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Entropy-based complexity of EEG microstates differentiates ADHD comorbidity in children with SeLECTS[☆]

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ABSTRACT

Self-limited epilepsy with centrotemporal spikes (SeLECTS) is frequently comorbid with attention-deficit/hyperactivity disorder (ADHD), yet objective neurophysiological markers for this comorbidity remain limited. In this work, we combined EEG microstate analysis with entropy-based complexity measures to characterize brain network dynamics and nonlinear signal irregularity during wakefulness and sleep. Resting-state 19-channel scalp EEG was recorded from 58 children with SeLECTS, including 38 in the SeLECTS+ADHD group and 20 in the SeLECTS-alone group. Four microstates were identified, and their temporal properties were quantified using time coverage and mean duration. Within each microstate, we computed global multivariate permutation entropy and assessed regional complexity using channel-level Shannon entropy and generalized entropy. Conventional microstate temporal parameters showed robust wake–sleep modulation in both groups but limited between-group discrimination. In contrast, microstate-resolved entropy measures showed clearer group differences, particularly during sleep, indicating reduced global complexity and localized regional alterations in the SeLECTS+ADHD group. Overall, these findings highlight a dissociation between microstate temporal dynamics and complexity features and suggest that sleep enhances the sensitivity of EEG complexity markers to ADHD comorbidity in SeLECTS. This integrated framework may provide candidate noninvasive biomarkers to support more objective identification of ADHD comorbidity in SeLECTS, pending validation in larger independent cohorts.

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1. Introduction

SeLECTS, formerly termed benign epilepsy with centrotemporal spikes, is the most common focal-onset epilepsy in childhood, estimated to account for 8%–25% of pediatric epilepsy diagnoses [1]. Although SeLECTS usually remits in adolescence, growing evidence suggests that up to half of affected children experience neurocognitive and behavioral difficulties, most notably ADHD [2]. ADHD occurs in 30%–65% of children with SeLECTS, substantially higher than the 5%–17% prevalence in the general pediatric population, and is associated with impaired social adjustment and reduced quality of life [3,4]. Given the high prevalence and clinical impact of ADHD comorbidity in SeLECTS, objective neurophysiological markers are needed to support timely identification and inform targeted clinical management.

Electroencephalography (EEG) has been widely used in the clinical evaluation of pediatric epilepsy [5]. In SeLECTS, syndromic diagnosis relies heavily on the characteristic pattern of centrotemporal spikes, which are markedly activated during sleep [6]. Despite their diagnostic value, these focal epileptiform discharges often fail to account for the substantial neurocognitive burden of SeLECTS, particularly its high comorbidity with ADHD. Timely identification of ADHD is essential for comprehensive care, yet behavioral screening is frequently confounded by seizure activity, cognitive side effects of anti-seizure medications, and reporting biases, underscoring the need for objective, physiology-based biomarkers [7]. Beyond conventional spike analysis, EEG contains rich information about brain network dynamics [8], which are increasingly recognized as central to the pathophysiology of both epilepsy and ADHD [9,10]. To capture the whole-brain dynamics of these networks, EEG microstate analysis has emerged as a powerful tool [11]. Microstates are quasi-stable scalp topographies that reflect the transient engagement of distinct large-scale neural networks [12,13]. Alterations in microstate temporal parameters, including mean duration, time coverage, and occurrence rate, have been reported across a range of neurological and psychiatric disorders, notably epilepsy [14] and ADHD [15], suggesting impaired network stability and reduced dynamic flexibility. Nevertheless, EEG studies specifically addressing comorbidity of SeLECTS and ADHD remain scarce, and EEG has yet to be fully leveraged for early clinical assessment. Identifying the EEG correlates of this comorbidity may therefore elucidate underlying neurophysiological mechanisms, thereby facilitating earlier detection and targeted interventions.

