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***Abstract*—Rapid-eye movement (REM) Sleep Behavior Disorder (RBD) is a parasomnia characterized by the loss of physiological muscle atonia during REM sleep, often resulting in dream enactment and violent behaviors. Idiopathic RBD is recognized as a prodromal marker of alpha-synucleinopathies, with conversion rates reaching 73–96% within 12–14 years. Despite its clinical relevance, diagnosis relies on polysomnography (PSG) in specialized sleep laboratories, limiting its accessibility and scalability, and its heterogeneity remains underexplored. To support remote health monitoring and early neurological assessment, this study proposes a sleep-stage-independent, unsupervised Machine Learning framework based on a single EEG channel collected during home-based PSG. In a dataset of 32 individuals with RBD, the method consistently identified two clusters with distinct neurophysiological profiles. These data-driven subgroups also showed differences in the REM Atonia Index (RAI), a quantitative marker of REM muscle atonia and RBD severity, supporting the interpretability of the identified neurophysiological patterns. These findings highlight the feasibility of low-complexity, unsupervised EEG analysis for characterizing RBD heterogeneity, suggesting the feasibility of scalable telehealth solutions for continuous monitoring and early detection of neurodegenerative risk.**

***Index Terms*—EEG, Unsupervised Learning, REM Sleep Behavior Disorder, RBD, Machine Learning**

I. Introduction

The growing demand for continuous, accessible, and patient-centered healthcare is promoting the development of telemedicine solutions capable of supporting diagnosis and monitoring outside traditional clinical environments. Sleep disorders remain particularly underserved in this transition, as gold-standard assessments rely on overnight polysomnography (PSG), a resource-intensive procedure entailing the recording of multiple biosignals during sleep and performed in specialized sleep laboratories, thus imposing instrumental, logistical, geographical, and economic barriers [1]. These challenges are especially critical for Rapid-eye Movement (REM) Sleep Behavior Disorder (RBD), a parasomnia featuring the loss of physiological muscle atonia during REM sleep. RBD affects approximately 1% of individuals aged 60 or older worldwide [2] and is recognized as one of the strongest prodromal markers of α-synucleinopathies, including Parkinson's Disease (PD) and Dementia with Lewy bodies (DLB) [3]–[5]. Since RBD often precedes overt neurodegeneration by several years [6], early detection and longitudinal monitoring are essential for timely clinical intervention, disease progression tracking, and potential enrollment in neuroprotective trials [3]. However, access to specialized sleep laboratories remains limited and unevenly distributed, leading to substantial delays in diagnosis and follow-up. Moreover, RBD evaluation requires manual scoring of muscle atonia and behavioral events during REM sleep from video-PSG recordings [7], a process that is labor-intensive, time-consuming process and inherently limits scalability, particularly for older patients who may face mobility constraints.

In this context, low-complexity, home-deployable monitoring solutions are becoming increasingly needed in order to ensure equitable access to diagnostic and prognostic assessments [1]. Recent advances in wearable sensing and Machine Learning (ML) have demonstrated the possibility to automatically detect RBD in real-world settings [8], [9]. In particular, single-channel electroencephalography (EEG) and automated data-driven analyses offer promising opportunities to reduce acquisition and scoring complexity [10]–[12], thus enabling scalable remote monitoring and supporting clinical decision-making. Despite this progress, the characterization of RBD heterogeneity, which might reflect distinct or overlapping pathophysiological trajectories and thus provide crucial insights into prodromal neurodegenerative patterns, remains underexplored. Specifically, assessing whether such variability can be captured outside laboratory condition represents a key step toward clinically meaningful telemonitoring of RBD.

This study proposes a sleep-stage-agnostic, unsupervised framework to assess the feasibility of exploring RBD heterogeneity from single-channel EEG acquired in home-based PSG recordings. By applying data-driven clustering to identify latent electrophysiological subgroups and clinical correlates, the proposed analysis investigates whether meaningful variability can be captured in real-world settings.

II. Materials

A. Subjects and Sleep Recordings

The study dataset included 32 subjects diagnosed with RBD (26 males, aged 62.2 $\pm$ 6.4 years) recruited at the Regional Sleep Centre of Molinette University Hospital (Turin, Italy).

