FULL-LENGTH ORIGINAL RESEARCH

Electroclinical findings of minor motor events during sleep in temporal lobe epilepsy

Loretta Giuliano, Denise Uccello, Daniela Fatuzzo, Greta Mainieri, Mario Zappia, and Vito Sofia

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Loretta Giuliano is a neurologist at the Neurology Clinic of the University of Catania.

Summary

Objective: It is well known that sleep-related motor seizures can originate from the temporal lobe. However, little is known about the clinical features of minor motor manifestations during sleep in patients with temporal lobe epilepsy. The main objective of our study was to verify the existence of minor motor events during sleep in patients with mesial temporal lobe epilepsy (MTLE) and to define their clinical features and electroencephalography (EEG) correlations.

Methods: We enrolled in the study patients with diagnosis of symptomatic MTLE and a group of healthy controls. All patients and controls underwent long-term video -EEG monitoring, including at least one night of nocturnal sleep. We analyzed all the movements recorded during nocturnal sleep of patients and controls and their electroencephalographic correlations.

Results: We analyzed the nocturnal sleep of 15 patients with symptomatic MTLE (8 males and 7 females; mean age ± standard deviation [SD]31.8 ± 14.9 years) and of 15 healthy controls (6 males and 9 females; mean age ± SD 32.8 ± 11.2 years). The analysis of movements during sleep revealed significant differences between groups, with the patients presenting significantly more movements in sleep than healthy controls (56.7 ± 39.2 vs. 15 ± 6.1; $p < 0.001$) with significant differences regarding oroalimentary automatisms, limb dystonia, straightening movements and gestural automatisms. EEG analysis showed that the proportion of movements preceded by EEG abnormalities was significantly higher in patients than in controls (57.8 ± 35.9 movements vs. 16.6 ± 13.4 movements; $p < 0.001$).

Significance: The results of our study demonstrated the presence of minor motor events during sleep in patients with MTLE, suggesting an epileptic origin of these episodes. The study of nocturnal sleep in MTLE patients is useful in helping the clinicians in the diagnostic and therapeutic workup of these patients.

KEY WORDS: Epilepsy, Sleep, Minor motor events.

The bidirectional interrelationships between sleep and epilepsy have been the subject of many scientific works[1–4] since the first demonstration by Gibbs and Gibbs in 1947.[5] It is well known that sleep influences the expression of seizures and that epilepsy itself can significantly alter the intrinsic structure of sleep.

Sleep-related motor seizures have always been considered a typical feature of nocturnal frontal lobe epilepsy (NFLE), an epileptic syndrome characterized by a large spectrum of nocturnal seizures with different motor behaviors, ranging from paroxysmal arousals to episodes of nocturnal wandering.[6–9] In NFLE, together with major episodes, minor motor events (MMEs) have been described, consisting of brief, stereotyped movements of the limbs, the trunk, or the head, occurring many times during nocturnal sleep.[10,11] However, although major episodes are proved to be of epileptic origin, it is still controversial whether the

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Department of Medical and Surgical Sciences and Advanced Technologies "G.F. Ingrassia," University of Catania, Catania, Italy
Address correspondence to Mario Zappia, Department of Medical and Surgical Sciences and Advanced Technologies "G.F. Ingrassia," Policlinico-Vittorio Emanuele, Via S. Sofia 78, 95123, Catania, Italy. E-mail: m.zappia@unict.it

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Key points

- Minor motor events during sleep occur more frequently in patients with MTLE than in healthy controls
- The most frequent movements found in sleep of patients with MTLE are oroalimentary automatisms, limb dystonia, straightening movements, and gestural automatisms
- These movements are associated with EEG abnormalities more frequently in patients than in controls

MMEs are causally linked to epileptiform discharges.[12–14] Recently the spectrum of nocturnal motor seizures has been extended with the definition of a new nosologic entity called "sleep-related hypermotor epilepsy" (SHE). SHE is characterized by seizures with characteristic motor features, occurring during sleep, that may arise from extrafrontal sites.[15] In fact, over the years it has been demonstrated that sleep-related motor seizures can also originate from the temporal lobe.[16–19]

