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AN UPDATE ON NON-EPILEPTIC SEIZURES: DIAGNOSIS AND BRIEF NOTES ON TREATMENT

Bernt A. Engelsen[1,2*],
Arne Gramstad[1,3],
Atle Lillebø[1],
Bjørn Karlsen[1]

[1]*Department of Neurology, University of Bergen, Haukeland University Hospital, N-5021 Bergen, Norway*

[2]*Department of Clinical Medicine, University of Bergen, Bergen, Norway*

[3]*Department of Biological and Medical Psychology, University of Bergen, Bergen, Norway*

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Abstract

Non-epileptic seizures (NES) are episodes that appear as epileptic seizures, but are not caused by epileptic discharges in the brain. The inclusion of NES in the differential diagnosis of epileptic seizures is important, as an accurate diagnosis is required for the administration of appropriate treatment. NES in the differential diagnosis may be classified as (1) physiologic or pathophysiologic NES, e.g. syncope, sleep disturbances, motor symptoms, migraine attacks, etc., and (2) psychogenic NES (PNES), e.g. affective disorders with anxiety or panic, dissociative disorders (somatoform or conversion) as well as depression or posttraumatic stress disorder (PTSD). PNES is the condition most frequently misdiagnosed as epilepsy. We report NES as experienced in our epilepsy monitoring unit (EMU) for adults in the neurology department of a university hospital in Norway. Our main emphasis is on PNES, highlighting the diagnostic procedures, currently recommended treatment options, and follow-up. A team approach with video-EEG monitoring and clinical observation by trained nurses, epileptologists and other personnel is preferred in the diagnosis and treatment of PNES. Evaluations are performed by a neuropsychologist and trained social worker. The EMU closely cooperates with cardiologists and sleep center specialists. They can also refer patients to psychosomatic medicine specialists. Components of the differential diagnosis addressed are syncope, motor symptoms, sleep disorders, migraine and other paroxysmal neurological symptoms.

Keywords

• Non-epileptic seizures • Psychogenic nonepileptic seizures • Epilepsy monitoring unit

Abbreviations

AED - Antiepileptic drugs
CFS - Complex focal seizures
ECG - Electrocardiography
EMU - Epilepsy monitoring unit
GTC - Generalized tonic clonic seizures
MSLT - Mean sleep latency test
MMPI - Minnesota Multiphasic Personality Inventory
NES - Non-epileptic seizures
PNES - Psychogenic NES
REM - Rapid eye movement (sleep)
SFS - Simple focal seizures
TGA - Transitory global amnesia
TIA - Transitory ischemic attack
WPSI - Washington Psychosocial Seizure Inventory

Introduction

Non-epileptic seizures (NES) resembling epileptic seizures may be grouped into physiologic or pathophysiologic NES, (e.g. syncopal attacks, paroxysmal motor symptoms, migraine, sleep disorders, acute alcohol or drug effects, etc.), and psychogenic NES (PNES) [1-7]. Correct diagnosis of NES is important for safe and appropriate treatment.

In our experience, 20 to 40% of patients evaluated at the epilepsy monitoring unit (EMU) have NES [8] (Table 1), and approximately 50% of these patients have PNES. We estimate that between 5% of outpatients with epilepsy and 20% or more treated for refractory epilepsy in tertiary epilepsy centers have PNES [3,5,9]. Epilepsy and PNES may coexist, but 50% or more of patients with convulsive PNES do not have epilepsy [12], and it is unlikely that more than 10 % of patients with PNES have additional epilepsy [12,13].

The nomenclature of PNES may vary, i.e. pseudo-seizures [1] or pseudo-epileptic seizures [14]. NES includes patients with PNES and we use the term NES to include all non-epileptic seizure phenomena (Table 1).

This overview of NES refers to experiences based on more than 1500 video-EEG recordings from 1995 to 2012. Patients were recruited from combined local, regional and inter-regional patient populations and supervised by the Department of Neurology at the University Hospital of Bergen in Norway.

The epilepsy monitoring unit (EMU)

The EMU is located in a full time neurological ward. Two rooms with two beds are dedicated to around-the-clock video-EEG monitoring (Nervus EEG software, v.5.71, Natus Europe GmbH, Munich, Germany) of patients 6 days per week.

The *epilepsy unit* (EU) is an interdisciplinary team of 3 senior physicians, a neuropsychologist, a social worker, an occupational therapist, a computer scientist and nurses with extensive experience in clinical neurophysiology and the managing and observation of epilepsy patients.

