

Childhood paroxysmal nonepileptic events

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ABSTRACT

We aimed to determine the types and clinical characteristics of paroxysmal nonepileptic events (PNEs) in children. During a 13-year period, 765 patients underwent long-term video-EEG monitoring, and 95 (12.4%) of them were identified to have PNEs. The most common diagnoses were conversion disorder, parasomnias, staring spells, movement disorders, and hypnic jerks. Paroxysmal nonepileptic events originated from physiologic or organic (43.2%) and psychogenic (56.8%) causes. Mean delay in diagnosis was 3.1 years. Mean ages at diagnosis were 8.8 and 13.8 years in physiologic or organic and psychogenic groups, respectively. A marked female predominance was seen in the psychogenic group, whereas males slightly predominated in the physiologic or organic group. In the physiologic or organic group, events were less frequent, longer in duration, and commonly manifested as subtle motor activity, whereas subtle and prominent motor activities were encountered equally in both groups. Concomitant epilepsy was present in 10.5% of the patients. Differences in clinical characteristics may be helpful in differentiating physiologic or organic PNEs in children from psychogenic PNEs.

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1. Introduction

Paroxysmal nonepileptic events (PNEs) are episodic changes in behavior, sensation, or consciousness that resemble epileptic seizures but are not associated with abnormal ictal cerebral electrophysiological discharges [1]. They may occur in all age groups. In most instances, the clinical history leads to the correct diagnosis, and ancillary testing serves as confirmation; in a subgroup of cases, however, an accurate diagnosis is one of the clinical problems faced by practitioners [2].

While psychogenic seizures and cardiac events comprise the majority of PNEs in adults, besides these disorders, a wide variety of physiologic and organic disorders including parasomnias (confusional arousals, sleep walking, sleep terrors, and nightmares), sleep-related movement disorders (periodic limb movements in sleep, nocturnal leg cramps, and rhythmic movement disorders), narcolepsy, benign paroxysmal nocturnal events (hypnic jerks and benign sleep myoclonus of infancy), breath-holding spells, Sandifer syndrome, and behavioral events can mimic seizures in children. There are no somatic causes for psychogenic PNEs, rather, they are somatic manifestations of psychologic distress [3]. The onset of psychogenic PNEs is typically in the adolescence or young adulthood period [4]. In a group of patients diagnosed with psychogenic PNEs, most patients experienced

the first episode between 10 and 19 years; however, correct diagnosis was established between 20 and 40 years [4,5].

Long-term video-EEG monitoring offers simultaneous assessment of both clinical events and cerebral electrical activity, so it has a great importance in differentiating epileptic seizures from nonepileptic seizures, as well as in seizure classification and presurgical evaluation [1,6–9]. Paroxysmal nonepileptic events are among the most common causes of treatment-refractory spells, and up to 43% of patients seen at pediatric epilepsy referral centers may have a paroxysmal nonepileptic disorder [2,10,11]. In a tertiary epilepsy center, the epilepsy diagnosis was disproved in 30% of children referred without any doubts about the epilepsy diagnosis [2]. The misdiagnosis of epilepsy has important consequences, including unnecessary exposure to antiepileptic drugs and unnecessary exposure to invasive interventions such as intubation and even to invasive therapeutic modalities such as vagal nerve stimulation implantation [12]. Early recognition and appropriate treatment of nonepileptic seizures can prevent significant iatrogenic harm and may result in a better outcome.

Data concerning the relative frequency and phenomenology of childhood PNEs by etiology are limited [6,7]. While staring episodes and unresponsiveness have been reported as more common manifestations in patients with physiologic or organic PNEs, prominent motor activity was more common in adolescents who mostly had psychogenic PNEs [6,13].

In this study, we aimed to evaluate the demographic features of children with PNEs and the nature, relative frequency, and clinical manifestations of the events documented in our pediatric long-term video-EEG monitoring unit during a 13-year period and to determine

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whether these features differ in children with PNEs that originated from organic or physiologic and psychogenic causes.