In this regard, a nosologic entity called "nocturnal temporal lobe epilepsy" has been coined in 1998 by Bernasconi et al., who well described the clinical characteristics of a group of temporal lobe epilepsy (TLE) patients with seizures occurring exclusively or predominantly during sleep.[20] In this study, seizures of nocturnal TLE, compared with frontal lobe seizures, have been found to be less frequent, not clustered, and with less hypermotor activity. However, subsequent studies demonstrated that nocturnal seizures of temporal lobe origin may have clinical features similar to those of frontal onset, being characterized by hypermotor events frequently recurring during sleep.[19] In particular, patients with mesial temporal lobe epilepsy (MTLE), usually associated with hippocampal sclerosis, have been more often described as manifesting peculiar behaviors during sleep such as sleepwalking, nightmares, frightened behaviors, or motor paroxysms, and showing characteristic electroencephalography (EEG) patterns such as fast activity synchronizations.[20–25] Some studies, focusing their attention on specific minor motor manifestations in MTLE, such as facial-wiping behaviors, demonstrated that they were more frequent in patients with MTLE than controls and usually occurred after a seizure.[25] However, these findings have been proven only during wakefulness until now.

Nevertheless, little is known about the clinical features of minor motor manifestations during sleep in MTLE and, to our knowledge, MMEs during sleep have never been described in these patients.

The main objective of our study was to verify the existence of MMEs of epileptic origin during sleep in patients with MTLE and to define their clinical features and their possible electroencephalographic correlations.

Methods

We enrolled in the study a group of patients with diagnosis of TLE made according to the 2001 International League Against Epilepsy (ILAE) criteria[26] evaluated in the epilepsy service of the Neurological Clinic of Catania. To have the certainty of the localization of the epileptogenic zone (EZ) in the mesial temporal lobe, we selected a subgroup of patients with diagnosis of symptomatic MTLE according to clinical, electroencephalographic, and neuroimaging evidences. In addition, we recruited a group of age-matched healthy controls, represented by subjects for which the diagnosis of epilepsy was excluded after clinical and electroencephalographic evaluation. The study was approved by the local ethics committee. Written informed consent was provided by all participants.

Demographic and clinical data of patients and controls were obtained through interviews with the patients and relatives, and by reviewing hospital charts in the group of patients. High-resolution magnetic resonance imaging (MRI) was performed for each patient (1.5-Tesla ACS-NT unit; Philips Medical Systems, Best, The Netherlands) and included inversion recovery (IR) and fluid-attenuated inversion recovery (FLAIR) sequences acquired in different planes, as well as T_1-weighted volumetric sequences. A neuropsychological evaluation was performed in patients and controls.

All subjects included in the study underwent long-term video-EEG monitoring (LTM), including at least one night of recorded nocturnal sleep. The recording was carried out using a minimum of eight EEG channels, placed according to the International 10–20 system. The sampling rate was 256 Hz. The low filter was set at 1.6 Hz and the high filter at 70 Hz; the sensitivity was switched between 50 and 200 μV/cm. Moreover, in almost all the subjects, two electrocardiographic derivations, chin and right anterior tibialis electromyography (EMG) and electrooculography electrodes were used.

LTM recordings were carefully analyzed independently by two experienced blinded observers (LG, DF) in order to evaluate every motor event during sleep. MMEs were identified as motor patterns of short duration (<4 s), occurring during sleep, with or without an electroencephalographic correlation.[11] Each motor event corresponding to these features was therefore standardized and assigned to a few distinct recurring patterns: hand-face movements, oroalimentary automatisms, limb dystonia, head rotation, pelvic movements, straightening movements, gestural automatisms, and other not defined movements (Fig. 1). The number of patients and controls performing each type of movement was computed. Moreover, the sleep stage in which each movement occurred was reported.

Afterwards, in both groups, the EEG traces were carefully analyzed by two independent observers (VS, GM).

