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Original Research

Inter-Hemispheric Asymmetry and Intra-Hemispheric Discrepancy of Electroencephalographic Activities in Patients With Idiopathic Rapid Eye Movement Sleep Behavior Disorder: An Exploratory Study

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Abstract

Background: In idiopathic rapid eye movement (REM) sleep behavior disorder (iRBD) patients, electroencephalogram (EEG) activities during REM sleep in some cortical regions are different from those in normal controls. This study aimed to examine inter-hemispheric asymmetry and intra-hemispheric EEG discrepancy in iRBD patients and normal controls during REM sleep. **Methods**: Polysomnographic recordings were carried out on 15 iRBD patients and 15 normal controls. The inter-hemispheric asymmetry and intra-hemispheric differences of EEG activities in the iRBD patients were compared with those in the normal controls during REM sleep. **Results**: During REM sleep, most of the powers of theta, alpha, sigma, beta, and gamma waves in the right cerebral hemisphere were significantly greater than those in the left cerebral hemisphere in both the iRBD patients and normal controls. The inter-hemispheric asymmetry was significantly larger in the central and occipital regions and generally smaller in the frontal region in the iRBD patients compared with the normal controls. In the iRBD patients and normal controls, the powers of theta, alpha, beta, and gamma bands during tonic and phasic REM sleep were highest in the frontal cortical region, followed by the central cortical region, and lowest in the occipital cortical region; sigma power during phasic, but not tonic, REM sleep fully followed this rule. **Conclusion**: In iRBD patients EEG activities are unevenly distributed, with an altered inter-hemispheric asymmetry that might be associated with changed bilateral neuronal differences compared with normal controls.

Keywords: electroencephalography; functional laterality; power spectral analysis; REM sleep behavior disorder

1. Introduction

Rapid eye movement sleep behavior disorder (RBD) is a parasomnia with a loss of muscle atonia accompanied by dream enactment behaviors during rapid eye movement (REM) sleep [1]. RBD predominantly occurs in males, and the onset age in most patients is 40–70 years old [2]. Violent behaviors during dreams range from mild physical convulsions, minor limb jerking and twitching to complex violent movements such as hitting walls and kicking legs, which can cause serious physical injuries to the patients themselves and/or their bedmates during REM sleep [2–4].

REM sleep can be further divided into phasic and tonic REM phases based on the presence absence of REMs or not and during phasic REM sleep, motor-behavioral episodes in RBD patients happen more frequently than during tonic REM sleep [5].

A notable clinical form of RBD with a relationship to REM sleep is idiopathic RBD (iRBD), which refers to RBD that occurs without any other obviously associated neurological diseases or triggers, such as the use of antidepressants [6]. It has been reported that approximately 80% iRBD patients develop neurodegenerative diseases within 10 years [7]. iRBD is also considered a strong predictor and an early sign for neurodegenerative alpha-synucleinopathies, such as Lewy body dementia (DLB), Parkinson's disease (PD) and multiple system atrophy [8, 9], although the pathophysiologic mechanisms underlying iRBD are still unclear. iRBD patients might have impairments in memory, executive function, and visuospatial ability [10–13], and up to 50% of iRBD patients have mild cognitive impairments (MCI) [11].

Compared to normal controls, iRBD patients show a higher metabolism in the premotor cortex and hippocampus and a lower metabolism in the occipital region [14], suggesting that a discrepancy in electroencephalogram (EEG) activities between different cortical regions within the same hemisphere may be exhibited in the iRBD patients. Inter-hemispheric differences in EEG activities in iRBD patients might differ from those in normal controls during REM

sleep. Furthermore, the cerebral hemispheres are not structurally or functionally identical in both sides [15–17]. During REM sleep, for example, EEG activity in the right side is more active than in the left [15,18]. In addition, it has been reported that the difference in alpha power between the left and right frontal cortical regions (frontal alpha asymmetry, FAA) might predict the level of anger in dream during REM sleep, and that people with a greater alpha power in the right frontal cortical region might be less able to control intense emotional states in dream, such as anger [19]. Since iRBD patients are often reported to have violent dreams, it is possible that iRBD patients might have a greater alpha asymmetry in EEG activities than normal controls. A previous study has shown that the brain networks of iRBD patients during the eyes-open resting state display higher topological properties in the left hemisphere when analyzed at the hemispheric level [20].

Since these various findings suggest both intra-hemispheric and inter-hemispheric EEG differences during REM sleep might be dissimilar between iRBD patients and normal controls, the study aimed to compare inter-hemispheric asymmetry and intra-hemispheric discrepancy in EEG activities during phasic and tonic REM sleep between iRBD patients and normal controls.

2. Materials and Methods

2.1 Participants

A total of 40 subjects, including 20 iRBD patients and 20 normal controls, were recruited and clinical examined at the Jiangxi Provincial People's Hospital. A total of 5 iRBD patients and 5 controls were eliminated due to failure to meet the inclusion criteria or having conditions in the exclusion criteria (see below). At the end, a total of 30 participants were enrolled in this study, including 15 iRBD patients and 15 normal controls.

The patients were recruited if they were 30–70 years old males diagnosed with iRBD based on the criteria defined in the International Classification of Sleep Disorders, Third Edition (ICSD-3) [1], and age-matched male normal controls were also included. Inclusion criteria included: (1) no insomnia, bruxism, narcolepsy, restless leg syndrome and any other sleep disorders; (2) no intake of anxiolytic, antidepressant or psychiatric medications within a month prior to polysomnographic (PSG) examination. The subjects were not allowed to take stimulants such as caffeinated beverages (e.g., tea and coffee) within 24 hours prior to the experiments. The patients and normal controls with one or more the following conditions were excluded: (1) apnea hypopnea index (AHI) $\geq$15; (2) primary and secondary lung diseases; (3) hypertension (Stage II or higher) and heart diseases with Functional Class II or worse specifically defined by the New York Heart Association; (4) poorly controlled diabetes mellitus with complications; (5) mental and neurological diseases; (6) brain injury; (7) alcohol or other substance abuse.

All iRBD patients were newly diagnosed during this assessment period and had not previously received any treatment for iRBD. The study included four patients with hypertension (Stage I), and three patients with well-controlled type II diabetes mellitus without complications. Patients with type II diabetes mellitus were primarily treated with metformin and none of the patients were taking medications known to significantly affect sleep architecture or EEG activity.

2.2 Polysomnographic Recording

Electroencephalographic (O1-A2, O2-A1, C3-A2, C4-A1, F3-A2, F4-A1), electrooculographic (EOG), and electrocardiographic (ECG) activities, as well as electromyographic (EMG) activities from bilateral mylohyoid, masseter, tibialis anterior and gastrocnemius muscles, as well as flexor and extensor radialis were simultaneously recorded with the Grael 4K PSG:EEG recording system (Compumedics Limited, Abbotsford, Victoria, Australia) as previously reported [21,22]. In addition, the participants' respiration was also recorded via a nasal pressure-monitoring cannula, an airflow thermistor, as well as thoracic and abdominal piezoelectric belts. Peripheral vascular oxygen saturation (SpO_2) was recorded with a pulse oximetry. In addition, body position was monitored by body position sensors, and snoring was recorded by snore sensors. All participants were monitored with audio-visual recorders during PSG recordings.

2.3 Data Analysis

All PSG data were initially analyzed using the Pro Fusion PSG 4 Software (Compumedics Limited, Abbotsford, Victoria, Australia) based on the standards developed by the American Academy of Sleep Medicine [23], and then analyzed with the Brainstorm program (Version 2, GNU GPLv2, McGill University, Montreal, QC, Canada). The EEG signals were digitized and filtered with a low- and a high-pass filter set at 70 Hz and 0.1 Hz, respectively [4], and a 50 Hz notch filter.

REM sleep was divided further into phasic and tonic REM phases based on the presence or absence of REMs, which were described as irregular, sharply peaked and conjugate eye movements recorded in the EOG derivations, with an initial deflection lasting shorter than 0.5 seconds [4,23]. Phasic REM epochs were defined as a 4-second segment with $\geq$2 consecutive REMs, while tonic REM epochs were defined as a 4-second segment in which no eye movements were detected. Epochs containing only a single REM were excluded to avoid transitional states [4]. In addition, phasic and tonic REM epochs were selected only if the intervals between the two types of REM phases were longer than 8 s to avoid possible mutual interference [24]. EEG signals contaminated by motion or EMG artifacts were excluded from further analysis.