2. Materials and methods

Video-EEG reports of 765 children who were admitted to the pediatric video-EEG monitoring unit at Gazi University Medical Faculty between October 1998 and May 2012 were reviewed. Pediatric Neurology Department of Gazi University Medical Faculty is a tertiary epilepsy referral center for children. An Institutional Ethical Committee approved the study. Children who had a clinical diagnosis of paroxysmal nonepileptic events (PNEs) were identified. Paroxysmal nonepileptic events were defined as paroxysmal changes in behavior, not associated with a seizure pattern on scalp EEG recordings. Only patients who had at least one of their typical episodes documented on video-EEG were included into the analysis. For children admitted more than once, only data from one admission were included into the study. Neonates were monitored in neonatal intensive care unit and excluded because of the specific characteristics of this age group. Children with clear clinical diagnosis of paroxysmal disorders such as syncope, migraine, breath-holding spells, tics, shuddering attacks, and parasomnias were not monitored and excluded from the study. Demographic, clinical, and video-EEG data including duration of symptoms prior to diagnosis, frequency and duration of events, previous and/or current use of antiepileptic medication, medical history in regard to premature birth, perinatal asphyxia, febrile convulsions, perinatal infections, and/or trauma, and semiology of events in regard to the type of motor activity were gathered. Prominent motor activity was defined as abrupt paroxysmal changes in motor activity that resembled an epileptic convulsion, such as focal or complex motor activity, generalized jerking, and generalized tremor. On the other hand, subtle motor activity was defined as paroxysmal benign events such as episodes of staring or daydreaming, generalized limpness, sensory symptoms, bursts of crying or shouting, or clearly stereotypical episodes with walking, running, rocking, or repetitive movements. Besides psychogenic seizures, a wide variety of physiologic and organic conditions can cause paroxysmal spells mimicking epileptic seizures [6,14]. Based on the etiology of the recorded events, we divided children with PNEs into psychogenic and physiologic or organic groups. Psychogenic PNEs included conversion reaction which was defined as abrupt paroxysmal changes in behavior or consciousness resembling an epileptic seizure which could be associated with secondary gain [3] and events originated from a psychiatric disorder. Physiologic or organic events included paroxysmal behavioral disorders which originated from an organic or physiologic etiology, such as sleep-related disorders, movement disorders, syncope, and staring spells.

Ictal and interictal EEG patterns were recorded by using a 32-channel digital video-EEG system (Telefactor Beehive System, Telefactor, Philadelphia, PA) which included automatic spike and seizure detection modules. Scalp electrodes were placed according to the International 10–20 System with additional bilateral inferior temporal chains. Entire interictal EEG patterns recorded during wakefulness and sleep and all clinical events identified by parents or caregivers or children were reviewed by one or more trained pediatricians on a daily basis for the presence of epileptiform discharges. Senior epileptologists (A.S., T.H.), together with one or more pediatricians, then reviewed each EEG and video segments of these episodes and determined whether they represented an epileptic or nonepileptic event. The diagnosis of the nonepileptic event was determined on the basis of its semiology and clinical characteristics and with the absence of any ictal epileptiform abnormalities on the EEG. The diagnosis of PNEs was made only if family members verified that the monitored events matched the patients' typical spells. A concomitant diagnosis of epilepsy was made if an epileptic seizure episode was also captured during the video-EEG monitoring and there were epileptiform abnormalities on the EEG.

Patients were monitored for 1 to 7 days, depending on whether sufficient numbers of the typical events occurred. Antiepileptic drugs were tapered or discontinued during the monitoring period as necessary in patients who were on treatment. In patients suspected of having psychogenic seizures, when typical episodes were not observed, an attempt was made to induce the event by verbal suggestion, hyperventilation, and/or the injection of 1- to 2-ml saline intravenously after obtaining parental consent. In 95 patients, the typical spells with no accompanying epileptic discharges on EEG recording were captured; in the remaining 27 patients, PNEs were diagnosed on the basis of clinical history and prolonged EEG recordings that did not show any epileptiform discharges.

Statistical analysis was performed using SPSS 15.0 for Windows. The Student *t* test, Pearson's χ^2 test, and Fisher's exact test were used to compare demographic and clinical characteristics between the two groups and considered statistically significant if the *p* values were <0.05.

3. Results

From October 1998 to May 2012, 765 children underwent long-term video-EEG monitoring, and 122 (15.9%) of them had a clinical diagnosis of PNEs. Ninety-five (12.4%) patients had at least one of their typical events during the monitoring and were included in the analysis. The remaining 27 patients whose habitual events were not captured during the monitoring were excluded from the study.

