Sleep Paralysis: Neurobiology, Circadian Regulation, and the Emerging Role of Biosensors in Diagnosis and Management

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Abstract- Sleep is an essential biological activity governed by sophisticated brain, harmone and body-clock interactions. Dysregulation gives rise to sleep pathologies including sleep atonia (SP), a parasomnia marked by temporary loss of voluntary muscle control and striking hallucinations at REM-wake boundaries. This article integrates findings from brain science, harmonal research and biomedical technology to deliver a holistic insight into sleep structure, neural control of REM and mechanisms underlying SP. Conventional approaches especially PSG still serves as the benchmark, yet are constrained by expense, availability and dependence on specialized labs. Progress in biosensors and AI supports comprehensive, unobtrusive physiological tracking such as brain activity (EEG), heart rate variability (HRV), and breathing patterns. Such advancements could aid in early SP recognition, anticipation of REM intrusions and live monitoring via connected systems. The study emphasizes triggers, sociocultural interpretations and treatment strategies, underlying how sensor-AI integration translates biological understanding into applied solutions. By combining neurobiology foundations with advanced engineering, it proposes routes to patient-centered affordable, and intelligent sleep management.

Keywords— REM atonia disorder, Sleep structure, biological clock, Neurosciences, endocrine signals, wearable sensors, Artificial Intelligence (machine learning), Sleep surveillance.

I.INTRODUCTION

Sleep serves as a core physiological function essential for physical rejuvenation, mental fortification, and psychological equilibrium. It is categorized into two primary states—non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep—which alternate every 90 to 110 minutes throughout the night [7][9]. During REM (Rapid Eye Movement) sleep, brain activity resembles that of wakefulness; however, the body experiences muscle atonia, a protective mechanism regulated by inhibitory neurotransmitters such as gamma-aminobutyric acid (GABA) and glycine that act on motoneurons [19][20]. This neuromuscular inhibition prevents the enactment of dreams and is controlled by overlapping inhibitory pathways in the pons and medulla, ensuring redundancy in motor suppression [19].

Sleep paralysis (SP) occurs when there is a disconnect between REM sleep atonia and wakefulness, resulting in a condition where the individual is aware but temporarily unable to move or speak [8][16]. Episodes typically arise during transitions between sleep and wakefulness—either hypnagogic (at the onset of sleep) or hypnopompic (upon waking)—and can last from a few seconds to several minutes [12][16]. Sleep paralysis is often accompanied by various hallucinations, which generally fall into three main categories: (a) intruder hallucinations, characterized by the sensation of being watched; (b) incubus hallucinations, which involve pressure on the chest and difficulty breathing; and (c) vestibular-motor hallucinations, which create feelings of floating, spinning, or out-of-body experiences [12][13][17]. Although typically non-threatening, SP can lead to considerable psychological distress, especially in recurrent instances, potentially resulting in anxiety,

fear of sleep, or difficulties in initiating sleep [8][13].

The neurophysiology of SP is characterized by a delicate balance among REM-on cholinergic circuits, REM-off monoaminergic pathways, and wake-promoting orexinergic systems within the hypothalamus [5][6][18][21]. An imbalance in neurotransmitters—particularly involving overactivation of serotonin 2A (5-HT_2A) receptors or excitatory pathways—can contribute to the phenomenon. to the perception-distorting and anxiety reactions during episodes [18]. Hormonal fluctuations also contribute; for instance, recent research indicates that variations in oestrogen and progesterone levels in women suffering from narcolepsy or hypersomnia correlate with changes in SP frequency, particularly around menstruation and menopause [4][5]. Factors such as sleep deprivation, disruptions in circadian rhythms, sleeping in a supine position, psychiatric comorbidities (e.g., PTSD, anxiety), and genetic predisposition may further enhance susceptibility[8][9][16].

Epidemiological research suggests an overall occurrence rate of 8–20% within the general population, with elevated rates observed among college students, shift workers, and individuals with psychiatric conditions [13][16][17]. In cases of narcolepsy, prevalence can reach as high as 30–50% [9][13]. The cultural significance of sleep paralysis varies worldwide—ranging from the “Old Hag” in Newfoundland to “Kanas Hibari” in Japan and “jinn” attacks in Middle Eastern cultures—highlighting the influence of cultural context on symptom perception and levels of discomfort [14][17].

