

# Differences in Cognitive Profile of Psychogenic Nonepileptic and Epileptic Seizure Patients

Anand Thaman<sup>1</sup>, Naina Sharma<sup>2</sup>

## ABSTRACT

Psychogenic non-epileptic seizures (PNES) are the episodes of involuntary movements, altered consciousness, sensations, and perceptions, which otherwise look similar to epileptic seizures (ES), but without any abnormal electrical activities in brain areas. PNES is generally considered psychological in origin; however, latest theoretical models suggest the mediation of cognitive-emotional vulnerabilities in these patients. Cognitive deficits especially in executive domains have been reported in previous studies, but findings are largely equivocal. The present study attempted to evaluate the comparative profile of two seizure groups along with the healthy control. Selected tests from NIMHANS neuropsychological battery were applied to measure the executive cognitive domains. Results of study found common (response inhibition/flexibility) as well as different (attention, verbal category fluency, and visual working memory) cognitive performances of two seizure groups. These findings confirmed the genuineness of cognitive deficits in PNES patients and urge the mental health professionals to consider these vulnerabilities while making interventional strategies.

**Keywords:** Executive cognitive functioning deficits, Neuropsychological profile, Psychogenic non-epileptic seizures.

*Indian Journal of Private Psychiatry* (2020); 10.5005/jp-journals-10067-0062

## INTRODUCTION

Psychogenic non-epileptic seizures (PNES) are the episodes of involuntary movements, alteration in consciousness sensation, and perception, which otherwise look similar to epileptic seizures (ES), but without any abnormal electrical activities in brain areas. PNES is a neuropsychiatric condition seen in both neurology and psychiatric clinics. About 20% of the patients who experience seizure of any kind receive final diagnosis of PNES.<sup>1</sup>

Where epilepsy is a neurological or biological condition, PNES was generally considered purely as psychological in origin. However, etiology of PNES is very complex and multidimensional. Various biopsychosocial factors have been explored and hypothesized to cause this phenomenon. These factors include biological vulnerabilities<sup>2-6</sup> such as childhood and early life traumas, sexual and physical abuse,<sup>7</sup> psychiatric psychopathology,<sup>8,9</sup> dysfunctional family environment,<sup>7,10</sup> and abnormal emotional processing.<sup>11</sup> According to the most accepted model, when distressful experiences or memories overwhelm, it de-stabilizes the already vulnerable cognitive and emotional system.<sup>12</sup> Thus, all components of mental faculties disintegrate and make the brain incapable to synthesize the incoming information. Sensory, motor, and cognitive systems become automated and ultimately manifest in the form of seizures.<sup>13,14</sup>

The above prepositions clearly indicate that various cognitive operations along with other factors mediate in developing PNES. In clinical practice too, patients with PNES bring various cognitive complaints like memory loss, delayed information processing, attentional deficits, numbness and freezing response during verbal expressions, poor control over emotions, etc. It suspects the clinician for possible brain pathology; thus, these patients undergo various neuropsychological evaluations.<sup>15</sup> Literature reporting cognitive findings in PNES patients is scarce and largely equivocal. Most of these studies explored limited domains and compared PNES patients with the ES group. Some reported better performance,<sup>16</sup> others found equal,<sup>17,18</sup> and yet other studies reported worst

<sup>1</sup>Psychological Services Unit, Manas Psychology Epilepsy and De-addiction Centre, Ludhiana, Punjab, India

<sup>2</sup>Department of Distance Education, Punjabi University, Patiala, Punjab, India

**Corresponding Author:** Anand Thaman, Department of Clinical Psychology, Manas Psychology Epilepsy and De-addiction Centre, Ludhiana, Punjab, India, Phone: +91 9855977790, e-mail: thaman.anand@gmail.com

**How to cite this article:** Thaman A, Sharma N. Differences in Cognitive Profile of Psychogenic Nonepileptic and Epileptic Seizure Patients. *Ind J Priv Psychiatry* 2020;14(2):62-67.

**Source of support:** Nil

**Conflict of interest:** None

performance on cognitive tasks.<sup>19,20</sup> Differences in these findings are attributed to the variability of etiology in both disorders.

Cognitive deficits in ES patients are explained in the context of organic brain conditions, such as structural damage or change after a head injury or infection, acquired malformations in various brain regions, effects of antiepileptic drugs (AED), and asymmetrical electrical activities in brain cortices due to seizures,<sup>21-23</sup> whereas literature suggest that cognitive deficits in PNES patients could be related to abnormal functioning of those brain areas that are responsible for emotions, executive control, and sensory-motor responses, such as prefrontal cortex, insula, inferior frontal gyrus, parietal cortex, and central sulcus.<sup>24</sup> Functional magnetic resonance imaging studies done on patients with PNES, conversion, and dissociative disorders support the "vulnerable cognitive emotional model," showing abnormal activities in areas that mediate attention process, concentration, memory, verbal abilities, and executive control.<sup>25,26</sup>

