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## Case Report

# Sporadic nocturnal frontal lobe epilepsy: A consecutive series of 8 cases

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## ABSTRACT

**Objective:** To present findings on a series of cases of sporadic nocturnal frontal lobe epilepsy (NFLE), a form of NFLE that is infrequently reported, in contrast to familial (autosomal dominant) NFLE. Both forms of NFLE need to be distinguished from parasomnias, nocturnal temporal lobe epilepsy, and other nocturnal disorders.

**Methods:** Eight consecutive cases of sporadic NFLE were evaluated at a sleep clinic in Taiwan. All patients had clinical evaluations, daytime waking and sleeping EEGs, brain MRIs, and overnight video-polysomnography (vPSG) with seizure montage.

**Results:** Gender was equal (four males, four females); mean age was 18.4 yrs (range, 7–41 yrs). Age of NFLE onset was by puberty. Premorbid history was negative for any neurologic, medical or psychiatric disorder. NFLE subtypes: nocturnal paroxysmal dystonia,  $n=6$ ; paroxysmal arousals,  $n=2$ . MRI brain scan abnormalities with clinical correlates were found in one patient. Daytime awake EEGs were negative for ictal/interictal activity in all patients, but two patients had daytime sleep EEGs with interictal epileptiform EEG activity. During vPSG studies, three of eight patients with NFLE seizure events had concurrent epileptiform EEG activity, and two patients had interictal epileptiform EEG activity during their vPSG studies. No case had a spontaneous remission. Anticonvulsant therapy was highly effective in all eight cases (>75% reduction in seizure frequency).

**Discussion:** These cases confirm that sporadic NFLE closely resembles familial NFLE, and comprises a set of distinct clinical manifestations, with variable intensity, and variable scalp EEG epileptiform abnormalities across sleep and wakefulness, which have previously been identified in Caucasian patients from Europe and North America.

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## 1. Introduction

In 1881, Gowers documented that 21% of epilepsy patients had seizures exclusively during sleep [1]. Subsequent studies of subjects with either partial or generalized seizures have

estimated that the relative incidence of seizures during sleep is from 7.5% to 30% [2].

Some types of sleep-related epilepsy present with bizarre behaviors or vocalization, but without convulsion-like movement, tongue biting or upward gaze. Extreme restlessness,

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excessive swallowing movements, nightmares, and sleep-walking may represent various phenomena of seizures that emerge from sleep [3]. Since nightmares and sleepwalking can be components of the nocturnal symptom complex, parasomnias can be incorrectly diagnosed as the basis for these sleep related behaviors and disturbed dreams, and then subsequently incorrectly treated. This scenario poses a major challenge for sleep clinicians. However, epilepsy with bizarre behaviors is usually caused by frontal lobe epilepsy (FLE) or temporal lobe epilepsy (TLE). FLE and TLE can be contrasted [4]. FLE presents with bizarre behaviors, frequent short attacks, rapid recovery, and substantial preservation of consciousness. Repetitive and stereotypical behaviors characterize the clinical presentation without change in scalp EEG; the aura and the postictal periods can be masked by sleep. This scenario further increases the risk of incorrectly diagnosing a parasomnia as the basis of these abnormal nocturnal behaviors.

On the basis of the different intensity, duration and features of the motor patterns, Provini et al. [5] classified the nocturnal FLE (NFLE) epileptic seizures into three groups, according to Montagna [6]: (1) Paroxysmal Arousals (PA) with brief (<20 sec) episodes in which patients suddenly open their eyes, raise their heads or sit up in bed with a bizarre posture of the limbs, staring around with a frightened or surprised expression, and sometimes screaming; they then return to sleep. (2) Nocturnal Paroxysmal Dystonia (NPD) with a longer duration (20 sec–2 min) and more complex behaviors characterized by wide-ranging, often violent, and sometimes ballistic movements, with dystonic posturing of the head, trunk and limbs, such as head rotation, torsion of the trunk and choreo-athetoid movements of the arms and legs, with vocalization. (3) Episodic Nocturnal Wandering (ENW) with duration of episodes lasting up to 1–3 min, for which the characteristic feature is stereotypic paroxysmal ambulation during sleep, often with agitation and accompanied by screaming and bizarre, dystonic movements.