The diagnosis of SP is primarily clinical, relying on the symptoms and history reported by the patient. Although polysomnography (PSG) is considered the gold standard for evaluating sleep disorders, it is rarely required for isolated SP unless narcolepsy or other complex parasomnias are suspected [10][11]. It is crucial to differentiate SP from nocturnal panic attacks, seizures, and REM sleep behavior disorder [8][13]. Traditional management focuses on patient education, reassurance, and lifestyle modifications aimed at improving sleep habits—such as maintaining regular sleep schedules, avoiding supine sleeping positions, and managing stress levels [12][13][16]. Cognitive Behavioral Therapy (CBT) tailored for SP is currently under investigation, and pharmacological treatments (e.g., SSRIs, TCAs) may be considered in particularly severe cases [8][18].

In recent years, advancements in wearable biosensor technology combined with artificial intelligence (AI) have opened new avenues for monitoring sleep disorders and. Commercial devices—primarily worn on the wrist—are capable of capturing various bodily parameters, including respiratory patterns, heart rate variability, and body movement, without the need for penetration [11]. AI algorithms, such as convolutional neural networks (CNNs), recurrent neural networks (RNNs), random forest models, and support vector machines (SVMs), enable computerized detection of sleep phases, identification of respiratory anomalies, and prediction of the onset of disorders [1][3][11]. Although the majority of current AI-wearable research focuses on sleep paralysis [1], the same biosensor-AI framework demonstrates significant potential for SP by identifying REM–wake dissociation events through continuous monitoring of physiological parameters [2][3]. IoT-enabled platforms, such as SleepSmart [3], illustrate this methodology by integrating biosensor data with machine learning to provide personalized recommendations and enhance sleep quality.

This review article intends to consolidate the interdisciplinary knowledge surrounding sleep paralysis, addressing: (a) normal sleep architecture; (b) neurobiological and hormonal regulation; (c) the pathophysiology and mechanisms that underlie SP; (d) influences of circadian rhythm and sleep timing; (e) risk factors and triggers; (f) existing diagnostic and monitoring techniques; (g) the rise of biosensors and AI-driven wearable technology; and (h) their prospective applications in predicting, detecting, and managing SP. By synthesizing insights from neuroscience, endocrinology, epidemiology, cultural studies, and biomedical engineering, this work aims to bridge the gap between process-level understanding and technological advancements in tackling this rare sleep disorder.

III. UNDERSTANDING SLEEP: STAGES AND ARCHITECTURE

Sleep is a intricate and recurring physiological essential for physical recovery, psychological regulation, and emotional balance. It is typically divided into two main types: non-rapid eye movement (NREM) sleep and rapid eye movement

(REM) sleep, which is an option throughout the night in predictable patterns ([7] [9]).

A) Sleep Cycles and Structure

On average, an individual experiences 4–6 complete rest cycles each night, with each cycle lasting approximately 90–120 minutes. The ratio of NREM and REM stages varies across these cycles — deep NREM sleep is predominant during the initial phases, while REM periods increase in span and frequency as morning approaches ([10]; [7]).

This configuration, known as sleep organization, is usually assessed through a sleep study —multi sensor recording technique that observes neural activity, oxygen levels, heart rate, and muscle tension. Recent developments in internet-connected and portable sleep assessment devices allow for Realtime assessment, external monitoring of sleep architecture in home environment ([2] [11][1]).

B) NREM Sleep Phases

NREM sleep is comprised of three distinct phases, each characterized by unique electroencephalographic (EEG) patterns and physiological characteristics:

Stage N1 (Light Sleep): A short phase that signifies the transition from wakefulness to sleep, marked by diminished alpha rhythms and gentle, slow eye movements ([7]).

Stage N2: This stage, characterized by sleep spindle and characteristic EEG bursts, is the most prevalent, making up the initial half of total sleep duration, and is crucial for information stabilization (Smith & Mong, 2019 [5]).

Stage N3 (Slow-Wave or Deep Sleep): This stage, dominated by delta wave activity, is vital for bodily healing, endocrine regulation, and disease resistance ([6]; [10]).

C. REM Sleep

REM sleep is associated with intense dreams, rapid eye movement, and significant paralysis of motor inhibition — a result of chemical and receptor interactions that inhibit the action of spinal motor neurons ([19] [18]). This stage is crucial for emotional stability, learning, and neural plasticity ([5]). Interruptions in the typical atonia related to REM sleep can result in sleep atonia, where an individual remains unable to move despite being partially wakefulness ([8]; [16]).