Other explanation for the variable cognitive profile in PNES patients is attributed to psychiatric symptomology, such as

somatization, posttraumatic stress disorder, anxiety, and depression, which also involved emotional and cognitive elements.<sup>27</sup> These psychopathologies bear prolonged emotional distress that affects cognitive functioning.<sup>28</sup> Because PNES patients show high incidents of psychiatric comorbidity,<sup>29</sup> deficits in performance on various neuropsychological tasks designed to measure cognitive functions may have correlation with those interactional factors that underlying already prevailing psychopathology in these patients.<sup>30</sup> It has also been noticed that PNES patients are generally misdiagnosed for ES and prescribed for AED for a longer duration, which may also have cognitive side effects.<sup>31</sup>

The existing literature emphasized the need for comprehensive neuropsychological assessment in PNES patients, which should focus on executive cognitive functions concerning attention, working memory, visuospatial task, language fluency, task shifting, response selection, and inhibition. The findings should be synthesized, and a clear difference in terms of cognitive variability in two seizure groups should be portrayed. These findings may be helpful in making differential diagnosis and developing interventional or remedial psychological strategies as a part of the treatment plan for these patients. Keeping in view the above objectives, the current study has been designed to compare the cognitive profile of PNES, ES, and healthy control groups. It was hypothesized that PNES patients will show more cognitive deficits as compared to ES and control groups.

## MATERIALS AND METHOD

The current study was conducted at a reputed neuropsychiatric clinic of xxxxxx. This study was dully approved by the ethical committee of the xxxxxxxxxxxxxxxx. The sample of the study consisted of 150 patients/participants divided equally (50) into three groups, that is, PNES, ES, and healthy control.

### Inclusion criteria

- Confirmed diagnosis of PNES/ES given by neurologist/neuropsychiatrist supported by video electroencephalogram (EEG) /EEG/radiological examination and history of symptoms.
- Minimum 2 seizure episodes with one episode within the last 15 days.
- Age between 18 and 40 years.
- Minimum accepted qualification was X and able to sign written consent.

### Exclusion criteria

- Coexisting condition (PNES/ES).
- History of congenital or developmental disorder
- Psychosis, bipolar, mania, obsessive-compulsive disorder, or major depression as a primary diagnosis
- Seizures occur solely due to alcohol or drug consumption or withdrawal.

### Matched control healthy sample

Participants in this group have been recruited from the community after matching sociodemographic characteristics with PNES patients and whose score was less than 10 on PGI health questionnaire.<sup>32</sup> Written consent was taken from all the subjects.

Tools used are as follows:

- (a) Semi-structured interview pro forma

Semi-structured interview pro forma was devised to collect information on sociodemographic variables, including Kuppuswamy's socioeconomic status scale.<sup>33</sup>

(b) NIMHANS battery of neuropsychological assessment  
NIMHANS battery of neuropsychological assessment<sup>34</sup> has been developed at the National Institute of Mental Health and Neurosciences, Bangalore, to meet the cross-cultural need of neuropsychological assessment in Indian settings. In the present study, 7 tests were selected to assess the executive functions, that is, attention, working memory (visual and auditory), verbal fluency (phonetic and category), and response inhibition. The battery possesses good factorial and constructs validity and also carries good sensitivity for predicting cognitive deficits.

The description of tests is given below:

- Color trail test*: Task requires focused attention, sequencing, and selecting targeted stimuli while ignoring others.
- Digit vigilance test*: It requires sustained attention to complete the task while filtering the targets that are not required.
- Controlled verbal oral test*: To measure the phonemic fluency
- Animal naming test*: To measure the category fluency
- N-back verbal memory test*: To assess the verbal working memory
- N-back visual memory test*: To assess the visual working memory
- Stroop test*: To measure the cognitive flexibility and response inhibition

## Statistical Analysis

The whole data were analyzed using the Microsoft Excel (2007) advanced statistical packages, that is, MegaStat, real statistics, and XLSAT. Descriptive statistics, such as frequencies, mean, median, range, and standard deviation, were calculated for categorical and continuous variables. Normality of the data was checked using visual inspection of histograms, QR plots, skewness, and kurtosis. Many of the variables were found skewed and violated the assumption of normality. Hence, nonparametric statistical techniques, such as chi-square test, KruskalWallis, and MannWhitney U test, were used to analyze the data. Level of significance was set as  $p < 0.05$ .

## RESULTS

Table 1 shows the sociodemographic profile of the three groups, that is, PNES, ES, and control. Both PNES and ES groups consisted of more number of females (PNES = 37; ES 33). The number of married patients in the PNES group (33) was higher, whereas the unmarried number was found higher in the ES group(30).

Three groups on variables such as the type of family, nature of the habitat, education, and socioeconomic status were noncomparable. No statistically significant difference was found on any of the variables except marital status ( $\chi^2 = 12.6; p < 0.05$ ). Thus, it was assumed that these sociodemographic variables would have a consistent effect in three groups on cognitive variables measured during the study.