* *E-mail: bernt.engelsen@helse-bergen.no*

The staff in the EU participates in the general neurological service of the department.

In patients with suspected PNES using antiepileptic drugs (AED), these drugs are tapered off as we attempt to provoke seizures with sleep deprivation, hyperventilation, flickering lights, physical exercise (including ergometer-cycling) and other patient-specific stressors or known seizure-triggering mechanisms. However, we do not use saline, other injections, or any other specific technique to provoke seizures [15,16].

In a preliminary investigation of 938 patients evaluated between 1995 and 2004, 60.7% had epilepsy, and 39.3% had NES. Within the group of NES patients, approximately 50% had established or assumed PNES and 16% had syncope. Of the 338 patients admitted to the West Haven epilepsy center during a three-year period, 84 patients (22%) had NES. From these 84 patients, 34 were classified as hysterical seizures and 9 as psychiatric disorders (derealisation, episodic loss of control), i.e. 43 total cases were PNES. Another 15 patients had "drug toxicity", 10 suffered cerebral ischemias, 5 demonstrated movement disorders, 4 experienced vascular headaches and there were 7 cases of sleep disorders [17]. Another EMU video-EEG confirmed 32.3% PNES, of which 5.5% had combined PNES and epilepsy [18].

Further complicating diagnosis is the registration of artefacts on EEG recordings (Table 2).

Table 1. Differential diagnoses of non-epileptic seizures.

Syncope
Drinking syncope (Cold Coca-cola!)
Coughing (tussive) syncope / Micturition- / Pain syncope
Malignant vasovagal syncope
Orthostatic syncope
Cardial syncope
AV-block with asystolia
Migraine
Hypoglycemia / hyperventilation
Sleep disorders
Pavor nocturnus / Nightmares
Sleep apnea
Nonepileptic myoclonus
Non-REM arousal disorder
REM sleep behavioural disorder
Paroxysmal brain stem dysfunction in multiple sclerosis
Paroxysmal motor disorders
Paroxysmal dystonia in cerebral palsy
Paroxysmal kinesiogenic choreoathetosis
Myoclonic dystonia
Spasms, tics (Tourette), dystonia, tremor, chorea
Valproate induced hyperammonic flapping tremor in epilepsy
TIA / TGA / various types of vertigo, including Ménière's disease
Toxic-metabolic event, incl. alchohol/drug use / "blackout"
Psychogenic non-epileptic seizures (PNES)
Swoon
Convulsive-like attacks
Tonic- / posturing / incl. opisthotonus
Dissociative fugue
Dissociative and perceptive distorsions in schizophrenia
Rage attacks / PTSD / panic attacks / psychosis
Factitious disorders / malingering / Munchhausen by proxy

All conditions have been diagnosed in our epilepsy monitoring unit.

PTSD: post traumatic stress disorder, TIA: Transitory ischemic attack, TGA: Transitory global amnesia.

Table 2. EEG artefacts or factors inducing seizure-like EEG abnormalities.

Midtemporal theta waves of drowsiness
Positive 14- and 6 Hz spikes
Hypnagogic hypersynchrony
Fluctuation of temporal background activity; Wicket rhytms; 6-11 Hz
Chewing (eating, swallowing)
Muscle tension artefacts; recordings of muscle potentials
Blinking- / nystagmus-induced artefacts
Rhythmic (epilepsy-like) artefacts caused by rhythmic movement
Voluntary / -involuntary movements / tics
Electrode artefacts; due to loose electrodes and high impedance
Sweating under the electrode
Infrequent artefacts due to electronic equipment
Bed-side or mobile telephone, use of PC, etc.
Grounding problems / head-box related artefacts

Pathophysiological NES

Syncope

The frequency of syncope varies between 7% and 25% in different populations of healthy persons and patients. The diagnostic work up includes a thorough medical history, supine and erect blood-pressure monitoring, baseline ECG and continued ECG monitoring, carotid sinus massage, echocardiograms, and TILT-table testing with isoproterenol or nitroglycerine provocation [19]. The most important prognostic factor is the establishment of any underlying cardiac cause and its treatment. In syncope, motor phenomena include myoclonic jerks (5-20 seconds), brief convulsion-like movements, irregular muscle twitching or tonic movements and muscle atonia. EEG findings in syncope are associated with the attenuation or the loss of electro cerebral activity [20].