Each participant underwent a full-night PSG recording acquired in their home environment using a portable PSG system (*Somté PSG*, Compumedics), recording 3 EEG channels, 1 electromyography channel (EMG), and 1 electrocardiography channel (ECG). The home-based data acquisition framework was adopted to reduce patient burden, support real-world data collection, and evaluate the feasibility of lightweight monitoring systems for RBD. All procedures were conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the A.O.U. Città della Salute e della Scienza di Torino (Approval No.: 00384/2020).Written informed consent for participation in the observational study was obtained from all subjects.

B. Signals

From the collected signals, a single EEG channel (C3, or C4 when C3 was unavailable) was selected for the analysis; this choice ensured continuity with prior experimental protocols [10], while supporting non-intrusiveness. In addition to EEG, the chin EMG channel (recorded from the mylohyioid muscle) was used to compute the REM Atonia Index (RAI) [13], [14], which served as the clinical reference measure of RBD severity. No other PSG signals were included in the present analysis. This reduced-channel configuration was adopted to assess the feasibility of a low-complexity, scalable monitoring system suitable for deployment beyond the sleep laboratory and compatible with wearable monitoring environments.

III. Methods

A. Data Pre-processing

Minimal preprocessing was applied to all signals, to preserve compatibility with pervasive and wearable monitoring scenarios. The EEG recordings were bandpass filtered (0.5–40 Hz) through a cascade of two zero-phase, anti-causal Butterworth filters to attenuate slow drifts and high-frequency noise. The EMG signal was bandpass filtered (10–100 Hz), following established international guidelines [7] and to allow for the computation of the RAI, as further detailed in Section III-E.

B. Feature Extraction from EEG

A comprehensive multi-domain feature extraction procedure was applied to the EEG signal, incorporating commonly used descriptors from the literature [15]–[18]. Features were grouped into four domains: time domain (TD), frequency domain (FD), time-frequency domain (TFD), and non-linear dynamics (NLD), to capture morphological, spectral, non-stationary and non-linear properties of sleep EEG, reflecting the neurophysiological mechanisms of the signal. The full list of extracted features is reported in Table I. Features were computed over 30-second epochs to align with the American Academy of Sleep Medicine (AASM) scoring criteria. For FD, TFD, and NLD descriptors, 2-second sub-epochs were used, to ensure local signal stationarity. The resulting feature distributions were then summarized using statistical descriptors (Mean, Standard Deviation, 75$^{\text{th}}$ percentile, and Main Mode), each employed as a distinct feature in the dataset.

TABLE I
FEATURES EMPLOYED IN THE STUDY, ACCORDING TO THEIR DOMAIN. ◇: ADAPTED FROM CITED STUDY.

Feature and Domain	**Reference**
Time Domain	
Mean, mode, STD, skewness, kurtosis, maximum and minimum values, Range	◇ [16]
Zero Crossing Rate	◇ [15]
Hjorth Parameters (activity, mobility, complexity)	◇ [19]
Hjorth-based parameters (sparseness, irregularity, spectrum bandwidth, average-zero-up, crossing period, average-peak-to-peak period)	◇ [17]
Percentiles (25, 75), interquartile range	◇ [16]
Form, Crest, and Impact factors	[17]
Coastline	[19]
Frequency Domain	
Absolute Power Spectrum, Mean Power Spectrum	◇ [16]
Peak Frequency, Mean Frequency (Spectral Centroid), Median Frequency (SEF50), SEF25, SEF75, SEF95, SEFd75-25, SEFd95-25, SEFd95-50	◇ [16], [17], [19]
Spectral Crest, Spectral Entropy, Renyi's quadratic Entropy	[15]
Frequency (EEG sub-bands)	
Max Value, Relative Power Spectrum, Mean Power Spectrum, Absolute Power Spectrum	◇ [16], [17]
Spectral Centroid, Spectral Spread, Spectral Skewness, Spectral Kurtosis, Variance of Central Frequency	◇ [17]
Peak Frequency, Median Frequency (SEF50), SEF95, SEFd95-50	◇ [19]
Spectral Entropy, Renyi's quadratic Entropy	◇ [15]
Time-Frequency	
6 levels Discrete Wavelet Analysis (DWT), db4 (Mean, Standard Deviation, Coastline, Ratio of absolute mean values of adjacent sub-bands)	◇ [16]
Non Linear	
Detrended Fluctuation Analysis	[15]
Higuchi Fractal Dimension	◇ [16]
Sample Entropy, Taiger-Kaiser Energy Operator (Mean, Standard Deviation, Skewness, Kurtosis, Maximum value)	◇ [15], [17]