Table 2 shows the comparison of scores of the three groups on measured cognitive domains, that is, focused attention (time taken on trails 1 and 2), sustained attention (digit vigilance test: time taken and number of errors), verbal working memory (N-back 1 and N-back 2: number of hits and errors), visual working memory (N-back 1 and N-back 2: number of hits and errors), and response inhibition/ mental flexibility (Stroop effect).

Results of the study found three groups were significantly different on focused attention (both trail 1 and trail 2:  $p < 0.001$ ), sustained attention (both hits and errors:  $p < 0.001$ ), category verbal fluency ( $p < 0.05$ ), visual working memory (N1 hits:  $p < 0.001$ ; errors:  $p < 0.01$ , N2 errors:  $p < 0.05$ ), and response inhibition (Stroop

**Table 1:** Comparison of sociodemographic profile of patients with PNES, ES, and healthy control group

S. No.	Variable	PNES	ES	Control	$\chi^2/f$ value
1.	Gender				
	Male	13	17	13	1.04
	Female	37	33	37	
2.	Age	28.7 ± 6.1	29.6 ± 6.84	29.2 ± 5.4	0.09
3.	Marital status				
	Married	33	20	32	
	Unmarried	15	30	16	12.6*
	Divorced	1	0	1	
	Remarried	1	0	1	
4.	Family type				
	Nuclear	29	36	35	2.88
	Joint	21	14	15	
5.	Type of habitat				
	Rural	12	14	11	
	Urban	26	27	25	1.54
	Semi-urban	12	9	14	
6.	Education				
	High school	13	8	11	2.51
	10+2	17	19	14	
	Graduation/PG	20	23	25	
7.	Socioeconomic status				
	Upper	6	3	5	12.51
	Upper middle	29	35	42	
	Lower middle	12	11	3	
	Upper lower	3	1	0	

\* $p < 0.05$ 

effect:  $p < 0.001$ ). However, no statistically significant difference was observed in the domains of phonemic verbal fluency and auditory working memory.

In pair-wise comparison of PNES and ES groups, it was found that PNES took significantly more time on a complex task (trail 2) involving focused attention ( $p < 0.05$ ), committed significantly more errors on sustained attention task ( $p < 0.001$ ) and less efficient in category fluency ( $p < 0.01$ ), but showed better performance in simple visual working memory (N-1) as compared to ES group ( $p < 0.001$ ). In the second pair comparison between PNES and control groups, it was observed that the PNES group performed significantly lower on both focused and sustained attention ( $p < 0.001$ ), committed significantly more errors on complex (N-2) visual working memory tasks, and showed deficits on response inhibition ( $p < 0.001$ ). In the third paired comparison between ES and control groups, the ES group took more time on the task requiring sustained attention ( $p < 0.001$ ) and performed lower on the task involving focused attention ( $p < 0.01$ ), visual working memory ( $p < 0.05$ ), and response inhibition ( $p < 0.001$ ).

## DISCUSSION

Current research is mainly interested in evaluating the comparative cognitive profile of two seizure groups. Based on the recent trends in neuropsychological assessment, the research was guided to view cognitive impairments in the context of an integrated cognitive-emotional model.<sup>12,30</sup> In other words, only those cognitive domains had been explored, which are presumed to be directly related to emotional processing.

Results of this study found more focused attention deficits in PNES than that in ES group, especially while performing on a complex task that needed attention on target stimuli while ignoring the distractors. It indicated difficulty in the selection process while exerting less control on the bulk of information due to interference. Similarly, on sustained attention task, PNES patients displayed significant filtration deficits (in terms of more errors) than ES and healthy control groups, which also requires appropriate selection, executive control, and sustained attentional effort throughout the task.

These results are at par with previous studies that confirmed the presence of nonspecific attentional deficits in PNES patients. Two studies reported scores of PNES patients significantly below the normal range on neuropsychological battery including attention.<sup>4,35</sup> Another study by Strutt et al.<sup>17</sup> showed declined scores on executive task performance including attention and concentration in PNES patients. Similarly, Bakvis et al.<sup>28</sup> reported significant difficulty in avoiding distractors and greater interference effect in PNES patients even under normal conditions.

Attentional deficits in ES patients are attributed to the direct effect of seizures, associated neurodevelopmental conditions such as attention deficit hyperactivity disorder or learning disability, and effect of AED.<sup>36,37</sup> But in PNES patients, attentional deficits are associated with multiple factors, including biological, psychological, and emotional. Neuroimaging studies on PNES patients confirmed altered structural and functional connectivity in posterior parietal, prefrontal, insular, anterior cingulate, and association cortex of the brain.<sup>26,38</sup> All these areas are involved in optimal arousal, selection, and executive control necessary for the attentional process. PNES patients have a mechanism similar to dissociation<sup>12</sup> characterized by multiple streams of information processing instead of selective processing along with poor inhibitory control that results in the unequal allocation of attentional resources. In other words, some information becomes overly focused, while others remain completely ignored.<sup>39,40</sup>