Sleep disorders

Differentiating nocturnal epilepsy from parasomnias is generally straightforward; however, parasomnias are comprised of behavioural, autonomic and experiential phenomena occurring during non-REM and REM sleep. Video-EEG is a prerequisite for excluding epilepsy and sometimes polysomnography or mean sleep latency tests (MSLT) may be performed to differentiate

epileptic seizures from disorders of arousal in NREM sleep (confusional arousals, sleep terror) as well as REM sleep behavioural disorders (RSBD), sleep paralysis and narcolepsy. Sleep apnea also needs to be included in the differential diagnosis of tonic seizures.

PNES may occur during the "sleep phase", but EEG will show pre-ictal awakening [21]. An important differential diagnosis is frontal hypermotor seizures, which may occur without obvious epileptic EEG changes, but with a convincing semiology [22,23].

Other events

Visual symptoms of migraine, with or without dizziness, and affective symptoms or syncope have been observed in our EMU. Furthermore, symptoms of schizophrenia (derealisation), behavioural symptoms in mentally retarded or demented patients, episodic symptoms in multiple sclerosis, various motor symptoms such as non-epileptic myoclonias, tics and dystonias (Table 1) have been diagnosed in our referrals.

Numerous other conditions may elicit motor, psychic, perceptual or cognitive phenomena resembling epilepsy [6,7].

Psychogenic NES (PNES)

PNES may be due to different organic or non-organic disorders, and include virtually all the symptoms found in various conditions considered during the differential diagnosis of epileptic seizures [1,3,9,24,25] and this represents a major challenge for clinicians, patients and their families. Synonyms for this term include hysterical seizures, hystero-epilepsy, functional seizures or psychic seizures. However, the term psychic or psychogenic may alternatively be used for seizures triggered by an action of mind, or by cognition [26].

Epidemiology

The prevalence and incidence of PNES has not been firmly established. A population-based study in Iceland found a PNES incidence of 1.4/100.000, equal to 4% of that reported for epilepsy in persons age 15 years and above [27]. Alternatively, an incidence of 3/100 000 has been reported [28]. Considering various uncertainties, a prevalence of 2-30 per 100 000 has been suggested [29]. In general, PNES is more frequent in women than men, e.g. 71% [30] or 77% [31], but a more equal distribution has been reported with a 57% frequency in women [24].

In outpatients and hospitalized epilepsy patients, prevalence of PNES may range from 5 to 20 % [1,3,24,30,32,33].

In epilepsy patients hospitalized in Warsaw, 85 of 1083 had PNES, i.e. 7.8% [31]. Of these, 48 had PNES alone and 37 had mixed PNES and epileptic seizures [31]. In 20% of 46 patients with intractable seizures both PNES and epilepsy occurred [34]. Another study revealed that 23% had a mixed seizure disorder [24], whereas 37% of 41 patients with a discharge diagnosis of PNES from John Hopkins Hospital between 1971-74 revealed true epileptic seizures at a 5 year follow-up [30]. Conversely, 5 of 50 patients with definite PNES [35] also had EEG evidence of epilepsy. In a subsequent study, 13% of 110 patients with definite PNES also had evidence of epilepsy [12], and in 9.4% of patients EEG revealed inter-ictal epileptic discharges [13].

In an EEG monitoring unit similar to ours, 30 of 444 patients monitored during 1989-1992 had mixed seizure types (6.7%) [36]. This study revealed that 141 of 444 patients (31.8%) had PNES, 62 (14%) had epileptic seizures, 88 (19.8%) had inter-ictal epileptic abnormalities, 15 (3.4%) were classified as miscellaneous and 108 (24.3%) were normal [36].

Classification of PNES based on semiology

PNES may resemble any type of epileptic seizure, but the semiology may vary and unusual seizure semiology may in itself be an important indicator of PNES.

PNES may be classified in different ways: convulsive or non-convulsive seizures, or by more detailed classification according to consciousness and motor manifestations [3,24,37,38].

Diagnosis of PNES

The diagnosis of PNES is usually based on clinical criteria, e.g. description of the behavioural events or seizures (Table 3), with non-epileptic ictal and often normal pre- and post-ictal EEG. Ictal video-EEG monitoring of PNES has been performed in several studies [12,24,25,37,39-41]. When PNES patients are examined in the EMU many will have a seizure within 48 hours [42].