Sporadic (i.e. non-familial) NFLE is rarely reported, in contrast to familial NFLE, and therefore is poorly understood. We now report eight consecutive cases of sporadic NFLE, which also comprise the first case series from Taiwan (or any other Asian country), and provide clinical and vPSG findings, particularly vis-à-vis findings reported in Caucasian populations. The differential diagnosis, particularly focused on nocturnal temporal lobe epilepsy [7,8] and the parasomnias, will be discussed. One case in this series reported herein has previously been reported (patient 4) [9].

## 2. Materials and methods

Eight consecutive patients with equal 4:4 sex ratio and mean age of 18.4 yrs (range, 7–41) presented to the sleep clinic of one author (S-B Y) from July 2006 to November 2011 on account of nocturnal paroxysmal episodes suggestive of NFLE. These eight patients completed a comprehensive questionnaire covering lifetime sleep-wake, medical and

psychiatric history, and review of systems. The patients and, when applicable, their caregivers were interviewed. They also received a full neurological examination by a pediatric or adult neurologist. Routine daytime awake and sleep EEG recordings were also performed for these patients. Table 1 contains the clinical data, including results of brain MRIs.

An overnight, hospital-based, vPSG monitoring, utilizing standard recording and scoring methods [10], was then performed on these patients after discontinuation of anti-epileptic drugs for at least one day, except patient 8 who had recurrent attacks several times daily in wakefulness and sleep beginning shortly on the day of medication discontinuation. The PSG monitoring included an electrooculogram (EOG), expanded EEG (seizure montage) with a 1 cm/sec recording speed, submental and bilateral anterior tibialis electromyograms (EMGs), nasal-oral airflow, chest and abdomen respiratory effort, electrocardiogram, and continuous time-synchronized audiovisual recording.

## 3. Results

Seven of eight patients manifested one or more nocturnal attacks during vPSG monitoring. Patient 8 (who was maintained on anticonvulsant medication) did not have an attack, but she had interictal epileptiform discharges (spike and waves) during the overnight vPSG study, and attacks were viewed with event video home recording provided by her family. These eight cases were classified as PA (two cases) and NPD (six cases). All eight cases had sporadic NFLE, without any positive family history.

The nocturnal paroxysmal episodes had been present for up to 18 yrs (mean 9.6 yrs) before the current reported evaluation; age at presentation ranged from 7 to 41 yrs (mean 18.4 yrs). Mean duration of the seizure history was 9.6 yrs (range, 2–18 yrs). Seizure frequency in all eight patients was several attacks nightly, during nearly every night. All eight patients were unaware of their nocturnal motor manifestations, and so medical consultation was sought by their families who had observed the recurrent episodes. All eight patients had undergone neuroradiological examination, viz. brain MRI. Abnormalities with clinical correlates were detected in one case, involving a right orbitofrontal lobe cortical dysplasia in patient 4.

Two or more seizures with a stereotypic motor pattern were recorded in six of eight patients, and in one patient a single episode was recorded; patient 8 had no seizure episode attack during vPSG recording, but with clear-cut interictal epileptic EEG activity during the vPSG recording. Three patients also had occasional seizures during daytime wakefulness, similar to their seizures during sleep. In these three cases, however, daytime seizures were sporadic with low frequency.

Six PA episodes were recorded from two patients, lasting a mean of 11.0 sec in patient 5 and 11.5 sec in patient 6. In the PA episodes, the first movement usually involved the upper limbs: the patients suddenly raised their arms while asleep,

