



Temporal Lobe Epilepsy with Bitemporal Interictal Epileptiform Discharges: Effects of Sleep and Wakefulness

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Abstract

Introduction. Independent bitemporal interictal discharges are often found in patients with temporal lobe epilepsy. The likelihood of registering epileptiform activity (EA) is higher during sleep. Assessment of bitemporal interictal epileptiform discharges (BIEDs) with various discharge predominance ratio is used for presurgical evaluation of epilepsy patients and prediction of surgical outcomes.

Our **objective** was to determine the predominant side (PS) in patients with bitemporal epilepsy using the incidence of epileptiform discharges for each sleep stage.

Materials and methods. We analyzed 45 recordings of 10–24 h long-term video-EEG monitoring (LTM) in patients with bitemporal EA. For each recording, the total incidence of EA (IEA) and EA incidence for wakefulness and for each sleep stage were calculated individually. We also assessed the discharge predominance index (DPI) as a ratio of IEA in the predominant and contralateral sides for the entire recording and for each sleep stage.

Results. We observed an IEA increase with sleep deepening, with maximum values observed during N2 and N3 sleep stages. The minimum IEA values were recorded during REM sleep; nevertheless, most of the REM sleep discharges were detected on the PS. DPI values were the highest and the most stable during N2 and N3 stages.

Conclusion. The findings of our study demonstrate an increase in DPI values with non-rapid eye movement (NREM) sleep deepening in patients with bitemporal localization of EA. Despite the protective effects of REM sleep (i.e., reducing the likelihood of EA), it may be pivotal in lateralization of EA in patients with BIEDs. The PS is generally determined by a higher DPI during N2 and N3 stages.

Keywords: temporal lobe epilepsy with bitemporal discharges; sleep; electroencephalography; sleep stages

Ethics approval. All patients provided their voluntary informed consent to participate in the study. The study protocol was approved by the Ethics Committee of the Research Center of Neurology (Protocol No. 11-6/22, dated 21 December 2022).

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Височная эпилепсия с битемпоральными интериктальными разрядами: влияние сна и бодрствования

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Аннотация

Введение. У пациентов с височной эпилепсией нередко выявляются независимые битемпоральные разряды в межприступном периоде. Вероятность регистрации эпилептиформной активности (ЭА) увеличивается во сне. Наличие битемпоральных интериктальных эпилептиформных разрядов с различным соотношением количества разрядов по сторонам учитывается для определения исхода хирургического лечения.

Цель: на основании расчёта индекса эпилептиформных разрядов для каждой стадии сна выявить доминирующую сторону у пациентов с битемпоральной эпилепсией.

Материалы и методы. В исследование были включены 45 записей видео-ЭЭГ-мониторинга длительностью 10–24 ч у пациентов с битемпоральной ЭА. Для каждой записи рассчитывали общий индекс ЭА и индекс ЭА для бодрствования и каждой стадии сна отдельно. Также определяли индекс доминирования разрядов (ИДР) в процентах как соотношение разрядов на доминирующей и контралатеральной сторонах для всей записи и отдельно для каждой стадии.

Результаты. Отмечено увеличение индекса ЭА по мере углубления сна, максимальные значения выявлены в стадиях сна N2 и N3. Минимальное значение индекса ЭА было в фазе REM-сна, тем не менее в большинстве случаев разряды в REM-фазе выявлялись на доминирующей стороне. ИДР был наиболее высоким и стабильным в стадиях N2 и N3.

Заключение. Результаты исследования пациентов с битемпоральной локализацией ЭА свидетельствуют о возрастании ИДР по мере увеличения глубины не-REM-сна. Несмотря на то что REM-сон обладает протективными свойствами, снижая вероятность появления ЭА, для пациентов с битемпоральными интериктальными эпилептиформными разрядами он может иметь латерализующее значение. Доминирующая сторона в значительной степени определяется высоким значением ИДР в стадиях N2 и N3.