Fifty-four (56.8%) patients were determined to have psychogenic PNEs, and except for 2 children who were diagnosed with hyperactivity disorder and impulse control disorder, all patients in this group had conversion reactions (54.2%). The remaining 41 (43.2%) patients had physiologic or organic PNEs, and the more frequent diagnoses in this group included parasomnias (12.6%), staring spells (11.6%), movement disorders (10.5%), and hypnic jerks (6.3%). The distribution of PNE disorders diagnosed by video-EEG monitoring is presented in Table 1.

Demographic and clinical characteristics of patients are shown in Table 2. Of 95 patients, 54 (56.8%) were females and 41 (43.2%) were males. The percentage of females (66.7%) was higher in the psychogenic group, whereas there was a slightly higher proportion of males (56.1%) in the physiologic or organic group ($p=0.027$). Male predominance was more marked in patients with parasomnias, followed by patients with hypnic jerks and movement disorders (Fig. 1). When all patients were analyzed as a single group, the mean ages at the time of onset of symptoms and at the time of diagnosis were 8.5 ± 4.5 years and 11.6 ± 4.4 years, respectively. Mean age at onset was significantly lower in patients with physiologic or organic PNEs when compared with patients with psychogenic PNEs (4.9 ± 3.3 years in the physiologic or organic group, 8.8 ± 4.5 years in the psychogenic group, $p<0.001$). Similarly, mean age at diagnosis was significantly lower in patients

Table 1

Types of paroxysmal nonepileptic event disorders by diagnosis and age at onset for each disorder.

Disorders	Number	Mean age at onset \pm SD yr (range)
Psychogenic events	54 (56.8) ^a	13.79 \pm 2.81
Conversion disorder	52 (54.7)	14.05 \pm 2.49 (5–18)
Psychiatric disorder	2 (2.1)	7.00 \pm 2.82 (5–9)
Organic or physiologic events	41 (43.2)	8.82 \pm 4.53
Parasomnia	12 (12.6)	8.41 \pm 4.07 (3–15)
Staring	11 (11.6)	7.27 \pm 5.23 (2–16)
Movement disorder	10 (10.5)	11.00 \pm 4.49 (4–17)
Hypnic jerk	6 (6.3)	8.33 \pm 4.17 (3–15)
Syncope	1 (1.1)	8.00
Sleep-related breathing disorder	1 (1.1)	13.00
Total	95 (100)	11.65 \pm 4.39

SD, standard deviation; yr, years.

^a Values are numbers (%).

Table 2
Demographic and clinical characteristics of patients with paroxysmal nonepileptic events by etiology.

	Patients with organic or physiologic PNEs	Patients with psychogenic PNEs	Total	p
Number (%)	41 (43.2)	54 (56.8)	95 (100)	
F/M (ratio)	18/23 (1:1.3)	36/18 (2:1)	54/41 (1:1.3)	0.027
Mean age at onset \pm SD (range) n = 94	4.87 \pm 3.30 (1–13)	11.35 \pm 3.01 (4–16)	8.53 \pm 4.49 (1–16)	<0.001
Mean age at time of VEEG diagnosis \pm SD (range) n = 95	8.82 \pm 4.53 (2–17)	13.79 \pm 2.81 (5–18)	11.65 \pm 4.39 (2–18)	<0.001
Mean duration of symptoms prior to diagnosis \pm SD (range) n = 94	3.92 \pm 3.81 (0–16)	2.49 \pm 2.18 (0–10)	3.11 \pm 3.07 (0–16)	0.024
AED treatment (%) n = 85	23 (67.6)	37 (72.5)	60 (70.6)	0.627
Concomitant epilepsy (%) n = 95	4 (9.8)	6 (11.1)	10 (10.5)	0.402
Abnormal interictal EEG (%) n = 95	7 (17.1)	4 (7.4)	11 (11.6)	0.129
Duration of events (%) n = 89				
0–2 min (%)	25 (67.6)	17 (32.7)	42 (47.2)	0.001
>2 min (%)	12 (32.4)	35 (67.3)	47 (52.8)	0.001
Frequency of events (%) n = 89				
\geq 1 per day	28 (75.7)	19 (36.5)	47 (52.8)	<0.001
Less than daily	9 (24.3)	33 (63.5)	42 (47.2)	<0.001
Medical history (%) n = 95				
Unremarkable	26 (63.4)	36 (66.7)	62 (65.3)	0.742
Febrile convulsions	4 (9.8)	6 (11.1)	10 (10.5)	0.554
Trauma	2 (4.9)	7 (13.0)	9 (9.5)	0.164
Perinatal asphyxia	9 (22.0)	4 (7.4)	13 (13.7)	0.041
CNS infections	0	1 (1.9)	1 (1.1)	0.568
Family history of epilepsy (%) n = 95	5 (12.2)	6 (11.1)	11 (11.6)	0.559