**Table 1 – Clinical data, N=10 patients.**

Patient	Sex	Age of latest visit, yr	Age of initial visit, yr	Age of seizure onset, yr	Duration of seizure history (yrs)	trigger factor	Personal antecedents	Seizure type	Neurological image (MRI)	Positive response to antiepileptic drugs	FLEP score (+ numbers)
1	M	15	9	5	10	Negative	Negative	NPD	Unremarkable	Yes, oxcarbazepine 600 mg bid and topiramate 25 mg hs	6
2	M	14	8	2	12	Protracted exercise	Negative	NPD	Unremarkable	Yes, carbamazepine 200 mg qd & 300 mg hs and topiramate 50 mg bid	4
3	M	7	2	1	6	Negative	Negative	NPD	Unremarkable	Yes, carbamazepine 200 mg hs	5
4	F	12	8	8	4	Negative	Negative	NPD	Right orbitofrontal lobe cortical dysplasia	Yes, oxcarbazepine 300 mg bid and topiramate 25 mg bid	6
5	F	41	41	23	18	Upper respiratory viral infections	Negative	PA	Unremarkable	Yes, carbamazepine 400 mg bid	4
6	M	7	7	5	2	Negative	Negative	PA	Unremarkable	Yes, valproate 300 mg bid and levetiracetam 500 mg bid	3
7	F	33	30	20	13	Menstrual related	Negative	NPD	Unremarkable	Yes, carbamazepine 200 mg bid or lamotrigine 300 mg hs	4
8	F	18	15	6	12	Negative	Negative	NPD	Unremarkable	Yes, acetazolamide 250 mg bid, oxcarbazepine 300 mg hs, and clonazepam 0.5 mg tid	7

or assumed a dystonic posture of one hand. Twenty-eight NPJ episodes were recorded from six patients, lasting from 22 to 55 sec (mean  $36.8 \pm 8.3$  sec). There was a mean 4.7 NPJ episodes per vPSG recording. Seizures appeared between 3 and 278 min after sleep onset (mean 150.1 min). 94.1% (32/34) of the episode attacks appeared during NREM sleep, with 88.2% of the attacks during light (stage N1-N2), and 5.9% during deep sleep (stage N3). Only 5.9% of seizures occurred during REM sleep. Mean seizure duration in the six NPJ patients ranged from 11.0 sec to 41.0 sec, and mean seizure duration in the two PA patients was 11.0 and 11.5 sec, respectively.

All eight cases had normal wake scalp EEG activity. In six patients (75%), sleep EEG was completely normal. Only two patients (25%) showed focal frontal epileptic abnormalities. EEG recordings during the attacks failed to disclose ictal epileptic activity in four cases. In such cases, EEG was often masked by muscle artifacts or characterized by an abrupt transition to wake activity or light sleep, occasionally (patient 6) preceded by a K-complex. EEG recordings during the attacks did record ictal activity in three cases: a burst of sharp waves in patient 1 (Fig. 1); background EEG activity suppression followed by a burst of sharp waves in patient 4; and continuous rhythmic sharp activity (2-6 Hz) for 50 sec, followed by slow-wave activity (1-2 Hz) in patient 5. The first EEG modifications were either a diffuse flattening of background activity (1 case), or sharp activity (four cases). In one patient (patient 8), the interictal EEG was characterized by spike-and-waves activity (Fig. 2). Autonomic hyperactivity was remarkable in many cases: tachycardia (four cases) and sustained tachypnea with tachycardia (one case) appeared synchronously with seizure onset or accompanied the movement artifacts. The supplementary video files contain examples of the behavioral correlates of seizure events recorded during the vPSG studies for patients #1, 3-7; patient #2 had suboptimal video quality, and patient #8 did not have any seizure during the vPSG study, as described. Screaming was also present in patients #4,7, vocalizations were also present in patients #1, 3, 5, and prominent tachypnea was also present in patient #1.

Supplementary material related to this article can be found online at <http://dx.doi.org/10.1016/j.slsci.2014.09.016>.

FLEP (Frontal Lobe Epilepsy and Parasomnias) scale scores were calculated for each patient, according to the method of Derry et al. [11,12]. An FLEP score  $\geq 1$  strongly supports the diagnosis of NFLE and FLEP score of  $\leq 0$  strongly supports the diagnosis of a parasomnia (viz. NREM sleep arousal parasomnia, such as sleepwalking, sleep terrors, confusional arousals). The reported findings of Derry et al. were independently confirmed by Manni et al. who found that the sensitivity of the FLEP scale as a diagnostic test for NFLE was 71.4%, the specificity was 100%, the positive predictive value was 100%, and the negative predictive value 91.1% [13]. The authors emphasized that the FLEP scale was particularly suited in distinguishing NFLE from NREM parasomnias more than in distinguishing NFLE from REM sleep behavior disorder.