Table 1. General demographic and sleep characteristics of the study population.

	iRBD patients	Normal controls	*p*
Sex	15 Males	15 Males	
Age (years)	40 (35–56)	42 (38–51)	NS
Total sleep time (min)	421.33 ± 64.74	428.30 ± 42.35	NS
WASO (min)	70.73 ± 39.99	37.67 ± 20.59	<0.01
Sleep efficiency (%)	83.07 ± 7.83	90.47 ± 4.24	<0.01
Sleep stage (%)			
N1	12.11 ± 5.49	9.23 ± 4.13	NS
N2	53.87 ± 6.56	52.70 ± 9.76	NS
N3	15.80 ± 9.82	18.52 ± 8.79	NS
REM	18.22 ± 4.82	19.55 ± 4.53	NS
Sleep stage (min)			
N1	51.17 ± 23.37	38.47 ± 15.01	NS
N2	227.23 ± 43.99	225.27 ± 46.31	NS
N3	66.97 ± 43.38	80.20 ± 41.24	NS
REM	75.97 ± 21.44	84.37 ± 23.10	NS
Microarousal index (events/h)	14.40 (11.10–20.80)	10.70 (6.40–15.50)	NS
SB index (events/h)	0.80 (0.40–1.90)	0.80 (0.40–3.40)	NS
PLMI (events/h)	1.70 (0.00–9.90)	4.90 (0.50–10.10)	NS
AHI (events/h)	6.11 ± 4.81	7.93 ± 4.53	NS

Normally distributed variables are reported as mean ± standard deviation (SD) and skewed variables are reported as the median (IQR).

AHI, Apnea hypopnea index; N1–N3, Non-rapid eye movement sleep stage 1–3; NS, not significant; PLMI, Periodic limb movement index; REM, rapid eye movement sleep; SB, sleep bruxism; WASO, wake after sleep onset; iRBD, idiopathic rapid eye movement sleep behavior disorder.

Phasic and tonic REM sleep in the iRBD patients were further categorized according to the presence or absence of excessive transient muscle activity (ETMA), which was defined as EMG bursts lasting 0.1–0.5 seconds and with amplitudes at no less than two times of the EMG activities with muscle atonia, or the lowest EMG amplitude during non-rapid eye movement (NREM) sleep if no muscle atonia was present during REM sleep [23]. In the iRBD patients and normal controls, 50 4-second phasic epochs (25 with and 25 without ETMA) and 50 4-second tonic epochs (25 with and 25 without ETMA) were randomly selected per participant. Power spectral analysis of the epochs of EEG signals was carried out using the Welch's method with 1-second windows and 50% overlap [4].

The ratio of duration of REM sleep without atonia (RSWA) to the total duration of REM sleep. A higher percentage of REM sleep showing increased muscle tone (tonic) or excessive transient activity (phasic) was used to indicate more severe pathology. The correlation of EEG measures with the ratio of RSWA/REM was analyzed.

The absolute and relative power of five specific EEG frequency bands specifically theta (4–8 Hz), alpha (8–12 Hz), sigma (12–15 Hz), and beta (15–30 Hz) [4], as well as gamma (30–48 Hz) were calculated based on linked mastoid reference (A1 + A2)/2. The 30–48 Hz gamma band, rather than the 30–50 Hz range as used in the previous study [4], was analyzed to avoid including alternating current (50 Hz) noise. Delta waves during REM sleep were not analyzed due to possible contamination with EOG signals.

Inter-hemispheric EEG asymmetry was defined as the difference in EEG power of the same waves during REM sleep in the frontal, central and occipital regions between left and right side, and expressed as inter-hemispheric EEG asymmetry score (INTER-EEG-A-Score), which was calculated by using the following formula:

INTER-EEG-A-Score = (Power $_{Right}$ − Power $_{left}$)/(Power $_{Right}$ + Power $_{left}$) [25,26].

In contrast, intra-hemispheric discrepancy referred to the difference in the power of certain EEG waves between any two cortical regions in the same cortical hemisphere.

2.4 Sample Size Estimation

Sample size was estimated based on the results of the preliminary data from four iRBD patients and four normal controls with G*Power (version 3.1.9.7, http://www.gpower.hhu.de/; α set at 5% and β at 20%), and eleven iRBD patients and eleven normal controls were estimated to be needed. In addition, after the experiments, the statistical power of each test with a p-value $\geq$ 0.05 was calculated and confirmed to be desired (i.e., $\geq$0.8) in this study.

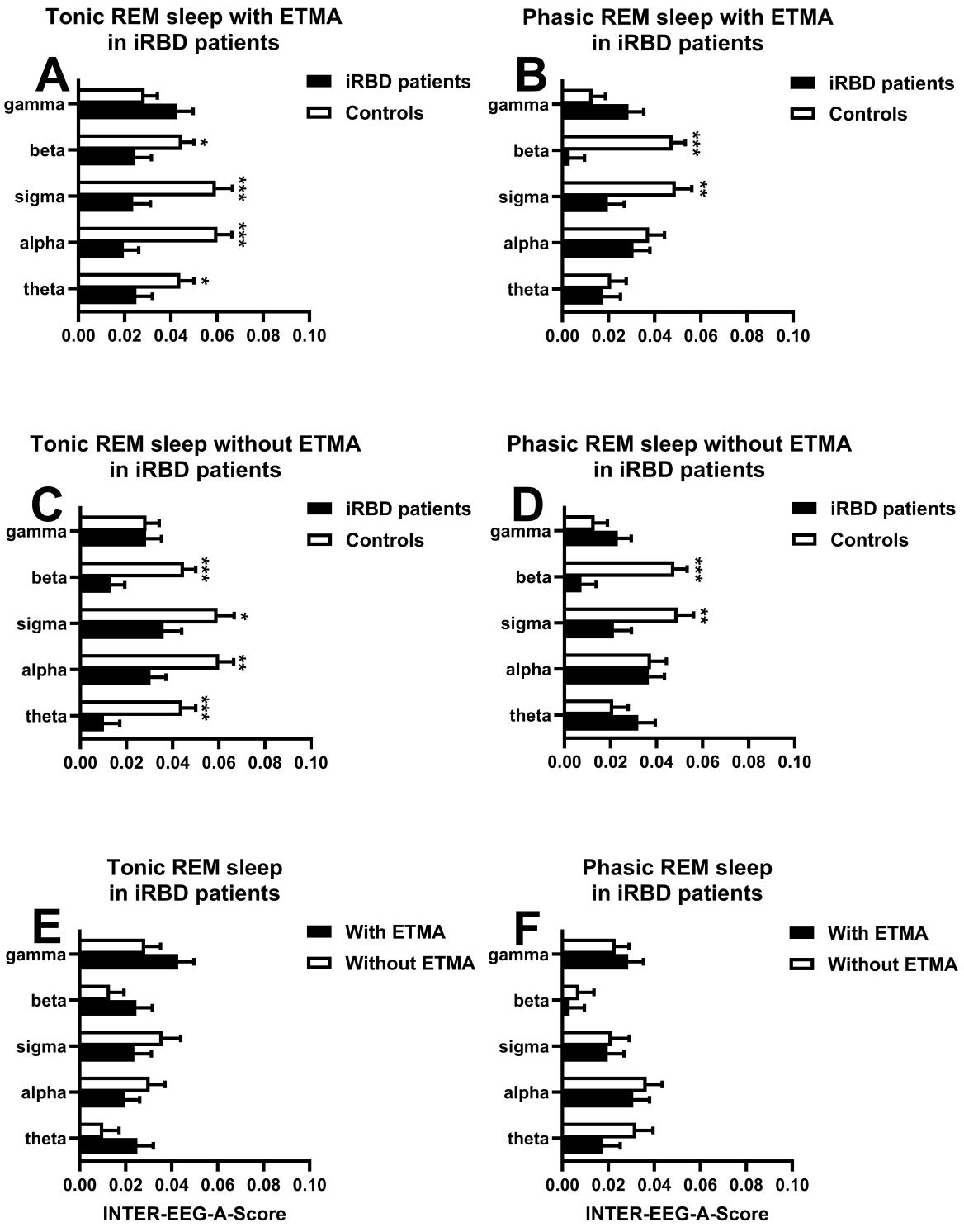

Fig. 1. INTER-EEG-A Scores in the frontal regions in the iRBD patients and normal controls during REM sleep. (A–D) INTER-EEG-A Scores in the frontal regions during tonic (A,B) and phasic (C,D) REM sleep in the iRBD patients and normal controls. (E,F) Inter-hemispheric EEG asymmetry Score (INTER-EEG-A-Score) in the frontal regions during tonic and phasic REM sleep with and without excessive transient muscle activity (ETMA) in the iRBD patients. Data are reported as mean ± SEM. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