Complementing microstate analysis, entropy-based methods quantify the non-linear complexity and predictability of neural dynamics. These approaches have been widely utilized to extract EEG features for clinical tasks [16,17]. Measures such as multivariate permutation entropy provide a window into the richness and flexibility of brain dynamics [18]. Reduced neural complexity has been linked to constrained brain states, such as those in sleep [19], while altered complexity is increasingly recognized as a feature of neurodevelopmental disorders [20]. Recent studies highlight that entropy metrics can capture discriminative non-linear neural dynamics beyond the reach of conventional statistical or time-frequency features, such as in seizure classification [21]. More pertinently, integrating nonlinear entropy metrics with linear microstate parameters has proven highly effective in yielding robust biomarkers for identifying abnormal brain dynamics in conditions like insomnia [22] and obsessive-compulsive disorder [23]. By embedding entropy calculations within the microstate framework, we achieve a unified spatiotemporal view, assessing not only which network states are active and for how long, but also the complexity of the underlying neural activity during each state's engagement. Therefore, the present study leverages this integrated approach to uncover distinct neurophysiological signatures of comorbidity in SeLECTS, aiming to identify novel, objective candidate EEG biomarkers that support robust clinical differentiation.

In this study, we analyze resting-state EEG recorded during both wakefulness and sleep in children with SeLECTS, comparing those with and without comorbid ADHD. Our analytical pipeline begins with microstate analysis to extract four classes and quantify their temporal dynamics using mean duration and time coverage. Subsequently, to capture nonlinear signal complexity within each microstate, we compute global multivariate permutation entropy and map channel-level Shannon and generalized entropies. Finally, by systematically comparing these linear and nonlinear features between two groups and across the two recording conditions, we aim to comprehensively characterize the EEG alterations associated with ADHD comorbidity in SeLECTS.

2. Methods and materials

The schematic overview is shown in Fig. 1. We analyzed EEG recordings from 58 children with SeLECTS. For each participant, we extracted resting-state EEG segments from two recording conditions, wakefulness and sleep. We then applied a two-branch analytic framework. First, we performed microstate analysis using k-means clustering to identify four microstates (MS1–MS4) and quantified their temporal dynamics using mean duration and time coverage. Second, we assessed nonlinear signal complexity within each microstate by computing multivariate permutation entropy (MvPE) and channel-level Shannon entropy (SE) and generalized entropy (GE). Finally, we compared these measures between diagnostic groups, across the two conditions, and across electrode sites to characterize global and regional EEG alterations associated with ADHD comorbidity.

2.1. Subjects and neuropsychological evaluation

Children diagnosed with SeLECTS at the Department of Neurology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, were prospectively and consecutively enrolled between February 1, 2023 and October 31, 2024. Diagnosis was established from seizure semiology and electroencephalographic findings consistent with the 2022 International League Against Epilepsy (ILAE) framework for self-limited focal epilepsies in childhood [24]. Only cases with complete clinical documentation and technically adequate EEG recordings obtained in both wakefulness and sleep were eligible. Written informed consent was obtained from a parent or legal guardian for all participants; assent was additionally obtained from children aged

