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Psychogenic non-epileptic seizures, prospective clinical experience: diagnosis, clinical features, risk factors, psychiatric comorbidity, treatment outcome

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ABSTRACT – In our study, we evaluated 249 patients with refractory seizures using video-EEG monitoring. In this sample, we identified 56 (22.5%) patients with psychogenic non-epileptic seizures – PNES only. Spontaneous seizures were recorded in 49 (87%) patients with PNES. Suggestive seizure induction using intravenous saline placebo was successful in 77.1% of induced PNES cases. Disease duration prior to PNES diagnosis was quite long. Prolonged past and current intake of high number of different antiepileptic drugs was also typical for these patients. We evaluated ictal PNES semiology. Whereas ictal EEG was normal in all PNES patients, interictal EEG was abnormal in 46.4%. Brain MRI was abnormal in 30.4%. Personality disorders were the most frequent psychiatric co-morbidity (in 44.6% of PNES patients), emotionally unstable (borderline) personality disorder was predominant (in 32.1% of PNES patients). Risk factors for epilepsy misdiagnosis and PNES manifestation are discussed. Therapeutic outcome after two years of combined treatment (psychopharmacotherapy and/or psychotherapy) is presented; approximately one third of patients were seizure-free following two years of treatment, one third of patients were responders (≥ 50% reduction in seizure frequency) and one third did not respond to treatment.

Key words: psychogenic non-epileptic seizures, dissociative seizures, video-EEG monitoring, MRI findings, psychiatric co-morbidity, therapeutic outcomes

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Patients with psychogenic non-epileptic seizures (PNES) represent 20 to 30% of patients referred to epilepsy centers as having refractory seizures (Benbadis and Hauser, 2000; Lancman *et al.* 2001). The differential diagnosis between PNES and epilepsy in daily neurological practice is difficult. Seizures can be very frequent, with dramatic manifestations, including generalized convulsions, falls and injuries. Interictal EEG and MRI abnormalities in PNES may be also a risk factor for misdiagnosis of epilepsy. Some of these patients are at times admitted to intensive care units with the diagnosis

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of "cluster epileptic seizures" or "status epilepticus" and intensively treated, including ventilatory assistance. Management of PNES as epileptic seizures can lead to significant iatrogenic harm (Gröppel *et al.* 2000; Reuber and Elger, 2003). Early correct diagnosis of PNES is important to avoid these hazards and to provide the patients with appropriate treatment, usually combined therapeutic procedures (psychopharmacotherapy and/or psychotherapy). These problems represent significant challenge to medical community thus far. Video-EEG monitoring, including suggestive seizure induction seems to be the most effective diagnostic tool and gold standard in seizure disorders differential diagnosis (Bazil *et al.* 1994; Devinsky, 1994; Benbadis *et al.* 1994; Benbadis *et al.* 2000).

The aim of our prospective study was to evaluate, in a group of patients with PNES, various aspects of diagnosis and treatment: video-EEG diagnosis including suggestive seizure induction using intravenous saline placebo, clinical profile, interictal EEG, MRI, psychiatric co-morbidity, possible risk factors for PNES manifestation and therapeutic outcome after two years of treatment.

Methods

We evaluated 249 patients admitted to our video-EEG monitoring unit for refractory seizures in 2001-2003. All these patients underwent video-EEG monitoring (continuous 24 hours video-monitoring, 18 EEG channels, 10-20 system, one ECG channel, Alien commercial system) including usual activation methods and/or suggestive seizure provocation (in approved patients with signed patient consent). On the video-EEG, we evaluated whether the seizures were "typical" spontaneous seizures, epileptic and/or PNES. Diagnosis of PNES with dissociative origin was made in patients with recorded spontaneous and/or provoked seizures and negative ictal EEG.

In these patients, other parameters, including ictal seizure semiology, interictal and ictal EEG, brain MRI, neurological and somatic co-morbidity, treatment with antiepileptic drugs, personality profile and other potential psychiatric co-morbidities (classified according to ICD-10 system) were also evaluated by an experienced psychiatrist and/or psychologist. Risk factors for epilepsy misdiagnosis and for PNES manifestation were identified.

Patients with epileptic seizures only, with both epileptic seizures and PNES, unclear diagnosis, no recorded seizures even after suggestive seizure provocation, patients with panic attacks only and patients with somatic non-epileptic seizures or sleep disorders were excluded from further evaluations.