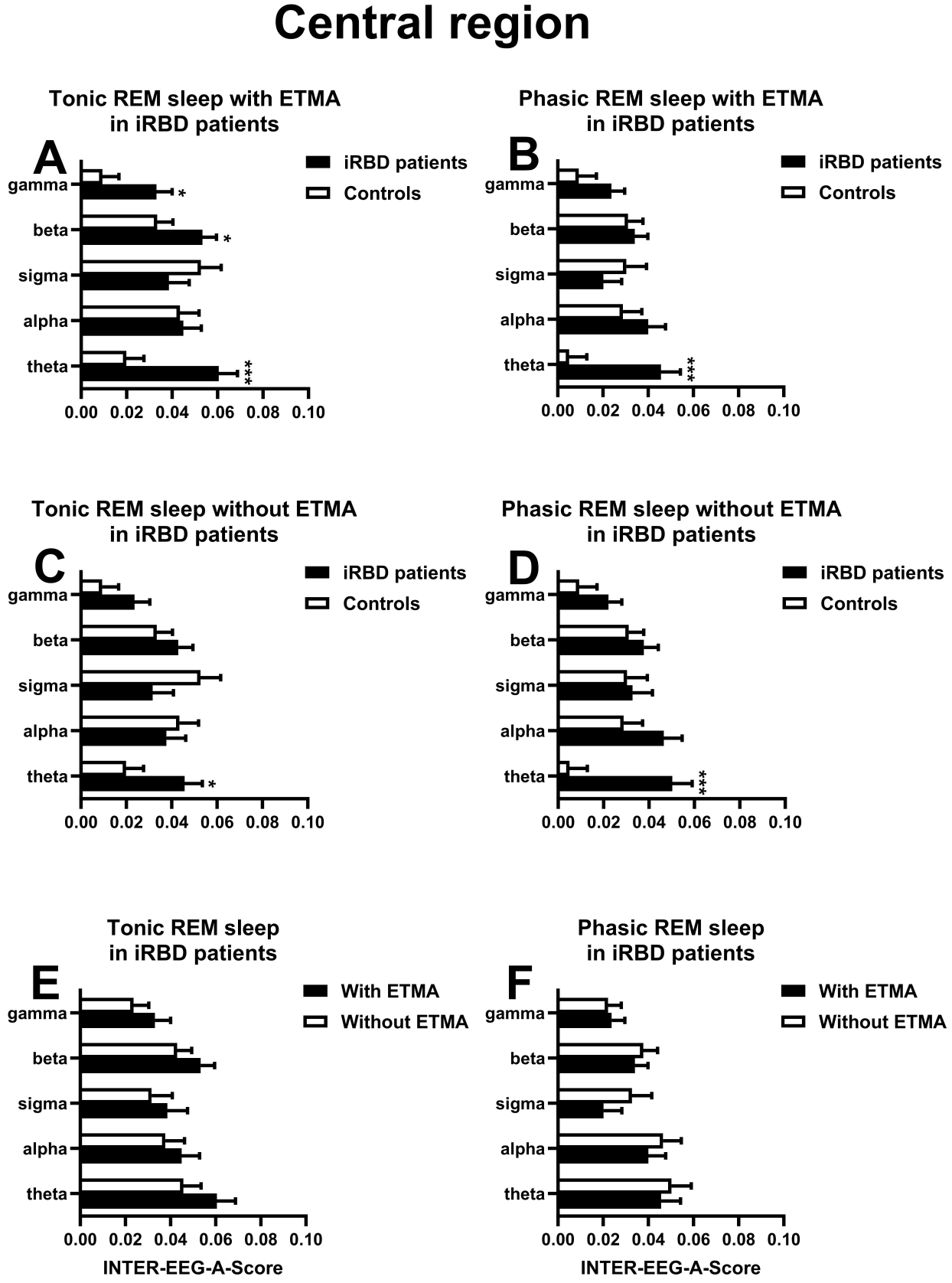

Fig. 2. EEG powers and INTER-EEG-A Scores in the central regions in the iRBD patients and normal controls during REM sleep. (A–D) INTER-EEG-A Scores in the central regions during tonic (A,B) and phasic (C,D) REM sleep in the iRBD patients and normal controls. (E,F) INTER-EEG-A Scores in the central regions during tonic and phasic REM sleep with and without ETMA in the iRBD patients. Data are reported as mean ± SEM. *$p < 0.05$, ***$p < 0.001$.

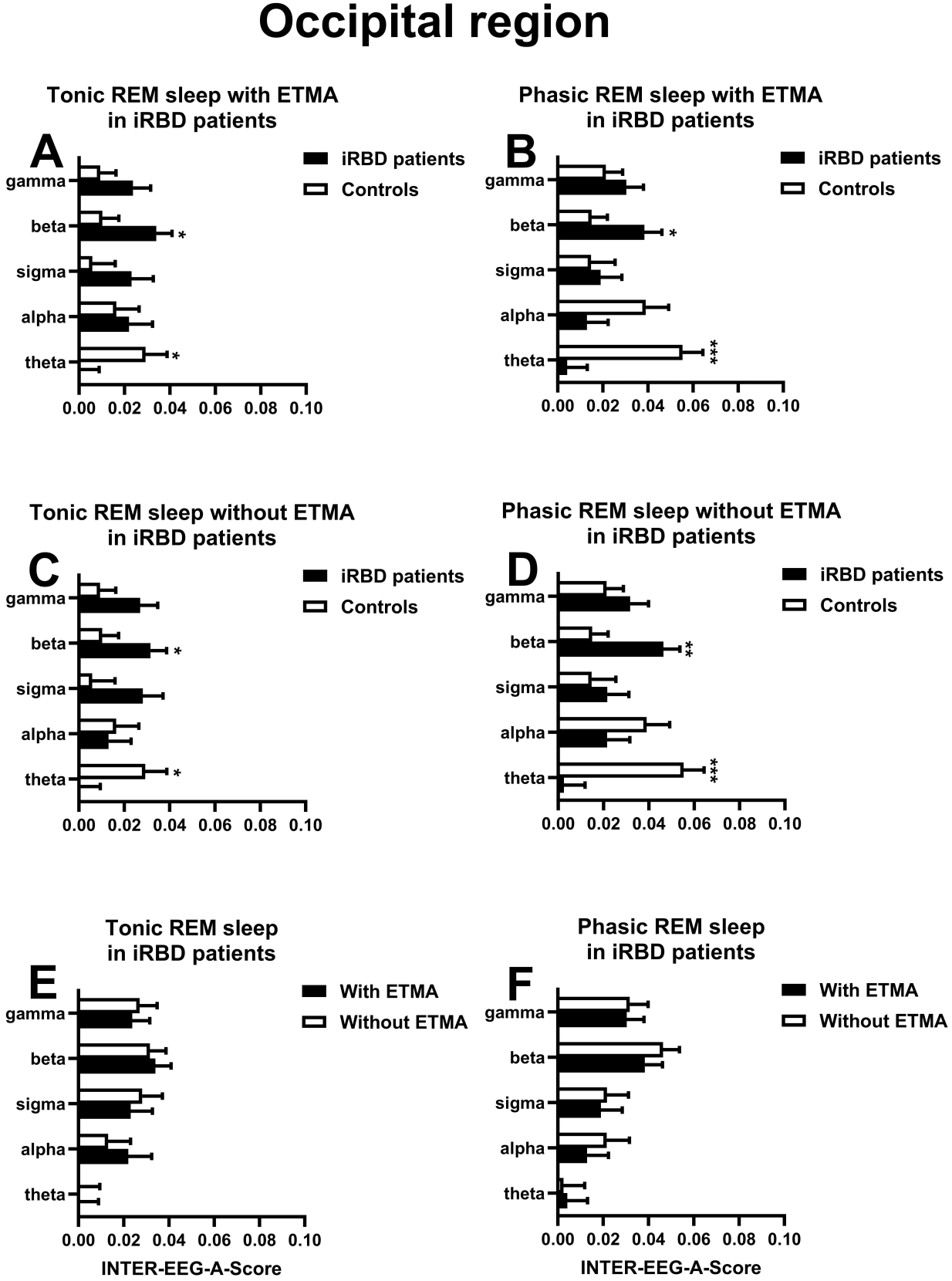

Fig. 3. EEG powers and INTER-EEG-A Scores in the occipital regions in the iRBD patients and normal controls during REM sleep. (A–D) INTER-EEG-A Scores in the occipital regions during tonic (A,B) and phasic (C,D) REM sleep in the iRBD patients and normal controls. (E,F) INTER-EEG-A Scores in the occipital regions during tonic and phasic REM sleep with and without ETMA in the iRBD patients. Data are reported as mean ± SEM. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

2.5 Statistical Analysis

Prior to any statistical analysis, normality for all variables was examined with Shapiro-Wilk tests. Normally distributed data were reported as mean ± standard deviation (SD) unless indicated otherwise, and the paired *t*-test or Student's *t*-test was used wherever appropriate. In addition, two-way repeated measures ANOVA followed by post-hoc Bonferroni tests was used to examine inter-hemispheric asymmetry and intra-hemispheric discrepancy in EEG activities. Skewed data are reported as the median (IQR) and the Mann–Whitney U test and the Wilcoxon signed-rank test were used wherever appropriate. The SPSS Statistics 25.0 software package (IBM, Armonk, NY, USA) was used to perform statistical analysis. Supplementary scatter plots were generated to display individual data points for each event by using GraphPad Prism (version 10.1.2, GraphPad Software, San Diego, CA, USA). $p < 0.05$ was considered as statistical significance.