Ключевые слова: височная эпилепсия с битемпоральными разрядами; сон; электроэнцефалография; стадии сна

Этическое утверждение. Исследование проводилось при добровольном информированном согласии пациентов. Проведение исследования было одобрено локальным этическим комитетом при ФГБНУ «Научный центр неврологии» (протокол № 11-6/22 от 21.12.2022).

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Introduction

Focal temporal lobe epilepsy (TLE) is the most common form of structural epilepsy and one of the most common neurological diseases, diagnosed in 1/3 of patients with epilepsy [1]. In 30% of cases, it is drug-resistant, so the patients become candidates for a surgical treatment [2]. Long-lasting focal TLE increases the probability of development of secondary

epileptogenic focus in the contralateral cerebral hemisphere. EEG data demonstrate that the prevalence of bitemporal interictal epileptiform discharges (BIEDs) in patients with focal TLE can reach up to 60% [3, 4].

When both temporal lobes are involved, the predominance of epileptogenic focus is challenging to determine, and bilateral resection options are limited due to the risk of the Kluver–

Bucy syndrome. The search for clinical and neurophysiological markers of the predominant side (PS) in TLE patients with bitemporal discharges is of particular interest to achieve favorable surgical outcomes. It has been previously shown that the effectiveness of surgical treatment in patients with BIEDs correlates with higher discharge predominance index (DPI) on one side, and the most favorable outcomes were observed in patients with over 80–90% of unilateral discharges [5, 6].

In patients with focal TLE, the likelihood to find epileptiform activity (EA) is largely determined by the level of wakefulness and depth of sleep [7, 8]. EA is more often detected during sleep, especially in slow-wave sleep. Thus, an absolute count of discharges is insufficient without considering the duration of wakefulness and individual stages of sleep. To this end, incidence of epileptiform activity (IEA) may be employed, which is calculated as the number of discharges occurring over a specified time interval (e.g., 1 h) during which the patient has been either awake or asleep. So, the absolute count of discharges without indicating wakefulness or sleep stage at the time of EEG recording may not suffice.

The **objective** of the study was to assess the variability of DPI calculated with IEA values depending on the level of wakefulness and sleep depth in TLE patients with bitemporal discharges.

Materials and methods

Out of a total of 2086 patients who underwent long-term video-EEG monitoring (LTM) in the laboratory of the Research Center of Neurology between February 2018 and February 2024 [9], the recordings of 10–24 h LTM in 1063 patients with temporal lobe localization of EA were selected. BIED were registered in 203 cases. To identify the structural causes of epilepsy, brain magnetic resonance imaging (MRI) was performed using Magnetom Prisma 3T (Siemens Healthineers, Germany).

Inclusion criteria [9]:

- 1) bitemporal EA;
- 2) recording of all stages of sleep and wakefulness;
- 3) manual count of epileptiform discharges without using the algorithm of automatic discharge detection;

- 4) total number of BIEDs ≥ 10 ;
- 5) brain MRI performed according to the HARNESS-MRI protocol using 3T MR scanner.

Non-inclusion and exclusion criteria [9]:

- 1) recordings with extratemporal EA;
- 2) recordings with ictal patterns, due to their possible effects on IEA;
- 3) recordings with more than 2000 discharges due to their challenging manual labeling;
- 4) DPI $< 60\%$.

We analyzed 45 recordings of 20 females and 25 males aged 25–67 years (median 44.6 years). The disease duration at the time of admission ranged from 6 months to 43 years (median 29.8 years), and the age of epilepsy onset ranged from 4 months to 67 years (median 14.9 years). We performed surface EEG with scalp electrodes according to the international 10–20 system, with additional inferior temporal chain (F9, F10, T9, T10, P9, P10) and with simultaneous one-channel electrocardiogram recording.[9] Equipment used for the recording: Xltek Brain Monitor (Natus, USA) and BE Plus LTM amplifier (EBNeuro, Italy). At the beginning of study and after morning awakening, patients underwent activation procedures with eye closure, intermittent photic stimulation, and hyperventilation for 5 min.