In patients with diagnosed PNES, existing antiepileptic drug treatment was withdrawn (except from the patients in whom specific treatment with antiepileptic drugs was indicated for psychiatric reason) and psychiatric treatment and/or psychotherapy were introduced. Treatment outcome was evaluated after two years. Based on a patient-held seizure frequency records, mean monthly PNES seizure frequency was calculated for baseline period (3 months before PNES diagnosis). During the treatment period (2 years) seizure frequency was recorded and evaluated individually in each patient. Psychiatric assessments were conducted at the time of PNES diagnosis and during the subsequent treatment period.

When the diagnosis of PNES was confirmed, the first therapeutic step in all patients was to inform the patient of the diagnosis, provide educational interview with the patient with an explanation of principles of dissociation, potential triggers, symptoms and treatment options. As part of this interview, video- records of their seizures were presented to the patients.

The second step involved introduction of SSRI antidepressants (sertraline, citalopram, paroxetine) in patients in whom SSRIs were indicated. SSRIs were prescribed in doses normally used for treatment of anxiety or depressive disorders. Treatment with a low dose of an atypical antipsychotic (risperidone, olanzapine, quetiapine, ziprasiodone) was started in patients, in whom no clear benefit from treatment with antidepressants was evident. In patients in whom anticonvulsants with positive mood stabilizing or anxiolytic effects were used before the verification of PNES aetiology (lamotrigine, carbamazepine, pregabaline), treatment with these anticonvulsants was reintroduced even after establishing the dissociative origin of seizures. Specific dynamically-oriented psychotherapy was introduced in patients in whom psychological origin of seizures was clearly established.

Results

PNES only, psychogenic non-epileptic seizures of dissociative origin, was diagnosed in 56 (22.5%) of all 249 monitored patients (*table 1*). Both epileptic and PNES were diagnosed and confirmed on the video-EEG in 3 (1.2%) cases, and were suspected, based on the personal history, in 8 (3.2%) patients.

Spontaneous PNES were recorded in 49 of PNES-diagnosed patients. In the remaining 7 patients, PNES were only diagnosed following suggestive seizure induction. Typical seizures were induced in 37 (77.1%) of 48 induced cases, provocation was negative in 11 (22.9%). Eight patients did not provide consent with seizure induction. All patients tolerated PNES induction well, with no delayed negative experience.

Disease duration prior to PNES diagnosis, age and gender of the patients, seizure frequency per month, mean number of different used antiepileptic drugs in the past therapeutic history and currently, and other PNES patients' characteristics are presented in *table 1*.

Neurological co-morbidity was found in 7 patients (12.5%). Two patients (3.6%) had epilepsy in family his-

Table 1. Clinical characteristics of PNES patients (n = 56).

Characteristics	
Gender	
Females	39 (69.6%)
Males	17 (30.4%)
Age (years, ± SD)	29.6 ± 10,1
Disease duration (years, ± SD)	6.7 ± 3.4
Seizure frequency per month, prior to PNES diagnosis (mean, ± SD)	10.8 ± 10.8
Number of antiepileptic drugs (AEDs) in past history (mean, ± SD)	4.4 ± 1.7
Number of currently used AEDs (mean, ± SD)	2.7 ± 1.2
Video-EEG monitoring duration, PNES (days, ± SD)	3.7 ± 2.2
Number of recorded seizures (mean, ± SD)	3.6 ± 3.3
Seizure duration (minutes, ± SD?)	6,2 ± 4,1
Spontaneous PNES (patients, %)	49 (87%)
Suggestive seizure provocation done (patients, %)	48 (85.7%)
- positive (patients, %)	37 (77.1%)
- negative (patients, %)	11 (22.9%)
Epilepsy in family history (patients, %)	2 (3.6%)
Neurological co-morbidity (patients, %)	7 (12.5%)

Table 2. Seizure semiology (frequency of individual symptoms) in PNES patients (n = 56, frequency > 5%).