D. Regulation of Sleep Architecture

The coordination of sleep stages is influenced by the biological clock (which regulates the timing of sleep) and the sleep pressure (which manages the need for sleep). Hormonal signals such as melatonin, cortisol, estrogen, and progesterone play a role in modulating the timing, distribution, and depth of various sleep stages ([9][6]). For example, fluctuations in reproductive hormones in women can affect the onset of REM sleep and the amount of deep sleep experienced ([4]).

E. Clinical and Technological Insights

Disruptions in standard sleep architecture are linked to various diseases, including sleeplessness, hypersomnia, narcolepsy (sudden sleep attacks), and parasomnias (abnormal sleep behaviors) ([10] [11]). New AI-powered portable technologies are capable of detecting subtle anomalies in sleep patterns, which may assist in the early identification of brain related disorders ([1] [3]). These systems utilize machine learning algorithms that evolve over time, facilitating individualized tracking of sleep health. *(Fig.1.)*

Fig.1. Typical sleep cycle showing alternating NREM and REM stages across an 8-hour night. Deep NREM occurs earlier, while REM becomes longer toward morning."

IV. NEUROBIOLOGY AND HARMONAL REGUALTION OF SLEEP

Sleep is governed by a complex interaction of brain circuits, neurotransmitters, and hormones. Two major processes regulate sleep: the homeostatic mechanism, which builds sleep pressure during wakefulness, and the biological clock, controlled by the suprachiasmatic nucleus (SCN), which aligns sleep with the diurnal cycle [5–7][9].

The regulatory hub is central: the VLPO promotes sleep by inhibiting arousal centres, while the ascending reticular activating system (ARAS) sustains wakefulness [5][6]. REM sleep arises when REM-on acetylcholine producing cells activate the cortex and induce muscle paralysis, while REM-off monoaminergic neurons fall silent [18–21]. In sleep atonia, this balance fails—awareness returns but atonia persists [8][18].

Principles of neurotransmitters include:

- GABA & Glycine – suppresses motor activity (NREM, REM atonia) [19][20]
- Acetylcholine – drives REM cortical activity [18][21]
- Serotonin & Norepinephrine – facilitate NREM/wake, suppressed in REM [18][21]
- Orexin – secures wake; deficiency linked to narcolepsy & SP [6][18]

Hormones add another layer:

- Melatonin – night release, initiates sleep [4][5]
- Cortisol – stress Harmone urge, prepares waking [4]
- Growth hormone – released in deep sleep for repair [5]
- Reproductive hormones – oestrogen & progesterone influence REM/NREM balance; variations increase SP risk in women [4][6]

Genetic regulators (CLOCK, PER, BMAL1) also fine-tune biological rhythms [21]. Maladaptation in these pathways contributes to disorders like insomnia, narcolepsy, REM sleep behaviour diseases, and sleep paralysis.

V. SLEEP PARALYSIS: DISEASE MECHANISM AND PROCESSES

Sleep paralysis (SP) is a transient disorder where an individual, during the transition into sleep or upon waking, experiences an inability to move despite being fully conscious. This condition is frequently accompanied by vivid perceptual illusions and a significant sense of anxiety ([12] [16]). SP can occur independently or in conjunction with other sleep disorders such as insomnia ([8] [13]).

a. Disease Mechanism Summary

The disorder arises from a discrepancy between the neurological systems that regulate REM sleep-related muscle paralysis and those that control wakefulness. During normal REM sleep, the brain induces muscle paralysis to prevent physical actions during dreams, primarily through the suppression of spinal motor neurons via neurotransmitters such as amino acid neurotransmitters and gamma-aminobutyric acid (GABA) ([19] [18]). In SP, this paralysis persists into waking consciousness, resulting in the paradoxical experience of being mentally alert yet physically immobile ([20] [14]).

b. REM Paralysis and Neurotransmitter Processes

In typical REM cycles, the subcoeruleus nucleus and related pontine networks activate inhibitory spinal interneurons that release amino acid neurotransmitters and GABA, effectively inhibiting skeletal muscle movement ([19]). Brainstem cholinergic systems sustain REM-related cortical activation, while serotonergic and noradrenergic pathways are largely inactive. In SP, an unusual persistence or premature reactivation of these inhibitory networks leads to muscle paralysis coinciding with wakefulness [18]