Seizure semiology differ between studies, i.e. convulsion-like PNES [37], or CFS-like PNES [41]. In one study, motor phenomena were the most frequent symptom of PNES [40], whereas in another study, unresponsiveness without motor phenomena was the most common symptom [24]. An extremely wide range of seizure events with bizarre and unusual behaviour were reported in 42 episodes in 6 patients [41]. Moreover, seizure manifestations reported by patients and witnesses reveal major heterogeneity [43]. However, in one

Table 3. Diagnostic support for psychogenic non-epileptic seizures (PNES).

1. Clinical semiology or symptomatology
   - Violent, irregular, and variable motor symptoms
   - "Out-of phase" clonic movements in upper or lower extremity (i.e. movements in arms/legs either not synchronous, or in opposite directions)
   - Side to side head movement
   - Swoon/abreactive attacks/behaviour/ictal crying
   - Closed eyes (resistance against manual opening)
   - The indifference; "massive seizures, no concern"
   - Often longer duration of seizures (minutes)
   - Rapid postictal recovery of responsiveness
2. Non-epileptiform ictal EEG, and normal pre- / postictal EEG
   - Seizures initiated under sleep associated with wake EEG
3. Seizures are refractory to treatment
4. Patient reveals signs or symptoms of psychiatric disease or psychological dysfunction/disturbance
5. Diagnostic support by additional test
   - Lack of postictal rise in serum prolactin
   - Lack of altered serum creatine kinase (GTC), blood gases etc.
   - Signs of personality disturbances in MMPI
6. Late start of seizures (e.g. > 15-20 years of age)

study of exclusive PNES, all of the 27 patients revealed consistent seizure semiology during all recorded seizures [25].

The clinical differentiation between epileptic seizures and PNES is often inaccurate [39], but some symptoms do suggest PNES [44] (Table 3). Authors of an ictal clinical cluster analysis stated: "We believe that neither a single symptom nor the combination of symptoms in this cluster are sufficient for a definite differential diagnosis". The most frequent symptom in this study was trembling of upper extremities, seen in 14 of 27 patients (51.9%) [25].

We strongly support a note of caution since PNES may be difficult to differentiate from some frontal lobe epileptic seizures [22,23,45]. We propose a list of clinical symptoms which may be more frequently observed in PNES than in epilepsy (Table 3). Noticeably, the delay from onset of PNES to correct diagnosis in one study was 7 years [46].

When PNES occur as convulsive seizures, motor phenomena such as out-of-phase movements of extremities, side-to-side head movements, forward pelvic thrust and opisthotonus are often seen [24,25,37,40,41]. The frequency of such symptoms varies, and they may be infrequent in some patients with PNES. In non-convulsive PNES, the seizures may start with swoon [12,47] or an abreaction according to Betts nomenclature [47]. Atonic seizures with fall may occur.

PNES are usually of longer duration than corresponding epileptic seizures. Mean generalized tonic clonic seizures (GTC) duration in patients with epilepsy was 70 seconds (range; 50-92 seconds), whereas the mean GTC-like PNES lasted 134 seconds (range: 20-205 seconds) [37]. Correspondingly, Theodore established a mean of 62 seconds based on 120 secondary GTC in 47 patients with epilepsy [48]. Thus, seizure duration may not differ statistically between epileptic GTC and PNES, but when seizures last more than 2 minutes one might conduct a closer analysis of the clinical semiology. There may be semi-purposeful movements mimicking automatisms in up to 52% of seizures [40]. Incontinence and injuries may occur in PNES [12,49], as well as tongue biting [49,50]. Most data concerning injury are, however, based on answers from patients [49,50]. In our experience patients rarely hurt themselves during PNES. In fact, none of the more than 1500 patients observed since 1995 had any major injuries. This corresponds to observations made by Brown *et al*. [51].

Onset of PNES is often later than epilepsy in comparable groups, i.e. they often start after 20 years of age [10,12,50]. In one study the highest age-specific incidence of PNES was between 15-24 years of age [27]. Children may have NES and PNES [52].

There is often no typical aura, but patients may report subjective nonspecific phenomena such as dizziness and headache [40]. The onset of seizures in PNES may be gradual. In provoked or induced PNES, the patients often keep their eyes closed [53], and cyanosis is generally not observed. In patients with PNES there is often a history of non-responsiveness to treatment and signs or symptoms of underlying psychiatric or psychological disturbance [4] (Table 3).