Ictal PNES symptoms	n (%)
Closed eyelids with resistant lid opening	37 (66.1%)
Trembling, rapid limb tremor	30 (53.6%)
Preictal "pseudosleep"	22 (39.3%)
Asynchronous "hyper-motor" limb movements, out-of-phase	19 (33.9%)
Opisthotonus	16 (28.6%)
Pelvic trusting, rhytmic pelvic movements	15 (26.8%)
"Aura", preceding other symptoms	14 (25.0%)
Head movements, side-to-side head shaking	13 (23.2%)
Clonic limb movements	12 (21.4%)
Tongue (tip), lip or buccae biting	10 (17.9%)
Unresponsiveness, areactivity, "staring" - without any motor symptoms	9 (16.1%)
Atonia with unresponsiveness	6 (10.7%)
Sounds, screams, vocalization or crying	5 (8.9%)
Subjective, sensory changes only	5 (8.9%)

tory and one patient suffered from serious diabetes mellitus, diagnosed a few months prior to first PNES occurrence (*tables 4* and 7).

Four patients (7.1%) had a history of "pseudo-status epilepticus" and two were treated with ventilatory assistance (3.6%).

Seizure semiology in PNES patients was evaluated and is presented in *table 2*. Most frequent symptoms were motoric. Non-motoric symptoms were also frequent. Tongue (tip), lips or buccal biting injuries were found in 10 patients; there were no other physical injuries and no urinary incontinence (*table 2*). Thirteen patients (23.2%) had non-stereotypical, varying semiology from more than one cluster: psychogenic motor seizure, psychogenic minor motor or trembling seizures and psychogenic atonic seizures (Gröppel *et al.* 2000).

While ictal changes were lacking on EEGs of all PNES patients, interictal EEG was abnormal in 26 (46.4%) of the 56 patients with the presence of interictal epileptiform abnormity in 12 (21.4%) cases, non-specific, slow abnormality was present in 19 (33.9%) patients' EEGs and both epileptiform and slow abnormality in 5 (8.9%) patients. Nevertheless, no patient had spike-and-wave, or polyspike-and-wave abnormality. Types and localization of EEG abnormalities are presented in *table 3*. Diagnosis of both PNES and epileptic seizures of frontal lobe origin was confirmed in one patient by the means of positive ictal SPECT correlate in suspected localization of epileptic seizures. Ictal EEG and interictal SPECT were negative in this patient.

Brain MRI was abnormal in 17 (30.4%) PNES patients. Individual MRI findings are presented in *table 4*.

Personality disorders were the most frequent psychiatric co-morbidity and were found in 25/56 (44.6%) patients *(table 5)*. The different types of personality disorders present within the sample are summarized in *table 6*; emotionally unstable (borderline) personality disorder was predominant. Other psychiatric co-morbidities were present in 31/56 (55.4%) patients *(table 5)*; anxiety disorders (panic disorder, generalised anxiety disorder and post traumatic stress disorder) and depression were frequent. The most frequent potential risk factors associated with PNES in patients' psychiatric history are presented in *table 7*.

Overall, the effect of combined therapeutic procedures (psychopharmacotherapy and/or psychotherapy) was moderate. After 2 years of treatment, 16/56 (28.6%) pa-

Table 3. Type and localization of interictal EEG abnormalities in PNES patients (n = 56).

Type/Localization	Regional n (%)	Bilateral n (%)	Generalized/diffuse n (%)
Non-epileptiform abnormality	6 (10.7%)	11 (19.6%)	2 (3.6%)
Epileptiform abnormality (sharp waves or spikes, no spike and wave complexes or polyspikes)	4 (7.1%)	5 (8.9%)	3 (5.4%)

Table 4. MRI abnormalities in PNES patients (n = 56).

Total number of abnormal MRIs	**17 (30.4%)**
Unspecified gliosis	8
Cavum septi pellucidi	2
Multiple sclerosis	2
Arnold-Chiari malformation	1
Lateral ventricular lipoma	1
Subarachnoideal cyst	1
Brain contusion	1
Cortical atrophy	1

Table 5. Psychiatric co-morbidity in patients with PNES of dissociative origin (n = 56).

Psychiatric co-morbidity	**n**	**%**
Personality disorders	25	44.6
Anxiety disorders (panic disorder, generalised anxiety disorder, post-traumatic stress disorder)	17	30.4
Depressive disorder	5	8.9
Somatization disorder	3	5.4
Munchhausen syndrome	2	3.6
Schizoaffective disorder	1	1.8
Eating disorder	1	1.8
Mental retardation	1	1.8
Malingering	1	1.8
Total	56	100

Table 6. Personality disorders (n = 25).