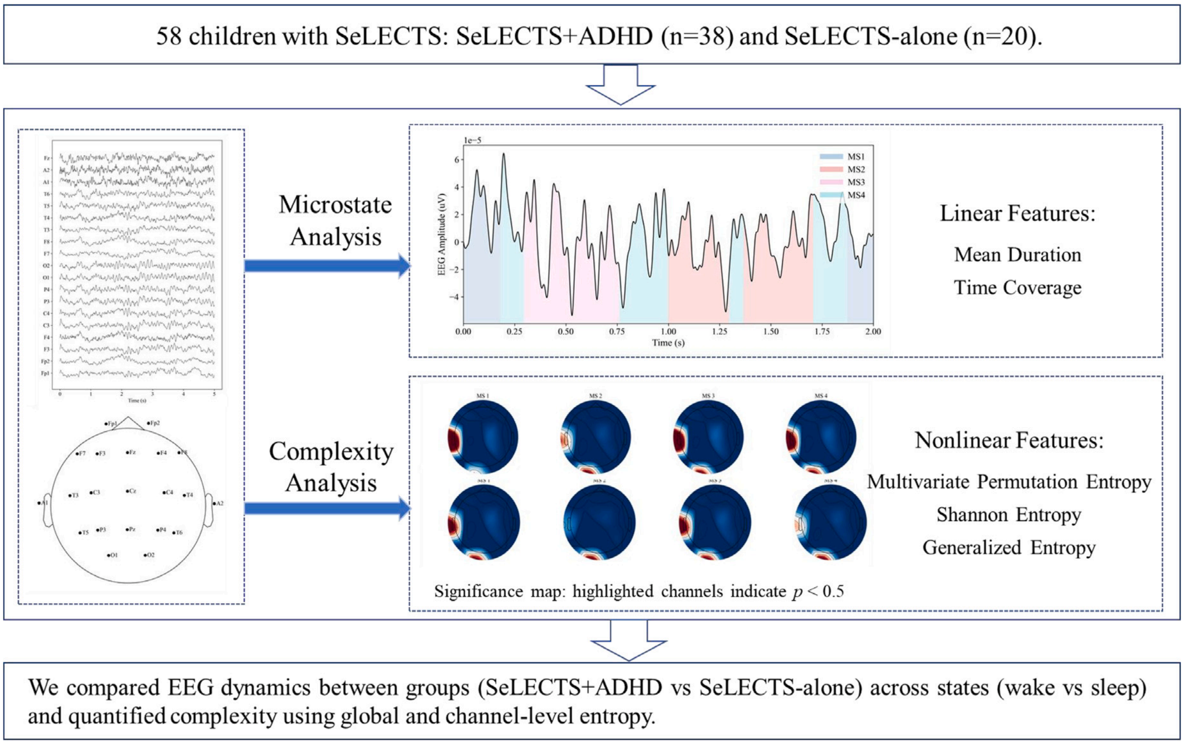

Fig. 1. Overview of the analytical framework.

≥8 years when developmentally appropriate. The study was approved by the Institutional Ethics Committee of Tongji Hospital and conducted in accordance with the Declaration of Helsinki. Exclusion criteria included: (1) evidence for an alternative epilepsy syndrome; (2) comorbid neurological or psychiatric disorders; (3) severe cognitive impairment precluding reliable cooperation during EEG acquisition.

Attention-deficit/hyperactivity symptomatology was assessed within one month of EEG acquisition using the Swanson Nolan and Pelham Rating Scale Version IV (SNAP-IV) parent or caregiver report [25], ensuring that the recorded EEG corresponded to the ADHD comorbidity status. The 26-item instrument comprises three domains—inattention, hyperactivity or impulsivity, and oppositional defiance—each rated on a four-point scale from 0 (not at all) to 3 (very much). Domain scores were calculated as the mean of all items within that domain, with higher scores indicating greater symptom severity. Operational thresholds were defined as follows: a mean domain score of 0 to 1.0 was considered normal, 1.1 to 1.5 borderline, 1.6 to 2.0 moderate, and greater than 2.0 severe. Children were classified as SeLECTS+ADHD group if any domain score was ≥ 1.6, or if a score between 1.1 and 1.5 was accompanied by at least five items rated 2 or 3 in that domain. All other children were assigned to the SeLECTS-alone group.

2.2. Participant characteristics and EEG acquisition

A total of 58 children who met all eligibility criteria were included in the analysis. All participants were right handed as determined by the Edinburgh Handedness Inventory following the scoring procedures of Oldfield [26]. Based on SNAP-IV parent/caregiver ratings, participants were assigned to either the SeLECTS+ADHD group (n = 38) or the SeLECTS-alone group (n = 20). The overall cohort comprised 36 males and 22 females, with a male-to-female ratio of 1.64:1. At the time of the study, the mean age of the participants was 11.45 ± 2.71 years, and their mean age at seizure onset was 7.43 ± 2.01 years. The median duration of epilepsy at enrollment was 40.0 months (interquartile range 27.75–68.50). All participants were classified as right-handed according to the Edinburgh Handedness Inventory. As summarized in Table 1, the two groups were well-matched. There were no significant differences between the two groups in terms of gender distribution, age, age at seizure onset, epilepsy duration, seizure types, or exposure to anti-seizure medication (all $p > 0.05$).