C. Statistical Analysis, Feature Normalization, and Dimensionality Reduction

After feature extraction, all features were standardized using z-score normalization to ensure comparability with different units and dynamic ranges and to prevent features with larger absolute values from disproportionately influencing the subsequent dimensionality reduction process. Then, to obtain a compact and informative subset of features suitable for lightweight and potentially wearable-oriented analysis pipelines, Principal Component Analysis (PCA) was applied to the set of features. PCA is a non-parametric linear dimensionality-reduction method that identifies orthogonal components capturing the greatest variance in the data. By projecting the original feature space onto these principal components (PCs), it reduces redundancy and highlights the sources of inter-subject variability. The cumulative explained variance of the ordered components was employed to quantify the effectiveness of the dimensionality reduction, and a scree plot was used to identify the most informative PCs. The component loadings were then inspected,

and the absolute contribution of each feature to each PC was computed.

D. Unsupervised Characterization through Clustering

Three unsupervised clustering methods were explored, namely: K-Means, Agglomerative, and Spectral Clustering. Clustering quality was assessed using three complementary internal validation indices: the Silhouette score, the Calinski-Harabasz (CH) and Davies-Bouldin (DB) indices. The Silhouette score evaluates the consistency of each sample within its assigned cluster by considering intra-cluster cohesion and inter-cluster separation. It ranges from -1 to 1, with values closer to 1 representing well-separated and compact clusters. Similarly, the CH index measures the ratio of between-cluster variance to within-cluster variance; although not presenting fixed bounds, larger CH values are associated with higher cluster cohesion. Finally, the DB index quantifies the average similarity between each cluster and its most similar neighbor; lower DB values indicate greater inter-cluster separation and more homogeneous cluster structure.

E. REM Atonia Index

The RAI [14] was computed and used as a clinical correlate and validation parameter, due to its established robustness as an indicator of REM sleep without atonia [20]. The RAI quantifies the extent of muscular atonia during REM sleep and is derived from the submental EMG signal, evaluated in 1-second epochs. Following the procedure introduced by Ferri et al. in [13], [14], the raw EMG signal was first bandpass filtered (10–100 Hz, as described in III-A) and rectified. The average amplitude of each epoch was then computed, and the RAI was defined as the proportion of epochs with amplitude below 1 μV. The index ranges from 0 to 1, with values near 1 indicating physiological atonia; values below 0.8 are strongly indicative of impaired chin EMG atonia during REM sleep and therefore serve as a clinical marker of RBD [14], [20]. For each participant, the RAI was computed across all REM periods of the night.

F. Clinical Validation and Statistical Tests

As introduced earlier, to clinically characterize the identified clusters, the RAI was used as the reference physiological marker, given its established reliability in quantifying chin EMG atonia during REM sleep and its relevance for RBD phenotyping.

Statistical analyses were conducted to assess whether the clusters differed according to their RAI distributions. First, the Shapiro-Wilk test was used to evaluate normality within each cluster. Given the presence of non-normal distributions, the primary group comparison was conducted using the Mann-Whitney U test. Then, to quantify the magnitude of the group difference, two effect size measures were computed: Cohen's d and the rank-biserial correlation (r). These metrics allow for the contextualization of the statistical findings and support the interpretation of cluster separation observed in the reduced PCA feature space.

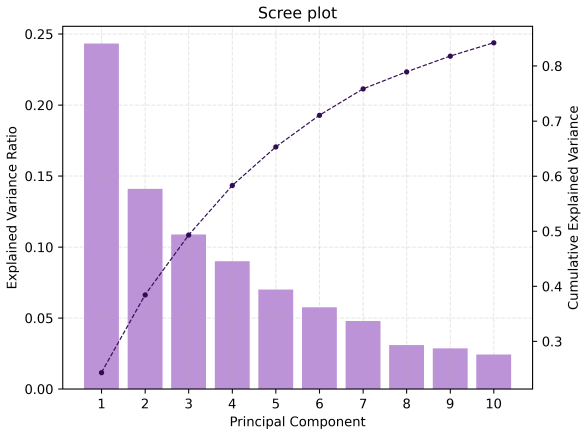

Fig. 1. Scree Plot of the first 10 principal components, reporting the explained variance ratio for each component, and the cumulative variance.

