Detection of REM Sleep Behavior Disorder based on EEG–EMG Coupling methods

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Content

1 Introduction

1.1 Background of RBD Detection

Rapid Eye Movement Sleep Behavior Disorder (RBD) is a parasomnia disorder that is defined by a loss of the normal muscular atonia of the REM sleep, hence resulting in complex and often violent enactments in the dreams. Idiopathic RBD (iRBD), that is presented without any secondary causes, has received an increasing scholarly interest due to its close relationship to a -synucleinopathies. The long-term prospective studies prove that the iRBD is one of the most stable prodromal indicators of Parkinson Disease (PD), and Dementia with Lewy Bodies (DLB) and Multiple System Atrophy (MSA) [1], [2]. Postuma et al. (2015) noted that over half of iRBD patients develop neuro-degenerative synucleinopathy in a 1015-year period, whereas recent reviews suggest conversion rates of 8090 per cent with longer follow-up [1], [2], [3]. At the same time, the manifestations of RBD are observed in approximately 40 percent of PD patients and reach up to 70 percent-90 percent in patients with DLB or MSA [4], which contributes to supporting the fact that it is a good biomarker of early progression of neurodegenerative diseases.

The gold standard for RBD diagnosis is the detection of REM Sleep Without Atonia (RSWA) using overnight polysomnography (PSG) [5]. Traditional diagnostic approaches rely on visual scoring of electromyographic (EMG) activity, with criteria such as the Montreal Method defining tonic and phasic EMG patterns based on amplitude thresholds and event durations [6]. Subsequent refinements, including normative multi-muscle EMG thresholds proposed by the SINBAR group, have improved diagnostic standardization by establishing robust definitions for tonic and phasic RSWA across larger cohorts [7]. Despite these advances, significant inter-scorer variability remains, prompting efforts to validate automated RSWA scoring tools, such as Ikelos-RWA and other algorithms demonstrating increasing agreement with expert manual scoring [6], [8]. These developments highlight both the importance and the current limitations of EMG-based RBD assessment.

1.2 Problem Statement

Current automated diagnostic instruments of RBD are mainly based on the REM-phase EMG (e.g.- SINBAR rule, RBDtector, Ikelos-RWA etc.), that report a high sensitivity and specificity in identifying classic RSWA. However, the approaches effectively measure peripheral muscle activity and fail to provide the upstream dysregulation patterns throughout the whole cortical-brainstem-spinal cord motor inhibition pathway. Specifically, at times RSWA is subtle or EMG quality is sub-optimal, the sensitivity of these instruments to prodromal or early RBD is not determined [8].

EEG studies have shown that, in patients with iRBD, there is widespread cortical impairment, which is characterised by slowing of the EEG, loss of peak frequency, changes in the 1/f spectral slope and loss of REM -phase, -inhibition. These neurophysiological alterations are associated with strong correlations with cognitive decline and the risk of conversion into a -synucleinopathy. These EEG measures however are mainly indicative of diffuse cortical impairment, and do not have a particular correlation to the occurrence of particular motor release behaviours in the state of REM sleep, thus limiting their diagnostic specificity to the behavioural phenotypes of RBD [3].

A few studies have used corticomuscular coherence (CMC) to establish significant increases of 2 -band EEG -EMG coupling in iRBD patients in REM sleep. Such a measure of the degree of coupling is linked to the intensity of clinical symptoms, which suggests that abnormal cortical motor drive could be a core pathophysiological process in RBD. However, the available EEG-EMG coupling literature suffers limitation due to limited sample sizes, offline, and lack of a full evaluation framework, quantitative thresholds, and a testable pipeline that would be compatible with standardised automated EMG RSWA quantification instrumentations.

1.3 Project Aim

To overcome these drawbacks, studies made more recently are progressively combining the EEG and EMG measurements to investigate cortico-muscular communication in the REM sleep [9]. Coherence-based analyses measure the synchronisation between the oscillations of cortex and the muscles, which would give a direct measure of information transfer between the peripheral motor output and the central nervous system. There is empirical evidence of increased corticomuscular coherence in patients with iRBD compared to control groups indicating which reflect a compensatory or pathological change in the dynamics of the motor network even at an early or preclinical phase [9], [10]. Therefore, these measures can serve as neurophysiological red flags of dysfunctional motor inhibition circuits, which are the hallmarks of synucleinopathy-related RBD.

The current project will seek to describe EEG-EMG patterns of coupling in RBD and determine whether they can improve diagnoses and, therefore, help identify high-risk individuals early on and understand the aspects of the neural mechanisms underlying RSWA. This method attempts to bridge the gap between the conventional EMG scoring and a mechanistic and biomarker-based system to explain the pathophysiology of RBD.

2. Literature Review

2.1 review of existing RBD detection

2.1.1 Traditional visual scoring methods

Early diagnosis of RBD primarily relied on manual observation of abnormally increased electromyographic (EMG) activity during the REM sleep phase in sleep physiotherapy (PSG)[11]. In the initial clinical experience phase, there were no unified quantitative standards; sleep specialists assessed the degree of loss of REM sleep dystonia based on whether the chin muscle tone was significantly higher than normal. Some early studies attempted to establish qualitative interpretation methods. For example, Montplaisir et al. [12] proposed the first scoring method based on chin EMG in 1992, classifying EMG activity per 30-second epoch of REM sleep into two categories: tonic (sustained high amplitude) and paroxysmal (brief EMG bursts), and found that RBD patients exhibited significantly more of both types of EMG activity than normal individuals. Subsequently, the "Montreal method" (developed by the Montplaisir team) introduced specific diagnostic thresholds in 2010[13]: if more than 30% of the 30-second segments during REM sleep show tonic electromyography (EMG) thickening, or more than 15% of the 3-second microsegments show paroxysmal EMG activity, then abnormal atonia during REM sleep can be diagnosed.

Applying these thresholds, the accuracy in distinguishing patients with idiopathic RBD from normal controls can reach approximately 82%–84%. During the same period, other sleep medicine centers also developed their own interpretation criteria. For example, the Mayo Clinic in the United States and centers in Innsbruck and Barcelona in Europe proposed criteria based on their own case experience, and subsequently validated the diagnostic efficacy of EMG indicators in their studies.[14]

In 2012, the Innsbruck and Barcelona group (SINBAR)[7] published standardized REM electromyography (EMG) values based on 30 patients with recurrent sleep deprivation (RBD) and 30 controls

The SINBAR criteria are characterized by adding multiple muscle channels, such as the upper limbs (e.g., biceps brachii or wrist flexors), in addition to chin EMG, and defining specific calculation methods for REM EMG activity. For example, if chin EMG "tonicity" persists for more than 10% of the entire night's REM sleep time, or if 3-second micro-segments of "paroxysmal" activity account for more than 16%, it suggests the possibility of RBD[10]. These results have been incorporated into the American Academy of Sleep Medicine (AASM) handbook and the international RBD research group guidelines, becoming standardized criteria for human interpretation[14].

It is worth noting that manual interpretation of multiple muscle leads has been proven superior to chin EMG alone: the SINBAR study found that limb EMG abnormalities are

more sensitive to RBD, improving the detection rate compared to chin EMG alone. Current international guidelines[15] also recommend recording multiple EMG channels of the chin and upper and lower limbs during video PSG assessment of RBD to improve diagnostic reliability.

Overall, traditional manual scoring methods have significant value in RBD diagnosis, but they suffer from drawbacks such as being time-consuming and having significant subjective differences among interpreters. Historically, different laboratories have used different standards, limiting the comparability of interpretation results. These limitations have driven the emergence of quantitative indicators and automated analysis methods.

2.1.2 Quantitative Indices and Automated Algorithms

To quantify the degree of loss of electromyographic inhibition during the REM phase, researchers have introduced various quantitative indicators. The most representative of these is the REM Atonia Index (AI)[16]. AI quantifies the degree of muscle tone inhibition by calculating the overall amplitude distribution of the chin muscle electromyography signal during REM sleep. The value ranges from 0 to 1, with values closer to 1 indicating more complete muscle tone inhibition (atonicity). Ferri et al. improved the calculation method of AI by first rectifying and averaging the electromyographic signal, and then subtracting the local low-noise baseline to more reliably reflect the actual electromyographic activity.