The final column in Table 1 contains the FLEP scores for the eight patients. Although the supplementary videos for patients 4, 5, 6 may not have been as compelling as for the

other patients, their FLEP scores of +6, +4, and +3 strongly supported the diagnosis of NFLE.

#### 4. Follow-up and response to antiepileptic drugs

Based on prior reported experience [14,15], carbamazepine was the drug of first choice in these patients. Due to safety considerations [16], oxcarbamazepine was also the initial treatment of choice. Six patients received carbamazepine at a dosage varying from 200 to 500 mg/day in monotherapy (two cases) or polytherapy (two cases, combined with topiramate or lamotrigine). Three patients received oxcarbamazepine at a dosage varying from 300 to 1200 mg/day in polytherapy (two combined with topiramate, and the other one combined with acetazolamide and clonazepam). Patient 6 was referred from a pediatric neurologist and was still under control with sodium valproate therapy, 300 mg bid and levetiracetam, 500 mg bid. These medications reduced the frequency of nocturnal seizures by at least 75% and abolished any occasional diurnal attack by more than 90%. None of the patients had any adverse effects or complaints related to treatment.

The long duration for diagnosing NFLE (a mean 3.4 yrs extending up to 6 yrs), and the long duration for initiating anticonvulsant therapy without any prior attempts at therapy, can be explained as follows: (i) the NFLE attacks occurred predominantly during nocturnal sleep, and often did not call attention to their presence; (ii) the bizarre movements were not typical seizure symptoms for most neurologists; (iii) and very few neurologists in Taiwan have any training in sleep medicine. Consequently, these patients were usually considered to have non-specific nocturnal events that would eventually resolve spontaneously.

#### 5. Discussion

Our case series of sporadic (non-familial) NFLE, which to our knowledge is the first reported case series of NFLE from Taiwan or from any other Asian country, corresponds closely to previously reported sporadic and familial NFLE among Caucasian patients in Europe and North America. In regards to any possible differences in epidemiologic frequency of NFLE in Taiwanese and other Asians compared to Caucasian populations, a PubMed search did not reveal any prevalence data for any of these groups, most likely because relatively few NFLE cases have been reported. Also, in regards to the question of whether any forms of epilepsy are different between Taiwanese and Caucasian populations (viz. epidemiology and clinical characteristics), a PubMed search did not reveal any identified differences.

Our findings reinforce how NFLE should always be suspected in the presence of paroxysmal nocturnal motor events characterized by a high frequency of same-night or inter-night recurrence, persistence beyond puberty into adulthood, quasi-extrapyramidal features, agitated behavior and remarkable stereotypy of the attacks. Although EEG recordings during the attacks failed to disclose ictal epileptic



