70
- [4] Goldstein LH et al. Cognitive behavioural therapy for adults with dissociative seizures (CODES): A pragmatic, multicentre, randomised controlled trial. *The Lancet Psychiatry*. 2020;**7**(6):491-505
- [5] Rawlings GH, Reuber M. Health care practitioners' perceptions of psychogenic nonepileptic seizures: A systematic review of qualitative and quantitative studies, no. April 2018
- [6] LaFrance WC Jr, Reuber M, Goldstein LH. Management of psychogenic nonepileptic seizures. *Epilepsia*. 2013;**54**(1):53-67. DOI: 10.1111/epi.12106. PMID: 23458467
- [7] Bodde NM, Brooks JL, Baker GA, Boon PA, Hendriksen JG, Aldenkamp AP. Psychogenic non-epileptic seizures—diagnostic issues: a critical review. *Clin Neurol Neurosurg*. 2009;**111**(1):1-9. DOI: 10.1016/j.clineuro.2008.09.028. Epub 2008 Nov 18. PMID: 19019531
- [8] Baroni G et al. Variables associated with co-existing epileptic and psychogenic nonepileptic seizures: A systematic review. *Seizure*. 2016;**37**:35-40
- [9] Hill SW, Gale SD. Predicting psychogenic nonepileptic seizures with the personality assessment inventory and seizure variables. *Epilepsy & Behavior*. 2011;**22**(3):505-510
- [10] Jimenez XF, Bautista JF, Tilahun BS, Fan Y, Ford PJ, Tesar GE. Epilepsy & behavior bridging a clinical gap in psychogenic nonepileptic seizures: Mental health provider preferences of biopsychosocial assessment approaches. *Epilepsy & Behavior*. 2016;**56**:149-152
- [11] Elliott JO, Charyton C. Biopsychosocial predictors of psychogenic non-epileptic seizures. *Epilepsy Research*. 2014;**108**(9): 1543-1553
- [12] Sahaya K, Dholakia SA, Sahota PK. Psychogenic non-epileptic seizures: A challenging entity. *Journal of Clinical Neuroscience*. 2011;**18**(12):1602-1607
- [13] Stonnington CM, Barry JJ, Fisher RS. Conversion disorder. *The American Journal of Psychiatry*. 2006;**163**(9): 1510-1517
- [14] Davis BJ. Predicting nonepileptic seizures utilizing seizure frequency, EEG, and response to medication. *Eur Neurol*. 2004;**51**(3):153-156. DOI: 10.1159/000077287. Epub 2004 Mar 12. PMID: 15017116
- [15] Reuber M. Psychogenic nonepileptic seizures: Answers and questions. *Epilepsy & Behavior*. 2008;**12**(4):622-635
- [16] Jenkins L, Cosgrove J, Chappell P, Kheder A, Sokhi D, Reuber M. Neurologists can identify diagnostic linguistic features during routine seizure clinic interactions: results of a one-day teaching intervention. *Epilepsy & Behavior*. 2016;**64**:257-261

- [17] LaFrance WC, Baker GA, Duncan R, Goldstein LH, Reuber M. Minimum requirements for the diagnosis of psychogenic nonepileptic seizures: A staged approach: A report from the International League Against Epilepsy Nonepileptic Seizures Task Force. *Epilepsia*. 2013;**54**(11):2005-2018
- [18] McKenzie P, Oto M, Russell A, Pelosi A, Duncan R. Early outcomes and predictors in 260 patients with psychogenic nonepileptic attacks. *Neurology*. 2010;**74**(1):64-69
- [19] Hall-Patch L et al. Acceptability and effectiveness of a strategy for the communication of the diagnosis of psychogenic nonepileptic seizures. *Epilepsia*. 2010;**51**(1):70-78
- [20] Duncan R. Psychogenic nonepileptic seizures: Diagnosis and initial management. *Expert Review of Neurotherapeutics*. 2010;**10**(12):1803-1809
- [21] Mellers JDC. The approach to patients with 'non-epileptic seizures'. *Postgraduate Medical Journal*. 2005;**81**(958):498-504
- [22] Shen W, Bowman ES, Markand ON. Presenting the diagnosis of pseudoseizure. *Neurology*. 1990;**40**(5):756
- [23] Baslet G, Seshadri A, Bermeo-Ovalle A, Willment K, Myers L. Psychogenic non-epileptic seizures: An updated primer. *Psychosomatics*. 2016;**57**(1):1-17
- [24] Lundahl B et al. Motivational interviewing in medical care settings: A systematic review and meta-analysis of randomized controlled trials. *Patient Education and Counseling*. 2013;**93**(2):157-168
- [25] Goldstein LH et al. Cognitive-behavioral therapy for psychogenic nonepileptic seizures: A pilot RCT. *Neurology*. 2010;**74**(24):1986-1994
- [26] Barry JJ, Wittenberg D, Bullock KD, Michaels JB, Classen CC, Fisher RS. Group therapy for patients with psychogenic nonepileptic seizures: A pilot study. *Epilepsy & Behavior*. 2008;**13**(4):624-629
- [27] Mayor R, Howlett S, Grünewald R, Reuber M. Long-term outcome of brief augmented psychodynamic interpersonal therapy for psychogenic nonepileptic seizures: Seizure control and health care utilization. *Epilepsia*. 2010;**51**(7):1169-1176
- [28] Salmon P, Al-Marzooqi SM, Baker G, Reilly J. Childhood family dysfunction and associated abuse in patients with nonepileptic seizures. *Psychosomatic Medicine*. 2003;**65**(4):695-700
- [29] Baslet G, Dworetzky B, Perez DL, Oser M. Treatment of psychogenic nonepileptic seizures. *Clinical EEG and Neuroscience*. 2015;**46**(1):54-64
- [30] Duncan R, Anderson J, Cullen B, Meldrum S. Predictors of 6-month and 3-year outcomes after psychological intervention for psychogenic non epileptic seizures. *Seizure*. 2016;**36**:22-26
- [31] Reuber M, House AO. Treating patients with psychogenic non-epileptic seizures. *Current Opinion in Neurology*. 2002;**15**(2):207-211