Besides AI, electromyographic activity density is also a commonly used quantitative parameter. For example, "tetanic density" refers to the proportion of high-amplitude, sustained electromyographic activity occurring for more than half of the 30-second REM phase; "paroxysmal density" refers to the proportion of short bursts of electromyographic activity occurring in 3-second micro-segments during the REM phase. The density of "any activity" is a comprehensive statistical measure of the overall proportion of any type of electromyographic abnormality.[11]

The thresholds of ≥30% for tonic and ≥15% for paroxysmal attacks recommended by Montplaisir et al. [13]are essentially these density indicators, and practice has proven that these thresholds are highly effective in clinical diagnosis.

To improve diagnostic accuracy, new combined and quantitative indicators have been introduced in recent years. For example, the SINBAR index adds the percentage of "arbitrary activity" of chin electromyography to the percentage of "paroxysmal activity" of forearm electromyography, and proposes a 32% cutoff value to distinguish RBD.

In addition, the duration of electromyographic bursts was also used for quantification: the average duration of a single paroxysmal electromyographic activity during REM phase was often longer in RBD patients (approximately 0.65 seconds for the mentalis muscle,

compared to approximately 0.2–0.3 seconds in the control group; approximately 0.79 seconds for the arm muscles, compared to shorter durations in the control group).[14]

In summary, these quantitative indicators provide an objective measure of the degree of loss of REM muscle tone, which can be used to determine the diagnostic threshold and as an assessment tool for the severity or progression of RBD.

2.1.3 Automated algorithms

With the development of computing technology, numerous studies have begun to explore automated algorithms to detect abnormal electromyography (EMG) during REM sleep. A typical method is rule-driven threshold detection: programs are written according to manual standards (such as SINBAR or Montreal thresholds) to automatically determine whether EMG values exceed twice the background level in each 30-second or 3-second segment, and for how long, thereby statistically analyzing the density of tonic and paroxysmal activity. This method is equivalent to formulating the manual interpretation process, thus its results are comparable to manual standards. Frauscher et al. [17] a software integrated with a PSG device that can automatically calculate the "arbitrary, paroxysmal, and rigid" indices of electromyography (EMG) of the mentalis and forearm muscles according to the SINBAR criteria. It is worth noting that purely automated algorithms are prone to false positives when electromyography artifacts are present. Frauscher et al. found that after technicians spent approximately 5 minutes quickly checking and removing obvious artifacts, the software's false positive rate decreased significantly by more than 40%, indicating that combining manual correction with automated algorithms can improve reliability.

Besides the rule-based thresholding method, some studies have employed feature engineering and machine learning to extract various statistical or frequency domain features from electromyography (EMG) signals, and then used classification algorithms to identify recurrent partial dysplasia (RBD). For example, it is reported promising results using data-driven algorithmic models to train EMG data[18].

In summary, the rule-driven and feature engineering approach demonstrated the feasibility of automatically quantifying REM electromyography abnormalities in this study, which can significantly reduce manual workload and improve objectivity consistency. However, these algorithms use different feature sets and discrimination criteria, resulting in poor consistency among different methods.

2.1.4 Open Source Standardized Tools

To promote the standardization of RBD electromyography analysis, researchers have developed open-source tools, among which the RBDtector software is a representative example[11]. The tool can read EU EDF sleep data format and automatically analyze

electromyographic signals from three channels: the chin (mentalis muscle), the upper limb (wrist flexor FDS), and the lower limb (tibialis anterior muscle TA). RBDtector strictly follows the SINBAR rules to detect electromyographic events in each channel, quantifying REM phase electromyographic activity into three categories: "tonic," "paroxysmal," and "arbitrary."

In preliminary verification, the RBDtector results showed a high degree of agreement with human scoring, and the correlation R^2 of most electromyographic parameters between humans and the machine exceeded 0.8, especially for paroxysmal and voluntary movements of the forearm muscles, where the correlation reached over 0.9.

RBDtector analyzed data from 174 independent subjects, showing that the electromyographic indices calculated by RBDtector were significantly higher in the RBD group than in the control group. Receiver operating characteristic (ROC) curve analysis revealed that combining the percentage of voluntary mentalis muscle activity with voluntary forearm muscle activity as the criterion yielded the best discriminative effect. This indicates that data-driven methods can optimize thresholds and improve detection rates without sacrificing specificity. Besides RBDtector, other teams have released open-source or shared tools for RSWA analysis, but their impact is limited or they are restricted to specific formats. Overall, RBDtector is the first fully validated open-source RBD electromyographic analysis tool that adheres to international standards[19], representing a significant step towards the standardization of REM atonicity quantification.

2.1.5 Performance Evaluation and Challenges of Automated Algorithms

The widespread use of automated algorithms in clinical practice is still facing a plethora of challenges. First of all, the subjectivity inherent in human scoring makes it difficult to conduct an algorithmic evaluation. Clinical specialists tend to show low inter-rater reliability in understanding rapid eye movement sleep without atonia (RSWA). Besides, algorithmic discrepancies: When different threshold paradigms are used, such as the Montreal or SINBAR criteria; although each of the individual algorithms can be very accurate, the results may not be comparable with each other. This has thus created a pressing demand to have a globally agreed standard or a common evaluation system that can be used to directly compare algorithm performance. Thirdly, automated analysis can be challenged by signal contamination and co-morbid conditions. Electromyographic channels also contain some of the most susceptible sources of artifacts, such as electrocardiographic interference, electrode motion, electromechanical noise, and snoring induced chin vibrations. A possible solution is to incorporate modules of automated artifact detection in the analytic pipeline. Empirical experiments show that the error rates can be significantly reduced by simple rule-based filters that either block high-amplitude

cardiac artifacts or localized artifacts of high frequency. Moreover, concomitant sleep disorders are common in the patients with the rapid eye movement sleep behavior disorder (RBD), and among them, obstructive sleep apnea (OSA). OSA may induce common micro-arousals, intermittent muscle tone restorations, thus boosting electromyographic action of REM and producing pseudo-RSWA.

2.2 EEG Biomarkers in RBD

In the clinical practice, the most commonly used modality to identify recurrent idiopathic brain dysregulation (RBD) is not electroencephalography (EEG), due to the fact that the hallmark of RBD is perturbed muscle tone, which is best measured with electromyography. However, EEG can give significant information on the activity of the central nervous system and cortical dysfunction, thus explaining the pathophysiological processes. The present review analyzes the salient EEG characteristics related to RBD, areas of clinical importance, and limitations intrinsic to them.

2.2.1 Cortical EEG Abnormalities

2.2.1.1 Global EEG Slowing

Additional to muscular activity, electroencephalography shows mild but extensive cortical functional change in persons who experience recurrent idiopathic brain disease (iRBD). Among the most noticeable ones is a generalized deceleration of the EEG, similar to the spectral pattern of early-stage Parkinson's disease (PD) or dementia with Lewy bodies (DLB) [20]. There are quantitative EEG studies that have indicated increased theta-delta (4-8 Hz 0.5-4 Hz) band power with corresponding decrease in the prevalent alpha rhythm frequency in iRBD patients [21]. This non-focal gradual slowing is indicative of mild, diffuse cortical impairment or a preclinical encephalopathic condition. The degree of baseline EEG slowing further has prognostic importance because the degree of slowing has been related to cognitive loss and a higher probability of advancing to clinically significant synucleinopathy [22]. Rodrigues Braz et al. showed that patients with iRBD whose background EEG rhythms were slow were better off to develop mild impaired cognitive or DLB over the course of time [22]. Therefore, resting EEG results can be considered as early biomarkers of the neurodegenerative load in RBD.

2.2.1.2 Spectral Slope decrease

In addition to the traditional frequency-band analysis, more recent studies have investigated neural noise and excitation-inhibition balance through the description of non-periodic 1/f spectral slope of EEG signal in RBD. The spectral slope is an index of cortical excitability or arousal state. A flatter slope represents comparatively elevated

power of the high-frequency, which signifies extensive cortical activity or noise, whilst steeper slope depicts supremacy of low-frequency power [23]. The steepest slope is observed in the case of healthy people during the REM and deep sleep and gradually grading to a shallow sleep during the wakefulness, hence coinciding with the increased cortical activity when awake. In RBD, 1/f slope changes are well quantified, but there has been a paucity of studies that consolidate evidence of altered spectral dynamics that are in line with aberrant excitability. It is important to note that, RBD participants show a weakened inhibited response to high frequency (b) EEG activity during REM and this essentially flattens the REM EEG spectrum as compared to normal sleepers [24]. Such reduction of the usual low to high frequency ratio of power during REM is an indicator of an increased cortical excitability. Combined, these 1/f -like changes could be due to neurochemical aberration, and could possibly create an ambience of motor disinhibition. Overall, increased neural excitability is directly reflected in flatter 1/f spectral slope, which supports the idea that pathophysiology-related abnormal spectral slopes are closely interconnected with RBD.