Type of personality disorders	**n**	**%**
Emotionally unstable (borderline) personality disorder	18	72
Dependent personality disorder	3	12
Organic personality disorder	2	8
Avoidant personality disorder	1	4
Schizotypal personality disorder	1	4
Total	25	100

Table 7. Risk factors in psychiatric history in PNES patients (n = 56).

Risk factor	**n**	**%**
Problems in family relations (divorce, death of a family member, traumatic relationship with an important family member, etc.)	16	28.6
Reaction to onset of serious somatic illness (neurological co-morbidity; multiple sclerosis 2 pts, brain contusion 1 pt, Arnold-Chiari malformation 1 pt, migraine 2 pts, diabetes mellitus 1 pt)	8	14.3
Sexual or physical abuse in childhood or early adulthood	6	10.7
History of uncontrolled aggressive behaviour	5	8.9
Substance abuse or dependency	4	7.1
History of head injury without somatic consequences (no brain contusion, normal CT, MRI finding)	3	5.4
Job loss	1	1.8
Absence of obvious risk factors	13	23.2
Total	56	100

Table 8. Therapeutic outcome over the past 12 months in PNES patients following two years of treatment (n = 56).

Therapeutic outcome over the past 12 months	**n**	**%**
Seizure free	16	28.6
Responders (> 50% seizure reduction but not seizure free)	19	33.9
No response	18	32.1
Lost to follow up	3	5.4
Total	56	100

tients were seizure free for at least the last 12 months, 19/56 (33.9%) were responders (reduction of seizures by at least 50%), 18/56 (32.1%) patients did not respond to any treatment and 3/56 patients (5.4%) were lost to follow up *(table 8)*. Four non-responders (7.1%) with no evidence of epilepsy additional to PNES were prescribed, by neurologists other then our center neurologists, a "new" anticonvulsive medication. These patients remained non-responders.

The video-presentation of PNES seizures and education were somewhat effective: we experienced immediate reduction of seizures by at least 50% (responders) in 4/56 (7.1%) patients, and consequent absence of seizures in 3/56 (5.4%) patients; these 3 patients remained seizure-free after two years without any other specific treatment, except from routine neurological and psychiatric follow-up examinations.

The best pharmacological treatment results were found in the group of PNES patients suffering from co-morbid anxiety or depressive symptoms, and treated with antidepressants. Following relief of anxiety or depression we noted ≥ 50% seizure reduction (responders) in 7/56 (12.5%) patients, and absence of seizures in 5/56 (8.9%) patients *(table 9)*. The combined treatment with SSRI and low dose of atypical antipsychotic (risperidone, olanzapine, quetiapine, ziprasidone) in resistant patients had moderate effect (*table 9*).

The most problematic group to treat were the patients suffering from PNES with co-morbid personality disorders, predominantly emotionally unstable (borderline) disorder. In these patients, we first introduced treatment with SSRI antidepressants and, in case of the lack of response, a

Table 9. Positive therapeutic outcome over the past 12 months in PNES patients with different psychiatric co-morbidity after 2 years of treatment (n = 56).

Type of patients	Explanation, education, video-EEG presentation	SSRI	SSRI + AAP
PNES patients with co-morbid anxiety or depressive disorder (n = 22)	1 SF 2 responders	5 SF 7 responders	2 SF
PNES patients with a personality disorder (n = 25)	1 SF 1 responder	2 SF 3 responders	3 SF 4 responders
Other PNES patients (n = 9)	1 SF 1 responder	1 responder	1 SF 1 responder

combination of an SSRI and low doses of atypical antipsychotic were prescribed. The best results it this group of patients were achieved with a combination of an SSRI and low doses of atypical antipsychotics *(table 9)*.

Anticonvulsants with positive mood stabilizing or anxiolytic effect were continued in 5/56 (8.9%) patients, two of whom suffered from emotionally unstable (borderline) personality disorder.

Specific dynamically-orientated psychotherapy was introduced in 16/56 (28.6%) patients; in all patients this was in combination with psychopharmacological treatment. Positive additive effect of psychotherapy was observable in approximately half of the patients.