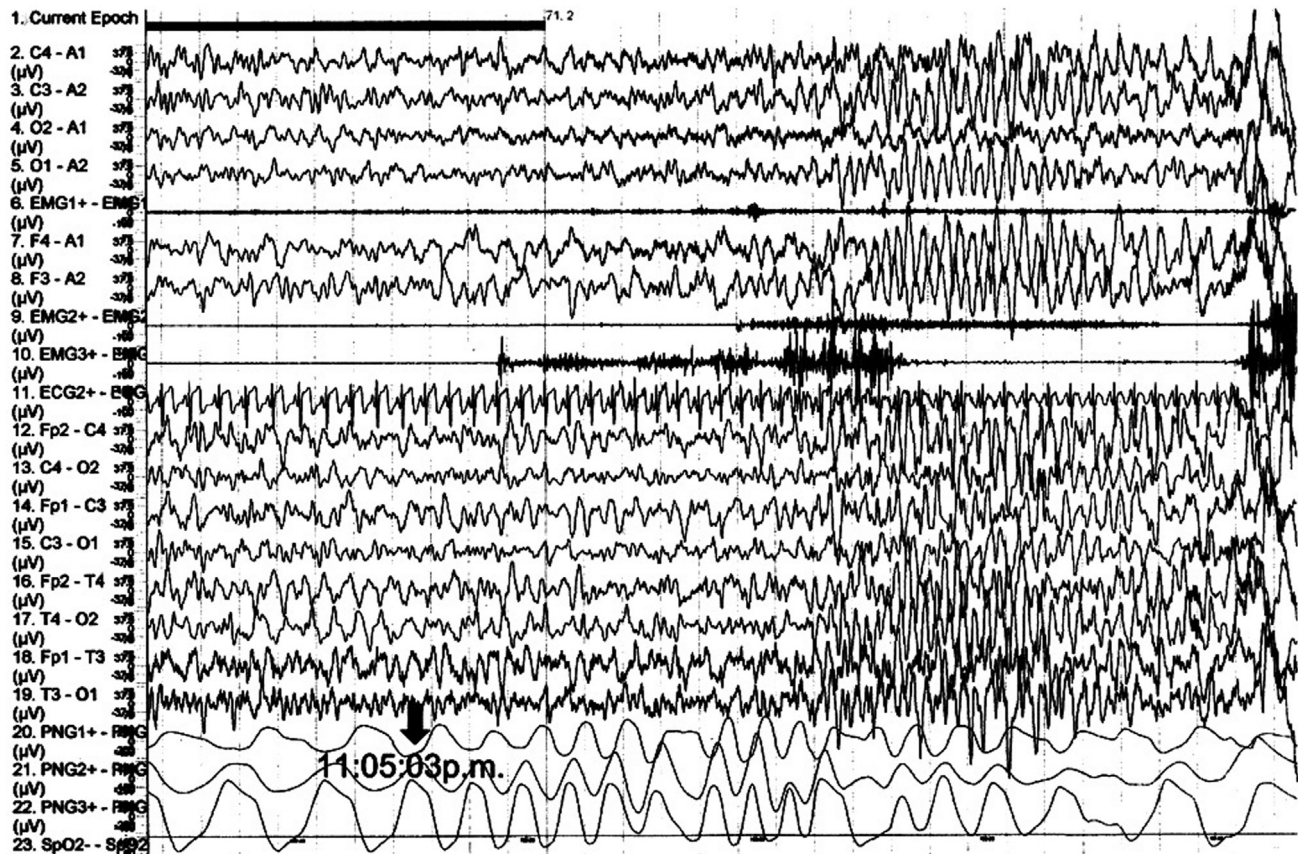


Fig. 1 – Nocturnal PSG (30s epoch) during stage 2 sleep and the emergence of tachypnea following the four limb dystonic posturing. Black solid arrow indicates the event onset time (11:05:03 p.m.), and the event duration persists for 36 sec. EEG montage (channels 12–19) shows with a run of sharp waves during the event episode. The electrooculogram (channel 7–8) indicates no rapid eye movement. The electrocardiogram (channel 11) shows an increase in heart rate during the episode attack. Channels 20–23 represent the nasal/oral airflow, chest respiratory effort, abdomen respiratory effort and O<sub>2</sub> saturation, which do not show any sleep apnea or oxygen desaturation, but an increase in respiratory rate beginning with the onset of the episode attack.

activity in four cases, and three cases also had no interictal findings, we concluded that the events were seizures because of the stereotyped movements and behaviors, and also because of the robust response to anti-epileptic medications. The absence of scalp EEG ictal epileptic activity during the NFLE seizure attacks in some patients can be explained by the seizure focus being located in deep brain regions, as previously discussed [5].