2.2.2 REM Sleep Oscillatory Dynamics

2.2.2.1 Failure of Beta Suppression

Sometimes under normal circumstances, the REM sleep sometimes begins with a distinctive EEG pattern; elevated theta, decreased muscle tone, and relative inhibition of power within the beta (1530Hz) band relative to during wakefulness [24]. This beta-decay is an indication of a decrease of cortical arousal during the REM, thus protecting the dream state. One of the characteristic abnormalities observed in the patients of the RBD is the failure to sufficiently suppress the activity of beta during the REM, especially at the stage of the REM-sleep [25]. The frontal cortex is the part of the brain, which empirical data point to as the area that has the most beta dysregulation in RBD [26]. Considering the centrality of the frontal cortex in motor control, it is likely over-excited in REM and, as such, over-excitation of the frontal cortex may trigger sleepwalking. Essentially, long-lasting beta power during REM i.e. failure to attain normal beta decay is a characteristic EEG dynamic phenomenon of RBD. This effect is an indication of the loss of the cortical soothing effect of normal REM sleep and thus the association of abnormal beta persistence with the pathogenesis of sleepwalking.

2.2.2.2 Slow-Wave Activity

Furthermore, slow-wave activity (SWA, power <4 Hz) in RBD patients exhibits an unusual trajectory at night. Normally, SWA gradually decreases during subsequent sleep cycles as steady-state pressure dissipates. In iRBD patients, the typical SWA decrease is not observed in the second half of the night, possibly due to cortical hyperexcitability or

reduced synaptic amplitude during sleep [32,33]. Microscopic analysis of pre-dreaming EEG in RBD patients by Kim et al. [27] revealed that RBD patients failed to exhibit the normal pre-REM muscle tone EEG pattern. In RBD patients, cortical EEG does not "decline" before movement; instead, if a decline occurs, it is observed as an increase, which may reflect a surge in cortical drive that triggers dreams. Date et al. noted the existence of an unstable transitional state in which protective inhibition fails and motor network dysfunction occurs [28].This seems to be related to the deficit in patients with RBD, who have significantly lower frontal slow wave power at the start of the night, thus reducing the space for further decline. Since slow waves are fundamental to synaptic renormalization and memory consolidation, weakened SWA dynamics indicate impaired neuroplasticity in RBD. Sunwoo et al. [29] recently provided very conclusive evidence of morphological abnormalities in slow waves during naps in RBD patients, including reduced amplitude and slope.

2.2.2.3 Microstructural Abnormalities

Rapid eye movement sleep behavior disorder (RBD) alters not only spectral power but also the microstructure of REM sleep, especially before the occurrence of abnormal behavior. High-resolution analysis of the 60 seconds before significant somnambulism in RBD patients showed a surge in frontal cortex delta wave power, along with spikes in high-frequency (γ) power in the frontal and occipital regions [28]. At the same time, whole-brain frequency functional connectivity (interregional synchrony) was significantly enhanced before the occurrence of somnambulism. Essentially, premotor EEG showed simultaneous enhancement of cortical resting rhythm (delta waves) and hyperarousal markers (β-γ activity). This high-low frequency mixture suggests unstable or fluctuating arousal state or transient microarousing or state transitions before the release of complex motor activity. These microstructural abnormalities may be triggers for motor events in RBD. Other studies supporting this view have used REM sleep microstructural analysis (e.g., periodic alternation pattern CAP [10]) to study iRBD and found that certain arousal oscillation patterns during REM sleep may be associated with neurodegenerative risk.

2.2.2.4 Clinical Significance

In summary, EEG findings in rapid eye movement sleep behavior disorder (RBD) consistently indicate diffuse cortical dysfunction and imbalance of REM sleep network activity. Slowing of background rhythm, reduced alpha wave peak, altered 1/f spectral slope, and REM-specific beta wave enhancement all indicate changes in the patient's brain. In a pioneering study, blinded quantitative EEG analysis was performed on iRBD patients with baseline cognitive impairment; after approximately 2.5 years of follow-up,

those who later developed mild cognitive impairment showed significantly slower baseline EEG (increased theta-delta waves in the central/occipital leads), while those who maintained normal cognition did not [30]. Furthermore, event-related EEG during REM sleep revealed that the ratio of slow to fast wave power in the occipital lobe during REM sleep could predict phenotypic transformation of iRBD with considerable accuracy (AUC≈0.75). Its performance in predicting neurodegenerative outcomes is superior to clinical indicators, such as subjective RBD severity[31].

2.3 EEG-Based RBD Detection

Rapid eye movement sleep behavior disorder (RBD) is characterized by muscle atrophy and loss during REM sleep, resulting in dream-like behaviors. There is growing interest in electroencephalographic (EEG) biomarkers for RBD. This interest stems from the recognition that isolated RBD (iRBD, idiopathic RBD without obvious neurological disease) is often an early manifestation of underlying neurodegenerative synucleinopathy.

Therefore, following standard EMG-based RBD testing, EEG measurements are being investigated to measure the physiological effects of RBD on the brain and potentially improve early diagnosis and risk stratification for conversion to PD or DLB.

In the following sections, we review the major EEG findings in RBD. We begin with the broad pattern of EEG slowing and alpha peak frequency reduction observed in RBD, then discuss REM sleep-specific spectral anomalies. Next, we examine studies of EEG microstructure and connectivity in RBD – including microstates, network coherence, and complexity metrics as potential biomarkers. We then highlight applications of machine learning and deep neural networks that use EEG-only data for RBD detection or prognostication. We also compare EEG findings across idiopathic RBD, PD patients with RBD, and DLB, noting commonalities and differences. Finally, we consider the limitations of incorporating EEG into mainstream RBD diagnostics and discuss future prospects for EEG-based RBD detection.

2.3.1 Machine Learning and Deep learning method

As databases of electroencephalograms (EEGs) from patients with recurrent brain degeneration (RBD) continue to expand, researchers are turning to machine learning (ML) and deep learning to automatically detect RBD or predict its outcomes using only EEG features (excluding muscle or eye-motor pathways). The motivation lies in the potential of machine learning algorithms to discover complex, multivariate patterns in EEGs, thereby distinguishing RBD from normal sleep or predicting which RBD patients will develop Parkinson's disease/Lewy body dementia. This is particularly attractive for developing screening tools or decision support systems that go beyond what human experts can discern from EEGs.

Early machine learning research focused on classical classifiers using artificially designed electroencephalogram (EEG) features. For example, studies have trained support vector machines or random forests on feature sets such as EEG spectral features, coherence values, or entropy measures to distinguish between patients with rapid eye movement sleep behavior disorder (iRBD) and controls. Deep learning can automatically learn feature representations from raw or minimized EEGs, usually by converting EEG signals into images and using convolutional neural networks (CNNs). Ruffini et al. [32] obtained resting-state EEG segments from a large number of iRBD patients and healthy controls and tracked which iRBD patients later developed Parkinson's disease (PD) or Lewy body dementia (DLB). The network was able to distinguish between healthy controls and iRBD patients who later developed neurodegenerative diseases with about 80% accuracy and an area under the curve of 87% when using the optimal single-channel spectrogram input. Compared with controls, RBD converts showed more frequent theta bursts and relatively fewer alpha bursts on EEG. Deep networks independently rediscovered the feature of EEG slowing, providing a new solution for feature design.

Another application is predicting the phenotypic transition time of iRBD. The best predictive performance was achieved using only EEG features extracted from PSG during REM sleep. Although the authors' proposed EEG slowing feature to explain iRBD patient stratification has not yet been validated on a large scale, they implemented a fully automated transition prediction model based on PSG[33].

In the diagnostic field, machine learning can automatically detect rapid eye movement sleep behavior disorder (RBD) from electroencephalography (EEG). Classifiers trained based on EEG features and heart rate or activity recorder features have been validated to distinguish RBD patients from controls with encouraging accuracy (typically between 80% and 90%) [34]. However, many of these studies include electromyography (EMG) or motion signals, as motor activity is a hallmark of RBD. Pure EEG methods are more challenging. With the increasing prevalence of deep learning models such as convolutional neural networks (CNNs), recurrent neural networks (RNNs), and Transformers in EEG analysis, we can expect more optimized detection algorithms that may capture subtle RBD features that standard polysomnography (PSG) scores cannot identify.