PNEs, paroxysmal nonepileptic events; F, female; M, male; AED, antiepileptic drug; SD, standard deviation; VEEG, video-EEG.

with physiologic or organic PNEs when compared with patients with psychogenic PNEs (11.4 \pm 3.0 years in the physiologic or organic group, 13.8 \pm 2.8 years in the psychogenic group, $p < 0.001$). In all patients, the mean duration between the onset of symptoms and establishment of the diagnosis was 3.11 \pm 3.0 years. Delay in diagnosis was significantly longer in the group with physiologic or organic PNEs when compared with the group with psychogenic PNEs (3.9 \pm 3.8 years in the physiologic or organic group, 2.5 \pm 2.2 years in the psychogenic group, $p = 0.024$). Thirteen (13.8%) patients were diagnosed within one year of onset of symptoms; in the remaining patients, diagnosis had been delayed for more than 1 year.

According to the clinical description reviewed on the video-EEG reports, there was no significant difference in regard to the type of motor activity between the two groups. Subtle motor activity was encountered in 63.4% of patients with physiologic or organic PNEs and in 50% of patients with psychogenic PNEs ($p = 0.192$). While stereotypic movements and staring episodes were the most common types of subtle motor activity seen in patients with physiologic or organic PNEs,

generalized jerking was the leading type of prominent motor activity in the psychogenic group (Table 3).

In 91 (95.7%) patients, typical episodes occurred spontaneously during monitoring. In the remaining 4 patients, who were all in the psychogenic group, events occurred only in response to provocative intravenous saline injection. Duration of events was 2 min or less in 67.6% of patients with physiologic or organic PNEs and in 32.7% of patients with psychogenic PNEs ($p = 0.001$). The majority of children with hypnic jerks and staring episodes had events lasting \leq 2 min (Fig. 2). The frequency of events was one or more per day in 75.7% of patients with physiologic or organic PNEs and in 36.5% of patients with psychogenic PNEs ($p < 0.001$). Comparisons of female/male ratio, age at onset of symptoms, delay in diagnosis, duration and frequency of events between patients with physiologic or organic and psychogenic PNEs are presented in Fig. 3.

Before video-EEG monitoring, 60 (70.6%) children had the diagnosis of epilepsy, and on discharge, 50 patients were found to have no epilepsy. In the past or at the time of admission, 37 (72.5%) patients

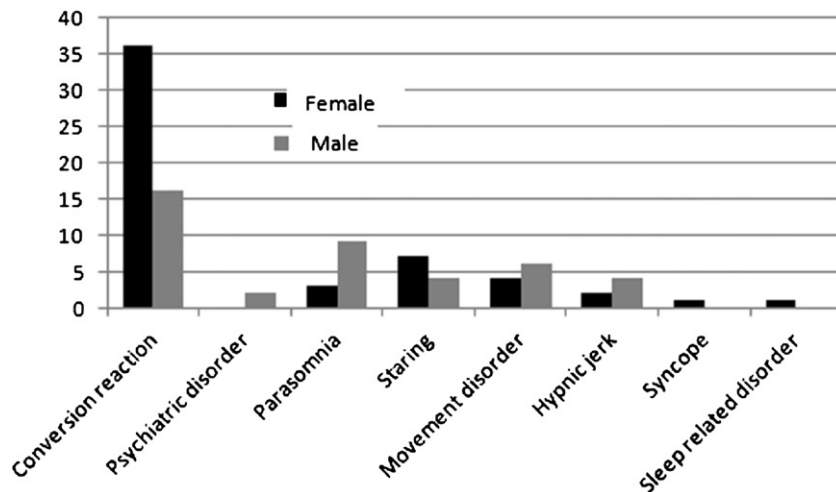


Fig. 1. Distribution of paroxysmal nonepileptic disorders by sex.

Table 3

Clinical description of paroxysmal nonepileptic events by etiology.