Discussion

Our study confirms high proportion of PNES patients with dissociative non-epileptic seizures in the population of patients treated for refractory epilepsy. Mean age and sex distribution in our sample, with predominating female population, were typical of PNES patients. These findings are consistent with previously published data (Devinski *et al.* 1996; Benbadis and Hauser, 2000; Litwin and Cardeňa, 2000; Lancman *et al.* 2001).

Both epileptic and PNES were confirmed by the means of video-EEG in only a smaller number of evaluated patients. Nevertheless, this combination of seizures was suspected in other patients in our sample based on their personal history. Spontaneous PNES were recorded in 87% of PNES-diagnosed patients. Suggestive seizure induction using intravenous saline placebo was highly positive (77.1% of induced patients) and effective for PNES diagnosis confirmation (Bazil *et al.* 1994; Devinsky, 1994; Benbadis *et al.* 1994; Benbadis *et al.* 2000 ; Dericioglu *et al.* 1999). These findings are in line with previously published data (Lancman *et al.* 2001; Benbadis *et al.* 2000). All patients tolerated PNES induction well, with no consequent subjective negative experience or damaging effect on the physician-patient relationship. We consider video-EEG monitoring, including suggestive seizure induction, to be the most effective and safe diagnostic tool in seizure disorder differential diagnosis.

The most frequent ictal symptoms were motoric, while non-motoric symptoms were also rather frequent (Lancman *et al.* 2001). Tongue (tip), lips or buccal biting were relatively more frequent than we expected. Large proportion of patients had non-stereotypical, varying semiology from more than one of the previously defined clusters: psychogenic motor seizure, psychogenic minor motor or trembling seizures and psychogenic atonic seizures (Gröppel *et al.* 2000). Our study included all PNES patients, not only those with consistent seizure semiology, as was the case in the above cited study. This is why we did not use the seizure classification based on the analysis of symptom clusters (Gröppel *et al.* 2000). This classification assumes relatively stereotypical semiology of PNES, almost identical to the description of epileptic seizures. However, the results of our study suggest, that pleomorhic, non-stereotypical and varying semiology may be more typical for patients with PNES. Recognition of this fact could become one of the most important factors for PNES diagnosis.

Disease duration prior to establishing the diagnosis of PNES was, in our opinion, surprisingly long. As was the prolonged intake of high number of different antiepileptic drugs in the patients' history and currently. Moreover, 4 patients (7.1%) had a history of "pseudostatus epilepticus", and 2 (3.6%) were treated with ventilatory assistance. These data are alarming from many points of view: the delay between the first manifestation of PNES and correct diagnosis remains unacceptably long, patients with PNES are treated inappropriately with anticonvulsants for a long time, and some patients are treated with inappropriate emergency interventions. Our findings are consistent with published data (Gröppel *et al.* 2000; Reuber and Elger, 2003). Management of PNES as epileptic seizures can lead to significant iatrogenic harm (Reuber and Elger, 2003). Early correct diagnosis of PNES is important to avoid these hazards and provide the patients with appropriate treatment, usually with combined therapeutic procedures. These issues have so far been challenging.

MRI and EEG abnormal findings were, according to patients' medical reports, considered by general practitioners and neurologists as significantly suggestive of "epilepsy diagnosis" in most of our PNES patients. Therefore, we perceive these MRI and interictal EEG abnormalities as important "organic" risk factors for "epilepsy misdiagnosis". Some authors did not find interictal epileptiform abnormalities in all PNES patients with consistent seizure semiology (Gröppel *et al.* 2000). Our findings, in a methodologically distinct study, are different. In agreement with our findings, other authors have described EEG and brain abnormalities in PNES patients (Lelliot and Fenwick, 1991; Reuber *et al.* 2002, Reuber *et al.* 2002a,b).

Our study has also indicated potential factors in patient history that might have contributed to the development of PNES. Most frequently in our study these were: family and relationship problems, history of sexual and physical abuse in childhood and conversion of uncontrolled aggression. Furthermore, a reaction to manifestation of a serious neurological and other somatic illness was identified as a potential underlying PNES precipitating factor and may thus be of importance. No obvious risk factors were identified in only 23.2% of our sample. Somatic and psychogenic risk factors for PNES manifestation have also been described in other studies (Conder and Zasler, 1990; Devinski *et al.* 1996; Abubakr *et al.* 2003).