The high rate of sustained treatment efficacy is gratifying for the patients (once they are informed of their disorder), their families, and their physicians. The most commonly effective anticonvulsants, as first-line therapy, in our series were carbamazepine and oxcarbamazepine, but also topiramate was effective in two patients, and several other anticonvulsants were used with benefit. Topiramate was reported to be highly effective therapy in a series of 24 consecutive NFLE patients presenting to a sleep disorders center in Italy, with 15 cases being sporadic NFLE and 9 cases being familial NFLE [17]. Nearly 90% of topiramate-treated patients (as monotherapy or add-on therapy) became either seizure-free or had >50% reduction in seizure frequency. Mean age of the cases in this series was  $29.3 \pm 10.4$  yrs; mean age of NFLE onset was  $14.6$

$\pm 10.5$  yrs (range, 4–40 yrs). Video-PSG documented a wide spectrum of NFLE behaviors, ranging from repeated stereotypic brief motor attacks to prolonged attacks with complex and bizarre behaviors. All recorded seizure episodes occurred during N2 and N3 sleep. Waking EEG was normal in all patients, and epileptiform abnormalities were documented during sleep in two patients. Therefore, all the clinical features in this case series of 24 Caucasian patients from Italy closely match the findings in our case series of eight Taiwanese patients.

In regards to the differential diagnosis, there was no evidence supporting the diagnosis of sleep-related myoclonic seizures, or any form of sleep-related (benign) myoclonus condition. Parasomnias need to be carefully considered in the differential diagnosis of NFLE [18]. For example, clues for distinguishing nocturnal seizures from sleep terrors include the following [18]: (i) nocturnal seizures have a brief, stereotypical presentation; (ii) with frontal lobe seizures, events usually occur from sleep (NFLE), with bizarre hypermotor activity, but without altered consciousness upon awakening; (iii) sleep terrors rarely occur >1 time per night, and very rarely occur nightly, and almost never occur nightly for months or years; (iv) with sleep terrors, there is confusion