IV. Results

A. Feature Inspection and PCA

A total of 212 features were extracted across the four considered domains (TD, FD, TFD, and NLD), as detailed in Section III-B. Each feature was summarized using four statistical descriptors, resulting in an initial dataset of 848 variables. To limit redundancy and reduce noise, pairwise correlations were examined, and features with strong inter-correlation ($|\rho| > 0.8$) were removed. This step ensured that the resulting feature set preserved the most informative and non-redundant predictors for subsequent dimensionality reduction through PCA. Then, as detailed in Section III-C, PCA was applied to derive a compact representation of the feature space. The first three PCs explained 24.3%, 14.1%, and 10.9% of variance respectively, accounting for a cumulative 49.3% (Figure 1). Although the variance captured by each PC seems moderate, this pattern is consistent with the distributed information content of EEG features [21], particularly in unsupervised, stage-agnostic approaches such as this, without prior class structure guiding feature selection. Inspection of the scree plot showed diminishing returns after PC3; hence, the first three PCs were retained for the subsequent clustering analysis. This choice provided a balance between dimensionality reduction and the retention of neurophysiologically meaningful variability, while avoiding the curse of dimensionality in the following unsupervised characterization.

B. Clustering Analysis and Neurophysiological Patterns

K-means clustering was applied to the first three PCs, and the optimal number of clusters was identified using the elbow method, which indicated k=2 as the best solution (Figure 2). This two-cluster configuration yielded moderate but satisfactory clustering performance (Silhouette: 0.41, CH: 18.1, DB: 1.22). The stability of this structure was supported by the convergent results of agglomerative and spectral clustering, both of which produced similar partitions and comparable metrics (Table II). These findings suggest that the underlying

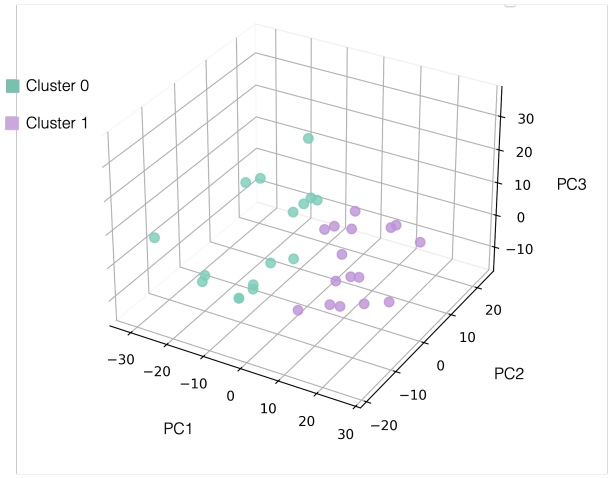

Fig. 2. Two-cluster configuration obtained through the K-Means algorithm.

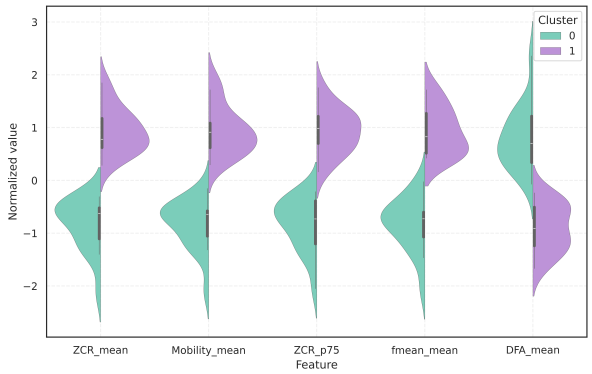

Fig. 3. Distribution of the top-5 features contributing to Principal Component 1 in the two clusters.

subgroup organization is robust across different unsupervised approaches.

Although the Silhouette scores were moderate (0.38 ± 0.03 across the three methods), such results might suggest the continuous and overlapping nature of biological phenotypes and neurophysiological variability, particularly in disorders with heterogeneous phenotypes such as RBD.

Despite this moderate overlap in PCA space, the clusters exhibited clear differentiation when examined through their constituent EEG features. Figure 3 illustrates the five features with the strongest absolute loadings on PC1 (24% explained variance), namely: Zero Crossing Rate (ZCR, mean and 75th percentile), Mobility, Mean Frequency, and DFA exponent. All displayed statistically significant differences between clusters ($p < 0.05$), demonstrating that the identified groups reflect meaningful and interpretable neurophysiological distinctions, observable through lightweight, interpretable EEG features. In more detail, Cluster 0 was characterized by lower-frequency activity (reflected by reduced ZCR, Mobility, and Mean Frequency) and increased signal regularity. Conversely, Cluster 1 exhibited higher spectral activity and reduced long-range temporal correlations (lower DFA). These patterns collectively suggest the presence of two distinct EEG profiles within the RBD cohort, despite partial overlap in the latent PCA space.