3. Results

3.1 General Demographic and Sleep Characteristics of iRBD Patients and Normal Controls

No significant difference ($p > 0.05$) existed between the iRBD patients and normal controls in age, total sleep time, microarousal index, sleep bruxism index, apnea-hypopnea index, periodic leg movement index, and percentage of the sleep time spent in each sleep stage (Table 1). However, compared with the normal controls, the iRBD patients had a significantly lower sleep efficiency ($p < 0.01$) and a significantly longer wake after sleep onset time ($p < 0.01$).

3.2 Inter-Hemispheric and Intra-Hemispheric and EEG Activity Differences in the iRBD Patients and Normal Controls

EEG powers of theta, alpha, sigma, beta and gamma waves in the right frontal, central and occipital regions were significantly greater ($p < 0.05$–0.001) than those in the corresponding left cortical regions during tonic and phasic REM sleep (regardless of ETMA) in the iRBD patients and normal controls, with the following exceptions. In the iRBD patients, no significant bilateral difference ($p > 0.05$) was found in the frontal theta and occipital alpha power during tonic REM without ETMA, frontal beta power during phasic REM sleep with and without ETMA, occipital theta power during phasic REM sleep without ETMA, and occipital alpha power during phasic REM sleep with ETMA. In contrast, in the normal controls, no significant bilateral difference was found in the occipital sigma, beta and gamma power, the central gamma power during tonic REM sleep, and the central theta power, occipital sigma power as well as the frontal and central gamma power during phasic REM sleep ($p > 0.05$) (Tables 2,3, and **Supplementary Figs. 1–3**).

During tonic REM sleep (regardless of ETMA) in the iRBD patients and normal controls, the powers of theta, beta, and gamma waves in the frontal region were significantly greater than those in the ipsilateral central ($p < 0.001$) and occipital regions ($p < 0.001$), and the powers of these frequency bands were significantly greater in the central region ($p < 0.001$) than those in the ipsilateral occipital region (Table 2). Similarly, alpha and sigma power were significantly greater in the frontal and central regions ($p < 0.001$) than in the ipsilateral occipital region (Table 2). In contrast, the power of alpha and sigma waves in the frontal regions were not significant differently ($p > 0.05$) from those in the ipsilateral central region, but alpha and sigma power was significantly greater ($p < 0.05$–0.001) in the left frontal region than in the left central region during tonic REM sleep with ETMA in the iRBD patients (Table 2).

During phasic REM sleep (regardless of ETMA) in the iRBD patients and normal controls, the powers of all five EEG frequency bands analysed in the frontal region were significantly greater than those in the ipsilateral central ($p < 0.001$) and occipital regions ($p < 0.001$), and the powers of these frequency bands in the central region were significantly greater ($p < 0.001$) than those in the ipsilateral occipital region (Table 3).

3.3 Comparisons of EEG Activities Between Left and Right Cerebral Hemispheres in the iRBD Patients and Normal Controls

In the frontal region, during tonic REM sleep regardless of ETMA, inter-hemispheric EEG asymmetry scores of theta, alpha, sigma and beta waves were significantly smaller in the iRBD patients compared to normal controls ($p < 0.05$–0.001), while no significant difference was found in gamma waves ($p > 0.05$) (Fig. 1A,C). In contrast, inter-hemispheric EEG asymmetry scores of sigma and beta waves were significantly smaller during phasic REM sleep regardless of ETMA, while no significant difference was found in those scores of theta, alpha and gamma waves during phasic REM sleep regardless of ETMA ($p > 0.05$) (Fig. 1B,D).

In the central region, inter-hemispheric EEG asymmetry scores of beta and gamma waves during tonic REM sleep with ETMA, not during tonic sleep without ETMA, were significantly greater in the iRBD patients than those in normal controls ($p < 0.05$). In contrast, inter-hemispheric EEG asymmetry scores of theta waves during tonic and phasic REM sleep (regardless of ETMA) were significantly greater in the iRBD patients than those in normal controls ($p < 0.05$–0.001). While no significant difference was found in the inter-hemispheric EEG asymmetry scores of alpha and sigma waves during phasic and tonic REM sleep (regardless of ETMA) ($p > 0.05$) (Fig. 2).

In the occipital region, inter-hemispheric EEG asymmetry scores of beta waves during tonic and phasic REM sleep (regardless of ETMA) were significantly greater in

Table 2. Comparisons of the power of EEG waves in dB between left and right hemispheres during tonic REM sleep.

Tonic REM sleep	With ETMA in iRBD patients		Without ETMA in iRBD patients		Normal controls	
	Left	Right	Left	Right	Left	Right
Theta						
Frontal	17.07 ± 2.23	17.29 ± 2.13$^{\#\#\#}$	16.89 ± 2.14	16.98 ± 2.07	16.06 ± 1.94	16.45 ± 1.91$^{\#\#\#}$
Central	16.00 ± 2.27***	16.55 ± 2.06$^{***,\#\#}$	15.99 ± 2.13***	16.41 ± 1.88$^{***,\#\#\#}$	15.34 ± 1.89***	15.52 ± 1.72$^{***,\#}$
Occipital	14.05 ± 2.55***,§§§	14.05 ± 2.41***,§§§	14.22 ± 2.54***,§§§	14.22 ± 2.32***,§§§	13.50 ± 2.10***,§§§	13.77 ± 2.07$^{***,\S\S\S,\#\#\#}$
Alpha						
Frontal	15.12 ± 2.27	15.30 ± 2.34$^{\#}$	14.86 ± 2.30	15.13 ± 2.30$^{\#\#\#}$	14.24 ± 1.87	14.77 ± 1.85$^{\#\#\#}$
Central	14.67 ± 2.40***	15.07 ± 2.36$^{\#\#\#}$	14.65 ± 2.40	14.99 ± 2.30$^{\#\#\#}$	14.19 ± 2.14	14.58 ± 2.01$^{\#\#\#}$
Occipital	13.99 ± 3.10***,§§§	14.2 ± 3.05$^{***,\S\S\S,\#\#}$	14.11 ± 3.11***,§§§	14.24 ± 2.96***,§§§	13.62 ± 2.59***,§§§	13.77 ± 2.28$^{***,\S\S\S,\#}$
Sigma						
Frontal	11.71 ± 2.23	11.92 ± 2.25$^{\#}$	11.59 ± 2.36	11.91 ± 2.41$^{\#\#\#}$	11.38 ± 1.75	11.91 ± 1.78$^{\#\#\#}$
Central	11.42 ± 2.35*	11.76 ± 2.16$^{\#\#\#}$	11.54 ± 2.47	11.82 ± 2.35$^{\#\#\#}$	11.33 ± 2.07	11.80 ± 2.13$^{\#\#\#}$
Occipital	10.61 ± 2.64***,§§§	10.83 ± 2.71$^{***,\S\S\S,\#\#}$	10.66 ± 2.77***,§§§	10.92 ± 2.7$^{***,\S\S\S,\#\#\#}$	10.75 ± 2.55***,§§§	10.81 ± 2.37***,§§§
Beta						
Frontal	17.84 ± 1.98	18.06 ± 1.86$^{\#\#\#}$	17.62 ± 1.78	17.74 ± 1.69$^{\#}$	17.26 ± 1.83	17.66 ± 1.91$^{\#\#\#}$
Central	16.62 ± 1.70***	17.09 ± 1.79$^{***,\#\#\#}$	16.56 ± 1.78***	16.94 ± 1.66$^{***,\#\#\#}$	16.78 ± 2.11***	17.08 ± 2.08$^{***,\#\#\#}$
Occipital	14.14 ± 1.96***,§§§	14.44 ± 1.75$^{***,\S\S\S,\#\#\#}$	14.10 ± 1.96***,§§§	14.38 ± 1.63$^{***,\S\S\S,\#\#\#}$	14.75 ± 1.83***,§§§	14.84 ± 1.78***,§§§
Gamma						
Frontal	14.59 ± 2.44	14.97 ± 2.53$^{\#\#\#}$	14.21 ± 2.21	14.47 ± 2.21$^{\#\#\#}$	13.81 ± 1.98	14.06 ± 1.88$^{\#\#\#}$
Central	13.32 ± 1.59***	13.61 ± 1.75$^{***,\#\#\#}$	13.08 ± 1.50***	13.29 ± 1.57$^{***,\#\#\#}$	12.99 ± 1.73***	13.07 ± 1.74***
Occipital	11.01 ± 1.70***,§§§	11.23 ± 1.29$^{***,\S\S\S,\#\#\#}$	10.84 ± 1.68***,§§§	11.09 ± 1.30$^{***,\S\S\S,\#\#\#}$	11.12 ± 1.69***,§§§	11.20 ± 1.56***,§§§

All values were shown as mean ± SD and two-way repeated measures (RM) ANOVA followed by post hoc Bonferroni tests were used for comparisons.