Attentional deficits in PNES are also explained in terms of faulty arousal mechanism that is similar to trauma-related disorders.<sup>41</sup> These patients oscillate between the state of hyper and hypoarousal before, during, and after the attack, making it difficult to integrate the incoming cortical information that is possible only when the system is in an optimal zone of arousal.<sup>12,42</sup> Other causes of attentional deficits in PNES patients can be due to the associated psychopathology in PNES patients<sup>43</sup> and emotional dysregulation mechanism.<sup>19,44</sup> Due to active suppression and cognitive avoidance, the attentional process in PNES patients becomes biased, which disrupts selection and switching (in other words cognitive control) more difficult during information processing.<sup>45</sup>

On verbal fluency tasks, three groups were found to be equal on phonemic fluency; however, PNES patients performed significantly below than ES for category fluency, which indicates deficits in lexical control in verbal expressions. These results are partially in accordance with previous findings. For example, Fargo et al.<sup>46</sup> in their study found no difference between PNES and ES on category fluency, but TLE patients reported significant phonemic fluency deficits. However, the same study found verbal skill deficits related to expressions (Boston Naming Test) in PNES patients. These results were replicated in another study by Black et al.<sup>47</sup> that reported comparable differences between PNES and ES on rapid word generation test. Contrary to this, Binder et al.<sup>19</sup> found more verbal skill deficits in ES as compared to PNES. A meta-analysis study by Metternich et al.<sup>48</sup> also found significant impairments

**Table 2:** Comparison of cognitive deficits in patients with PNES, ES, and healthy control group ( $N = 50$  in each group)

S. No.	Cognitive domain	Test name	PNES (median/ range)	ES (median range)	Control (median range)	H value	Pair-wise comparison of three groups (U value)		
							PNES vs ES	PNES vs control	ES vs control
1.	Focused attention	Trail 1	56.50 (18–146)	50.50 (18–102)	30.50 (23–85)	15.05***	1163.5	747***	788***
		Trail 2	107.50 (48–204)	94.50 (47–198)	69.00 (43–133)	32.78***	1008.5*	461.5***	657***
2.	Sustained attention	Digit vigilance test	526.50 (292–855)	528.00 (310–786)	463.50 (276–876)	17.61***	1206	711***	738.5***
		No. of errors	37 (7–93)	24 (7–59)	21 (5–48)	22.50***	706.5***	624***	1104
3.	Verbal fluency	Phonemic fluency	6.15 (3.33–11.3)	6.00 (2–9)	6.50 (2.33–16.3)	2.672	1045	1223	1047
		Category fluency	14 (6–23)	16.50 (5–22)	15 (11–24)	7.386*	874**	1032	1043.5
4.	Verbal working memory	N-back 1	9 (5–9)	9 (7–9)	9 (5–9)	2.691	1152	1141	1044
		No. of errors	0 (0–3)	0 (0–8)	1 (0–4)	2.186	1154	1052	1153
		N-back 2	7 (2–9)	7.50 (3–9)	7 (2–9)	3.725	1019.5	1238	1013.5
5.	Visual working memory	No. of errors	3 (1–9)	3 (0–8)	3 (0–7)	0.452	1210	1151	1201
		N-back 1	7 (1–9)	6 (3–9)	8 (3–9)	17.54***	777.5***	1187	701***
		No. of errors	3 (0–17)	5 (1–11)	2 (0–10)	11.34**	902**	1164	778.5***
6.	Response inhibition	N-back 2	3 (0–7)	3 (0–8)	3 (0–8)	2.51	1043	1238	1073
		No. of errors	12 (5–19)	11 (4–17)	9.50 (3–21)	6.40*	1152	895**	997*
		Stroop test	273 (106–634)	264 (118–609)	186 (67–293)	47.24***	1113	362***	425***

\* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ 

in focal epileptic (especially TLE) patients on both phonemic and category fluency.

Category fluency is more related to executive control located in frontal lobe areas, but these deficits are mostly seen in TLE due to unknown reasons.<sup>49</sup> These deficits were not found in the current study as classification of ES patients was not done according to the type of epilepsy. On the contrary, comparatively low performance of PNES patients on category fluency can be due to known executive control deficits in these patients.<sup>50</sup> Category fluency is also closely related to the attention process,<sup>51</sup> which is generally impaired in PNES patients, and the same had been confirmed in the current research.