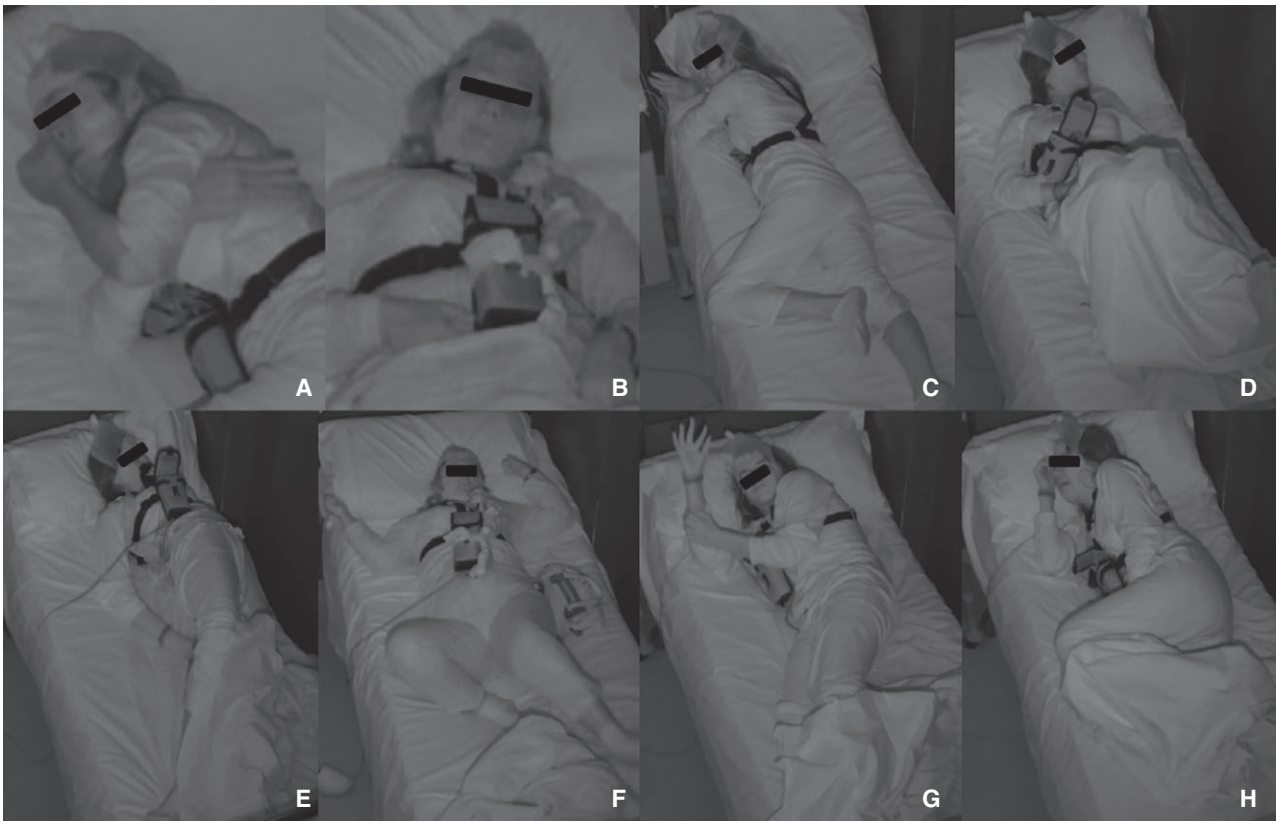

Figure 1.
Recurring motor patterns during sleep: (**A**) hand-face movement; (**B**) oroalimentary automatism; (**C**) limb dystonia; (**D**) head rotation; (**E**) pelvic movement; (**F**) straightening movement; (**G**) gestural automatism; and (**H**) other movements.

The EEG features were standardized in a few recurring patterns: rhythmic spikes or sharp waves, rhythmic or monomorphic slow activity (theta or delta), polymorphic slow activity (theta or delta), low voltage fast activity (LVFA) and EEG flattening[27] and other not specified activities. The EEG recordings were analyzed considering the following time frames: 2 min before the motor event and during each motor event. A proportion was calculated between the number of movements and the associated EEG abnormalities in patients and controls so that the comparisons were made between the number of movements preceded by EEG abnormalities in patients and controls. Moreover, a comparison was made between the movements with and without EEG abnormalities in the group of patients.