C. *Neuroendocrine and Circadian Influences:*

Hormonal fluctuations, particularly in estrogen and progesterone levels, can affect the initiation of REM sleep and the intensity of paralysis, which may account for the increased incidence of sleep paralysis (SP) during specific menstrual phases in women experiencing sleep disorders or hypersomnia ([4][6]). Disruptions in circadian rhythms and inadequate sleep are recognized triggers, as they disrupt the precise timing of transitions between REM sleep and wakefulness ([9]).

d. Hallucinatory Encounters

SP frequently includes experiences of intruders, incubus, and vestibular-muscular (V-M) illusions, which are thought to arise from the overlap of REM dream content with waking consciousness ([8][16]). Neuropharmacological studies suggest that the activation of serotonin 2A receptors may enhance the clarity and emotional resonance of these experiences ([18]). Imaging and brain structure research indicates that these illusions correlate with heightened activity in the temporoparietal junction and other cortical regions associated with body representation and threat assessment ([15]).

e. Genetic and Neurological Factors

Genetic predispositions play a role in the likelihood of experiencing SP, as variations in genes that govern REM-related neurological pathways and circadian regulation can disrupt sleep-wake transitions ([21]). Neurological disorders such as sleep disorders, idiopathic hypersomnia, and certain psychiatric conditions are linked to an increased frequency of SP episodes ([10] [13]).

f. Technological Insights into Mechanism Detection

AI-powered wearable sleep monitors and IoT-enabled polysomnography can detect irregularities in REM-wake boundaries and prolonged episodes of paralysis, providing significant insights into SP occurrences outside of controlled laboratory environments ([2]) With machine learning combination, these devices can recognize subtle physiological markers that may forecast SP onset.

VI. CIRCADIAN RHYTHMS AND STANDARD SLEEP TIMING

Biological clock are ~24-hour natural body rhythms regulated by the suprachiasmatic nucleus (SCN) / brains master clock in the hypothalamus, aligning bodily functions — including the sleep–wake cycle — with photoperiod changes [9][21]. Light signals from melanopsin-containing retinal ganglion cells(light sensitive retinal cells) reach the SCN via the Retin hypothalamic tract, regulating clock genes [21].

The SCN coordinates melatonin secretion from the pineal gland, peaking at night to induce sleep [4][6], while cortisol surges in the early morning to promote alertness [4]. Growth hormone (GH) is released during slow-wave sleep, and reproductive hormones (estrogen, progesterone) influence REM-NREM distribution, with variations affecting uninterrupted and SP risk [4][6].

In healthy adults, sleep generally initiates between 10 PM–12 AM, sustaining for 7–9 hours, moving through NREM and REM every 90–110 minutes [7][9][19][20]. Chronotype (morningness/eveningness) influenced by heredity and surrounding [21].

Biological rhythm mismatch — from shift work, jet lag, irregular schedules, or excessive nighttime light — disturbs REM regulation and increases SP vulnerability [9][8][16]. Other triggers include lack of sleep, lying on the back, stress, and genetic predisposition [8], [12], [13][21]. Cultural interpretations, such as *kanashibari* in Japan or "jinn" in Middle Eastern beliefs, may worsen discomfort and further disturb sleep timing [14], [16].

Regular sleep routines, managed light, and wearable/IoT-based supporting systems [1][3], [11] can help balance biological clocks and decrease SP occurrence.

VII. DETERMINANTS INFLUENCING THE SLEEP WAKE CYCLE AND PROVOKING SLEEP PARALYSIS

The sleep cycle is controlled by a complex interaction among biological clock, sleep pressure, and harmonal control systems. Disruptions in these mechanisms can contribute to onset of sleep paralysis (SP) — a abnormal sleep event marked by brief muscle atonia at sleep onset or during awakening, frequently accompanied by sensory misperceptions and intense fear [8, 12, 13, 16].

a. Circadian Rhythm Alterations

The biological pacemaker, coordinated by the suprachiasmatic nucleus (SCN) within the hypothalamus, synchronized periods of sleep and alertness with the day-night cycle [9]. desynchronization caused by night shift work, jet lag, or irregular routines–wake patterns interferes with melatonin and delays REM onset, thereby increasing likelihood of REM intrusion into wakefulness — a core cause underlying SP [9, 10, 16]. Conditions such as shifted sleep-wake disorder and circadian rhythm disorder further intensify vulnerability [9].