Findings on working memory deficits in ES patients are incongruent with previous literature,<sup>52–54</sup> however, these studies do not specify the type of working memory impairments. Few researches on visual/spatial memory deficits confirmed their presence in temporal and frontal lobe epilepsies<sup>55,56</sup> due to hippocampal-based pathology and reduced structural connectivity between posterior visual and prefrontal cortex during and after seizure activities.<sup>47,57</sup> Nonspecific working memory deficits have also been reported in PNES patients and are usually correlated to psychopathology, attentional deficits as well as seizure-related characteristics, such as the age of onset and duration.<sup>28,47,58</sup> On the contrary, O'Brien et al.<sup>59</sup> in their study found spatial working memory deficits in PNES patients as compared to healthy control. The presence of visual memory impairments in PNES patients

could also be the result of attentional and decision-making deficits leading to discrimination and recognition difficulties for visual material.<sup>60,61</sup>

Response inhibition, which is an aspect of executive control, was found to be affected in both ES and PNES groups. These results are similar to a comparative study by Celick et al.<sup>49</sup> that reported higher Stroop interference time in both PNES and ES groups as compared to healthy control. In another study, Turner et al.<sup>62</sup> found no difference in response inhibition between ES, PNES, and combined ES/PNES groups; however, these results were not compared with the healthy control group. Other studies on response inhibition also found deficits in patients with frontal and temporal epilepsies.<sup>63,64</sup> Deficits of response inhibition in ES patients have been associated with seizures-related activities that either over-activate or reduce connectivity between left frontal and temporal cortex areas that mediate executive control.<sup>63</sup> In PNES patients, abnormal functional connectivity of insular and cingulate region that is associated with executive control, set-shifting, attention, and response selection can be attributed to low performance.<sup>17,47</sup> Therefore, both ES and PNES may have a common brain mechanism that suggests poor executive control causing similar semiology in terms of automaticity in behavioral and motor symptoms during seizures.

The findings of the current study have both therapeutic and diagnostic values. Unlike ES patients, cognitive deficits in PNES patients are nonspecific and associated with psychological and emotional factors. Thus, suitable psychological interventions

like relaxation therapy, stress management, CBT, and emotional processing therapies can be designed to reduce the underlying distress. It will benefit these patients by improving overall cognitive functioning and quality of life. On the contrary, variable characterization of cognitive deficits in two groups may differentiate the type of seizures and can be supportive in taking a clinical decision about diagnosis during a conflicting situation.

Like every study, this research also had some limitations. Only a limited numbers of tests were used for cognitive evaluation. It is suggested that a more comprehensive evaluation is required for better characterization and portraying systematic profile in PNES and ES groups. More sophisticated and computer-based robust tests should be applied for precise measurement of pattern and type of deficits in the vast area of cognitive functioning. Many variables like baseline cognitive measures, duration of seizures, age of onset, dynamism in cognitive characteristics, grouping of patients according to the type of epileptic seizures, etc., which had been ignored in the current study, can be considered in a more rigorous study design. Finally, these findings must be verified objectively through neurobiological evidence before their generalization. Despite these limitations, the current study added useful data and partially confirmed the already hypothesized cognitive-emotional model behind PNES.

## CONCLUSION

Results of the current study found both common and different patterns of cognitive deficits in PNES and ES groups. Attentional deficits, verbal category fluency, and visual memory deficits were the key differences, whereas executive control in terms of response inhibition was found common among both seizure groups. The above findings confirmed the cognitive vulnerabilities as suggested in PNES models and emphasize the need to consider these deficits 'genuine' in these patients. Understanding cognitive differences will supplement the decision for differential diagnosis and directs the mental health professionals to use psychosocial intervention in alleviating distress and improving well-being of these patients.

## ACKNOWLEDGMENT

We are thankful to Dr. Rajeev Gupta, Consultant Psychiatrist, Manas Clinic and Manas Hospital, Ludhiana, for allowing us to conduct research at his center and providing valuable guidance from time to time.