2.3.2 Limitations and Future Potential of EEG

Despite numerous studies demonstrating EEG abnormalities in RBD, EEG has not yet become a mainstream clinical tool for the diagnosis or prognosis of RBD. The diagnostic criteria for RBD remain focused on EEG-polysomnography (EPSG) of loss of muscle tone during rapid eye movement (REM) and behavioral manifestations. On average, cognitively normal iRBD patients have essentially normal awake EEG spectral

characteristics, which may be caused by a variety of conditions (e.g., Alzheimer's disease, cerebrovascular changes, drug effects) [35]. In addition, detecting microstructural or connectivity EEG indicators often requires advanced offline analysis, which presents certain technical challenges. Furthermore, conditions such as obstructive sleep apnea (OSA) can cause slowed EEGs due to hypoxemia or sleep fragmentation. Most EEG markers reflect generalized cortical dysfunction rather than specific neural events during REM sleep that lead to complex movements. Slowed alpha wave power or increased theta wave power suggests the presence of overall encephalopathy, but does not directly explain why patients kick or punch during sleep [26].

Therefore, while EEG abnormalities help us better understand RBD as a brain disease and may help identify high-risk patients, they cannot serve as specific diagnostic criteria for the unique behavioral phenotypes of RBD. This has prompted researchers to explore EEG-EMG coupling, directly studying the relationship between brain waves and muscle activity, in order to bridge the gap between the central and peripheral manifestations of RBD.

2.4 EEG–EMG Coupling Analysis in RBD

2.4.1 Corticomuscular Coupling

A promising approach to understanding and detecting RBD is to analyze corticomuscular coherence (CMC). This is a frequency domain measurement of the coupling between electroencephalogram (EEG) signals (cortical activity) and electromyogram (EMG) signals (muscle activity). CMC effectively quantifies how synchronized oscillations in the brain drive muscle activation. It is classically used in studies of motor control during wakefulness, with significant coherence in the β band (15–30 Hz) reflecting communication between the corticospinal cord and stable motor outputs such as maintaining posture or constant force.

In rapid eye movement (REM) sleep, healthy individuals should generally have little sustained brain-muscle coupling because voluntary motor commands are blocked by atonic neural circuits in the brainstem. However, in idiopathic RBD, the presence of movement indicates that this isolation has failed. Jung et al. first provided evidence of abnormal CMC in REM sleep in patients with RBD: using central EEG leads (C3/C4) and EMG of the jaw and leg muscles, they found significantly increased β band coherence in 11 untreated iRBD patients [45,46]. In healthy sleepers, coherence at ~20 Hz was close to zero (as expected in REM), but was significantly elevated in RBD patients (mean CMC ≈ 0.1–0.2 range, p ~0.07 control group) [45,46]. This suggests that cortical oscillations are abnormally propagated to muscles during REM in RBD subjects, consistent with the detection of neural signal leakage in RBD.

Subsequent larger-scale studies have confirmed and expanded this finding. Choi et al. analyzed 105 individuals and confirmed that REM β coherence was significantly elevated in RBD patients [36]. Interestingly, Choi et al. found that patients with RBD who also had Parkinson's disease (either PD or DLB) had REMCMC levels similar to those in patients with idiopathic RBD. This result may indicate that elevated CMC is a marker of the RBD state itself, rather than PD. These results support the possibility that the degree of electromyography insensitivity may increase with the progression of neurodegenerative disease.

2.4.1 Coherence Analysis Methods

2.4.1.1corticomuscular coherence (CMC)

EEG–EMG coupling is commonly quantified through corticomuscular coherence (CMC), defined as:

$$C_{xy}(f) = \frac{|\langle X(f)Y^*(f)\rangle|^2}{\langle |X(f)|^2\rangle\langle |Y(f)|^2\rangle}$$

where X(f) and Y(f) represent the complex spectral components of EEG and EMG signals at frequency f, respectively. Cxy(f) ranges from 0 to 1 and measures the degree of linear synchronization between the two signals in the frequency domain. This approach is widely applied in studies of motor control and sleep neurophysiology [37][38].

In RBD research, analyses typically focus on EEG electrodes over the central region (C3/C4 or Cz) coupled with EMG signals from the submental or limb muscles. For instance, Jung et al. [9] used central leads with leg or chin EMG, while Choi et al. [36] incorporated both the Muscle Atonia Index (MAI) and cortico-cortical coherence (CCC) for a comprehensive assessment.

Notably, high–temporal-resolution studies capturing second-by-second CMC dynamics immediately before and after dream-enactment episodes remain scarce, representing an important direction for future investigation.

2.4.1.2 muscle atonia index (MAI)

Advanced coherence analyses have also been explored. Choi et al. (2021) introduced complementary metrics such as the muscle atonia index (MAI)

$$MAI = \frac{N_{\text{low EMG}}}{N_{\text{total}}}$$

MAI is used to measure the degree of muscle tone inhibition during REM sleep and is a core quantitative indicator of REM sleep behavior disorder.

2.4.1.3 Corticocortical Coherence (CCC)

CCC measures synchronous activity between different brain regions and is an important indicator for evaluating the connectivity of cortical networks. For any two-channel EEG signals x(t) and y(t), calculate their frequency domain coherence:

$$CCC(f) = \frac{\left|P_{xy}(f)\right|^2}{P_{xx}(f)P_{yy}(f)}$$

The CCC variation with frequency is a spectral curve, which is not conducive to statistical analysis. Therefore, averaging the coherence values of a certain frequency band (such as α, β, γ) forms a scalar characteristic.

$$CCC_{\text{band}} = \frac{1}{F}\sum_{f \in \text{ band}} CCC(f)$$

Patients often exhibit hyper-synchronization or desynchronization in certain frequency bands. Using the band CCC band, the network strength between different groups can be directly compared.

Corticocortical Coherence (CCC) is essentially a channel-to-frequency matrix, where each channel pair has a coherence value at each frequency. This means that a single subject would result in a huge and high-dimensional network data structure. Directly using this high-dimensional data can negatively impact model performance. Therefore, a global metric is needed to reflect the overall state of the functional network.

$$CCC_{\text{global}} = \frac{1}{N(N-1)}\sum_{i \neq j} CCC_{ij}$$

Typically, it is easier to reveal significant differences between groups than a single CCC.

2.4.2 Clinical impaction

Increased cortical coherence in REM provides some objective evidence that cortical abnormalities “drive” muscle activity in RBD. Sunwoo et al. (2019) reported using EEG source imaging that the motor cortex of iRBD was more activated in phase REM (rapid eye movement and muscle twitching phase) than in controls [9]. Due to the incomplete brainstem tone system, synchronized cortical output actually leads to muscle activation (and thus has measurable coherence). Secondly, the coherence of the β band increases before vigorous movement, and there are anecdotal observations that EEG-EMG coherence briefly spikes a few seconds before major RBD behavior, but more high

temporal resolution studies are needed to confirm this. A more recent analysis by Date et al. suggests certain EEG/EMG changes in the 5–10 seconds before the event, but more work is needed to verify repeatable warning signals of RBD onset [28]. If confirmed, such markers may be valuable for real-time prediction or sleep-loop interventions, although this remains speculative.

From a clinical perspective, coherence intensity may serve as a severity index, reflecting the intensity of abnormal cortical-muscle interactions. For example, in Choi's study, higher REM cell counts were associated with more severe RBD behavior. Coherence may also help distinguish true RBD from increased muscle tone caused by other factors. For instance, other sleep disturbances or normal REM without heart failure in older adults may show increased electromyographic activity. Therefore, combining coherence with standard electromyographic indicators may be more specific for true RBD (brain-driven active dreaming) than for diseases involving passive muscle twitching.

Some unresolved questions include whether longitudinal changes in coherence track disease progression or PD conversion. Currently, there is no large longitudinal cohort data showing whether increased coherence predicts phenotypic conversion. Given the high concordance in iRBD patients, it is unclear whether it further increases as clinical Parkinson's disease approaches. Existing evidence suggests that CMC is an early-established but plateauing feature. As previously mentioned, REMCMC in Parkinson's disease patients is not higher than in RBD patients without Parkinson's. Therefore, coherence is more likely a marker of the RBD state itself, rather than a marker of gradual progression. Nevertheless, its presence indicates a fundamental pathophysiological disorder in the motor inhibitory network.