C. REM Atonia Index across Clusters

As described previously, given that RBD clinical heterogeneity remains currently poorly characterized, the RAI was employed as a candidate clinical correlate of the identified neurophysiological subtypes (Section III-E). Indeed, it serves as a strong predictor of REM sleep without atonia, a hallmark feature of RBD pathophysiology.

Figure 4 displays RAI distributions in the two clusters, showing two distinct patterns. Cluster 0 featured higher RAI values with larger variability (0.342 ± 0.341; median: 0.22), whereas Cluster 1 showed lower and more compact values (0.196 ± 0.28; median: 0.07). This pattern suggests that Cluster 0 encompasses a broader spectrum of atonia impairment, while Cluster 1 represents a more homogeneous subgroup with consistently reduced atonia.

Statistical testing was conducted to formally evaluate these distributional differences. Normality tests (Shapiro-Wilk) indicated non-normal distributions in both groups (Cluster 0: W=0.866, p=0.019; Cluster 1: W=0.731, p=0.0005), leading to subsequent non-parametric statistics. The Mann-Whitney U test indicated a trend toward higher RAI values in Cluster 0, although it did not reach conventional significance (U=156.0, p=0.289). Effect sizes suggested a small-to-moderate difference between clusters (rank-biserial correlation $|r| = 0.22$, Cohen's $d = 0.47$). The modest effect size and statistical outcomes suggest a genuine, though subtle, difference in atonia levels in the two underlying neurophysiological subtypes; a post-hoc power analysis indicated that the current sample size provided approximately 25% power to detect an effect at α=0.05, suggesting that the study was underpowered to establish significance, despite observable distributional differences (Figure 4). Overall, the distinct central values and variability profiles across clusters suggest potentially meaningful clinical heterogeneity in REM atonia patterns. While these findings remain preliminary, they support the feasibility of identifying RBD subtypes through home-based EEG analysis and highlight the need for larger cohorts to strengthen statistical conclusions.

TABLE II
CLUSTERING PERFORMANCE OF THE EXPLORED METHODS, IN TERMS OF SILHOUETTE SCORE, CALINSKI-HARABASZ AND DAVIES-BOULDIN INDICES.

Algorithm	Silhouette	CH score	DB score
K-Means	0.41	18.1	1.22
Agglomerative	0.35	16.7	1.28
Spectral	0.38	15.98	1.23

V. DISCUSSION

REM sleep behavior disorder presents with substantial clinical heterogeneity in symptom severity, progression rates, and

risk of conversion to synucleinopathies. Yet, the neurophysiological mechanisms underlying this variability remain poorly characterized and understood, partly because conventional assessments focus primarily on the evaluation of REM sleep without atonia [7], potentially overlooking subtler neurophysiological dimensions that distinguish patient subgroup. Moreover, such approaches require cumbersome instrumentation that limits accessibility and large-scale or longitudinal remote monitoring.

This study was designed as a feasibility investigation, aiming to assess whether home-based, whole-night, single-channel EEG recordings acquired without laboratory supervision could capture meaningful neurophysiological heterogeneity in patients with RBD, by applying unsupervised ML methods without imposing *a priori* clinical classifications. Indeed, by relying on unobtrusive EEG systems suitable for repeated home use, this framework enables longitudinal and continuous assessment of sleep-related neurophysiological patterns in real-world conditions, overcoming the episodic and resource-intensive nature of laboratory PSG. The adopted clustering approach consistently identified a two-cluster configuration across three independent algorithms (K-Means, agglomerative, and spectral clustering), with comparable evaluation metrics (Silhouette score: 0.38 ± 0.03). Although moderate, these values align with the nature of biological phenotypes in clinical populations, that follow continuous, rather than discrete, distributions. Furthermore, the presented outcomes highlight the promising nature of the identified subtypes, particularly given the robust statistical differences observed at the feature level (Figure 3). Five features emerged as the strongest contributors for clustering: ZCR (mean and 75^{th} percentile), Mobility, Mean Frequency, and DFA exponent, and exhibited robust and statistically significant differences between clusters (p <0.005), indicating that the subtypes reflect coherent and physiologically interpretable differences in cortical activity during sleep. Indeed, the obtained feature patterns suggested a dissociation between spectral content and temporal organization, thus possibly describing two neurophysiological subtypes. Cluster 1, characterized by elevated high-frequency activity and reduced long-range temporal correlations, may reflect more profound alterations in more advanced disruption of sleep-regulating networks. Conversely, Cluster 0 featured slower activity with preserved temporal structure, indicating maintained sleep architecture despite the presence of RBD, potentially representing earlier disease stages. The larger internal variability of Cluster 0 further supports the possibility that this subgroup may encompass a broader clinical spectrum or include latent subtypes that were not fully resolved with the current sample size. Finally, the relationship between the identified EEG subtypes and the RAI was investigated. A preliminary visual inspection (Figure 4) revealed distinct distributional characteristics: Cluster 0 exhibited higher RAI values with greater variability, while Cluster 1 showed lower values and a more constrained distributions. Although formal statistical testing revealed no significance, with effect sizes in the small-to-moderate range, post-hoc power analysis revealed that the non-significant result likely reflects insufficient sample size rather than lack of association. Indeed, the observed effect size (Cohen's d of 0.47) suggested the presence, though subtle, of a relationship between cortical EEG patterns during sleep and muscle atonia, that requires larger cohorts to be definitely characterized. Furthermore, since the RAI primarily reflects brainstem function, the partially independent behavior of cortical and subcortical EEG features might suggest distinct pathophysiological trajectories within RBD, that need further investigation, possibly with multimodal datasets.