In accordance with the recommendations of the American Association of Sleep Medicine (2017) [10], sleep scoring was done manually at 30 second epochs. REM sleep was scored based on oculomotor artifacts in frontal leads (under electrodes Fp1–F7, Fp2–F8), myographic artifacts in EEG channels, and EEG waveforms specific for REM sleep. We used traditional designations for sleep and wakefulness stages: N1 – sleep stage 1; N2 – sleep stage 2; N3 – sleep stage 3 (slow-wave sleep); REM – REM sleep; Wake – wakefulness [9]. A sample hypnogram with EA labels is presented in Figure 1.

The number of BIEDs during the recording ranged from 11 to 1920 discharges (median 299.6 discharges). For each recording, after building a hypnogram with BIED labels, we assessed the total IEA (the ratio of total number of discharges to the duration of recording in hours), IEA for wakefulness and each



Fig. 1. Hypnogram with EA labels in the right (top row) and left (bottom row) temporal regions.

sleep stage (the ratio of number of BIEDs to the duration of stage in hours) [9]. The side with the predominance of discharges during the entire recording was defined as the PS, the opposite side was defined as the contralateral side (CS).

The DPI (%) for the entire recording and for wakefulness and each sleep stage was calculated using the formula: (number of discharges on PS)/(number of discharges on both sides) × 100. The amplitude of discharges was measured in the average reference montage. The highest discharge amplitude on each side was selected regardless of the level of vigilance (sleep and wakefulness).

To evaluate the DPI trends associated with specific sleep stages and wakefulness, additional normalization was performed via calculation of relative DPI for each stage on the PS (the ratio of the DPI of each stage to the total DPI).

When EEG data were compared with brain MRI findings according to the HARNESS-MRI protocol, potentially epileptogenic findings were identified in 17 cases: 9 cases of hippocampal sclerosis; 2 benign tumors associated with long-term epilepsy (LEAT); 3 meningiomas; 2 cases of temporal encephalocele; 1 cerebral hemiatrophy. Non-specific changes (cerebral microangiopathy, poststroke changes, and venous anomalies) not corresponding to EA localization were found in 23 cases. In 5 cases focal and diffuse changes in the brain were absent.

Results

In 21 patients the discharges predominated in the left temporal region and 24 patients had a right temporal predominance. Analysis of the BIED amplitude revealed that in 32 (71.1%) cases the maximum amplitude of discharges corresponded to PS. Bitemporal slowing was observed in 27 EEG recordings, unilateral slowing was detected in 13 recordings (11 out of 13 recordings on PS), and in 5 recordings slowing was absent.

The distribution of EA in temporal regions across wakefulness and sleep stages is presented in Table 1. During wakefulness epileptiform discharges were found in 33 (73.3%) cases.

Table 1. Distribution of EA in temporal regions across sleep stages and wakefulness, n (%)

Sleep stage	Recordings with EA	Recordings with BIEDs	Recordings with EA on PS	Recordings with EA on CS
Wakefulness	33 (73,3%)	17 (37,8%)	32 (71,1%)	18 (40,0%)
N1	25 (55,6%)	11 (24,4%)	22 (48,9%)	14 (30,4%)
N2	45 (100,0%)	42 (93,3%)	45 (100,0%)	42 (93,3%)
N3	44 (97,8%)	36 (80,0%)	44 (97,8%)	36 (80,0%)
REM	19 (42,2%)	7 (15,5%)	18 (40,0%)	8 (17,8%)

In 32 (71.1%) cases EA was detected on PS and in 18 (40%) it was found on CS (Table 1).

Table 2 presents the IEA ratios for the sleep stages, where BIEDs were detected, as well as the distribution of EA across wakefulness and sleep stages. The patients, whose recordings showed no significant differences in IEA values between two sides, were referred to a separate group. The PS/CS discharge ratios > 40% and < 60% were considered approximately equal.