* $p < 0.05$, *** $p < 0.001$: comparisons of EEG power between the frontal and central regions, and between the frontal and occipital regions.

§§§ $p < 0.001$: comparisons of EEG power between the central and occipital regions.

$^{\#}$ $p < 0.05$, $^{\#\#}$ $p < 0.01$, $^{\#\#\#}$ $p < 0.001$: comparisons of EEG power between analogous regions in bilateral hemispheres.

EEG, electroencephalogram; ETMA, excessive transient muscle activity; dB, decibel.

Table 3. Comparisons of the power of EEG waves in dB between left and right hemispheres during phasic REM sleep.

Phasic REM sleep	With ETMA in iRBD patients		Without ETMA in iRBD patients		Normal controls	
	Left	Right	Left	Right	Left	Right
Theta						
Frontal	18.90 ± 2.00	19.06 ± 1.98$^{\#}$	18.27 ± 1.95	18.55 ± 1.93$^{\#\#\#}$	17.96 ± 1.92	18.16 ± 1.92$^{\#\#}$
Central	17.60 ± 2.32***	18.01 ± 2.25$^{***,\#\#\#}$	16.92 ± 2.18***	17.37 ± 2.01$^{***,\#\#\#}$	16.50 ± 2.20***	16.55 ± 1.92***
Occipital	14.84 ± 2.62***,§§§	14.88 ± 2.53***,§§§	14.57 ± 2.56***,§§§	14.59 ± 2.45***,§§§	13.98 ± 1.96***,§§§	14.49 ± 2.10$^{***,\S\S\S,\#\#\#}$
Alpha						
Frontal	16.22 ± 2.21	16.49 ± 2.24$^{\#\#\#}$	15.68 ± 2.12	16.00 ± 2.14$^{\#\#\#}$	15.41 ± 1.84	15.74 ± 1.85$^{\#\#\#}$
Central	15.31 ± 2.31***	15.66 ± 2.17$^{***,\#\#\#}$	14.80 ± 2.20***	15.22 ± 1.98$^{***,\#\#\#}$	14.69 ± 2.02***	14.97 ± 1.82$^{***,\#\#\#}$
Occipital	14.42 ± 3.10***,§§§	14.54 ± 2.86***,§§§	14.09 ± 3.11***,§§§	14.30 ± 2.90$^{***,\S\S\S,\#\#}$	13.85 ± 2.31***,§§§	14.20 ± 2.38$^{***,\S\S\S,\#\#\#}$
Sigma						
Frontal	12.00 ± 1.89	12.18 ± 1.90$^{\#}$	11.98 ± 2.09	12.18 ± 1.99$^{\#}$	11.81 ± 1.61	12.26 ± 1.69$^{\#\#\#}$
Central	11.42 ± 2.11***	11.60 ± 1.85$^{***,\#\#\#}$	11.47 ± 2.21***	11.76 ± 2.02$^{***,\#\#\#}$	11.40 ± 1.90***	11.66 ± 1.69$^{***,\#\#\#}$
Occipital	10.49 ± 2.48***,§§§	10.68 ± 2.36$^{***,\S\S\S,\#}$	10.56 ± 2.45***,§§§	10.77 ± 2.44$^{***,\S\S\S,\#\#}$	10.29 ± 2.01***,§§§	10.42 ± 1.92***,§§§
Beta						
Frontal	17.79 ± 1.57	17.82 ± 1.38	17.82 ± 1.69	17.89 ± 1.50	17.56 ± 1.82	17.98 ± 1.90$^{\#\#\#}$
Central	16.45 ± 1.49***	16.75 ± 1.43$^{***,\#\#\#}$	16.55 ± 1.58***	16.88 ± 1.57$^{***,\#\#\#}$	16.59 ± 1.96***	16.86 ± 1.65$^{***,\#\#\#}$
Occipital	14.27 ± 1.85***,§§§	14.62 ± 1.53$^{***,\S\S\S,\#\#\#}$	14.20 ± 1.88***,§§§	14.62 ± 1.60$^{***,\S\S\S,\#\#\#}$	14.84 ± 1.70***,§§§	14.97 ± 1.63$^{***,\S\S\S,\#}$
Gamma						
Frontal	14.48 ± 1.91	14.74 ± 1.93$^{\#\#\#}$	14.44 ± 2.02	14.65 ± 1.98$^{\#\#\#}$	14.27 ± 1.53	14.38 ± 1.56
Central	13.21 ± 1.28***	13.42 ± 1.34$^{***,\#\#\#}$	13.20 ± 1.33***	13.40 ± 1.39$^{***,\#\#\#}$	13.18 ± 1.55***	13.26 ± 1.40***
Occipital	11.39 ± 1.67***,§§§	11.66 ± 1.28$^{***,\S\S\S,\#\#\#}$	11.13 ± 1.71***,§§§	11.42 ± 1.22$^{***,\S\S\S,\#\#\#}$	11.67 ± 1.73***,§§§	11.87 ± 1.80$^{***,\S\S\S,\#\#}$

All values were shown as mean ± SD and two-way RM ANOVA followed by post hoc Bonferroni tests were used for comparisons.

*** $p < 0.001$: comparisons of EEG power between the frontal and central regions, and between the frontal and occipital regions.

§§§ $p < 0.001$: comparisons of EEG power between the central and occipital regions.

$^{\#}$ $p < 0.05$, $^{\#\#}$ $p < 0.01$, $^{\#\#\#}$ $p < 0.001$: comparisons of EEG power between analogous regions in bilateral hemispheres.

the iRBD patients than those in normal controls ($p <$ 0.05–0.01). In contrast, inter-hemispheric EEG asymmetry scores of theta waves during tonic and phasic REM sleep (regardless of ETMA) were significantly smaller in the iRBD patients than those in normal controls ($p < 0.05$–0.001). While no significant difference was found in the inter-hemispheric EEG asymmetry scores of alpha, sigma and gamma waves during phasic and tonic REM sleep (regardless of ETMA) ($p > 0.05$) (Fig. 3).

In addition, no significant difference was found in inter-hemispheric EEG asymmetry scores of all five frequency bands in the frontal, central, and occipital regions between REM sleep with and without ETMA in the iRBD patients ($p > 0.05$) (Figs. 1,2,3).

3.4 Comparisons of EEG Power During REM Sleep Between the iRBD Patients and Normal Controls

As reported in Table 4, during tonic REM sleep with ETMA in the frontal cortical region, the iRBD patients exhibited a significantly greater EEG power in theta, alpha, sigma, beta and gamma waves in the left side, and theta, alpha, beta, and gamma waves in the right side than that in the normal controls ($p < 0.05$–0.001). In contrast, during tonic REM sleep without ETMA, the iRBD patients exhibited a significantly greater EEG power in theta, alpha, beta and gamma waves in the left frontal cortical region, and theta, alpha, and sigma waves in the right frontal region than that in the normal controls ($p < 0.05$–0.001).

During tonic REM sleep with ETMA in the central cortical region, the iRBD patients exhibited a significantly greater EEG power in theta, alpha and gamma waves in the left and right side than that in the normal controls ($p <$ 0.01–0.001). In contrast, during tonic REM sleep without ETMA, the iRBD patients exhibited a significantly greater EEG power in theta and alpha waves in the left and right central regions ($p < 0.05$–0.001).

During tonic REM sleep with ETMA in the occipital cortical region, the iRBD patients exhibited a significantly greater EEG power in theta and beta waves in the left side, and alpha, beta, and theta waves in the right side than that in the normal controls ($p < 0.05$–0.001). In contrast, during tonic REM sleep without ETMA, the iRBD patients exhibited a significantly greater EEG power in theta, alpha and beta waves in bilateral occipital regions, and gamma waves in the left occipital region than that in the normal controls ($p <$ 0.01–0.001).