## REFERENCES

1. Kotsopoulos IA, de Krom MC, Kessels FG, et al. The diagnosis of epileptic and non-epileptic seizures. *Epilepsy Res* 2003;57:59–67. DOI: 10.1016/j.epilepsyres.2003.10.014.
2. Westbrook LE, Devinsky O, Geocadin R. Non-epileptic seizures after head injury. *Epilepsia* 1998;39:978–982. DOI: 10.1111/j.1528-1157.1998.tb01447.x.
3. Pakalnis A, Paolicchi J. Psychogenic seizures after head injury in children. *J Child Neurol* 2000;15:78–80. DOI: 10.1177/088307380001500202.
4. Cragar DE, Berry DTR, Fakhoury TA, et al. A review of diagnostic techniques in the differential diagnosis of epileptic and nonepileptic seizures. *Neuropsychol Rev* 2002;12:31–64. DOI: 10.1023/a:1015491123070.
5. Reuber M, Fernandez G, Helmstaedter C, et al. Are there physical risk factors for psychogenic non-epileptic seizures in patients with epilepsy? *Seizure* 2003;12:561–567. DOI: 10.1016/s1059-1311(03)00064-5.
6. Silver LB. Conversion disorder with pseudoepileptic seizures in adolescence: a stress reaction to unrecognized and untreated learning disabilities. *J Am Acad Child Psychiatry* 1982;21:508–512. DOI: 10.1016/s0002-7138(09)60803-7.
7. Krawetz P, Fleisher W, Pillay N, et al. Family function in subjects with pseudoepileptic seizures and epilepsy. *J Nerv Ment Dis* 2001;189:38–43. DOI: 10.1097/00005053-200101000-00007.
8. Jones SG, O'Berien TJ, Adams SJ, et al. Clinical characteristics and outcome in patients with PNES. *J Psychosom Med* 2010;72:487–497. DOI: 10.1097/PSY.0b013e3181d96550.
9. Senevirantne U, Briggs B, Lowenstern D, et al. The spectrum of psychogenic non-epileptic seizures and comorbidities seen in epilepsy monitoring unit. *J Clin Neurosci* 2011;18:361–363. DOI: 10.1016/j.jocn.2010.07.120.
10. Lesser RP. Psychogenic Seizures. *Neurology* 1996;46:1499–1507. DOI: 10.1212/wnl.46.6.1499.
11. Robert NA, Reuber M. Alteration of consciousness in psychogenic nonepileptic seizures: emotion, emotion regulation and dissociation. *Epilepsy Behav* 2014;30:43–49. DOI: 10.1016/j.yebeh.2013.09.035.
12. Baslet G. Psychogenic non-epileptic seizures: a model of their pathogenic mechanism. *Seizure* 2011;20:1–13. DOI: 10.1016/j.seizure.2010.10.032.
13. Bowman ES. Why conversion seizures should be classified as a dissociative disorder. *Psychiatr Clin North Am* 2006;29:185–211. DOI: 10.1016/j.psc.2005.10.003.
14. Kyuk J, Van Dyck R, Spinhoven P. The case for a dissociative interpretation of pseudoepileptic seizures. *J Nerv Ment Dis* 1996;184:468–474. DOI: 10.1097/00005053-199608000-00003.
15. Prigatano GP, Hill SW. Cognitive complaints, affect disturbances and neuropsychological functioning in adults with psychogenic non-epileptic seizures. In: LaFrance WC and Schachter SC (eds) *Non-epileptic Seizures: culture, cognition and personality cluster*. London: Cambridge Press, 2018, pp. 158–164.
16. Darne DL, Williamson DJ, Stroup ES, et al. Cognitive impairment is not equal in patients with epileptic and psychogenic nonepileptic seizures. *Epilepsia* 2006;47:1879–1886. DOI: 10.1111/j.1528-1167.2006.00611.x.
17. Strutt AM, Hill SW, Scott BM, et al. A comprehensive neuropsychological profile of women with epileptic and psychogenic non-epileptic seizures. *Epilepsy Behav* 2011;20:24–28. DOI: 10.1016/j.yebeh.2010.10.004.
18. Swanson SJ, Springer JA, Benbadis SR, et al. Cognitive and psychological functioning in patients with non-epileptic seizures. In: Gate JR, Rowan AJ (eds) *Non-epileptic seizures*. Boston, MA: Butterworth-Heinemann, 2000, pp. 124–137.
19. Binder LM, Kindermann SS, Heaton RK, et al. Neuropsychological impairment in patients with nonepileptic seizures. *Arch Clin Neuropsychol* 1998;13:513–522. Available at: <https://pubmed.ncbi.nlm.nih.gov/14590635/>
20. Bortz JJ, Prigatano GP, Blum D, et al. Differential response characteristics in nonepileptic and epileptic seizures patients on a test of verbal learning and memory. *Neurology* 1995;45:2029–2034. DOI: 10.1212/wnl.45.11.2029.
21. Holmes GL. Cognitive impairment in epilepsy: the role of network abnormalities. *Epileptic Disord* 2015;17:101–116. DOI: 10.1684/epd.2015.0739.
22. Hermann B, Meador KJ, Gaillard WD, et al. Cognition across the lifespan: antiepileptic drugs, epilepsy or both? *Epilepsy Behav* 2010;17:1–5. DOI: 10.1016/j.yebeh.2009.10.019.
23. Kleen JK, Scott RC, Holmes GL, et al. Hippocampal interictal epileptiform activity disrupt cognition in humans. *Neurology* 2013;81:18–24. DOI: 10.1212/WNL.0b013e318297ee50.
24. Van der Kruis SJ, Jagannathan SR, Bodde NM, et al. Resting state network and dissociation in psychogenic non-epileptic seizures. *J Psychiatr Res* 2014;54:126–133. DOI: 10.1016/j.jpsychires.2014.03.010.
25. Van der Kruis SJ, Bodde NM, Vaessen MJ, et al. Functional connectivity of dissociation in patients with psychogenic