All participants underwent approximately 24-hour EEG monitoring using a 19-channel digital system (Natus Neuroworks, USA). Scalp electrodes were placed according to the international 10–20 system, with signals referenced to linked mastoids and sampled at 512 Hz. Following an initial 3-minute hyperventilation test, continuous EEG was recorded during wakefulness and sleep state. Finally, two certified epileptologists independently reviewed each recording to confirm the presence of EEG features consistent with a SeLECTS diagnosis.

Table 1
Comparison of clinical characteristics between two groups.

Index	SeLECTS+ADHD group (n=38)	SeLECTS-alone group (n=20)	Statistical value/P value
Gender [male, n(%)]	25 (65.79)	11 (55.00)	0.648/0.421
Handedness [right, n(%)]	38 (100)	20 (100)	/
Age ($\bar{x} \pm s$, year-old)	11.35 ±2.80	11.63 ±2.58	0.368/0.714
Age of onset ($\bar{x} \pm s$, year-old)	7.42 ±2.19	7.47 ±1.65	0.097/0.923
Disease course [M(Q_1, Q_3), month(s)]	36.00 (26.50, 70.50)	41.50 (36.25, 64.00)	–0.761/0.447
Seizure types [n(%)]			1.048/0.592
Focal aware or impaired awareness seizures only	20 (52.63)	10 (50.00)	
Focal to bilateral tonic-clonic seizures	14 (36.84)	6 (30.00)	
Both of the above two types	4 (10.53)	4 (20.00)	
ASMs [n(%)]			5.724/0.057
ASM monotherapy	13 (34.21)	13 (65.00)	
LEV	2 (15.38)	6 (46.15)	
OXC	2 (15.38)	3 (23.08)	
LCM	3 (23.08)	3 (23.08)	
VPA	4 (30.77)	0 (0.00)	
PER	1 (7.69)	0 (0.00)	
LTG	1 (7.69)	1 (7.69)	
ASM polytherapy	22 (57.89)	7 (35.00)	
two kinds of ASM	13 (59.09)	6 (85.71)	
three kinds of ASM	9 (40.91)	0 (0.00)	
four kinds of ASM	0 (0.00)	1 (14.29)	
Not yet taken ASM	3 (7.89)	0 (0.00)	

Note: There were no significant differences between SeLECTS+ADHD group and SeLECTS-alone group in the above clinical variables. ($P > 0.05$) ASM: anti-seizure medication; LEV: Levetiracetam; OXC: Oxcarbazepine; LCM: Lacosamide; VPA: Valproate acid; PER: Perampanel; LTG: Lamotrigine

For subsequent quantitative analysis, the EEG recordings were preprocessed using the MNE-Python environment [27]. Preprocessing steps included re-referencing the signal to the common average and applying a zero-phase bandpass filter (0.5–70 Hz). Following this, segments of EEG from wakefulness and sleep were selected. These data underwent careful manual review for the rejection of segments contaminated by movement, muscle, or ocular artifacts. The remaining artifact-free segments were then concatenated to yield approximately 4 min of analyzable signal from both the wake and sleep states for each participant.

2.3. EEG microstate analysis

Microstate analysis was used to characterize subsecond whole-brain topographic dynamics in artifact-free EEG during wakefulness and sleep. Analyses were implemented in custom Python routines built on MNE-Python. Following standard procedures, scalp field strength at each time point was quantified by the global field power (GFP), defined as the spatial standard deviation of voltages across electrodes [28]. Topographic maps at local GFP maxima were retained because they capture time points with high field strength and representative topographic configurations.

GFP-peak maps from all participants and both conditions were pooled and submitted to a k-means clustering procedure to derive a common set of microstate templates. A four-class solution was selected a priori, in line with the commonly used microstate model in resting-state EEG studies. The resulting templates (MS1-MS4) were then back-fitted to each participant's EEG by spatial correlation. At each time point, the class with the highest correlation was assigned.