In addition, during tonic REM sleep with ETMA, the iRBD patients exhibited a significantly greater ($p < 0.05$–0.001) EEG power in gamma waves in bilateral frontal, central, and occipital regions, beta waves in the bilateral frontal region, alpha waves in the left frontal and theta waves in the right frontal region than that during tonic REM sleep without ETMA.

As shown in Table 4, during phasic REM sleep with ETMA in the frontal cortical region, the iRBD patients exhibited a significantly greater EEG power in theta, alpha waves in the left and right sides, gamma waves in the right side compared to the normal controls ($p <$ 0.01–0.001). In contrast, during phasic REM sleep without ETMA, the iRBD patients showed a significantly greater EEG power in beta waves in the left frontal regions, and theta and gamma waves in the right frontal region than that in the normal controls ($p < 0.05$).

During phasic REM sleep with ETMA in the central cortical region, the iRBD patients exhibited a significantly greater EEG power in theta and alpha waves in the left and right sides than that in the normal controls ($p < 0.001$). In contrast, during phasic REM sleep without ETMA, the iRBD patients exhibited a significantly greater EEG power in theta waves in bilateral central regions, ($p < 0.05$–0.001).

During phasic REM sleep with ETMA in the occipital cortical region, the iRBD patients exhibited a significantly great EEG power in theta and beta waves in bilateral sides, and alpha and gamma waves in the left side than that in the normal controls ($p < 0.05$–0.001). In contrast, during phasic REM sleep without ETMA, the iRBD patients showed a significantly greater EEG power in beta and gamma waves in bilateral occipital regions, theta waves in the left occipital region, and sigma waves in the right occipital region than that in the normal controls ($p < 0.05$–0.001).

In addition, during phasic REM sleep with ETMA, the iRBD patients showed a significantly greater ($p < 0.05$–0.001) EEG power in theta and alpha waves in bilateral frontal and central regions, theta waves in bilateral frontal region, alpha waves in the left occipital region, and gamma waves in bilateral occipital regions than that during phasic REM sleep without ETMA.

3.5 Correlation of EEG Activity Asymmetry With the Ratio of RSWA/REM Sleep

No significant correlation was found between EEG activity asymmetry of theta, alpha, sigma, beta, and gamma waves in frontal, central and occipital regions during REM sleep (with and without ETMA) with the ratio of RSWA duration/REM sleep duration ($p > 0.05$).

4. Discussion

iRBD is a common sleep movement disorder with an abnormal increase in muscle tone accompanied by dream enactment behaviors during REM sleep in patients without any neurological disease. In this study, the authors systematically investigated differences in EEG activities between bilateral cerebral hemispheres and between different cortical regions in the same hemisphere during REM sleep, and found there were intra-hemispheric discrepancy and inter-hemispheric EEG asymmetry during both tonic and phasic REM sleep in the iRBD patients and normal controls. In addition, compared to the normal controls, the iRBD patients exhibited a significantly greater inter-hemispheric asymmetry in EEG activities in the central and occipital regions,

Table 4. Comparisons of EEG power in dB during REM sleep between the iRBD patients and normal controls.

Tonic REM sleep	iRBD patients		Controls	p^*	$p^§$	$p\#$
	With ETMA	Without ETMA				
Left frontal region (F3)						
Theta	17.07 ± 2.23	16.89 ± 2.14	16.06 ± 1.94	<0.001	<0.001	NS
Alpha	15.12 ± 2.27	14.86 ± 2.30	14.24 ± 1.87	<0.001	<0.001	<0.05
Sigma	11.71 ± 2.23	11.59 ± 2.36	11.38 ± 1.75	<0.05	NS	NS
Beta	17.84 ± 1.98	17.62 ± 1.78	17.26 ± 1.83	<0.001	<0.01	<0.05
Gamma	14.59 ± 2.44	14.21 ± 2.21	13.81 ± 1.98	<0.001	<0.01	<0.001
Right frontal region (F4)						
Theta	17.29 ± 2.13	16.98 ± 2.07	16.45 ± 1.91	<0.001	<0.001	<0.05
Alpha	15.30 ± 2.34	15.13 ± 2.30	14.77 ± 1.85	<0.01	<0.05	NS
Sigma	11.92 ± 2.25	11.91 ± 2.41	11.91 ± 1.78	NS	NS	NS
Beta	18.06 ± 1.86	17.74 ± 1.69	17.66 ± 1.91	<0.01	NS	<0.01
Gamma	14.97 ± 2.53	14.47 ± 2.21	14.06 ± 1.88	<0.001	<0.01	<0.001
Left central region (C3)						
Theta	16 ± 2.27	15.99 ± 2.13	15.34 ± 1.89	<0.001	<0.001	NS
Alpha	14.67 ± 2.40	14.65 ± 2.40	14.19 ± 2.14	<0.01	<0.01	NS
Sigma	11.42 ± 2.35	11.54 ± 2.47	11.33 ± 2.07	NS	NS	NS
Beta	16.62 ± 1.70	16.56 ± 1.78	16.78 ± 2.11	NS	NS	NS
Gamma	13.32 ± 1.59	13.08 ± 1.50	12.99 ± 1.73	<0.01	NS	<0.01
Right central region (C4)						
Theta	16.55 ± 2.06	16.41 ± 1.88	15.52 ± 1.72	<0.001	<0.001	NS
Alpha	15.07 ± 2.36	14.99 ± 2.30	14.58 ± 2.01	<0.01	<0.05	NS
Sigma	11.76 ± 2.16	11.82 ± 2.35	11.80 ± 2.13	NS	NS	NS
Beta	17.09 ± 1.79	16.94 ± 1.66	17.08 ± 2.08	NS	NS	NS
Gamma	13.61 ± 1.75	13.29 ± 1.57	13.07 ± 1.74	<0.001	NS	<0.01
Left occipital region (O1)						
Theta	14.05 ± 2.55	14.22 ± 2.54	13.50 ± 2.10	<0.01	<0.001	NS
Alpha	13.99 ± 3.10	14.11 ± 3.11	13.62 ± 2.59	NS	<0.05	NS
Sigma	10.61 ± 2.64	10.66 ± 2.77	10.75 ± 2.55	NS	NS	NS
Beta	14.14 ± 1.96	14.10 ± 1.96	14.75 ± 1.83	<0.001	<0.001	NS
Gamma	11.01 ± 1.70	10.84 ± 1.68	11.12 ± 1.69	NS	<0.05	<0.05
Right occipital region (O2)						
Theta	14.05 ± 2.41	14.22 ± 2.32	13.77 ± 2.07	NS	<0.01	NS
Alpha	14.20 ± 3.05	14.24 ± 2.96	13.77 ± 2.28	<0.05	<0.05	NS
Sigma	10.83 ± 2.71	10.92 ± 2.70	10.81 ± 2.37	NS	NS	NS
Beta	14.44 ± 1.75	14.38 ± 1.63	14.84 ± 1.78	<0.01	<0.001	NS
Gamma	11.23 ± 1.29	11.09 ± 1.30	11.20 ± 1.56	NS	NS	<0.05
Phasic REM sleep						
Left frontal region (F3)						
Theta	18.90 ± 2.00	18.27 ± 1.95	17.96 ± 1.92	<0.001	NS	<0.001
Alpha	16.22 ± 2.21	15.68 ± 2.12	15.41 ± 1.84	<0.001	NS	<0.001
Sigma	12.00 ± 1.89	11.98 ± 2.09	11.81 ± 1.61	NS	NS	NS
Beta	17.79 ± 1.57	17.82 ± 1.69	17.56 ± 1.82	NS	<0.05	NS
Gamma	14.48 ± 1.91	14.44 ± 2.02	14.27 ± 1.53	NS	NS	NS
Right frontal region (F4)						
Theta	19.06 ± 1.98	18.55 ± 1.93	18.16 ± 1.92	<0.001	<0.05	<0.001
Alpha	16.49 ± 2.24	16.00 ± 2.14	15.74 ± 1.85	<0.001	NS	<0.001
Sigma	12.18 ± 1.90	12.18 ± 1.99	12.26 ± 1.69	NS	NS	NS
Beta	17.82 ± 1.38	17.89 ± 1.50	17.98 ± 1.90	NS	NS	NS
Gamma	14.74 ± 1.93	14.65 ± 1.98	14.38 ± 1.56	<0.01	<0.05	NS

Table 4. Continued.