*b. Neuroendocrine and Hormonal Modulators*Sleep regulation is closely linked to neuro-harmonal regulation, especially fluctuations in cortisol, melatonin, estrogen, and progesterone levels [5, 6, 7]. Females may exhibit heightened susceptibility to SP episodes during menstrual cycles, perimenopause, or postpartum intervals, where hormonal oscillations disrupt REM regulation [4, 6]. Increased night-time cortisol and reduced melatoninconcentrations have been linked to disrupted REM patterns, creating more opportunities for REM atonia to persist into wakefulness [5, 6].

c. Sleep Structure and REM Dysregulation

Normal sleep structure cycles between NREM and REM phases, with REM characterized by vivid dreaming and motor suppression via brainstem neural circuits [18, 19, 20]. SP manifests when brain arousal occurs without termination of REM-related atonia, typically due to disrupted REM–wake transitions [18, 19]. Contributing factors include sleep restriction, broken rest, and sudden sleep disorder, all of which destabilize REM activity and raise SP incidence [13, 16]

d. Neurological and Genetic Contributions

Neurophysiological function studies indicates that REM paralysis is maintained by glycine- and GABA-mediated inhibition of spinal motor neurons, influenced by brainstem messengers [19, 20]. Hereditary traits may predispose individuals to abnormal REM regulation [21]. Neural imaging findings also reveal modifications in parietal–temporal network connectivity during SP, potentially clarifying associated dream like experience's 15].

f. Behavioral and Environmental Stress Factors

Ongoing stress, irregular sleep patterns, and poor sleep practices sleep practices can disrupts circadian and homeostatic balance [8, 12, 16]. Overuse of caffine or alcohol consumption, extended night time scren time, and irregular bedtime schedules may delay REM onset and break up rest [10, 16]. Social stress are notably influential in triggering episodes among predisposed populations [14]. *Technological Developments and Surveillance Advancement* in wearable AI-driven sleep tracking and IoT-based monitoring allow detection of circadian disruptions, REM irregularities, and external triggers [1, 2, 3, 11]. Such technologies can help chart rhythms preceding SP events, enabling customized strategies [1, 3, 11].

Fig.2. Relationship between sleep efficiency and other dataset features

VIII. DIAGONOSIS AND MONITORING OF SLEEP DISORDERS

The identification of sleep disorders, including sleep paralysis (SP), relies initially on a combination of medical assessment, case history, and, when needed, clinical sleep tests[10][12][13]. In most cases, isolated SP is said to be through subjective reports, such as temporary atonia upon awakening, presence of sensory illusions, and associated anxiety, rather than through invasive testing [12] [13] [16]. Clinical interviews and standardized questionnaires, including the Sleep Paralysis Experiences and Phenomenology Questionnaire (SP-EPQ), are often used to assess frequency, triggers, and severity of episodes [12].The bench mark method for objective sleep assessment is polysomnography (PSG) or overnight sleep study, a comprehensive overnight test that monitors brain activity electroencephalography (EEG), eye movements (EOG) or ocular signal monitoring, muscle tone (EMG), heart rate (ECG), respiratory effort, oxygen saturation, and body position [10], [11]. PSG is most often used to diagnose narcolepsy, sleep paralysis, and parasomnias, but in SP, it is typically reserved for complex or recurrent cases where differentiation from other diseases is needed [10][11][13].

Alternative methods such as the Multiple Sleep Latency Test (MSLT) and Maintenance of Wakefulness Test (MWT) assess abnormal drowsiness and sleep–wake stability, helping to distinguish SP associated with narcolepsy from idiopathic or situational SP [10]. These chemical or laboratory-based tools, though have accuracy, but are costly, time-consuming, and require specialized facilities.In clinical practice, major focus is often placed on condition comparison, as SP can mimic or overlap with conditions such as night panic attacks, seizures, or REM sleep behavior disorder (RBD) [8], [12], [13]. Careful detection of symptom onset, duration, and associated behaviors is necessary to avoid misdiagnosis.Traditional management following diagnosis relies on patient education, reassurance of SP's harmless state, and behavioral adjustment strategies aimed at improving sleep hygiene (e.g., consistent schedules, reduced stress, side sleeping) [12], [13], [16]. Drug treatments(SSRIs, TCAs) are generally reserved for severe, occurring continuously, or narcolepsy-associated SP [8].Although PSG and MSLT remain the foundational methods, recent advancements in wearables and AI-enabled biosensors are creating opportunities for less invasive, affordable, and home-based monitoring solutions [1]–[3], [11].