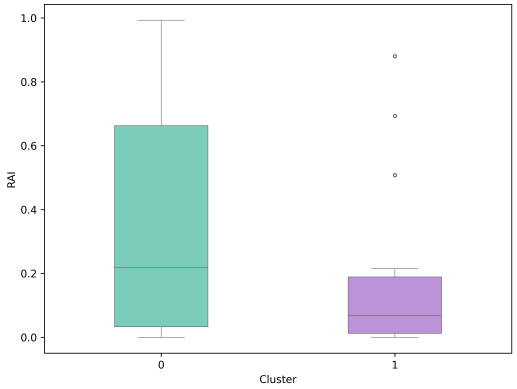

Fig. 4. Boxplots displaying the REM Atonia Index distribution in the two observed clusters.

To the best of the authors' knowledge, this study represents the first attempt to characterize RBD subtypes through unsupervised learning applied to home-recorded single-channel EEG; hence, a direct comparison with previous work is challenging. Nonetheless, the physiological characteristics emerging from the identified clusters (particularly the extent of muscle atonia, and the slowing of EEG frequency) align with findings from previous studies focusing on PD patients with comorbid RBD [22], thus supporting the validity of the obtained results.

Despite preliminary, the results of this pilot study highlight the potential of unsupervised, data-driven approaches for phenotype characterization in heterogeneous neurological disorders. Specifically, the use of home-based, whole-night recordings demonstrates the feasibility of monitoring RBD heterogeneity outside traditional laboratory settings, potentially enabling larger-scale studies with true ecological validity.

However, a few limitations require consideration.

First, the modest sample size limited statistical power for detecting subtle inter-cluster differences and prevented a stratified analysis. Hence, the identified subtypes should be further explored in larger, independent cohorts. Second, the moderate clustering metrics reflect the intrinsic heterogeneity and continuity of neurophysiological subtypes; future implementations should adopt mixture modelling or fuzzy clustering techniques to better capture subtler transitions and reveal latent subtypes. Third, while the single-channel approach was intentionally

adopted to demonstrate home-feasibility, it may overlook regional cortical dynamics. Future investigations should focus on the optimal sensor placement and explore alternative approaches based on other physiological metrics, such as heart rate variability or actigraphy. Finally, validation on a diverse cohort is needed to address cross-sectional analysis of the temporal stability of subtypes or their prognostic significance, and assess whether population characteristics (e.g., disease duration, comorbidities, or medication status) may influence generalizability.

Hence, future work will address the ecological validity of this framework on a larger cohort, possibly with longitudinal design, and explore whether subtypes can offer prognostic value for predicting conversion patterns to α-synucleinopathies (e.g., PD or DLB) or cognitive outcomes; preliminary investigations are currently ongoing.

VI. Conclusion and Future Work

This pilot study demonstrated that unsupervised monitoring of home-based, whole-night EEG recordings can identify distinct neurophysiological subtypes within the RBD population, providing an objective characterization of disease heterogeneity without requiring an extensive clinical infrastructure. Although preliminary, these findings highlight the potential of non-intrusive, low-complexity EEG markers as scalable tools for patient stratification. Indeed, the explored approach enables repeated, home-based monitoring of sleep physiology, allowing continuous real-world assessments and supporting the development of personalized follow-up and therapeutic strategies.

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