- nonepileptic seizures. *J Neurol Neurosurg Psychiatry* 2012;83:239–247. DOI: 10.1136/jnnp-2011-300776.
26. Ding J, An D, Liao W, et al. Abnormal functional connectivity density in psychogenic non-epileptic seizures. *Epilepsy Res* 2014;108:1184–1194. DOI: 10.1016/j.eplepsyres.2014.05.006.
  27. Kret ME, Ploeger A. Emotional processing deficits: a liability spectrum providing insight into comorbidity of mental disorders. *Neurosci Biobehav Rev* 2015;52:153–171. DOI: 10.1016/j.neubiorev.2015.02.011.
  28. Bakvis P, Spinhoven P, Putman P, et al. The effect of stress induction on working memory in patients with psychogenic nonepileptic seizures. *Epilepsy Behav* 2010;19:448–454. DOI: 10.1016/j.yebeh.2010.08.026.
  29. Diprose W, Sundram F, Menkes DB. Psychiatric comorbidity in psychogenic nonepileptic seizures compared with epilepsy. *Epilepsy Behav* 2016;56:123–130. DOI: 10.1016/j.yebeh.2015.12.037.
  30. Willment K, Hill M, Baslet G, et al. Cognitive impairment and evaluation in psychogenic non-epileptic seizures: an integrated cognitive-emotional approach. *Clin EEG Neurosci* 2015;46:42–53. DOI: 10.1177/1550059414566881.
  31. Reuber M, Fernandez G, Helmstaedter C, et al. Evidence of brain abnormality in patients with psychogenic non-epileptic seizures. *Epilepsy Behav* 2002;39:249–254. DOI: 10.1016/s1525-5050(02)00004-5.
  32. Wig NN, Verma SK. PGI health questionnaire N-1: a simple neuroticism scale in India. *Indian J Psychiatry* 1971;15:80–88. Available at: <https://www.indianjpsychiatry.org/article.asp?issn=0019-5545;year=1973;volume=15;issue=1;page=80;epage=88;aulast=Wig;type=0>
  33. Kohli C, Kishore J, Kumar N. Kuppuswamy's socio-economic scale-update for July 2015. *Int J Prev Curat Comm Med* 2015;1:26–28. Available at: [https://www.researchgate.net/profile/Jugal-Kishore-2/publication/312894228\\_Kuppuswamy%27s\\_Socioeconomic\\_Scale-Update\\_for\\_July\\_2015/links/5889d271458515701203616f/Kuppuswamy's-Socioeconomic-Scale-Update-for-July-2015.pdf](https://www.researchgate.net/profile/Jugal-Kishore-2/publication/312894228_Kuppuswamy%27s_Socioeconomic_Scale-Update_for_July_2015/links/5889d271458515701203616f/Kuppuswamy's-Socioeconomic-Scale-Update-for-July-2015.pdf)
  34. Rao SL, Shubbakrishna DK, Gopukumar K. *NIMHANS neuropsychology battery manual*, 1st ed. Bangalore: National Institute of Mental Health and Neurosciences, 2004.
  35. Kalogjera-Sackellares D, Sackellares JC. Intellectual and neuropsychological features of patients with psychogenic pseudoseizures. *Psychiatry Res* 1999;86:73–84. DOI: 10.1016/s0165-1781(99)00016-5.
  36. Schubert R. Attention deficit disorder and epilepsy. *Paediatr Neurol* 2005;32:1–10. DOI: 10.1016/j.pediatrneurol.2004.06.007.
  37. Williams AE, Giust JM, Kronenberger WG, et al. Epilepsy and attention-deficits hyperactivity disorder: links, risk, and challenges. *Neuropsychiatr Dis Treat* 2016;9:287–296. DOI: 10.2147/NDT.S81549.
  38. Mcsweeney M, Reuber M, Levita L. Neuroimaging studies in patients with psychogenic nonepileptic seizures: a systematic review. *Neuroimage Clin* 2017;16:210–221. DOI: 10.1016/j.nicl.2017.07.025.
  39. Dorahy MJ. The dissociative processing style: a cognitive organization activated by perceived or actual threat in clinical dissociators. *J Trauma Dissociation* 2006;7:29–53. DOI: 10.1300/j229v07n04\_03.
  40. De Ruiter MB, Phaf RH, Veltman DJ, et al. Attention as a characteristic of nonclinical dissociation: an event related potential study. *Neuroimage* 2003;19:376–390. DOI: 10.1016/s1053-8119(03)00099-5.
  41. Bakvis P, Roelofs K, Kuyk J, et al. Trauma, stress and preconscious threat processing in patients with psychogenic nonepileptic seizures. *Epilepsia* 2009;50:1001–1011. DOI: 10.1111/j.1528-1167.2008.01862.x.
  42. Ogden P, Minton K, Pain CK. *Trauma and the body: a sensorimotor approach to psychotherapy*. New York: W.W. Norton and Company, 2006.
  43. Brown RJ, Reuber M. Towards an integrative theory of psychogenic non-epileptic seizures (PNES). *Clin Psychol Rev* 2016;47:55–70. DOI: 10.1016/j.cpr.2016.06.003.
  44. Baslet G, Seshadri M, Bermeo-Ovalle A, et al. Psychogenic non-epileptic seizures: an update primer. *Psychosomatics* 2016;57:1–17. DOI: 10.1016/j.psym.2015.10.004.