In one MR-negative patient, EA was detected on the CS (Table 2). In N1 sleep stage, EA was detected in 25 (55.6%) recordings: 22 (48.9%) with EA on PS and 14 (30.4%) on CS (Table 1), while in 3 cases it was found on CS only (Table 2).

In sleep stage N2, EA was detected in 45 (100%) recordings: 45 (100%) with EA on PS and 42 (93.3%) on CS. In sleep stage N3, epileptiform discharges were found in 44 (97.8%) recordings on PS and in 36 (80%) recordings on CS. Only in one patient's recording EA was not found in N3, although detected in stage N2 (Table 1).

In the majority of recordings, BIEDs had the highest IEA during N3 stage (43 recordings; 95.5%), including 26 (57.8%)

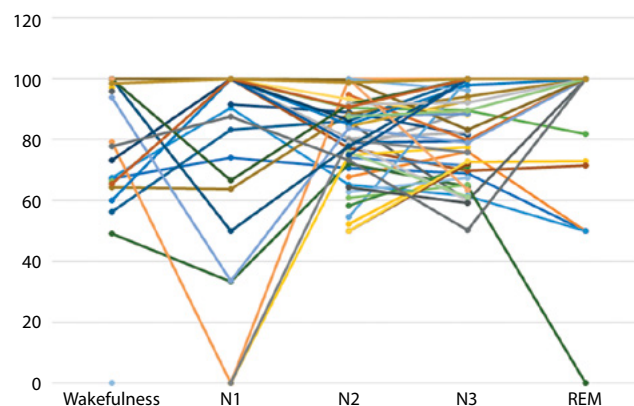


Fig. 2. Distribution of DPI values (%). The X-axis presents sleep and wakefulness stages, the Y-axis – normalized DPI values. DPI values < 50% indicate the predominance of discharges on CS.

Table 2. Distribution of EA across sleep stages and wakefulness, *n* (%)

Stage	EA on PS only	EA on CS only	Higher IEA on PS*	Higher IEA on CS	IEA with no significant differences between sides
Wakefulness	15 (33,3%)	1 (2,2%)	13/17 (76,5%)	2 (4,4%)	3 (6,7%)
N1	11 (24,4%)	3 (6,7%)	7/11 (63,6%)	5 (11,1%)	2 (4,4%)
N2	3 (6,7%)	0	37/42 (88,1%)	0	5 (11,1%)
N3	8 (17,8%)	0	33/36 (91,7%)	0	3 (6,7%)
REM	11 (24,4%)	1 (2,2%)	3/7 (42,9%)	1 (2,2%)	4 (8,9%)

Note. * — % of recordings with BIEDs during sleep or wakefulness.

recordings with BIEDs in both temporal regions. In 7 (15.5%) cases, EA with maximal IEA was recorded on the CS and in 10 (22.2%) cases – on the PS. In REM sleep EA was found in 19 (42.2%) recordings: in 18 (40%) cases on the PS and, in 8 (17.8%) cases on the CS, while in one recording EA was detected exclusively on the CS (Table 2).

We analyzed wakefulness and sleep stages with maximum IEA registered on PS during this study. The highest values were detected in the period of slow-wave sleep (N3 stage), amounting to 36 (80%) recordings. In 3 cases the leading stage was N2, in 5 cases – N1, in 1 case the maximum IEA was recorded during wakefulness. No cases with predominance of EA in REM sleep were detected.

Figure 2 shows the distribution of DPI values across wakefulness and each sleep stage. Maximum density of DPI values in N2 and N3 stages, where DPI was always > 50% (i.e., discharge predominance on PS) was of particular interest.