Types of events	Patients with organic or physiologic PNEs, n = 41	Patients with psychogenic PNEs, n = 54	Total n = 95	p
Prominent motor activity	15 (36.6) ^a	27 (50.0)	42 (44.2)	0.192
Generalized jerking or flailing	0 (0)	23 (42.6)	23 (24.2)	
Focal motor activity	6 (14.6)	1 (1.9)	7 (7.4)	0.192
Complex motor activity	9 (22.0)	2 (3.7)	11 (11.6)	
Generalized tremor	0 (0)	1 (1.9)	1 (1.1)	
Subtle motor activity	26 (63.4)	27 (50.0)	53 (55.8)	
Staring	11 (26.8)	8 (14.8)	19 (20.0)	
Generalized limpness	1 (2.4)	9 (16.7)	10 (10.5)	
Stereotypic movements	12 (29.3)	6 (11.1)	18 (18.9)	
Eye fluttering	1 (2.4)	0 (0)	1 (1.1)	
Eye deviation	1 (2.4)	0 (0)	1 (1.1)	
Subjective sensation	0 (0)	4 (7.4)	4 (4.2)	

PNEs, paroxysmal nonepileptic events.

^a Values are numbers (%).

with psychogenic PNEs and 23 (67.6%) patients with physiologic or organic PNEs were treated with one or more antiepileptic medications. Of all patients, 10 were diagnosed with concomitant epilepsy, 6 in the psychogenic group and 4 in the physiologic or organic group. Abnormal interictal EEG findings were seen in 7 patients with physiologic or organic PNEs and in 4 patients with psychogenic PNEs. Seven of 11 patients with interictal epileptiform activity had concomitant epilepsy. Only 4 patients in the physiologic or organic group and none in the psychogenic group without concomitant epilepsy had interictal epileptiform activity. Three patients with normal interictal EEG findings had the diagnosis of concomitant epilepsy. We noted a medical history of perinatal asphyxia in 13.7%, febrile convulsions in 10.5%, trauma in 9.5%, and CNS infections in 1.1% of patients. A history of perinatal asphyxia was more common in the group with organic or physiologic PNEs as compared with the group with psychogenic PNEs ($p=0.041$). There were no significant differences between the two groups in regard to history of febrile convulsions, trauma, and/or CNS infections. Family history of epilepsy was present in 11 (11.6%) of 95 patients, 5 in the physiologic or organic group and 6 in the psychogenic group ($p=0.559$). None of the patients who had concomitant epilepsy had a family history of epilepsy.

4. Discussion

Our data indicate that a wide variety of events in children mimics epileptic seizures, and some clinical features differ in children with physiologic or organic and psychogenic PNEs. Nonepileptic paroxysmal

events are quite commonly encountered in children. Although the diagnosis of most PNEs can accurately be established following a detailed history and clinical examination, in certain cases, differentiation from epileptic seizures may pose diagnostic difficulties [6,15]. Even experienced child neurologists may misdiagnose nonepileptic events as epilepsy [16]. The frequency of PNE episodes in children who underwent long-term video-EEG monitoring has ranged between 3.5% and 43% [7,11,17]. This wide range may reflect a referral bias. In our series, documented PNEs accounted for 12.4% of children admitted to the long-term video-EEG monitoring unit, a frequency similar to previous reports [6,9,17]. Since the study included patients referred for differentiation of epileptic and nonepileptic paroxysmal spells, seizure classification, and presurgical evaluation, our results do not show the actual incidence of PNEs in the general population.

Few studies have evaluated the frequency and clinical characteristics of PNEs in children that originated from organic or physiologic and psychogenic causes [1,6]. In our series, approximately 57% of documented PNEs were psychogenic in nature, and the remaining 43% originated from physiologic or organic causes. Except for 2 patients with hyperactivity disorder and impulse control disorder, conversion disorder accounted for all psychogenic PNEs. Unlike that of psychogenic events, etiology of the physiologic-organic events was heterogeneous and included parasomnias, staring spells, movement disorders, and hypnic jerks. In accordance with our findings, a previous study has reported that approximately half the documented PNEs were physiologic or organic in nature, and the remaining half comprised psychiatric disorders [6]. In another study, the percentage of children with organic or