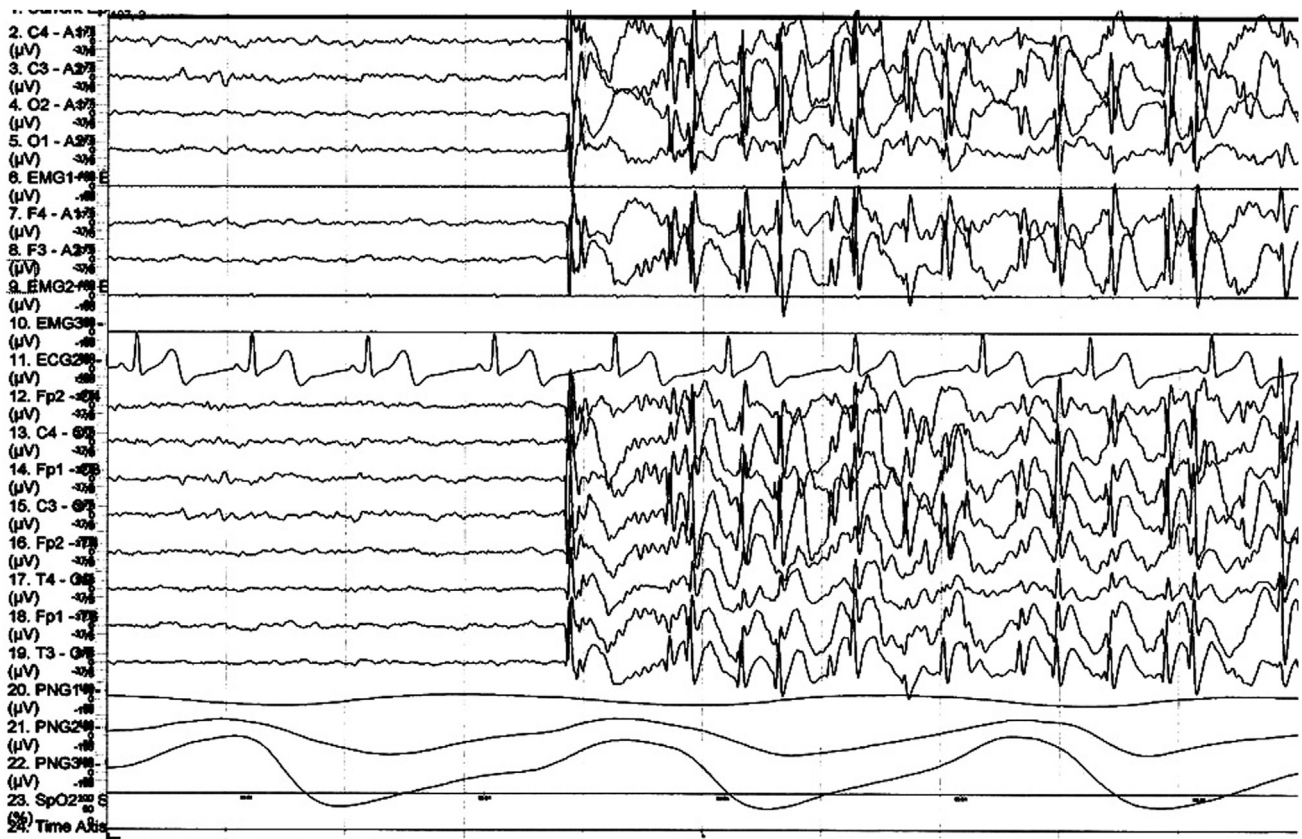


Fig. 2 – Nocturnal PSG (10s epoch) during N2 sleep and the emergence of epileptiform EEG discharge. EEG montage (channels 12–19) shows a run of spike-and-waves during an interictal period. The electrooculogram (channels 7–8) indicates no rapid eye movements. The electrocardiogram (channel 11) is unremarkable during the interictal epileptiform EEG discharge. Channels 20–23 represent the nasal/oral airflow, chest respiratory effort, abdomen respiratory effort and O<sub>2</sub> saturation, which do not show any sleep apnea, oxygen desaturation, or tachypnea.

and disorientation during an arousal, and episodes can last for minutes; (v) sleep terrors usually respond to bedtime benzodiazepine therapy.

The differential diagnosis of NFLE, nocturnal temporal lobe epilepsy, and parasomnias merits further elaboration. Sleep-related hyperkinetic seizures of temporal lobe origin were documented in three patients by means of long-term stereo-EEG investigations and surgical outcome [7]. In a retrospective study of 442 consecutive patients with drug-resistant hyperkinetic seizures that were surgically treated, 25 of these patients had sleep-related hyperkinetic seizures, of which 18 had a frontal lobe onset, and 7 had a temporal lobe onset [8]. The latter group of seven patients with temporal lobe origin had anamnestic and clinical features that mirrored the clinical features found in the 18 patients with frontal onset, with agitated movements, high seizure frequency and absent history of febrile convulsions. Therefore, the presence of sleep-related hyperkinetic seizures is not specific to NFLE, as they can also be found with nocturnal temporal lobe epilepsy.

The spectrum of minor motor events (MMEs), paroxysmal arousals, and major attacks during sleep in NFLE was carefully studied in three patients with drug-resistant NFLE who underwent stereo-EEG monitoring with implanted intracerebral electrodes [19]. This monitoring provided important and

relatively rare (on account of the intracerebral monitoring) insights into the clinical and neurophysiological manifestations of the episodes. The first patient, a 17-year-old male, had very frequent major motor attacks during NREM sleep, along with numerous MMEs lasting 1–2 sec that consisted of left head and eye deviation. Major attacks began with the same movements observed with MMEs followed by hypertonic posturing of the right arm and pelvic thrusting. This activity persisted approximately 40 sec and culminated with clonic movements of the right hand. During MMEs, the stereo-EEG showed a burst of rhythmic sharp activity within the supplementary sensorimotor area, the frontal and central cingulate gyrus and the posterior part of the middle frontal gyrus. After undergoing a left frontal corticectomy (which included the abnormal areas identified by stereo-EEG), the patient remained seizure-free during the reported 3-month follow-up period. The second patient, a 44-year-old woman with a positive family history of parasomnia, had almost exclusively nocturnal high-frequency seizures with rolling movements of the pelvis followed by hypertonic-hyperkinetic activity and sleeptalking with bruxism. At times, she had an aura with the sensation “as if the body moved around its axis.” Scalp video-EEG recorded hyperkinetic seizures during both NREM and REM sleep, and also rolling pelvic movements lasting several seconds, with sleeptalking and isolated, repetitive leg