  45. Gul A, Ahmad H. Cognitive deficits and emotional regulation strategies in patients with psychogenic nonepileptic seizures: a task switching study. *Epilepsy Behav* 2014;32:108–113. DOI: 10.1016/j.yebeh.2014.01.015.
  46. Fargo JD, Schefft BK, Dulay MF, et al. Confrontation naming in individuals with temporal lobe epilepsy: a quantitative analysis of praphasic error subtypes. *Neuropsychology* 2005;19:603–611. DOI: 10.1037/0894-4105.19.5.603.
  47. Black LC, Schefft BK, Howe SR, et al. The effect of seizure on working memory and executive functioning performance. *Epilepsy Behav* 2010;17:412–419. DOI: 10.1016/j.yebeh.2010.01.006.
  48. Metternich B, Buschmann F, Wagner K, et al. Verbal fluency in focal epilepsy: a systematic review and meta-analysis. *Neuropsychol Rev* 2014;24:200–218. DOI: 10.1007/s11065-014-9255-8.
  49. Jaimes-Bautista AG, Rodriguez-Camacho M, Martinez-Juarez I, et al. Semantic processing impairment in patients with temporal lobe epilepsy. *Epilepsy Res Treat* 2015;16:746745. DOI: 10.1155/2015/746745.
  50. Celik AO, Kurt P, Yener G, et al. Comparison of cognitive impairment between patients having epilepsy and psychogenic nonepileptic seizures. *Arch Neuropsychiatry* 2015;52:163–168. DOI: 10.5152/npa.2015.7290.
  51. Bizzozero I, Scotti S, Clerici FP, et al. On which abilities are category fluency and letter fluency grounded? A confirmatory factor analysis of 53 Alzheimer's dementia patients. *Dement Geriatr Cogn Disord Extra* 2013;3:179–191. DOI: 10.1159/000351418.
  52. Grippo A, Pelosi L, Mehta V, et al. Working memory in temporal lobe epilepsy: an event related potential study. *Electroencephalogr Clin Neurophysiol* 1996;99:200–213. DOI: 10.1016/0013-4694(96)95705-3.
  53. Krauss GL, Summerfield M, Brandt J, et al. Mesial temporal spikes interfere with working memory. *Neurology* 1997;49:975–980. DOI: 10.1212/wnl.49.4.975.
  54. Axmacher N, Elger CE, Fell J. Working memory related hippocampal deactivation interferes with long term memory formation. *J Neurosci* 2009;29:1052–1060. DOI: 10.1523/JNEUROSCI.5277-08.2009.
  55. Abrahams S, Morris RG, Polkey CE, et al. Hippocampal involvement in spatial and working memory: a structural MRI analysis of patients with unilateral mesial temporal lobe sclerosis. *Brian Cogn* 1999;41:39–65. DOI: 10.1006/brcg.1999.1095.
  56. Myatchin I, Lage L. Impaired spatial working memory in children with well controlled epilepsy: an event-related potential study. *Seizure* 2011;20:143–150. DOI: 10.1016/j.seizure.2010.11.005.
  57. Gleißner U, Helmstaedter C, Elger CE. Right hippocampal contribution to visual memory: a presurgical and post surgical study in temporal lobe epilepsy. *J Neurol Neurosurg Psychiatry* 1998;65:665–669. DOI: 10.1136/jnnp.65.5.665.
  58. Kent GP, Schefft BK, Howe SR, et al. The effects of duration of intractable epilepsy on memory function. *Epilepsy Behav* 2006;9:469–477. DOI: 10.1016/j.yebeh.2006.07.005.
  59. O'Brien, FM, Fortune GM, Dicker P, et al. Psychiatric and neuropsychological profiles of people with psychogenic nonepileptic seizures. *Epilepsy Behav* 2015;43:39–45. DOI: 10.1016/j.yebeh.2014.11.012.
  60. McNally KA, Schefft BK, Szaflarski JP, et al. Application of signal detection theory to verbal memory testing to distinguish patients with psychogenic nonepileptic seizures from patients with epileptic seizures. *Epilepsy Behav* 2009;14:597–603. DOI: 10.1016/j.yebeh.2009.01.012.
  61. Duff K, Schoenberg MR, Scott JG, et al. The relationship between executive functioning and verbal and visual learning and memory. *Arch Clin Neuropsychol* 2005;20:111–122. DOI: 10.1016/j.acn.2004.03.003.
  62. Turner K, Piazzini A, Chiensa V, et al. Patients with psychogenic non-epileptic seizures: video-EEG, clinical and neuropsychological evaluation. *Seizure* 2011;20:706–710. DOI: 10.1016/j.seizure.2011.07.001.
  63. McDonald CR, Delis DC, Norman AA, et al. Response inhibition and set shifting in patients with frontal lobe epilepsy or temporal lobe epilepsy. *Epilepsy Behav* 2005;7:438–446. DOI: 10.1016/j.yebeh.2005.05.005.
  64. Alvaarez-Alamilla J, Velasco AL, Rio-Portilla YD. Conflict processing and response inhibition in patients with temporal lobe epilepsy: fMRI study. *Epilepsy J* 2016;2:1000113. DOI: 10.4172/2472-0895.1000113.