From the resulting microstate sequences, we computed temporal parameters for each microstate and condition, focusing on time coverage and mean duration. Time coverage was defined as the proportion of analyzed time assigned to a given class, and mean duration was defined as the average length of contiguous segments assigned to that class. We prioritized the two measures because coverage captures the overall predominance of a microstate, whereas duration reflects its temporal stability once it is engaged. Metrics were computed separately for wakefulness and sleep for each participant and entered into subsequent statistical analyses at the group level and across conditions.

2.4. Entropy analysis

Nonlinear signal complexity within each microstate was quantified using three complementary entropy measures. Multivariate permutation entropy (MvPE) was computed as a global index to summarize multichannel temporal complexity and the diversity of joint patterns across distributed signals. To map regional variability, we calculated channel-wise Shannon entropy (SE) and Tsallis generalized entropy (GE), which provide spatially resolved indices of local irregularity. Specifically, SE measures the uncertainty of a probability distribution to capture the unpredictability of amplitude fluctuations at individual electrodes. Building upon this, GE generalizes SE through an adjustable parameter q, offering tunable sensitivity to rare versus frequent fluctuations in complex neurophysiological recordings. Together, these measures enable a comprehensive assessment of both global and regional alterations in EEG dynamics.

For each multichannel segment, we constructed delay-embedded vectors using an embedding dimension of $m = 4$ and a time delay of $\tau = 1$. These parameter values were selected based on established recommendations for electrophysiological signals, ensuring an optimal balance between adequately capturing the nonlinear dynamical complexity of EEG time series and avoiding artifacts from finite data length [29,30]. At each time step, we formed a joint ordinal pattern by ranking the embedded samples across the D channels, thereby preserving the relative ordering of multivariate observations. We estimated the probability distribution $P(\pi_i)$ of all distinct joint ordinal patterns π_i over the segment and computed MvPE as:

$$\mathrm{MvPE} = -\sum_{i=1}^{N_p} P(\pi_i)\,\log P(\pi_i),$$

where N_p denotes the number of unique joint patterns. Higher MvPE values indicate greater diversity of multivariate temporal patterns and higher global signal complexity.

For each channel, the amplitude distribution was estimated using equal-width histogram binning to obtain a discrete probability mass function $\{p_i\}_{i=1}^{n}$, with the number of bins determined using the Freedman–Diaconis rule. SE was then computed as

$$SE = -\sum_{i=1}^{n} p_i \log p_i, \tag{1}$$

where n denotes the number of bins and p_i is the probability that amplitudes fall within the ith bin. Higher SE indicates greater uncertainty in the channel amplitude distribution [31,32].

GE was computed using the Tsallis formulation

$$GE = \frac{1}{q-1}\left(1 - \sum_{i=1}^{n} p_i^q\right), \quad q \neq 1, \tag{2}$$

with a fixed q applied across all analyses to ensure comparability across channels and conditions, following previous applications of Tsallis entropy to EEG analysis [33,34].

2.5. Statistical analysis

Microstate temporal metrics were tested for within subject differences between wakefulness and sleep and for between group differences within each condition. Parametric or nonparametric tests were selected based on distributional checks.

Entropy analyses were conducted in a separate step. MvPE was computed as a global multichannel complexity index for each microstate and condition, and we evaluated both within subject wake-sleep changes and between group differences using the same testing scheme. For SE and GE, we computed channel-level values within each microstate. We first summarized these values at the subject level by taking the mean across channels and tested group and condition effects on these summary measures. When significant effects were observed, we performed channel-wise comparisons to localize electrodes contributing to the overall differences. Multiple comparisons across microstates, entropy metrics, and channels were controlled using Bonferroni correction.