EEG, neuropathology and psychiatry in patients with PNES

EEG findings may be pathologic in 19-50% of patients with PNES [3,30,54]. PNES patients may even reveal epileptiform potentials [55].
Conversely, some epileptic seizures may not reveal themselves by epileptic paroxysms in EEG tracings. Both frontal lobe [22,23] and deep temporal lobe seizures may be accompanied by restricted subcortical epileptic discharges. Simple focal seizures may occur with normal ictal EEG recordings. It is also vital to identify artefacts in EEG resembling epileptogenic changes (Table 2).

In a study from John Hopkins Hospital, 18 of 41 patients discharged with a diagnosis of PNES had coexisting organic neurological disorders [30]. Neuropsychological impairment is as frequent in patients with PNES as in patients with epilepsy [56]. The MMPI [57,58] has been used with neuropsychological tests in order to study whether the existence of cognitive impairment or personality disturbances may help to differentiate between PNES and epileptic seizures [51,56,59-61]. The results have been difficult to interpret. One study confirmed that personality characteristics of PNES patients whose attacks had either little affective display or prominent motor expression could not be distinguished from those of epileptic patients with generalized convulsive seizures [56]. Nevertheless, elevated scores in MMPI scales Hs (hysteria), Hy (hypochondriasis) [51,60] and Sc (schizophrenia), relative to D (depression), might give indirect support for PNES [51,56,59-61]. In some patients the D-scale may also be elevated in PNES, although this scale aids little in differentiating these patients from those with epileptic seizures [51].

In one study, routine EEG together with the profile of the MMPI-2-Hy scale and the duration in years since the first seizure produced an overall classification accuracy of 86.3% [60] with regard to PNES. Duration of PNES was shown to be a negative prognostic factor [62], while routine EEG itself did not appear to be a sufficient diagnostic tool, neither to obtain a good outcome nor to convince patients of their diagnosis [50].

PNES may be defined as a somatic communication of mental distress [63], or as somatoform symptoms as an expression of a traumatic experience. The latter does not have to be of a sexual nature [64], although sexual abuse may be an important factor [47,65,66].

The majority of PNES may be due to somatoform disorders [3,64]. The essential feature of somatization disorder is a pattern of recurring, multiple, clinically significant somatic complaints with pain symptoms, gastrointestinal symptoms, one sexual symptom and one pseudo-neurological symptom. Conversion disorders are symptoms or deficits affecting voluntary motor or sensory function, or seizures, and suggest a neurological or other general medical condition. Conversion disorders may be frequent; up to 50% in patients with PNES [59,64].

Panic attacks may occur in the context of several different anxiety disorders [67], including post-traumatic stress disorder (PTSD) [68]. PTSD is more prevalent in patients with PNES, but may occur in patients with epilepsy as well (37%) [68]. The essential clinical feature is a disruption in the usually integrated functions of consciousness, memory, identity, or perception of environment.

In our EMU, patients with schizophrenia or borderline psychosis have reported episodic delusions, detachment or bodily sensations

mimicking simple or complex partial seizures. Moreover, psychotic symptoms may occur post, inter or peri-ictally in patients with temporal lobe epilepsy [69], thus making video-EEG monitoring essential.

Factitious disorders and malingering may occur (e.g. to avoid duties, exams, military service, etc.). Munchhausen syndrome frequently presents as epileptic seizures, but is usually a non-epileptic condition [70].

A history of childhood sexual abuse may be found in patients with PNES [38,47,64,65,71-73]. The actual frequency is uncertain and can vary from 17% [10] to 70% of patients with a specific PNES type of "abreactive" seizures [47]. Noticeably, sexual abuse may also trigger epilepsy [66]. Flashback experiences may occur when the patient is alone in bed and may sometimes explain why PNES occur in bed at night [66]. Sexual abuse is important to disclose in order to start proper treatment [47]. Based on estimates described in [47], a history of sexual abuse may occur in 10-20% of the population, whilst severe penetrative abuse in the female population occurs in perhaps 5-10%. Some victims seem to recover completely from the abuse, but at least 20% of victims develop long-standing psychological complications [47].

Our policy has been neither to ask directly nor to infer the possibility of sexual abuse in individual patients. This difficult topic is sometimes uncovered during close communication between the patient or relatives with doctors, nurses, psychologists or other people whom the patients trust. Development of a trusting relationship is essential in this process.

Management and outcome of PNES

The management and treatment of PNES start with a firm diagnosis [4]. However, specific guidelines for treatment are lacking (74) and outcomes are not very good (4).