Statistical analysis

Statistical analysis was performed using STATA 12 software packages (version 12.0, College Station, TX, U.S.A.). Qualitative variables were described as percentages and quantitative variables as mean ± standard deviation (SD). The data were examined for normality using Shapiro-Wilk test. Pearson chi-square test (χ^2) or one-tailed Fisher's exact test were used to study categorical variables; comparisons between means were performed using unpaired *t*-test for parametric data and Mann-Whitney test for nonparametric data. Comparisons in the group of patients were made using Wilcoxon matched-pairs signed-ranks test. A $p < 0.05$ was set as the level of significance.

Results

A total number of 30 subjects was enrolled in the study: 15 symptomatic MTLE and 15 healthy controls. The main clinical characteristics of patients and controls are summarized in Table 1.

Table 1. Clinical characteristics of patients and controls

Variable	Patients (n = 15)	Controls (n = 15)
Age (years), mean ± SD	31.8 ± 14.9	32.8 ± 11.2
Sex M, n (%)	8 (53.3)	6 (40)
Family history of epilepsy, n (%)	1 (6.6)	2 (13.3)
Cognitive deficits, n (%)	11 (73.3)	0

n, number; SD, standard deviation; M, male.

Baseline characteristics of the two groups did not differ significantly except for the presence of cognitive deficits found in patients (seven patients with memory deficits and four patients with slight mental retardation) and not in controls ($p < 0.001$). The neurologic examination was normal in all patients and controls. The different types of seizures reported by the patients are shown in Table 2. Most of them presented behavioral arrest and automatisms (86.7%), with six patients manifesting an epigastric aura (40.0%), and generalized tonic–clonic seizures reported by six patients (40.0%). All of the patients and none of the controls were taking antiepileptic drugs (AEDs). The most used AEDs were carbamazepine, taken by seven patients (46.7%) and levetiracetam and topiramate, taken by five patients (33.3%). The AEDs taken by the patients and their MRI findings are shown in Table 2. Seven patients showed mesial temporal lobe sclerosis, one patient had cortical dysplasia in the mesial temporal lobe, one patient had a temporal glioma, and five patients showed other lesions located in the mesial temporal lobe. More specifically, one patient had hyperintensities located in both mesial temporal lobes; one patient had a hyperintense lesion located in a sulcus of the right temporal cortex; one patient showed asymmetry of the temporal horns with an increased volume and hyperintensity of the left amygdala; one patient had a hyperintense signal, due to gliosis, in the left temporal lobe and a hyperintense signal in the left insula. One patient presented only functional alterations, demonstrated by fluorodeoxyglucose positron emission tomography (FDG-PET) in both mesial temporal lobes; therefore, his epilepsy was considered of unknown etiology. The total number of MMEs was higher in patients (851) than in controls (225). The analysis of all the movements during nocturnal sleep revealed significant differences between patients and controls, with a higher number of patients showing oroalimentary automatisms, limb dystonia, straightening movements, and gestural automatisms, as shown in Table 3. Moreover, the comparison of the mean number of movements performed by patients and controls, considering only the actual number of subjects performing the movements, revealed significant differences with a mean total number of MMEs of 56.7 ± 39.2 in patients and 15 ± 6.1 in controls ($p < 0.001$), as well as regard to other single types of movements, such as left hand-face movement (7.9 ± 5.2 vs. 3.1 ± 2.7; $p = 0.002$) and pelvic movements (19.6 ± 24.7 vs. 1.6 ± 0.9; $p < 0.001$). These MMEs occurred more frequently during stage N2 of sleep both in patients (76.4%) and controls (62.8%). The proportion of movements preceded by EEG abnormalities was significantly higher in patients than controls (57.8 ± 35.9 vs. 16.6 ± 13.4; $p < 0.001$). The specific EEG characteristics associated with movements in patients and controls are shown in Table 4. Nonetheless, both in patients and controls, MMEs occurred more frequently without any EEG correlation.