Tonic REM sleep	iRBD patients		Controls	p*	$p^{\S}$	p#
	With ETMA	Without ETMA				
Left central region (C3)						
Theta	17.60 ± 2.32	16.92 ± 2.18	16.50 ± 2.20	<0.001	<0.05	<0.001
Alpha	15.31 ± 2.31	14.8 ± 2.20	14.69 ± 2.02	<0.001	NS	<0.001
Sigma	11.42 ± 2.11	11.47 ± 2.21	11.40 ± 1.90	NS	NS	NS
Beta	16.45 ± 1.49	16.55 ± 1.58	16.59 ± 1.96	NS	NS	NS
Gamma	13.21 ± 1.28	13.2 ± 1.33	13.18 ± 1.55	NS	NS	NS
Right central region (C4)						
Theta	18.01 ± 2.25	17.37 ± 2.01	16.55 ± 1.92	<0.001	<0.001	<0.001
Alpha	15.66 ± 2.17	15.22 ± 1.98	14.97 ± 1.82	<0.001	NS	<0.001
Sigma	11.60 ± 1.85	11.76 ± 2.02	11.66 ± 1.69	NS	NS	NS
Beta	16.75 ± 1.43	16.88 ± 1.57	16.86 ± 1.65	NS	NS	NS
Gamma	13.42 ± 1.34	13.4 ± 1.39	13.26 ± 1.40	NS	NS	NS
Left occipital region (O1)						
Theta	14.84 ± 2.62	14.57 ± 2.56	13.98 ± 1.96	<0.001	<0.001	<0.05
Alpha	14.42 ± 3.10	14.09 ± 3.11	13.85 ± 2.31	<0.01	NS	<0.01
Sigma	10.49 ± 2.48	10.56 ± 2.45	10.29 ± 2.01	NS	NS	NS
Beta	14.27 ± 1.85	14.20 ± 1.88	14.84 ± 1.70	<0.001	<0.001	NS
Gamma	11.39 ± 1.67	11.13 ± 1.71	11.67 ± 1.73	<0.05	<0.001	<0.001
Right occipital region (O2)						
Theta	14.88 ± 2.53	14.59 ± 2.45	14.49 ± 2.10	<0.05	NS	<0.05
Alpha	14.54 ± 2.86	14.3 ± 2.90	14.20 ± 2.38	NS	NS	NS
Sigma	10.68 ± 2.36	10.77 ± 2.44	10.42 ± 1.92	NS	<0.05	NS
Beta	14.62 ± 1.53	14.62 ± 1.60	14.97 ± 1.63	<0.01	<0.01	NS
Gamma	11.66 ± 1.28	11.42 ± 1.22	11.87 ± 1.80	NS	<0.001	<0.001

p*: REM sleep with ETMA in iRBD patients vs Normal controls.

$p^{\S}$: REM sleep without ETMA in iRBD patients vs Normal controls.

p#: REM sleep with ETMA in iRBD patients vs REM sleep without ETMA in iRBD patients. NS, not significant.

whereas the frontal region showed a significantly smaller asymmetry. These findings of the altered inter-hemispheric asymmetry in EEG activities of iRBD patients might reflect changes in the bilateral neuronal differences that normally exist in the left and right hemispheres.

4.1 Comparisons of EEG Activities During Tonic and Phasic REM Sleep in the iRBD Patients and Normal Controls

Tonic and phasic REM sleep occurs alternatively during REM sleep and might have different functions. Phasic REM sleep is mainly involved in the processing of internal motor, sensory and corticothalamic network information, promoting sleep stability and emotional regulation, and human subjects are not sensitive to changes in the surrounding environment during phasic REM sleep [5,27]. By contrast, tonic REM sleep is associated with a higher alertness to the surroundings and a lower threshold of awakening to protect the body from potential external dangers [27]. In addition, the differences between tonic and phasic REM sleep are also reflected in alterations in memory and sensory processing [5,27]. The memory and sensory processing functions of individuals are weakened to a large extent during phasic REM sleep, but partially recovered during tonic REM sleep [5,27].

Dream-enactment behaviors during phasic REM sleep, in RBD patients are directionally coherent with saccadic eye movements, implying active visual scanning of dream imagery [28,29]. The previous study has shown that pre-representative behavior (i.e., the 60 s prior to dream enactment behaviors) was related to an increased delta power in the left frontal cortex and gamma power in the right frontal and occipital cortex, and enhanced beta-band functional connectivity compared with background (i.e., a 60 s segment with the least submental muscle EMG activity) [30].

In this study, theta and alpha power in the frontal, central and occipital regions, and gamma power in the occipital region were found to be significantly greater during phasic REM sleep with ETMA than during phasic REM sleep without ETMA (Table 4). Gamma-band synchronization has been reported to play a crucial role in the transmission of visual information and in visuomotor integration [31,32]. In iRBD patients, phasic REM is associated with abnormal excitation of the motor cortex and altered high-frequency (beta/gamma) connectivity [4]. By contrast, during tonic

REM sleep with ETMA, beta power in the frontal cortical region and gamma power in the frontal, central and occipital cortical regions were found to be significantly greater than those during tonic REM sleep without ETMA (Table 4). In comparison to tonic and phasic REM sleep without ETMA, the EEG power changes observed during REM sleep with ETMA likely reflect a pathological overactivation of sensorimotor and associative networks, which may drive the occurrence of dream enactment behaviors [4].

In the current study, theta and alpha powers in the iRBD patients were generally greater than those in the normal controls during tonic REM sleep regardless of ETMA, except for no significant difference in alpha power in left occipital region in the iRBD during tonic REM with ETMA compared to the normal controls (Table 4). This feature is similar to previous findings in patients with obstructive sleep apnea, showing that percentage of total sleep time with SpO_2 <90% was significantly correlated with increased power of theta and alpha waves in REM sleep [33]. Increased alpha-synuclein accumulation was found in iRBD patients [34], Alpha-synuclein can combine with hemoglobin to form hemoglobin-α-synuclein (Hb-α-syn) complex, which has a high-affinity binding with oxygen that hinders oxygen release [35]. In addition, Hb-α-syn complex can reduce the functional hemoglobin in red blood cells and the free neuronal hemoglobin (nHb) in the mitochondria, interfering with cellular energy production and causing hypoxia [36]. However, whether the increased theta and alpha powers in the iRBD patients is due to hypoxia is unclear.

Sigma band activity has been reported to show relatively little variation during REM sleep [37], which aligns with the finding that sigma power did not differ significantly in most cortical regions (Table 4). Compared to the normal controls, beta and gamma power during phasic REM sleep and beta power during tonic REM sleep in the iRBD patients were significantly smaller in the occipital region. In contrast, beta and gamma power in the frontal region during tonic REM sleep and gamma power in the central region during tonic REM sleep with ETMA were significantly greater compared to the normal controls (Table 4). Cholinergic neurons in the brainstem suppress specific low-frequency oscillations, while high-frequency oscillations are related to increased acetylcholine release from the thalamus and cortex [38]. Rats with basal forebrain cholinergic lesions showed significantly decreased high-frequency EEG activities, particularly in the gamma band, compared to sham controls [39]. The widespread changes in beta and gamma power that are observed in iRBD patients likely reflects impaired functional integration within large-scale cortical networks, and may indicate early dysfunction of cholinergic system in the basal forebrain, which is critical for maintaining high-frequency oscillatory activity during REM sleep.

The nucleus basalis of Meynert (NBM), a primary source of cholinergic projections to the neocortex [40], receives inputs from many brainstem nuclei, such as the laterodorsal tegmental nucleus, the locus coeruleus and the pedunculopontine tegmental nucleus [41,42], and the functional connection between the NBM and the cerebral cortex has been used to evaluate the basal forebrain cholinergic system [43]. Studies have found that NBM projects to the occipital region of the brain, and that the functional connection between the NBM and the occipital region in iRBD patients is weaker than in normal controls [44]. Although the pathophysiologic mechanisms underlying iRBD are not fully clear, the pathogenesis of iRBD is considered to be associated with damages to the cholinergic neurons in the laterodorsal tegmental nuclei [45], which causes dysfunction in the thalamus, given that the primary sources of cholinergic projections to the thalamus come from the pedunculopontine tegmental nucleus and the laterodorsal tegmental nucleus [46].

4.2 Comparisons of EEG Activities in Bilateral Hemispheres and EEG Asymmetry Between the iRBD Patients and Normal Controls

The current study revealed that the EEG powers of almost all analyzed EEG waves in the right cerebral hemisphere were significantly greater than those in the left cerebral hemisphere in the iRBD patients and normal controls during tonic and phasic REM sleep (Tables 2,3). This is consistent with the previous study showing structural or functional differences in bilateral cerebral hemispheres, and more involvement of the right cerebral hemisphere in maintaining alertness than the left [15], as maintenance of a certain degree of vigilance at night is necessary [18].