Less than 50% of patients with PNES are seizure-free at follow-up ranging from 1 to over 5 years later [12,30,62,75,76]. When considering prognosis and outcome, one should differentiate between patients with PNES alone and those with PNES combined with epilepsy. In a publication of 56 patients with PNES only, 52% were seizure free at follow-up 18 months later [50]. This study revealed no significant difference in the outcome among patients based on any intervention [50]. Factors predicting a positive outcome in this study include acceptance of the PNES diagnosis, good general health and good occupational functioning [50]. Poor outcome has been associated with an IQ of less than 80, and a past history of violent behaviour [10]. Persisting PNES has been associated with longer duration of PNES before diagnosis, presence of additional psychiatric disease, but *not* with gender, psychotherapeutic treatments after diagnosis or the presence of epilepsy [62].

Thus NES, with the exception of PNES, are generally easy to treat once the diagnosis is set since the underlying disease may be easily treated, e.g. cardiac arrhythmias, migraine, narcolepsy, TIA or TGA, etc.

A few patients reported PNES following epilepsy surgery [77], but their seizures readily resolved after thorough video-EEG confirmation.

The most difficult therapeutic challenge is with patients in whom seizures are frequent, dramatic, unresponsive to AED or other medical treatments, and in which semiology and context suggest an underlying psychiatric or psychological disorder, i.e. PNES. Therapy should be individually designed [34] with a multidisciplinary approach.

Behavioural psychotherapy significantly reduced seizures in 7 of 9 patients during a four year follow-up [78]. In a controlled study, cognitive behavioural therapy was more effective than only standard medical care in reducing seizure frequency in PNES, although effect size was only moderate [79]. Supportive psychotherapy, occupational therapy and operant conditioning made 8 of 16 patients with PNES seizure free, and gave 3 patients relief with only occasional seizures, whereas 5 remained unchanged in another study [10].

PNES is often intermittent in that momentous life changes (e.g. new partner, marriage, established job, solving complex psychosocial problems etc.) may contribute to seizure-freedom for years. However, severe personality, anxiety, or neurotic problems need to be addressed in order to achieve lasting improvement. Patients should be met with an open, tactful and positive empathic attitude, but PNES should be ignored or treated by shielding when the diagnosis is established.

A therapeutic effect of AED on seizures does not necessarily exclude PNES, since patients with PNES may be easily influenced, or have affective disorders in which AED may alleviate symptoms [63]. Our experience is that any effect is temporary, and in general the loss or failure of AED effects may be an important symptom of PNES. A broad attitude towards psychological treatment must be taken because of the diversity of the psychopathological processes leading to PNES. Sometimes long-term treatment of serious psychopathology is indicated, but often more focused treatment strategies are sufficient. In many cases, family therapy or interventions directed at social or educational problems are indicated. Prescription of psychotropic medications may be warranted depending on comorbid diagnoses [74].

Brief clinical case notes

I.) A 17 year old patient presented with a one month history of episodes of dizziness, pallor, jerks and loss of consciousness, occasionally leading to falls with minor injuries. Video-EEG was inconclusive without any attack. A TILT table test was performed and blood pressure at 20 minutes was 123/77, with pulse frequency of 70. Five minutes after nitroglycerin provocation the blood pressure dropped to 69/31, with ECG rhythm of 100 rising to 150 during syncope. *Diagnosis*: Vasopressor response likely cause of syncope.

II.) A female patient had multiple convincing episodes of PNES, but also falls with minor head injuries. Several routine EEGs were normal. A video-EEG revealed focal epileptic seizure activity with very subtle semiology (CFS) followed by a PNES with classical side to side head movements and artefacts in EEG (Figure 1 a/b). *Diagnosis*: Epilepsy with frequent PNES.

III.) A male patient with acute prolonged GTC-like seizure and a brief response to