movements. Ictal scalp EEG showed low voltage fast activity localized over the fronto-central region. Intracerebral activity (video-EEG) was monitored concurrently with scalp EEG and documented many rolling pelvic movements related to arousal fluctuations during NREM sleep and lasting 3–10 sec. One major episode was recorded that started with rolling pelvic movements and progressed in <8 sec to a bilateral hypertonic-hyperkinetic seizure. Sleepwalking occurred with arousal after the pelvic movements. The third patient, a 31-year-old male, had almost exclusively sleep-related hyperkinetic seizures, along with sudden arousals from sleep with dystonic leg posturing; he was able to speak immediately after these seizures. Both scalp-EEG and four nights of video-stereo EEG (intracerebral monitoring) recorded motor events with variable intensity that ranged from PAs to major hyperkinetic seizures lasting 40 sec. PAs featured head elevation (always with open eyes) followed by truncal elevation, with the patient sitting up and looking around for a few seconds. All episodes occurred during NREM sleep. Prolonged episodes of PAs were followed by brief dystonic movements of the limbs and pelvic thrusting or fully developed bilateral hypertonic-hyperkinetic activity. Stereo-EEG showed that all PAs were correlated to a discharge of polyspikes localized over the dorsolateral cortex of the superior frontal gyrus. With the more intense episodes, the epileptic discharge started in the same regions, but with a higher amplitude and immediately involved the whole frontal region with a discharge of fast activity lasting about 40 sec. Right frontal corticectomy resulted in complete resolution of seizures during two years of follow-up. Thus, these three cases demonstrated the frontal epileptic origin of MMEs and supported prior postulations that the increasing complexity of ictal motor behavior could reflect a different length and spread of the discharge within the frontal lobe [6]. However, the absence of stereotypy of PAs, despite the morphological and topographic similarity of the epileptic EEG discharges, might suggest that the clinical features of PAs depend not only on the site of epileptic discharge but also on other variables such as the level of arousal from which the epileptic discharges occurred and the patient's body position [19]. This highlights how the motor phenomena in NFLE are not always easily distinguishable from NREM sleep parasomnias [20], and emphasizes the importance for using the FLEP scale in helping the diagnostic differentiation of NFLE from NREM parasomnias [11].

Other differential diagnoses includes psychogenic, non-epileptic seizures [21], and nocturnal sleep-related dissociative disorder [22–24]. The latter condition represents a “psychiatric parasomnia” emerging during EEG wakefulness during the sleep period, in which the patient is often an adolescent or young adult female with a history of being sexually abused at night, usually in bed either in the process of falling asleep or after being abruptly awakened by a sexual predator. For example, a young adult woman during vPSG displayed prolonged (>5 minutes) sexualized pelvic movements, with a crescendo build-up accompanied by painful moaning that eventually resembled the hyperkinetic pelvic thrusting seen in NFLE [23]. She also repeatedly demonstrated having her shoulders pinned to the mattress during the vPSG. This prolonged event occurred during sustained EEG wakefulness that had emerged during the wake-sleep

transition. When the episode stopped spontaneously, the patient told the attending sleep technologist that she had just been dreaming that her older sister was shoving a ruler in and out of her vagina (just as the sister had done during her childhood, besides pinning her shoulders to the bed), which was causing her a lot of pain. In the eight cases of sporadic NFLE reported herein, there was no history of prior sexual and/or physical assault, and no associated dreaming, and no suspicion for nocturnal dissociative disorder or any other psychopathology.

In conclusion, differentiating NFLE from nocturnal temporal lobe epilepsy, NREM sleep parasomnias, and other nocturnal conditions, requires a careful, comprehensive, and multi-modal clinical evaluation, given the peculiar (and even bizarre) presentation of the recurrent abnormal behaviors that often emerge in the context of a normal scalp EEG.

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