3. Results

3.1. Microstate temporal dynamics

As shown in Fig. 2, we examined microstate time coverage across MS1–MS4 using two sets of comparisons: (i) between-group differences within each condition and (ii) within-subject differences between wakefulness and sleep within each group. Within both groups, paired wake–sleep comparisons revealed a clear modulation of coverage, with higher coverage of MS1 and MS3 and lower coverage of MS2 and MS4 during sleep. In contrast, between-group comparisons within wakefulness or within sleep showed largely overlapping coverage distributions, and no between-group differences survived multiple-comparison correction across microstates.

We next examined mean duration (Fig. 3), which reflects how long each microstate persists once it appears. In both groups, mean duration was consistently higher during sleep than during wakefulness for MS1 to MS4, consistent with more sustained microstate dynamics during sleep. Between-group differences were generally small, but MS2 showed a notable divergence, with a significant group difference in mean duration during wakefulness.

Overall, these two figures demonstrate that microstate temporal dynamics are primarily differentiated by recording condition, whereas diagnostic group effects are limited when using linear timing metrics.

3.2. Nonlinear dynamic analysis

To assess whether comorbid ADHD is associated with altered EEG complexity in SeLECTS, we performed an entropy analysis within microstate segments. For each of the four microstates, we first computed MvPE as a global multichannel complexity index. We then computed SE and GE at the channel level to map spatially localized differences. This approach enables complementary evaluation of global and regional complexity changes across wakefulness and sleep.

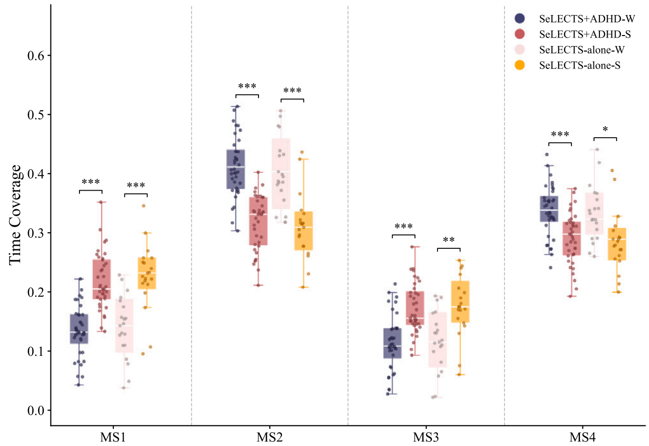

Fig. 2. Microstate time coverage in children with SeLECTS with and without comorbid ADHD during wakefulness and sleep. Participants: SeLECTS+ADHD ($n = 38$) and SeLECTS-alone ($n = 20$). Boxplots show the distribution across participants. Asterisks indicate significant differences (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$).

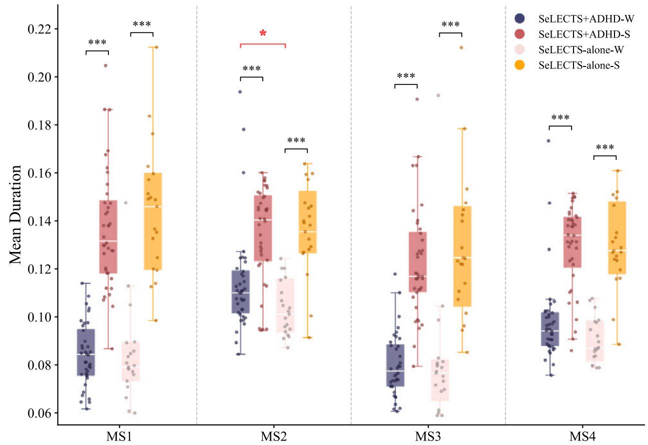

Fig. 3. Microstate mean duration in children with SeLECTS with and without comorbid ADHD during wakefulness and sleep. Participants: SeLECTS+ADHD ($n = 38$) and SeLECTS-alone ($n = 20$). Boxplots show the distribution across participants. Asterisks indicate significant differences (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$).