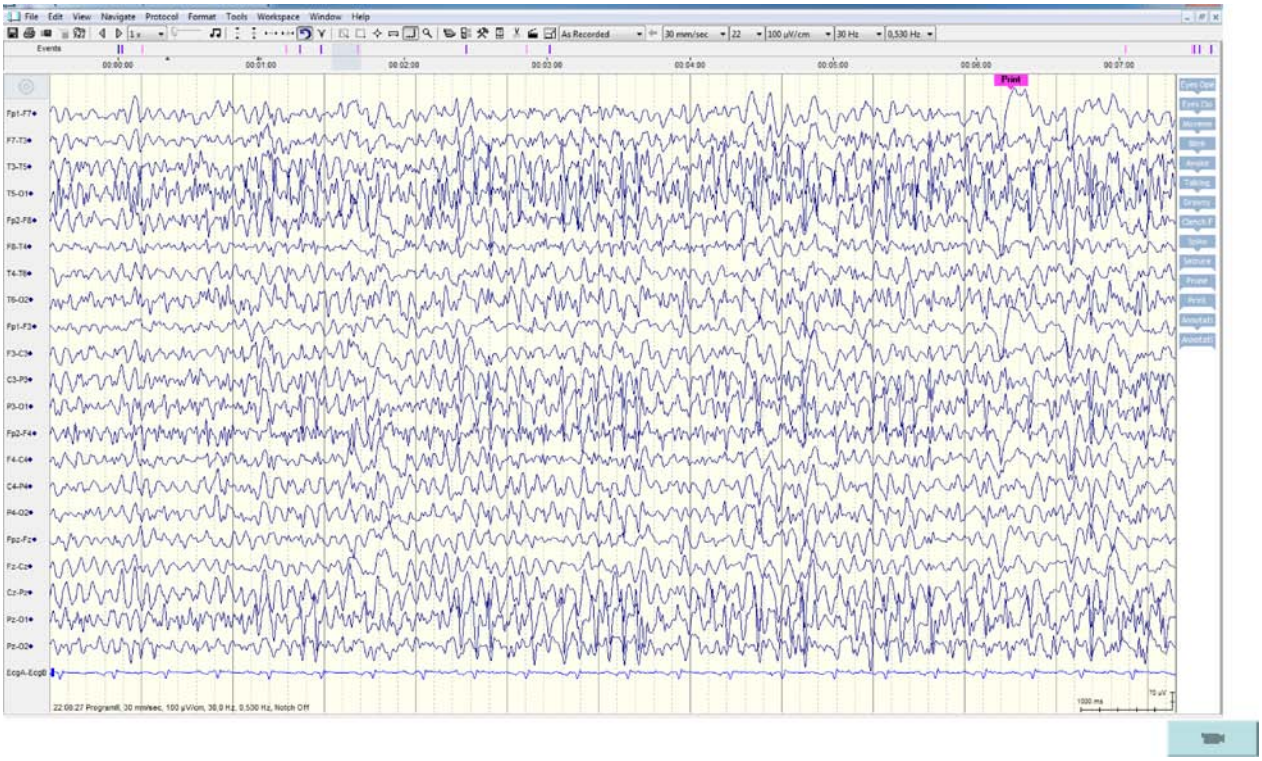

Figure 1a. Twenty-five year old female with definite PNES in earlier video-EEG. Early phase of 5-6 minutes with epileptiform activity in EEG and quiet CPS with open eyes and hypersalivation.