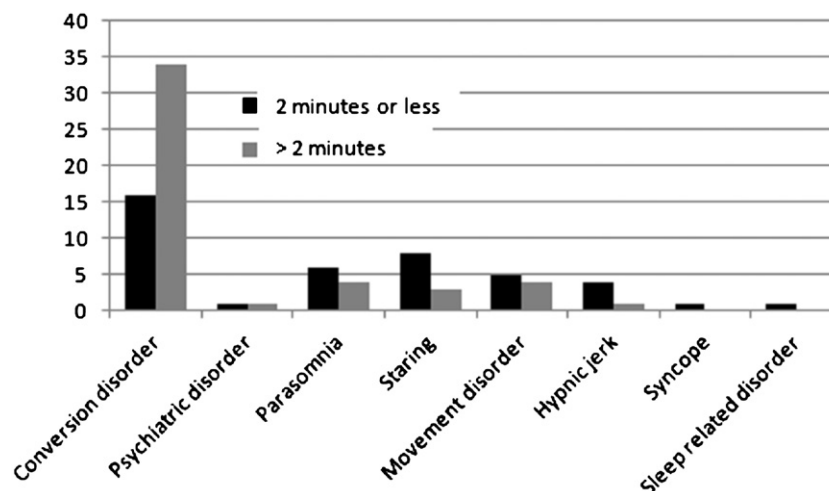


Fig. 2. Distribution of event duration by type of paroxysmal nonepileptic disorders.

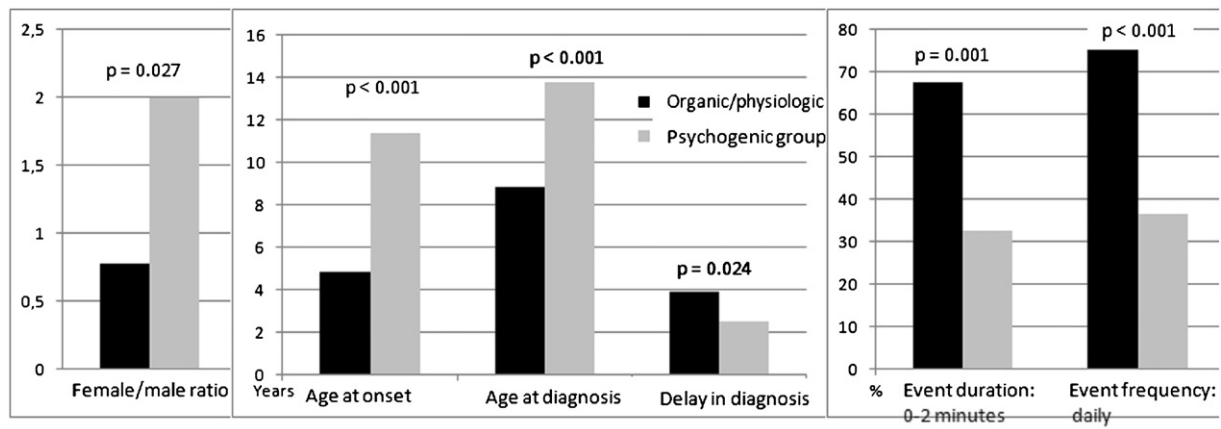


Fig. 3. The gender predominance of males, a younger age at onset of symptoms, shorter duration of events, and daily seizure frequency were found as differentiating features of physiologic or organic PNEs from psychogenic PNEs in children.

physiologic PNEs was found to be higher than that of patients with psychogenic PNEs [17]. In these studies, while most of the psychiatric events were conversion reactions, inattention or daydreaming episodes, hypnic jerks, stereotyped movements, parasomnias, and gastroesophageal reflux were more common diagnoses in patients with physiologic or organic events [6,17].

In the present study, the mean age at the time of onset of symptoms was 8.5 years. It is well established that PNEs typically manifest in the second or third decade of life [18,19] with a mean age of 12 years and 9 months [7]. When we excluded the patients with organic or physiologic events, our data showed similar results, with a mean age of 11.3 years. In accordance with a previous report [6], the patients with organic or physiologic PNEs were much younger, with a mean age of 4.8 years in our series.

We found an overall female predominance which has previously been reported in children, mainly in adolescents [1,6,7]. However this predominance was restricted to patients with psychogenic PNEs; on the contrary, we observed a slight male predominance in patients with organic or physiologic PNEs. Previously, a downward trend of female predominance was reported in younger ages [17,20]. In another study, males outnumbered girls in children younger than 12 years of age, a group which mainly consisted of patients with organic or physiologic PNEs [6].