2.4.3 Pathophysiological Model from coherence

Based on the above findings, we can conceptualize the pathophysiology of RBD as a dual process: the combination of cortical hyperactive motor circuits and brainstem inhibitory gating failure. Normally, during REM sleep, the cortex produces vivid dreams and even some motor commands, but the subdural nuclei (posterior inferior nucleus) in the pons inhibit spinal motor neurons. However, in iRBD, EEG and coherence evidence show enhanced cortical drive, with the motor cortex being hyperactive during REM. Neuroimaging has revealed signs of degeneration in the medullary inhibitory centers of RBD, while transcranial magnetic stimulation shows hyperexcitability of the motor cortex in early Parkinson's disease and RBD[20].

Therefore, RBD can be considered a network disorder extending from the cortex to the spinal cord. iRBD is an early stage of synucleinopathy, with the pathology starting in the brainstem and then spreading anteriorly to the cortical areas. Initially, symptoms (RBD) primarily reflected lower brainstem damage (loss of muscle tone), but changes in EEG

and subtle motor networks revealed progressive involvement of the upper brainstem, even before a clinical diagnosis of Parkinson's disease or DLB[38].

2.4.4 limitations and prospecting of coupling methods

There are still some obstacles to overcome in the translation of clinical practice. One of them is to determine the standard values and thresholds for EEG-EMG coupling measurements. Large-scale studies are needed to determine the “normal” level of coherence (or EEG beta power, etc.) during REM at different ages, and what threshold is optimal to distinguish erythrocytosis. To date, reported figures (e.g., coherence of RBD ~0.1 vs. control group ~0.01[9]) are based on small samples. Another challenge is to ensure that any new biomarker delivers value beyond what simple methods can achieve. For example, if RBD can be detected with 3E90% accuracy by automated EMG analysis alone, then additional EEG indicators must significantly improve sensitivity (e.g., early labeling of prodromal cases) or specificity (e.g., distinguishing RBD from mimicry conditions such as obstructive sleep apnea or periodic limb movement, which may also lead to REM EMG activity[9]).

Finally, future research should fully utilize both longitudinal and interventional studies. With disease modification trials planned for precursor synaptopathies, having quantifiable biomarkers such as EEG/EMG to track whether interventions slow neurodegenerative changes is crucial. Similarly, observing changes in coherence or EEG spectra in iRBD patients progressing to Parkinson's disease can validate these indicators as true premotor biomarkers. Advances in wearable sleep monitors and high-density EEG may even enable home EEG-EMG monitoring, making these biomarkers more readily available. In summary, the integration of EEG and EMG functions provides a more comprehensive window into understanding RBD. By simultaneously capturing both the "symptoms" (muscle activity) and "causes" (brain activity) of REM sleep behavior, this approach holds promise for improving early detection of RBD, refining prognostic predictions, and revealing the neural mechanisms between RBD and the progression of neurodegenerative diseases.

3. Progress Summary (Enhanced Version with Equations)

3.1 Research Planning

The project aims to evaluate EEG–EMG coupling as a potential early marker for RBD. Current RBD detection tools focus on EMG activity during REM. These tools cannot capture cortical involvement in the loss of motor inhibition. Recent studies suggest that beta-band corticomuscular coherence (CMC) may detect early abnormalities in the corticospinal and corticobulbar pathways.

The plan for this project was defined during the first semester.

The analysis framework includes EEG C3 and C4 channels and chin or forearm EMG. The data structure follows a REM–wake comparison. The coherence calculation uses short-time spectral estimation. Feature extraction focuses on beta-band coupling strength and directional patterns. This planning defines the scope for implementation in the next period.

3.2 Design and Development of System Components

A modular pipeline was designed to process PSG data and extract coupling features. The system contains preprocessing, segmentation, coherence computation and feature extraction.

3.2.1 Signal preprocessing design

EEG and EMG signals are pre-processed using standard digital filters. The band-pass filter used for beta-band preparation is:

$$x_\beta(t) = \text{Bandpass}(x(t), 13 - 30\text{Hz})$$

In addition, EMG baseline is computed using full-wave rectification and smoothing:

$$EMG_{\text{env}}(t) = \text{LPF}(|EMG(t)|)$$

3.2.2 REM epoch selection

A module was created to extract REM segments from the XML annotation files. This replaced the earlier attempt at automated REM detection, which was discontinued after confirming that scorer-provided annotations are more reliable.

3.2.3 Planned coherence module

CMC will be computed using the standard magnitude-squared coherence:

$$C_{xy}(f) = \frac{\left|P_{xy}(f)\right|^2}{P_{xx}(f)P_{yy}(f)}$$

where:

$P_{xx}(f)$ is the EEG power spectrum

$P_{yy}(f)$ is the EMG power spectrum

$P_{xy}(f)$ is the cross-spectrum

The calculation will use short windows with overlap:

$$C_{xy}^{(k)}(f) = \text{ Coherence } (x_k(t), y_k(t))$$

This design supports time-resolved coupling metrics.

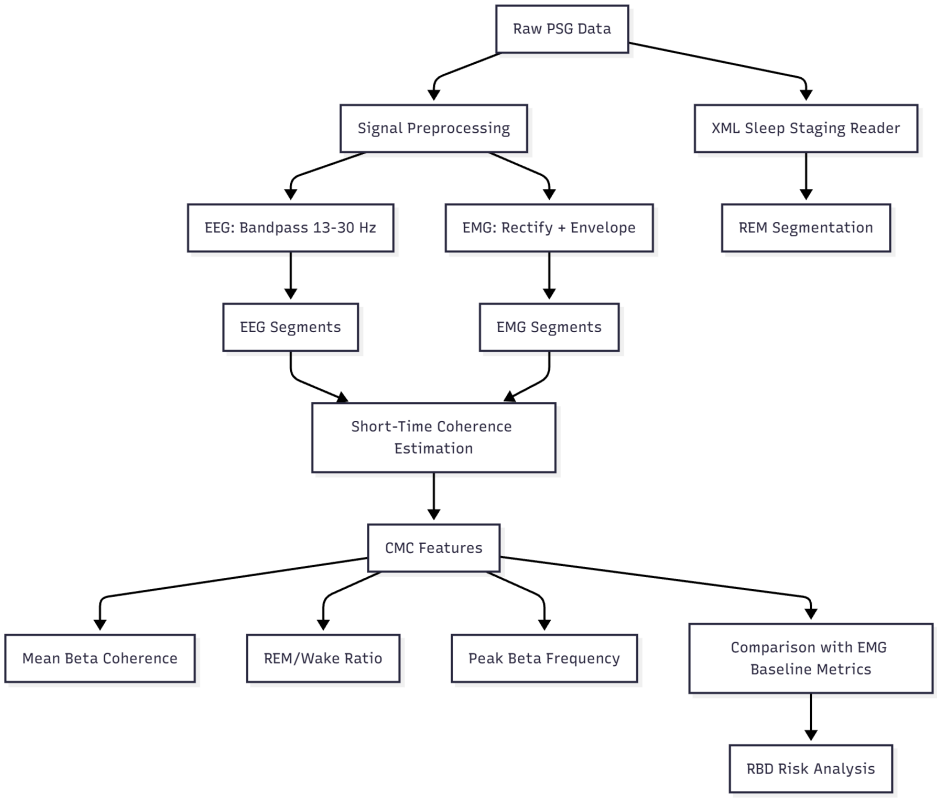

Figure1: pipeline for CMC project

3.3 Preliminary Testing

PSG Data Structure

```
======================================================================
DATA EXTRACTION
======================================================================

Extracting data from 0 to 100.0 seconds

EEG data extracted:
  Channels: ['F3', 'F4', 'C3', 'C4', 'M1', 'M2', 'O1', 'O2', 'E1', 'E2']
  Shape: (10, 102400)
  Sampling frequency: 1024.0 Hz

EMG data extracted:
  Channels: ['Chin 1', 'Chin 2', 'Chin 3', 'Leg/L', 'Leg/R']
  Shape: (5, 102400)
  Sampling frequency: 1024.0 Hz
======================================================================
```