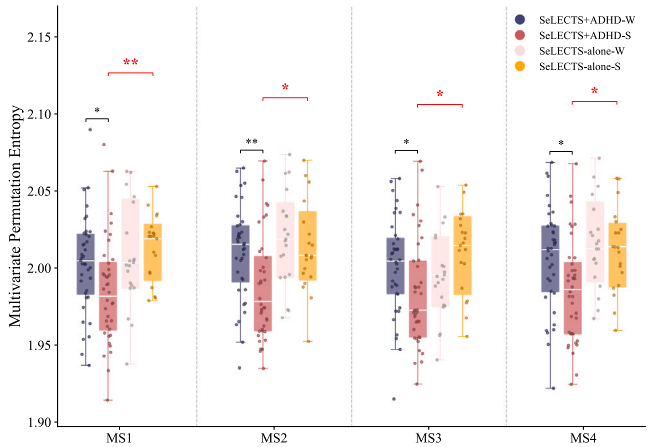

Fig. 4. Global multichannel complexity across microstates. Boxplots show MvPE values for MS1-MS4 in children with SeLECTS with and without comorbid ADHD during wakefulness and sleep. Participants: SeLECTS+ADHD ($n = 38$) and SeLECTS-alone ($n = 20$). Asterisks indicate significant pairwise differences (* $p < 0.05$, ** $p < 0.01$).

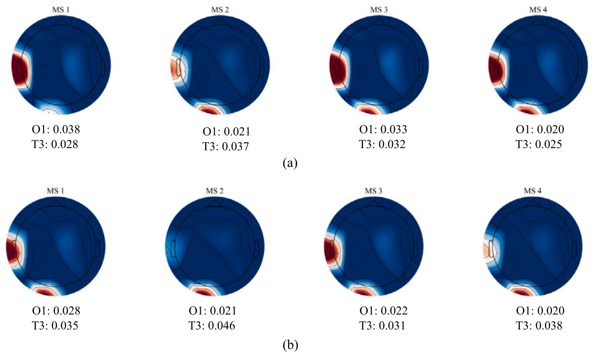

Fig. 5. Topographic maps of significant channel-level between-group differences in entropy measures during sleep across microstates MS1–MS4. (a) Shannon entropy; (b) generalized entropy. Highlighted regions mark the scalp locations of electrodes exhibiting significant differences.

3.2.1. Global complexity analysis

Fig. 4 summarizes the distribution of MvPE values across MS1–MS4 in children from the two groups.

First, between-group differences were evident primarily during sleep. Across all four microstates, the SeLECTS+ADHD group showed lower MvPE than the SeLECTS-alone group in the sleep condition, with the clearest separation observed in MS1 and significant between-group differences also present in MS2–MS4 after Bonferroni correction. These sleep-related group differences were accompanied by moderate-to-large effect sizes (Hedges' g ranged from -0.81 to -0.73 across MS1–MS4), indicating lower global complexity in the SeLECTS+ADHD group during sleep. In contrast, the corresponding wakefulness comparisons showed substantial overlap between groups and no significant between-group differences across microstates. Second, within-group comparisons revealed different wake-to-sleep modulation patterns across diagnoses. In the SeLECTS+ADHD group, MvPE decreased from wakefulness to sleep across all four microstates, indicating a consistent reduction in global complexity during sleep. By contrast, in the SeLECTS-alone group, MvPE remained relatively stable between wakefulness and sleep, with no significant state-related differences across microstates.

Taken together, the MvPE results indicate that global complexity differences associated with ADHD comorbidity are evident during sleep and are accompanied by a sleep-related reduction in MvPE that is specific to the SeLECTS+ADHD group.

3.2.2. Regional entropy mapping

To localize scalp regions contributing to group differences in complexity, we quantified channel-level entropy within each microstate using Shannon entropy and generalized entropy. For wakefulness and sleep separately, SE and GE were computed at each electrode, and between-group differences were tested within each microstate.

No electrodes showed significant between-group differences for either entropy measure during wakefulness. During sleep, however, significant effects were confined to two focal scalp sites, located over the left temporal and left occipital regions. This spatial pattern was consistent across microstates for both entropy measures, yielding a reproducible topographic signature as shown in Fig. 5. No other electrodes reached significance in any microstate for either measure.