We divided semiology of events into subtle and prominent motor activities as described by Patel et al. [7]. Both types of phenomenology were seen equally in the psychogenic group. Previously, unresponsiveness without motor activity was reported as the most common feature of psychogenic events, mainly in the adult population [21]. Another study has shown that tremor was the most frequent ictal motor sign in children with psychogenic PNEs [22]. In our series, generalized jerking was the most common semiology, followed by generalized limpness and staring episodes in the psychogenic group. Unlike that in the psychogenic group, subtle motor activity such as stereotyped movements and staring episodes was more common (although not significant) in the physiologic or organic group. In a recent study, it has been reported that nonepileptic events commonly manifested as subtle motor activity in children younger than 13 years [7].

The mean latency between onset of symptoms and determination of diagnosis has been reported to be very long in adults at 7.2 years [5]. In the pediatric population, however, this delay was much shorter [6,7,20]. In contrast to these reports, the time interval reaching the correct diagnosis was remarkably long in our series, at 3.27 years, and in only 13.5% of patients, was it less than one year. Delay in diagnosis was more marked in patients with organic or physiologic PNEs. Limited number of long-term video-EEG monitoring units in our country in the beginning of the 2000s may partly be responsible for this delay.

Psychogenic PNE episodes typically last longer than epileptic seizures [4]. In a recent study, it has been reported that the mean duration

of psychogenic PNEs in children was found to be longer (269 s) as compared with that of epileptic seizures (83 s) [22]. In our series, 67% of patients with psychogenic PNEs had events lasting more than 2 min, whereas the majority of patients in the physiologic or organic group experienced events lasting 2 min or less. Since children with clear clinical diagnosis of PNEs such as breath-holding spells, shuddering attacks, and simple tics were not monitored, we could not evaluate the features of these common disorders. Of children who underwent monitoring for differentiation of myoclonic jerks, 6 children were identified to have brief hypnic jerks. In accordance with our results, the mean duration of physiologic or organic events was reported as 1.6 s for shuddering and simple tics, 10 s for staring episodes, 11 s for sleep myoclonus, and 52 s for arousals [11]. These findings suggest that duration of events may help differentiate physiologic or organic PNEs from psychogenic ones. Nevertheless, video-EEG monitoring appears essential for differentiation of these relatively benign jerks from myoclonic seizures. Recently, it has been reported that frequency of events in adults was daily in 39% of patients with psychogenic PNEs [23]. In agreement with this report, 36% of children with psychogenic PNEs in our series had daily event frequency, whereas this percentage was significantly higher (76%) in the organic or physiologic group.

Comparable with previous studies reporting coexisting epilepsy in 9–46% of patients with PNEs [6,17,24], 10.5% of patients had concomitant epilepsy in our series, and 11.6% of patients had an epileptiform interictal EEG. Of our patients with confirmed PNEs, 70% were started on antiepileptic drugs. When we excluded 10 patients with concomitant epileptic seizures, we determined that 58% of patients were treated unnecessarily. Previously, unnecessary antiepileptic drug treatment was reported in 19–74% of patients with PNEs [6,7,21].

A family history of epilepsy has been reported in 25% of patients with PNEs [7]. In 11.6% of our cases, there was a family history of epilepsy. We also noted a medical history of perinatal asphyxia, febrile convulsions, trauma, and/or CNS infections in 35% of patients.

In limitations, we included a heterogeneous group of subjects who were referred not only for differentiation of epileptic and nonepileptic paroxysmal spells but also for seizure classification and presurgical evaluation. The patients in the study had treatment-refractory spells, and mild cases were possibly not monitored because of limited access to the monitoring. In a retrospective study, we were constrained by the information available in the medical records. However, a senior epileptologist (A.S.) participated in evaluation of all patients during the entire 13 years of the study period.

5. Conclusion

Unlike adults with nonepileptic events for whom the majority of events originated from psychogenic causes, in the pediatric population,

there is a wide spectrum of etiologies, both psychogenic and organic or physiological in nature. The gender predominance of males, a younger age at onset of symptoms, shorter duration of events, and daily seizure frequency appear to be the distinctive features for children with physiologic or organic PNEs as compared with psychogenic PNEs. There is no difference in regard to the type of motor activity between the two groups. In pediatric patients with treatment-refractory spells whose semiology and EEG findings are not clear and inconsistent with each other, long-term video-EEG monitoring is essential to differentiate nonepileptic events from epileptic seizures.

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