Figure 2: PSG Data structure

XML annotation：

```
======================================================================
XML ANNOTATION READING
======================================================================

Loading XML file: /Users/yanggi/Desktop/RBD/luzy_raw_data/C067/C067_EDF_Export.edf.XML
File exists: True

Epoch length: 30.0 seconds
Sleep stages found: 1215 epochs
  Total duration: 10.12 hours
  Time range: 0 - 36450.0 seconds

Scored events found: 606 events

Event types summary:
  SpO2 desaturation: 186
  Hypopnea: 181
  Arousal (ARO RES): 137
  Arousal (ARO SPONT): 90
  Obstructive Apnea: 8
  SpO2 artifact: 4

Events DataFrame created: (606, 6)

First few events:
...
  - events_df: Scored events as DataFrame
  - aligned_events: Events in extracted time range
  - epoch_length: Epoch length in seconds (typically 30)
======================================================================
Output is truncated. View as a scrollable element or open in a text editor. Adjust cell output settings...
```

Figure 3: XML annotation on labels and data remarks

EMG and EEG preprocessing：

```
============================================================
EMG FILTERING PIPELINE
============================================================

Filter parameters:
  Bandpass: 20.0-500.0 Hz
  Notch frequencies: [ 50 100 150 200 250 300 350 400 450 500] Hz
  Sampling frequency: 1024.0 Hz

Step 1: Applying bandpass filter (20.0-500.0 Hz)...
  ✓ Bandpass filter applied to 5 channels

Step 2: Applying notch filters...
  ✓ Notch filter applied at 50 Hz
  ✓ Notch filter applied at 100 Hz
  ✓ Notch filter applied at 150 Hz
  ✓ Notch filter applied at 200 Hz
  ✓ Notch filter applied at 250 Hz
  ✓ Notch filter applied at 300 Hz
  ✓ Notch filter applied at 350 Hz
  ✓ Notch filter applied at 400 Hz
  ✓ Notch filter applied at 450 Hz

============================================================
EMG FILTERING COMPLETE
...
  Original std: 0.000960
  Filtered std: 0.000118
  Reduction: 87.7%
============================================================
Output is truncated. View as a scrollable element or open in a text editor. Adjust cell output settings...
```

```
============================================================
EEG FILTERING PIPELINE
============================================================

Filter parameters:
  Bandpass: 0.5-40.0 Hz
  Notch frequencies: [ 50 100 150 200 250] Hz
  Sampling frequency: 1024.0 Hz

Step 1: Applying bandpass filter (0.5-40.0 Hz)...
  ✓ Bandpass filter applied to 10 channels

Step 2: Applying notch filters...
  ✓ Notch filter applied at 50 Hz
  ✓ Notch filter applied at 100 Hz
  ✓ Notch filter applied at 150 Hz
  ✓ Notch filter applied at 200 Hz
  ✓ Notch filter applied at 250 Hz

============================================================
EEG FILTERING COMPLETE
============================================================

Filtering effects (first channel F3):
  Original std: 0.000956
  Filtered std: 0.000088
  Reduction: 90.8%
============================================================
```

Figure 4: applying notch filter and BP filter on Preprocessing

Results of biosignals preprocessing

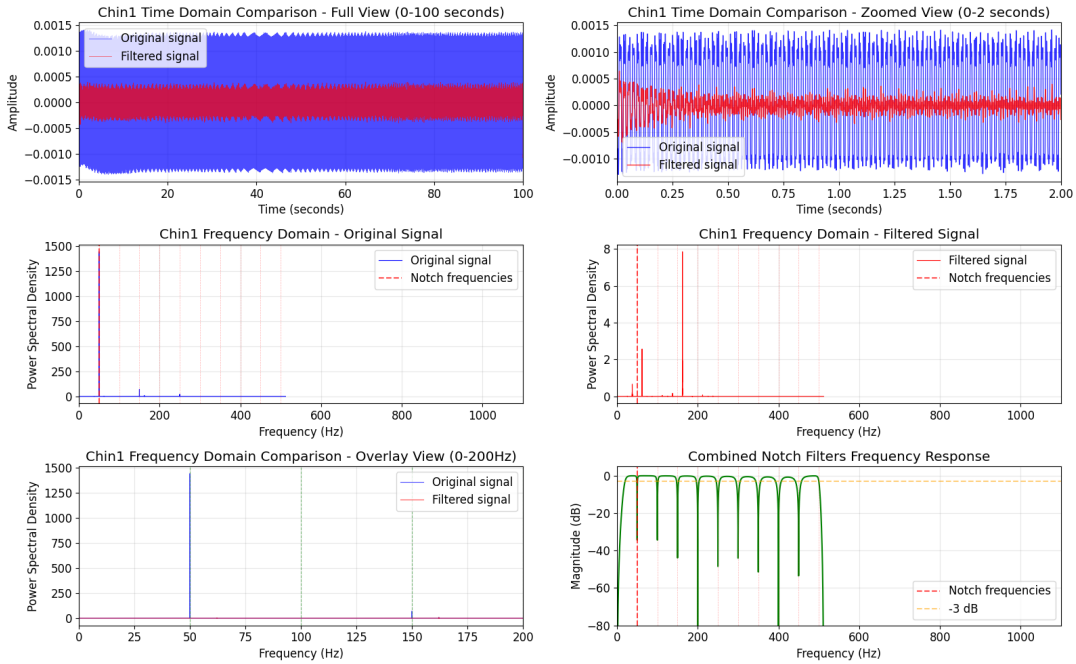

Figure 5: demonstration on filtering EMG channel(50Hz notch filter in pass band-20-500Hz), the noise from filtered signal reduce significantly.

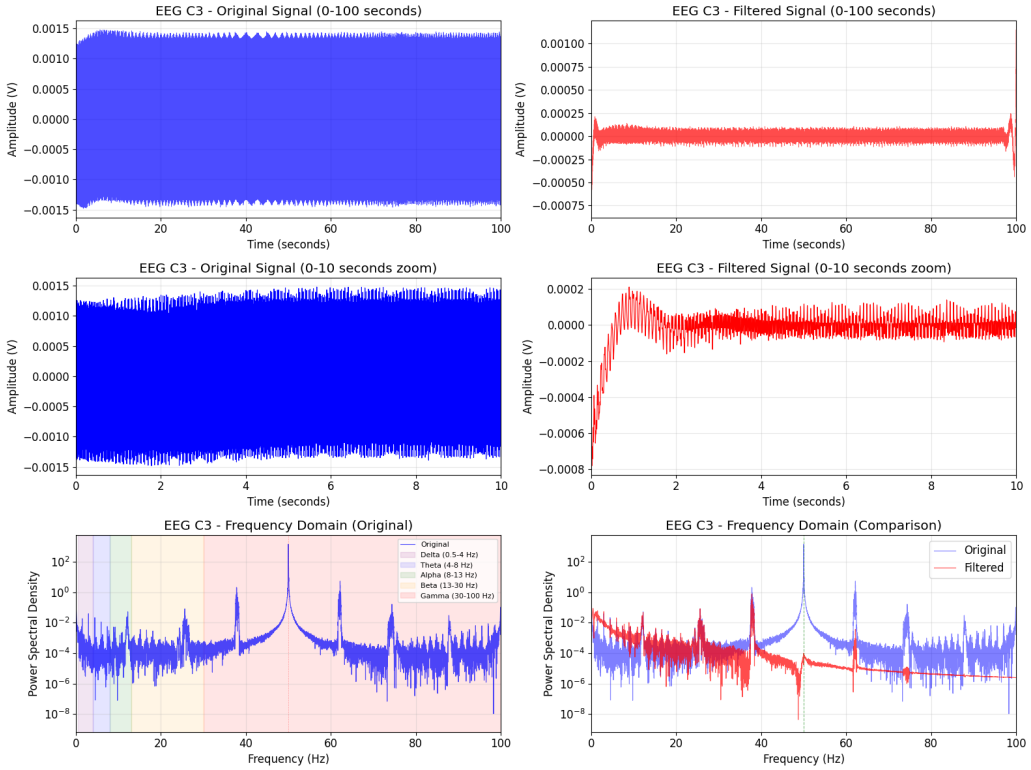

Figure 6: demonstration on filtering EEG channel(50Hz notch filter and pass band with 0.5-40Hz), the powerline interference from filtered signal reduce significantly.

CMC extraction is not yet completed. Most of the time was spent building stable preprocessing. Then several weeks were used to develop automatic REM detection, which was later removed after review. The current REM extraction system uses the XML scorer labels and is now reliable.