To ensure identification and appropriate interpretation of the above-mentioned potential risk factors for development of PNES, we recommend close neuro-psychiatric cooperation with correct neurological and psychiatric diagnoses.

Psychiatric disorders in our study were classified according to ICD-10 system. Personality disorders were the most frequent psychiatric co-morbidity, particularly the emotionally unstable (borderline) personality disorder. Similarly, this type of personality disorder has been reported in PNES patients by other authors (Bowman and Markand, 1996; Kalogjera-Sackellares and Sackellares, 1997; Kuyk *et al.* 2003). In a recently published study, personality disorder cluster A (paranoid, schizoid, schizotypical), classified according to DSM-IV system, was found as the most prevalent in the group of PNES patients (Kuyk *et al.* 2003). These types of personality disorders have been found in only a small proportion of patients in our study. In the study by Kuyk *et al.* (2003), personality disorders were assessed by self-reported questionnaire. Psychiatric diagnoses in our study were established on clinical bases, by psychiatrist and psychologist, both experienced in clinical diagnostics. Therefore, we believe our results are clinically valid and reliable. Similarly to reports by other authors (Kuyk *et al.* 2003), anxiety disorders (panic disorder, generalised anxiety disorder, post traumatic stress disorder) and depression were also frequent psychiatric co-morbidities in our sample.

After two years of treatment, the overall effect of combined therapeutic procedures (pychopharmacotherapy and/or psychotherapy) was moderate; when evaluated for the last 12 months, 28.6% of patients in our sample were seizure free, 33.9% were responders and 32.1% patients did not respond to treatment (5.4% of patients were lost to follow up). These findings support previous data indicating poor prognosis of PNES patients (Conder and Zasler, 1990; Reuber *et al.* 2003, Reuber and Elger, 2003).

Delivery of the diagnosis, video-presentation of PNES seizures and education formed the first step of therapy. They were well-tolerated but effective in only a small proportion of PNES patients. Only 5.4% of patients remained seizure-free after two years without any other specific treatment, except routine neurological and psychiatric follow-up examinations.

The best pharmacological treatment results were found in the subgroup of PNES patients suffering from co-morbid anxiety or depressive symptoms. In these patients, we have successfully used treatment with SSRI antidepressants and, in resistant patients, combined treatment with SSRI antidepressants and low doses of an atypical antipsychotic. This pharmacotherapy was well tolerated, without serious side effects. The most problematic group to treat was the patients suffering from PNES with co-morbid personality disorders, predominantly emotionally unstable (borderline) disorder. The best results in this group of patients were achieved with a combination of an SSRI and low doses of atypical antipsychotics.

Where anticonvulsants with positive mood stabilizing or anxiolytic effect were prescribed before the verification of PNES diagnosis (carbamazepine, lamotrigine, pregabaline), treatment with these anticonvulsants was continued after establishing PNES diagnosis. This was the case for 5 (9.3%) patients, two of whom suffered from emotionally unstable (borderline) personality disorder.

Specific dynamically-oriented psychotherapy was introduced in about one third of PNES patients. In all of them this was in combination with psychopharmacological treatment. Positive additive effect of psychotherapy we observed in approximately half of the patients.

Therapeutic outcome achieved in our study was consistent with other published data concerning PNES patients (McDate and Brown, 1992, Buchanan and Snars, 1993; Reuber *et al.* 2003); approximately one third of patients were seizure-free after two years, one third of patients were responders and one third of patients did not respond to treatment.

Conclusions

Our study contributes evidence for a topical and challenging paradigm: "If we consider 5 patients sitting around a table and treated for refractory epilepsy, it is very likely that one does not have epilepsy at all". The correct recognition of "who is who" and subsequent appropriate treatment is hitherto challenging task for epileptologists, even

those working in epilepsy centers operating with complex diagnostic and therapeutic tools. Early diagnosis and management of PNES patients is even more challenging task for general practitioners and primary care neurologists. To ensure appropriate early management of PNES patients; the "hidden" patients should be, as early as possible, consulted in epilepsy centers.
Appropriate early diagnosis and treatment of PNES patients are of the same urgency as, for example, early identification of epilepsy surgery candidates. □

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