Discussion

The results of our study demonstrated that the highest IEA values were characteristic of the slow-wave sleep stage in the temporal region of PSDH. N2 sleep stage was the most significant in recordings with no EA detected during the delta sleep. A comparison of sleep stages revealed that the maximum number of discharges in N3 stage were found in 80% of recordings for PS and in 71.3% for CS. The IEA increased with sleep deepening (maximum values were detected in N2 and N3 stages). The minimum IEA was obtained in REM sleep.

Earlier studies also showed that the highest IEA values were characteristic of the delta sleep [8, 9, 11–14]. The distinctive features of our study are the analysis of patients exclusively with bitemporal discharges, manual labeling of discharges throughout the entire recording, and count of discharges by each sleep stage. The PS was determined by the total number of discharges on the left or right side, whereas, for example, Z. Clemens et al. determined it by the seizure onset side [13]. In other studies, the discharge count was often performed on fragments with different duration (5–20 min for each stage of

sleep and wakefulness). High IEA in N2 and N3 stages can be explained by a high degree of neural synchronization in the cerebral cortex [15–17].

It is believed that EA in REM sleep is important for seizure-onset zone localization in TLE patients [18]. Our data partly confirm this statement. EA was recorded on PS in 19 (42.2%) cases and on CS in 8 (17.8%) cases. In 3 cases, IEA was higher on PS, while in 4 cases there was no significant difference between sides. In 1 case EA was found only in the contralateral temporal region (Table 2). There are conflicting opinions on this hypothesis. S. Singh et al. showed a lower localizing value of REM sleep compared to non-REM sleep when quantifying EA [14]. At the same time, it remains certain that REM sleep as a part of healthy sleep structure has protective effects, inhibiting not only EA but also epileptic seizures [19, 20].

While performing presurgical evaluation of patients with a drug-resistant focal TLE, one should take into account the lateralizing and localizing seizure symptoms. The data of ictal and interictal EEG and neuroimaging should correspond to each other [21]. However, in patients with BIEDs, which may occur due to secondary epileptogenesis often associated with a long-lasting epilepsy, it is necessary to assess IEA ratio in the temporal regions on both sides. Correlation between this ratio and surgical outcomes has been previously evaluated [5, 6]. The most favorable outcomes were achieved with a ratio of > 80% of discharges on the PS.

Various studies demonstrated that the prevalence of BIEDs varies in a wide range, from 21% [22] to 61% [3]. High variability of these data is primarily explained by the duration of recordings. The low prevalence values are associated with the relatively short duration of recordings. For example, in study [22], the duration of recording was ≤ 2 h. In study [3], on the contrary, patients with only unitemporal discharges detected by a routine EEG were initially selected. These patients were subsequently monitored daily and BIEDs were also detected in 61% of recordings.

The results of our study demonstrate not only an increase in likelihood of finding EA with the sleep deepening, but

also considerable variability in DPI values during wakefulness and in the light sleep. DPI in these stages is unreliable and may be misleading, whereas it is the most stable in a deep sleep.

The results of this study justify the need for LTM with delta sleep assessment in patients with suspected focal epilepsy, because standard EEG is a short-term screening method of diagnosis, and daytime EEG monitoring of no more than 4 h does not always allow reaching a deep sleep stage. In structural forms of focal epilepsy, it is also necessary to count discharges in temporal regions and compare these data with MRI epilepsy protocol findings to substantiate the need for surgical treatment and to predict favorable surgical outcomes.

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Conclusion

The findings of our study demonstrate an increase in DPI values with non-rapid eye movement (NREM) sleep deepening in patients with BIEDs. The ratio of IEA in a deep sleep is the most reliable indicator to determine the PS. Although REM sleep has protective properties reducing the likelihood of EA occurrence, it has significant localizing value for patients with BIEDs. In studies with short-term EEG recordings, the necessary depth of sleep is often not achieved, therefore reducing their diagnostic value. Count of epileptiform discharges in the right and left temporal regions is necessary to substantiate the need for surgical treatment and to predict favorable surgical outcomes in patients with focal TLE.

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