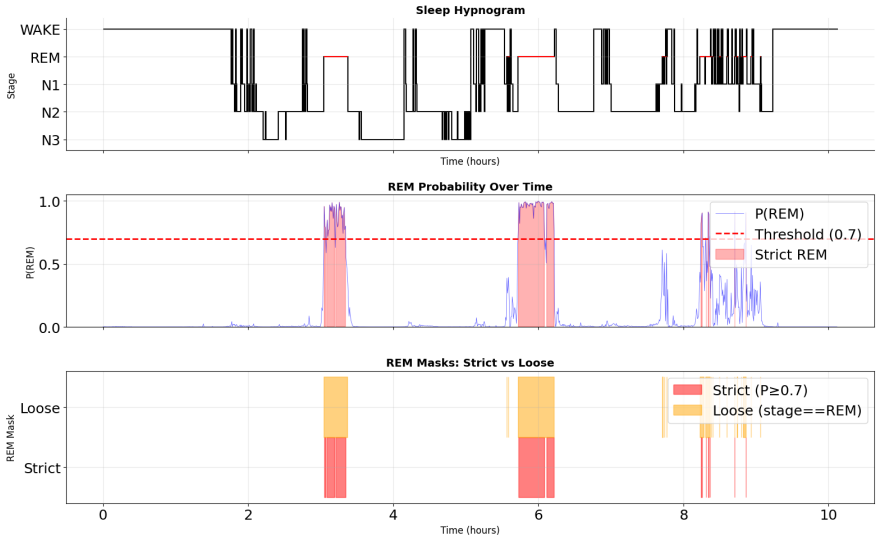

Figure 7: Replication of REM detection with YASA pipeline

The preprocessing step was necessary because the project compares two pipelines: baseline-EMG(like RBDtector) and pipeline-CMC. To make the comparison fair, the same preprocessing pipeline must be applied to both EEG and EMG.

The preprocessing module has been tested on multiple PSG recordings and produces clean and stable output. The system is now ready for CMC computation.

3.4 Data Analysis and Preparatory Work

A review of existing literature on RSWA scoring, EEG slowing, and corticomuscular coherence helped identify the most promising features for early RBD detection.

A statistical analysis plan was drafted for the next phase.
Group-level comparisons will focus on beta-band coherence in REM:

$$C_{\beta}^{REM} = \frac{1}{B}\sum_{f\in\beta} C_{xy}(f)$$

where B is the number of frequency bins in the beta band.
The REM-to-wake ratio is another planned metric:

$$R = \frac{C_{\beta}^{REM}}{C_{\beta}^{\text{Wake}}}$$

A peak frequency metric will identify shifts in maximum coupling:

$$f_{\text{peak}} = \arg\max_{f \in \beta} C_{xy}(f)$$

These formulas are part of the pipeline design and provide clarity for upcoming analysis steps.

3.5 Software Development

The preprocessing system has been fully implemented. It includes the filtering functions, rectification, segmentation and REM epoch extraction.

The EMG baseline module is complete. Next stage will go on building CMC software and launch comparison with CMC-based metrics.

Planned CMC features
Three main features will be included in the next stage:

1 Mean beta coherence

$$F_1 = C_\beta^{REM}$$

2 REM-to-wake coherence ratio

$$F_2 = R$$

3 Peak beta coherence frequency

$$F_3 = f_{\text{peak}}$$

These features will support both group-level analysis and individual-level profiling.

4. Research Plan

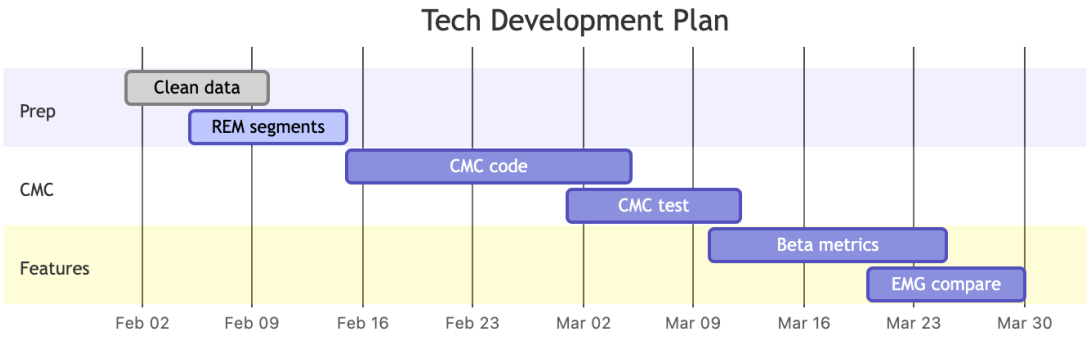

Figure 8: Plan on technical work

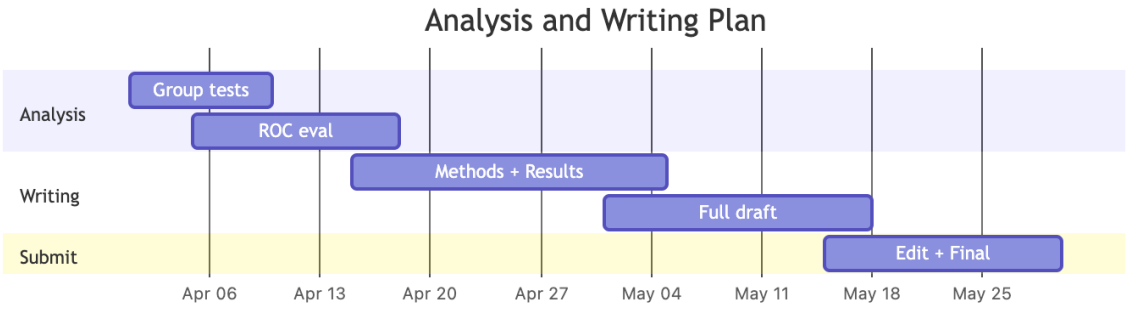

Figure 9: Plan on written work

Reference:

[1] R. B. Postuma, J.-F. Gagnon, J.-A. Bertrand, D. Génier Marchand, and J. Y. Montplaisir, “Parkinson risk in idiopathic REM sleep behavior disorder,” Neurology, vol. 84, no. 11, pp. 1104–1113, Mar. 2015, doi:.

[2] A. Roguski, D. Rayment, A. L. Whone, M. W. Jones, and M. Rolinski, “A Neurologist’s Guide to REM Sleep Behavior Disorder,” Front. Neurol., vol. 11, Jul. 2020, doi:.

[3] A. Stefani et al., “From mechanisms to future therapy: a synopsis of isolated REM sleep behavior disorder as early synuclein-related disease,” Molecular Neurodegeneration, vol. 20, no. 1, p. 19, Feb. 2025, doi:.

[4] P.-C. Chan, H.-H. Lee, C.-T. Hong, C.-J. Hu, and D. Wu, “REM Sleep Behavior Disorder (RBD) in Dementia with Lewy Bodies (DLB),” Behav Neurol, vol. 2018, p. 9421098, Jun. 2018, doi:.

[5] L. Leclair-Visonneau et al., “Contemporary diagnostic visual and automated polysomnographic REM sleep without atonia thresholds in isolated REM sleep behavior disorder,” J Clin Sleep Med, vol. 20, no. 2, pp. 279–291, Feb. 2024, doi:.

[6] M. Figorilli et al., “Comparison Between Automatic and Visual Scorings of REM Sleep Without Atonia for the Diagnosis of REM Sleep Behavior Disorder in Parkinson Disease,” Sleep, vol. 40, no. 2, p. zsw060, Feb. 2017, doi:.

[8] A. Papakonstantinou, J. Klemming, M. Haberecht, D. Kunz, and F. Bes, “Ikelos-RWA. Validation of an Automatic Tool to Quantify REM Sleep Without Atonia,” Clin EEG Neurosci, vol. 55, no. 6, pp. 657–664, Nov. 2024, doi:.

[7] B. Frauscher et al., “Normative EMG values during REM sleep for the diagnosis of REM sleep behavior disorder,” Sleep, vol. 35, no. 6, pp. 835–847, Jun. 2012, doi:.

[9] K.-Y. Jung et al., “Increased Corticomuscular Coherence in Idiopathic REM Sleep Behavior Disorder,” Front Neurol, vol. 3, p. 60, Apr. 2012, doi:.