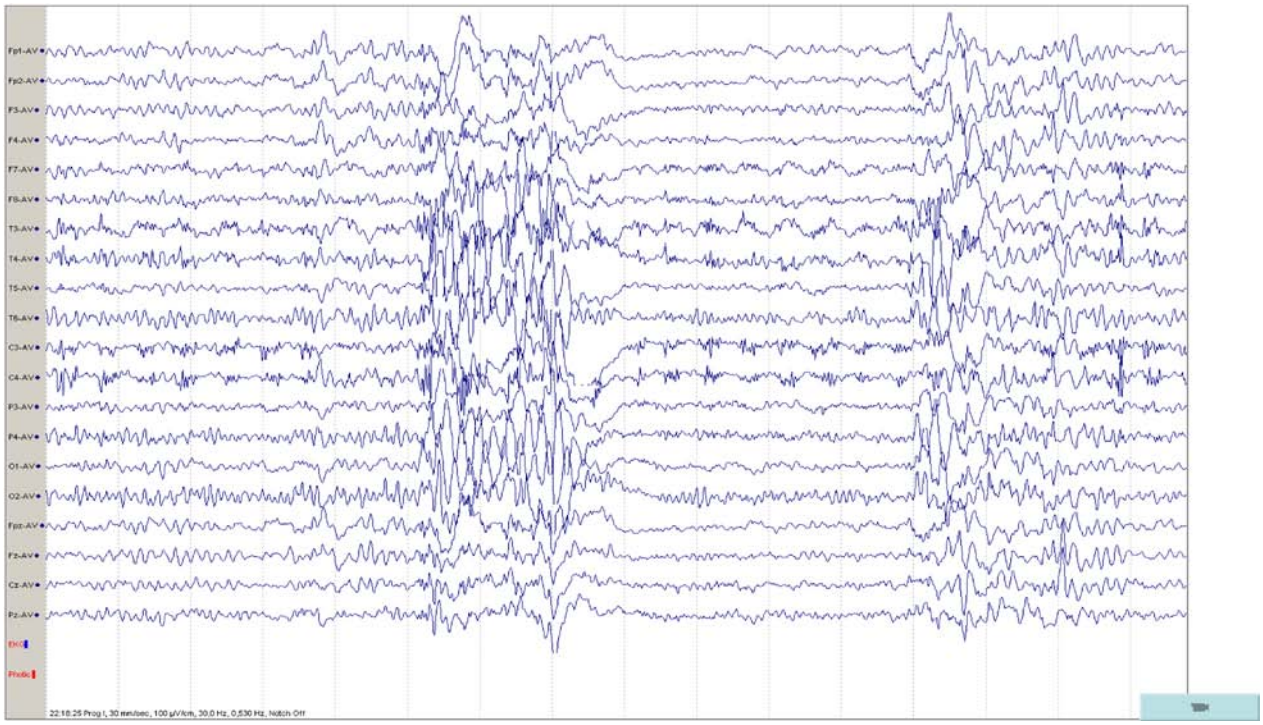

Figure 1b. Movement artefacts in EEG during PNES with side to side movement of the head very shortly following the epileptic seizure in Figure 1a.

diazepam. Video-EEG revealed irregular motor components suggestive of PNES and anxiety. Consultation with a psychologist from Dept. of Psychosomatic medicine revealed maladaptive reaction with prolonged anxiety and depression, with dissociative convulsions.

IV.) A 19 year old female presented with seizures with loss of consciousness almost weekly for six months. Signs and symptoms included irregular, partly arrhythmic jerking of the extremities, combined with nausea, loss of motor power and "apathy" for hours. These symptoms were enhanced by prior intake of moderate doses of alcohol. A 63 hour video-EEG with ECG electrode was normal, whereas the MMPI-2 revealed a classical "psychosomatic V" with elevations on the Hs and Hy scales. We concluded with a somatisation disorder, but a TILT test will be performed.

Concluding discussion

Both NES and PNES pose important diagnostic challenges, but in our experience a firm diagnosis can often be established. In approximately 10-15% of PNES patients, we may find co-occurring signs of epilepsy for which AED treatment may be prolonged. The specific features of PNES are explained to patients and caregivers to aid in establishing non-medical treatment.

A special challenge is the manifestation of simple phenomena such as local sensory disturbances, minor motor symptoms, simple visual phenomena, etc., which may occur as epileptic seizures without corresponding epileptic EEG changes. However, if these are not considered SFS or migraine aura symptoms, based on duration and accompanying symptoms, AED are usually tapered off safely [80]. Migraine auras are occasionally treated with AED, such as topiramate, valproate or lamotrigine, and tapered off at later consultations in the outpatient clinic.

PNES is usually diagnosed based on a combination of all available information, i.e. medical history with extensive tests and interviews of family or colleagues (if appropriate and with the consent of a patient). Video-EEG monitoring with seizure provocation is performed and patients are thoroughly informed beforehand. Invasive seizure provocation via intravenous injection is not utilized [15,16,81]. This decision is based more on practical than ethical reasons. In our case, the size of the patient population and the level of communication within patient groups and organizations are high, so that provocative measures would most likely become well known among the patients.

For diagnostic purposes we may perform additional investigations like neuropsychological evaluation, WPSI [82], MMPI-2 and additional peri-ictal tests like serum prolactin, creatine kinase, and blood-gas analysis.

In patients in whom we are able to establish a diagnosis of PNES we consequently taper off AED therapy. All patients are informed in detail about the diagnosis, and also about potential doubts regarding the rationale for the diagnostic decision. We may demonstrate seizure recordings for the patient and provide referrals to local psychiatrists or psychologists. If necessary, some patients may be followed by a psychologist within our department for a limited time. Patients with PNES are treated courteously with empathy; however, once the PNES diagnosis is established the PNES seizures are ignored. If collaboration is weak or insufficient for a thorough diagnosis we may conclude by using an observational diagnosis.

We are generally open to re-evaluating diagnoses when seizure semiology changes. Lastly, it is important to know all possible sources of EEG artefacts that may contribute to a false positive diagnosis of epilepsy (Table 2).

Conclusion

On a yearly basis we find that around 20-40% of patients in the EMU have NES, and 50% of these have PNES (Table 1). As an estimate, 10% of our patients with PNES also have epilepsy.

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