4. Discussion

In this study, we investigated EEG recorded during wakeful rest and sleep in children with SeLECTS with and without comorbid ADHD using an integrated microstate and entropy framework. Across both groups, microstate timing measures showed robust modulation from wakefulness to sleep, whereas between-group differences in linear timing features were limited. In contrast, microstate-based entropy measures provided clearer group differentiation during sleep, suggesting that sleep may amplify the sensitivity of nonlinear complexity features to ADHD comorbidity in SeLECTS.

The wake-to-sleep modulation of microstate timing observed here is consistent with prior work reporting that sleep is associated with longer microstate durations and more persistent temporal organization [35]. In this work, both time coverage and mean duration changed systematically from wakefulness to sleep in the SeLECTS+ADHD and SeLECTS-alone groups, indicating broadly similar state-dependent reorganization of microstate temporal structure. The relative scarcity of between-group effects in timing measures suggests that ADHD comorbidity is not strongly expressed in linear microstate occupancy or persistence measures under resting conditions.

By comparison, the complexity analyses revealed more pronounced condition-specific group differences. MvPE showed clear group separation during sleep but not during wakefulness, and the comorbidity group exhibited a stronger reduction in MvPE from wakefulness to sleep. Within the same microstate-defined network configurations, this pattern is consistent with multichannel dynamics becoming more regular or less diverse during sleep in the comorbidity group [36]. More generally, reduced neural complexity is often interpreted as a constrained dynamical repertoire, and these results highlight the value of complementing microstate timing with nonlinear complexity metrics that capture signal irregularity beyond occupancy and duration. Channel-level entropy mapping further indicated that sleep-related group differences were spatially heterogeneous rather than widespread.

Significant effects were confined to a focal left temporal site and a focal left occipital site across microstates and across both entropy measures. The consistency of this topographic pattern for Shannon entropy and generalized entropy suggests that the observed global complexity differences are not uniformly distributed across the scalp.

Taken together, our findings suggest a complementary feature for characterizing ADHD comorbidity in SeLECTS. Linear microstate timing features primarily reflect state modulation shared across groups, whereas microstate-based complexity measures, particularly during sleep, show greater sensitivity to diagnostic differences. Microstate segmentation provides a structured way to quantify complexity within comparable network-state configurations, which can improve interpretability when linking nonlinear dynamics to specific microstate classes.

Several limitations of the present study should be acknowledged. First, the modest sample size, combined with multiple-comparison correction, may have constrained statistical power and reduced sensitivity to smaller effects. Second, the use of a standard 19-channel EEG montage inherently limits spatial precision. Third, sleep was analyzed as a single, undifferentiated condition; future work is needed to determine whether the observed complexity alterations are specific to distinct sleep stages. Finally, because non-linear entropy estimates are sensitive to parameter selection and data length, these findings should be validated in larger, independent cohorts with high-density, stage-resolved, and source-informed EEG datasets.

5. Conclusions

In this work, we applied an integrated microstate and entropy framework to resting-state EEG recorded across wakefulness and sleep in children with SeLECTS, comparing those with and without comorbid ADHD. Microstate timing metrics exhibited robust wake-to-sleep modulation in both groups, while between-group differences in these conventional parameters were limited. By contrast, microstate-resolved entropy measures showed clearer group separation during sleep, with lower complexity in the SeLECTS+ADHD group at both the global level and the regional level. Overall, these results indicate that microstate-resolved complexity features, especially in sleep, provide complementary information beyond standard timing metrics and represent candidate EEG markers for differentiating ADHD comorbidity in SeLECTS.

CRediT authorship contribution statement

Qing Cai: Writing – original draft, Methodology. **Jianpeng An:** Writing – original draft, Methodology, Investigation, Conceptualization. **Mengying Wang:** Methodology, Data curation, Conceptualization. **Zhongke Gao:** Writing – review & editing, Writing – original draft, Methodology, Funding acquisition. **Matjaž Perc:** Writing – review & editing, Supervision, Methodology, Investigation. **Huicong Kang:** Writing – review & editing, Supervision, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

Data will be made available on request.

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