[10] Figorilli, M., Lanza, G., Congiu, P., Lecca, R., Casaglia, E., Mogavero, M. P., Puligheddu, M., & Ferri, R. (2021). Neurophysiological Aspects of REM Sleep Behavior Disorder (RBD): A Narrative Review. Brain sciences, 11(12), 1588. https://doi.org/10.3390/brainsci11121588

[11] M. Frauscher et al., “RBDtector: Open-source automated detection of REM sleep without atonia based on SINBAR criteria,” Sleep Medicine, vol. 80, pp. 123–132, 2022.

[12] O. Lapierre and J. Montplaisir, “Polysomnographic features of REM sleep behavior disorder: development of a scoring method,” Neurology, vol. 42, no. 7, pp. 1371–1374, 1992.

[13] J. Montplaisir et al., “Polysomnographic diagnosis of idiopathic REM sleep behavior disorder,” Movement Disorders, vol. 25, no. 13, pp. 2044–2051, 2010.

[14]S. J. McCarter et al., “Diagnostic thresholds for quantitative REM sleep phasic burst duration, phasic and tonic muscle activity, and REM atonia index in REM sleep behavior disorder with and without comorbid obstructive sleep apnea,” Sleep, vol. 37, no. 10, pp. 1649–1662, Oct. 2014, doi:.

[14] R. B. Berry et al., The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, Version 2.6. Darien, IL: American Academy of Sleep Medicine, 2020.

[15] M. Cesari et al., “Video-polysomnography procedures for diagnosis of rapid eye movement sleep behavior disorder and the identification of its prodromal stages: Guidelines from the International RBD Study Group,” Sleep, vol. 45, no. 3, art. zsab257, 2022.

[16] R. Ferri et al., “Improved computation of the atonia index in normal controls and patients with REM sleep behavior disorder,” Sleep Med, vol. 11, no. 9, pp. 947–949, Oct. 2010, doi:.

[39] T. Sasai-Sakuma et al., “Quantitative assessment of isolated rapid eye movement (REM) sleep without atonia without clinical REM sleep behavior disorder: clinical and research implications,” Sleep Medicine, vol. 15, no. 9, pp. 1009–1015, Sept. 2014, doi:.

[17]B. Frauscher et al., “Validation of an integrated software for the detection of rapid eye movement sleep behavior disorder,” Sleep, vol. 37, no. 10, pp. 1663–1671, Oct. 2014, doi:.

[18] M. Cesari et al., “Isolated REM Sleep Behaviour Disorder—Is Screening Possible?,” Journal of Sleep Research, vol. 34, June 2025, doi:.

[19]“Automated scoring of REM sleep without atonia using the open-source software RBDtector,” MDS Abstracts. Accessed: Nov. 16, 2025. [Online]. Available:

[20] M. Ferri, “EEG slowing as a hallmark of REM sleep behavior disorder,” Sleep Med. Rev., vol. 44, pp. 12–22, Feb. 2019, doi: 10.1016/j.smrv.2018.12.002.

[21] M. Figorilli, A. Stefani, and B. Frauscher, “Quantitative EEG markers in idiopathic RBD: α-peak frequency and cortical dysfunction,” Sleep, vol. 42, no. 8, p. zsz120, 2019, doi: 10.1093/sleep/zsz120.

[22] A. Rodrigues Braz et al., “EEG slowing predicts cognitive decline in idiopathic REM sleep behavior disorder,” Neurology, vol. 94, no. 10, e1084–e1093, Mar. 2020, doi: 10.1212/WNL.0000000000009161.

[23] N. Kozhemiako, D. Mylonas, J. Q. Pan, M. J. Prerau, S. Redline, and S. M. Purcell, “Sources of Variation in the Spectral Slope of the Sleep EEG,” eNeuro, vol. 9, no. 5, p. ENEURO.0094-22.2022, 2022, doi:.

[24] Valomon, A., Riedner, B.A., Jones, S.G. et al. A high-density electroencephalography study reveals abnormal sleep homeostasis in patients with rapid eye movement sleep behavior disorder. Sci Rep 11, 4758 (2021). https://doi.org/10.1038/s41598-021-83980-w

[25] M. L. Fantini et al., “Slowing of electroencephalogram in rapid eye movement sleep behavior disorder,” Ann Neurol, vol. 53, no. 6, pp. 774–780, June 2003, doi:.

[26]B. Baird et al., “Human Rapid Eye Movement Sleep Shows Local Increases in Low-Frequency Oscillations and Global Decreases in High-Frequency Oscillations Compared to Resting Wakefulness,” eNeuro, vol. 5, no. 4, p. ENEURO.0293-18.2018, 2018, doi:.

[27] A. Terzaghi et al., “Altered slow-wave activity homeostasis in REM sleep behavior disorder,” Clin. Neurophysiol., vol. 135, pp. 98–109, Jan. 2023, doi: 10.1016/j.clinph.2022.11.007.

[40] Y. W. Kim et al., “Cortical spectral dynamics preceding dream-enactment episodes in RBD,” Sleep, vol. 46, no. 4, p. zsad016, Apr. 2023, doi: 10.1093/sleep/zsad016.

[28] S. Date et al., “Polysomnographic features prior to dream enactment behaviors in isolated rapid eye movement sleep behavior disorder,” Clin. Neurophysiol., vol. 166, pp. 74–86, Oct. 2024, doi: 10.1016/j.clinph.2024.07.013.

[29] J.-S. Sunwoo et al., “Nonrapid eye movement sleep electroencephalographic oscillations in idiopathic rapid eye movement sleep behavior disorder: a study of sleep spindles and slow oscillations,” Sleep, vol. 44, no. 2, p. zsaa160, Feb. 2021, doi:.

[30] Iranzo A, Isetta V, Molinuevo JL, Serradell M, Navajas D, Farre R, Santamaria J: Electroencephalographic slowing heralds mild cognitive impairment in idiopathic REM sleep behavior disorder. Sleep Med 2010;11:534-539.

[31] Gong SY, Shen Y, Gu HY, Zhuang S, Fu X, Wang QJ, Mao CJ, Hu H, Dai YP, Liu CF. Generalized EEG Slowing Across Phasic REM Sleep, Not Subjective RBD Severity, Predicts Neurodegeneration in Idiopathic RBD. Nat Sci Sleep. 2022;14:407-418

[41] S. Schopp et al., "EEG alpha peak frequency: cognitive impairment severity marker in isolated REM sleep behavior disorder," npj Parkinsons Dis., vol. 11, no. 1, p. 185, July 2025, doi:.

[32] Ruffini, G., Ibañez, D., Castellano, M., Dubreuil-Vall, L., Soria-Frisch, A., Postuma, R., Gagnon, J. F., & Montplaisir, J. (2019). Deep Learning With EEG Spectrograms in Rapid Eye Movement Behavior Disorder. Frontiers in neurology, 10, 806.

[33] Cesari M, Portscher A, Stefani A, Angerbauer R, Ibrahim A, Brandauer E, Feuerstein S, Egger K, Högl B, Rodriguez-Sanchez A. Machine Learning Predicts Phenoconversion from Polysomnography in Isolated REM Sleep Behavior Disorder. Brain Sciences. 2024; 14(9):871.

[34] Salsone, M., Quattrone, A., Vescio, B., Ferini-Strambi, L., & Quattrone, A. (2022). A Machine Learning Approach for Detecting Idiopathic REM Sleep Behavior Disorder. Diagnostics (Basel, Switzerland), 12(11), 2689.

[35]Rodrigues Brazète, J., Montplaisir, J., Petit, D., Postuma, R. B., Bertrand, J. A., Génier Marchand, D., & Gagnon, J. F. (2013). Electroencephalogram slowing in rapid eye movement sleep behavior disorder is associated with mild cognitive impairment. Sleep medicine, 14(11), 1059–1063. https://doi.org/10.1016/j.sleep.2013.06.013

[36] G. S. Choi et al., "Can Corticomuscular Coherence Differentiate between REM Sleep Behavior Disorder with or without Parkinsonism?," J. Clin. Med., vol. 10, no. 23, p. 5585, 2021, doi: 10.3390/jcm10235585.

[37] G. S. Choi, "Corticomuscular and Corticocortical Coherence Analyses in Patients with REM Sleep Behavior Disorder with or without Parkinsonism (P4.279)," Neurology, vol. 86, no. 16 Suppl., p. P4.279, Apr. 2016.

[38] R. Nardone et al., "Neurophysiological insights into the pathophysiology of REM sleep behavior disorder: A review," Neurosci. Res., vol. 76, no. 3, pp. 106–112, Jul. 2013, doi: 10.1016/j.neures.2013.03.009.