Discussion

Epilepsy is strictly related to sleep, as epileptic seizures tend to occur more frequently during nocturnal sleep than in wakefulness. Moreover, patients with focal epilepsy usually show EEG paroxysmal discharges during sleep more frequently than during waking state with the greatest number of paroxysms occurring in the light sleep stages. These findings seem to suggest a close functional relationship between the neuronal mechanism underlying sleep and that of triggering epileptic seizures.[2,3]

To date, only one systematic description has been made about the clinical expression of TLE in relation to sleep.[20] However, many other reports supported the existence of a functional link between TLE and sleep.[16–18,24,28,29] Moreover, the electroencephalographic features of TLE during sleep have been described extensively.[30–32]

Nevertheless, to our knowledge, the presence of MMEs during sleep in patients with TLE and their correlation with EEG epileptiform discharges has never been explored until now.

The results of our study confirm the presence of stereotyped nocturnal motor events that are significantly more frequent in patients with MTLE than in healthy controls. In particular, we demonstrated a higher frequency of oroalimentary automatisms, limb dystonia, gestural automatisms, and straightening movements in the group of patients. Moreover, among patients who actually performed the movements, other patterns were found to be significantly more frequent in patients than controls: left hand-face and pelvic movements. Furthermore, we demonstrated that these MMEs during sleep in patients with MTLE could be associated with epileptiform EEG abnormalities.

These data can lead to various physiopathologic interpretations. The higher frequency of motor events in patients compared to controls, the stereotyped features of the movements, and their frequent correlation with a recurring EEG pattern weigh against their physiologic nature, suggesting an epileptic origin of these motor patterns.[11] However, the causative mechanism of these MMEs remains speculative, since they are present, with the same characteristics, also in normal subjects, mainly in relation to periodic fluctuations of sleep and arousal. In fact, periodic arousals during sleep, represented by the well-known cyclic alternating pattern (CAP), can be linked to the appearance of a wide spectrum of different physiologic and pathologic manifestations during sleep.[33] Therefore, it can be hypothesized that the occurrence of MMEs during sleep in patients with MTLE could be due to the higher recurrence of arousals in this group, linked to a peculiar feature of these patients whose nocturnal sleep has been reported to show an increased instability and fragmentation.[30,34,35] Moreover, the role of AEDs in inducing arousals cannot be ruled out in the group of patients. It is in fact well known that many AEDs have a destabilizing effect on sleep, possibly leading to an increased number of

Table 2. Main clinical and neuroradiologic characteristics of the group of patients

Patient's gender/age	Type of seizure				AEDs intake										MRI characteristics			
	Epigastric aura	Behavioral arrest and automatisms	GTCS	Other	CBZ	CLB	CNZ	LEV	LCM	LTG	OXC	PHT	PB	TPM	VPA	ZNS	Mesial sclerosis	Dysplasia	Glioma	Other lesions[b]
M/25	+	+	+		+					+					+		+			
M/25	+	+	+					+				+		+						
M/42	+	+	+					+						+	+		+			
M/19		+	+		+	+		+											+	
M/20				+[a]				+										+		
M/14	+	+			+												+			
F/37		+			+								+				+			
F/30		+			+											+	+			
F/42		+		+[a]	+				+											+
F/16		+												+						+
F/17	+	+			+												+			
F/51			+											+			+			
M/52	+	+					+				+									+
M/62		+	+					+	+						+					+
F/26		+					+							+						+

M, male; F, female; GTCS, generalized tonic–clonic seizures; AEDs, antiepileptic drugs; MRI, magnetic resonance imaging; CBZ, carbamazepine; CLB, clobazam; CNZ, clonazepam; LEV, levetiracetam; LCM, lacosamide; LTG, lamotrigine; OXC, oxcarbazepine; PHT, phenytoin; PB, phenobarbital; TPM, topiramate; VPA, valproic acid; ZNS, zonisamide.

[a]Nocturnal wandering in patient 5 and déjà-vu in patient 9.

[b]For the complete description, see text.

Table 3. Movements during sleep in patients and controls

Type of movement	Patients	Controls	p-Value†
All movements	15	15	1
Right hand-face	13	11	0.33
Left hand-face	15	13	0.24
Oroalimentary automatisms	12	4	0.005*
Right limb dystonia	7	0	0.003*
Left limb dystonia	7	0	0.003*
Head rotation	13	11	0.33
Pelvic movements	11	8	0.22
Straightening movements	15	11	0.05*
Gestural automatisms	8	2	0.02*
Other movements	7	6	0.5

*Statistically significant.
†Pearson's chi-square test or Fisher's exact test.