Hemispheric specialization, particularly in the prefrontal cortex, supports efficient cognitive processing by lateralizing functions such as attention, executive control, and emotional regulation [47]. In the frontal region, iRBD patients showed significantly reduced interhemispheric asymmetry in theta, sigma, and beta bands during tonic REM sleep regardless of ETMA, and in sigma and beta waves during phasic REM sleep regardless of ETMA (Fig. 1). Reduced frontal asymmetry may indicate a loss of hemispheric specialization. Under comparable cognitive task conditions, older adults typically show reduced lateralization of prefrontal activity relative to younger adults [47]. In contrast, elevated asymmetry in the central regions may indicate lateralized cortical hyperexcitability.

In the central region, the iRBD patients in the current study exhibited increased asymmetry in theta, beta, and gamma bands during tonic REM sleep with ETMA, and in the theta band during tonic REM sleep without ETMA, and in theta band during phasic REM sleep regardless of ETMA. This suggests that in the central region, the right hemisphere became relatively more active than the left in iRBD patients. In addition, the presence of ETMA appears to exacerbate the asymmetry in theta and beta bands

during tonic REM sleep. Although iRBD patients do not present with clinical motor symptoms, this pattern may reflect early-stage lateralized neurodegeneration. In the PD patients, symptom laterality is associated with sleep EEG asymmetry, particularly with greater slow-wave power observed in the hemisphere contralateral to the left, but not the right, dominant motor symptoms side [48]. Enhanced central asymmetry in iRBD patients might indicate early unilateral alterations in brainstem or basal ganglia circuits. However, the inter-hemispheric EEG asymmetry scores in the iRBD patients showed no significant difference between REM sleep regardless of ETMA in frontal, central and occipital cortical regions (Figs. 1,2,3). The findings indicate that inter-hemispheric imbalance may not be related to the limb movements.

In the occipital region, iRBD patients showed increased asymmetry in sigma and beta bands during tonic and phasic REM sleep with or without ETMA. As a central hub for cognitive processes, the thalamus regulates communication in the cerebral cortex, playing a critical role in memory, attention, and executive functions [49]. The disturbance of thalamo-occipital functional connection is related to cognitive deficits in various neurological diseases [50] and iRBD patients [51], and significantly weaker functional connection between NBM and the left occipital region in iRBD patients [40,52].

In this study, it was found there were no significant differences in EEG activity asymmetry of theta, alpha, sigma, beta, and gamma waves in the frontal, central and occipital regions between REM sleep with ETMA and without ETMA. In addition, no significant correlation was found between EEG activity asymmetry of these waves in all cortical regions during REM sleep (with and without ETMA) with the ratio of RSWA duration/REM sleep duration ($p > 0.05$). These findings suggest the ratio of RSWA/REM sleep per se might not be related to EEG activity asymmetry.

4.3 Comparisons of EEG Activities Between Different Regions on the Same Side of the Cerebral Hemisphere

In the iRBD patients and normal controls during tonic and phasic REM sleep, it was found that the EEG powers of theta, beta, and gamma bands in the frontal region were significantly greater than those in the central and occipital regions, and those in the central region were significantly greater than in the occipital region (Tables 2,3). The unique physiological functions of REM sleep, such as consolidation of emotional and procedural motor memory [53], might be taking place in the frontal cortex [54,55], which might result in more active EEG activity in the frontal region than other brain regions, as shown in the current study. During REM sleep, rhythmic waves of neuronal activities in the frontal region are dominated by theta and beta waves [56], and theta and beta waves in the anterior cingulate cortex and dorsolateral prefrontal cortex are also very active [56].

Theta waves were initially found in the hippocampus during spontaneous activity (e.g., walking or exploratory sniffing) and during REM sleep [53], but could also be recorded in many other cortical and subcortical structures [57]. The theta activity in the prefrontal lobe and its interaction with the hippocampus and amygdala during REM sleep may be important for the consolidation of emotional memory [56]. In addition, the theta rhythm during human REM sleep was more phasic than the tonic theta oscillations in rodents [58,59]. During REM sleep, beta waves have also been found to be active in the frontal and central regions [56]. This is in agreement with findings in the current study showing that EEG power of theta and beta waves in the frontal and central regions were significantly greater than those in the occipital region during tonic and phasic REM sleep.

Gamma waves are related to emotional processing, consolidation of memory, and dream recall [53,60,61], and may be a potential marker of the suppression of central adrenergic neurons participating in the encoding of emotionally salient events and activation in the amygdala-hippocampal system during REM sleep [61]. The amygdala is an emotional center of the brain, and the interaction between the amygdala and the hippocampus can regulate emotional recalls [60]. The inhibition of central adrenergic neurons during REM sleep plays a crucial role in emotional regulation, such as decreasing emotional intensity and defusing affective experiences [62].

Gamma as well as beta waves in the frontal region are associated with memory and emotional regulation [63,64], and may be interchangeable during memory regulation, because the EEG activities can switch from beta to gamma waves in a very short time [65]. It was shown in a previous study that no precise boundary occurs between beta and gamma waves, and that these waves might fluctuate simultaneously, such as increased EEG activities of both beta and gamma waves during cognitive processes involving memory [66]. It was shown in the current study that the EEG powers of theta, beta and gamma waves in the frontal region consistently were significantly greater than in the ipsilateral central and occipital cortical regions during tonic and phasic REM sleep (Tables 2,3). In addition, in the central region, these frequency bands were also significantly greater than in the ipsilateral occipital region.

The function of alpha and sigma waves during REM sleep is still not clear. Alpha and sigma waves mainly exist during NREM sleep, and high alpha activities may represent active preparation of the cerebral cortex for complex information processing, while sigma waves are associated with non-emotional memory consolidation during NREM sleep [67]. During tonic and phasic REM sleep, alpha and sigma powers in the frontal region were significantly greater than those in the central or occipital region, and those in central region were also significantly greater than in occipital region in the iRBD patients and normal

controls, forming a descending gradient (frontal > central > occipital) (Table 3). The long-range inter- and intra-hemispheric alpha and beta synchrony has been reported to be enhanced during tonic REM sleep, but decreased during phasic REM sleep [68]. During phasic REM sleep, cortical activity is largely disconnected from external environmental input, whereas during tonic REM sleep, sensory processing and attentional functions can still be partially maintained [5].

In short, in both iRBD patients and normal controls, interhemispheric asymmetry of EEG activities exists and EEG activities of theta, beta and gamma waves are unevenly distributed across different cortical regions of the same hemispheres during tonic and phasic REM sleep. In iRBD patients, inter-hemispheric asymmetry in EEG activities is significantly greater, which might reflect increased differences in neuronal activities between the left and right hemispheres.

4.4 Limitations

Although EEG activities were shown to be unevenly distributed across different cortical regions, and enlarged inter-hemispheric asymmetry in EEG activities in the iRBD patients, this study has some limitations. First of all, these included the selection of only male subjects. The rationale for this was because iRBD was more likely to occur in males than in females, and iRBD symptoms in males are more severe [2]. Therefore, gender differences in EEG activities during REM sleep could not be examined in this study. Second, the age range of the patients and controls was relatively narrow and the number of patients and controls was relatively small. Third, the cognitive functions of the iRBD patients were not systematically examined and the relationship between EEG activities during REM sleep and cognitive functions could not be investigated in this study. Fourth, patients with mental disorders have been excluded from this study and depressive comorbidity has been reported to be relatively common in iRBD [69]. Therefore, our findings may not be fully generalizable to iRBD populations with comorbid depression, and further studies in such populations are warranted. In addition, future studies could include more iRBD patients and controls, including females with a wider range of ages, and the cognitive functions of the iRBD patients will be systematically tested and long-term follow-up will be conducted.

Since the current study is cross-sectional study without follow-up data, EEG asymmetry in iRBD patients cannot serve as a predictive marker of disease progression or reflect the causal relationship of EEG asymmetry with the occurrence of iRBD.

5. Conclusion

In iRBD patients, EEG activities are unevenly distributed, with an altered inter-hemispheric asymmetry that might be associated with changed bilateral neuronal differences compared to normal controls.

Availability of Data and Materials

The data used to support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

ZZ, QL, XZ and DY contributed to the conceptualization and study design; ZZ, QL, QO, MW, CG, FY, and ZX acquired the data; ZZ, QL, XZ, and MW analyzed the data; ZZ, QL and DY drafted the original manuscript, reviewed and edited the final version. All authors have participated in drafting, revising, or critically reviewing the article, and approved the final version for publication. All authors agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The experimental protocol was reviewed and approved by the local Ethical Committee at the Jiangxi Provincial People's Hospital (No. 2020102) according to the ethical principles of the Declaration of Helsinki. Informed consent was obtained from all the subjects before participation.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/JIN47930.

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