Table 4. Proportion of movements associated with EEG abnormalities during sleep in patients and controls

	Percentage of movements associated with EEG abnormalities		
EEG pattern[a]	Patients	Controls	p-Value†
Before the movement	38.5 ± 24.4	16.6 ± 13.4	0.006*
Rhythmic spikes or sharp waves	12.3 ± 18.9	0	<0.001*
Rhythmic or monomorphic slow activity	4.8 ± 17	0	0.07
Polymorphic slow activity	1 ± 3.9	0	0.15
LVFA and EEG flattening	20.4 ± 13.0	16.6 ± 13.4	0.33
During the movement	19.3 ± 21.7	0	<0.001*
Rhythmic spikes or sharp waves	0.1 ± 0.5	0	0.37
Rhythmic or monomorphic slow activity	8.4 ± 16.3	0	<0.001*
Polymorphic slow activity	5.9 ± 9.7	0	0.003*
Other EEG activities	4.9 ± 18	0	0.07

LVFA, low voltage fast activity.
*Statistically significant.
†Mann-Whitney test.
[a]Values are percentages, mean ± SD.

arousals.[36,37] However, the EEG pattern shared by seizures and arousal, represented by EEG flattening, has been demonstrated to be present in both patients and controls without significant differences. Therefore, we cannot exclude in patients that some recordings of EEG flattening could be related to arousals. However, the presence only in the group of patients of other EEG epileptiform typical patterns in close relationship with the movements supports their epileptic nature.

Nevertheless, it cannot be excluded the incidental inclusion, in the group of patients, of interictal epileptiform discharges not related with the analyzed events. In this regard, in order to reduce this possibility, we included in the analysis only EEG patterns usually considered to be "paroxysmal."[27] Moreover, we analyzed only the EEG abnormalities occurring during or immediately before the motor events. Our choice to select a time frame of 2 min before the movement was based on a previous finding, demonstrating the occurrence of facial wiping behaviors as postictal manifestations, usually occurring within 2 min after the seizure.[25] Thus the hypothesis that these motors patterns could be an expression of visceral sensations frequently reported by MTLE patients cannot be ruled out. In fact, for example, all the movements directed to the face and the nose could reflect a reaction triggered by increased upper highway secretions, as well as all the gestural automatisms directed to the abdominal region could be linked to the presence of "epigastric sensation." Consequently another possible interpretation for these motor patterns can be represented by the recurrence, in MTLE patients, of postictal motor manifestations triggered by epileptiform discharges.[25]

Nevertheless, among patients, a higher number of movements seem to occur without any EEG pathologic correlation, suggesting that only a part of them can be considered linked to epilepsy itself. However, one cannot overlook the occurrence of paroxysmal motor events even in the absence of any visible scalp EEG abnormality, as frequently found in clinical practice.

Another important consideration is that not all the motor patterns found in our patients are typical manifestations of temporal lobe seizures. Except for oroalimentary automatisms, limb dystonia, and gestural automatisms, the other patterns found, such as straightening and pelvic movements are usually associated with a frontal lobe origin. However, it cannot be excluded either a propagation of the discharge from the temporal to the frontal lobe, explaining such motor features, or the disinhibition, by the epileptiform discharges, of phylogenetically ancient motor behaviors, with the activation of the so-called central pattern generators.[38]

In agreement with previous literature findings, we observed that these minor events occurred more frequently during stage 2 non–rapid eye movement (NREM) (N2) sleep, in accordance with the higher expression of interictal and ictal EEG alterations that usually occur in this stage of sleep.[31,39]

In conclusion, the results of our study show the existence of MMEs during sleep in patients with MTLE. Our findings suggest an epileptic origin of these events that seem to share similar significance of those seen in SHE. However, to clarify the exact nature of these phenomena, further studies with larger samples are needed. Until now, these events have been overlooked, with a negative impact on the management of these patients. The results of our work demonstrate that the study of nocturnal sleep in patients with MTLE could be useful in helping clinicians in the diagnostic and therapeutic workup of these patients.

Disclosure

None of the authors has any conflict of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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