IX. INTRODUCTION OF BIOSENSORS IN SLEEP MONITORING AND DIAGNOSIS

Traditional sleep assessment such as polysomnography (PSG) and multiple sleep latency tests (MSLT), though highly reliable, are limited by their cost, invasiveness, and laboratory dependence [10][13]. This has accelerated research into biosensor-enabled and wearable technologies that provide uninterrupted, non-intrusive, and real-world monitoring of sleep physiology [1], [2], [11].

A. Wearable Biosensors for Sleep Monitoring

Modern wearables—such as digital watches, smartwatches, and headbands—incorporate composite detector including motion sensors, photoplethysmography (PPG), electrodermal activity (EDA), and EEG monitors to track physiological markers like heart rate variability (HRV), oxygen level, respiratory rate, and sleep stage transitions [1], [11]. These devices simulate PSG by reconstructing sleep stages through computational models, thereby offering home based alternatives, economical substitutes for long-term monitoring.

B. IoT and Wireless PSG Systems

Advancements in IoT-based setups have enabled wireless polysomnography, which reduces patient discomfort and allows real-time cloud-based sleep data transmission [2]. For instance, Lin et al. developed an IoT-PSG framework that wirelessly records brain, muscle, and breathing signals while synchronizing them with cloud storage for clinician access [2]. This approach not only enhances diagnostic reach but also improves patient compliance compared to traditional wired PSG setups.

C. AI and Machine Learning in Biosensor Data Analysis

Artificial intelligence (AI) is playing a transformative role in interpreting biosensor outputs. Algorithms such as convolutional neural networks (CNNs), recurrent neural networks (RNNs), random forest models, and support vector machines (SVMs) have been successfully applied to automatic sleep staging, respiratory event detection, and anomaly prediction [1], [3]. Systems like Sleep Smart, an IoT-enabled continual learning algorithm, demonstrate how biosensor–AI integration can provide personalized feedback and optimize sleep health dynamically [3].

D. Applications in Sleep Paralysis and Related Disorders

While most biosensor research has focused on obstructive sleep apnea and insomnia, their utility extends to parasomnias like sleep paralysis (SP) [8], [12], [16]. Continuous biosensor monitoring can detect REM–wake dissociation, abnormal HRV patterns, or respiratory irregularities that precede SP episodes. Moreover, AI-driven platforms can correlate subjective reports with objective physiological data, bridging the gap between patient experience and quantifiable markers [1], [3], [11].

E. Advantages and Limitations

Biosensors offer several advantages:

- Scalability and portability for home use
- Continuous monitoring over weeks/months
- Patient comfort compared to wired PSG
- Integration with smartphones/IoT platforms for personalized care

However, limitations include reduced accuracy compared to PSG, challenges in signal noise reduction, and regulatory validation requirements before clinical adoption [11].

X. APPLICATION OF BIOSENSORS IN UNDERSTANDING AND MANAGING SLEEP PARALYSIS

Recent progress in wearable biosensors, artificial intelligence (AI), and Internet of Things (IoT)-enabled devices have opened new prospects for observation sleep rhythms, identifying early indicators of sleep paralysis (SP), and developing individualized therapies. Sleep paralysis arises from irregular REM–wake shifts and disturbances in circadian and neuroendocrine control, making it highly suitable for detection through continuous physiological observation [8, 12, 13, 16].

a. Wearable AI-Based Biosensors

Smart wearables integrated with AI algorithms can track multi-dimensional biosignals, including heart rate variability (HRV), electroencephalography (EEG), skin conductance, and oxygen saturation. These metrics allow the identification of slight

forerunners to REM intrusion and nocturnal arousals associated with SP [1]. A scoping review highlights how wearable AI can detect irregular REM beginning latency and irregular sleep, enabling predictive modeling of SP episodes [1]

b. IoT-Enabled Polysomnography and Continuous observation:

Conventional polysomnography is limited to clinical environments; however, IoT-based wireless polysomnography systems now provide home-based, immediate observation of brain and autonomic activity [2]. By continuously capturing EEG, EOG, EMG, and cardiovascular activity, these systems can detect REM dyscontrol and shifts where muscle atonia persists into wakefulness—defining traits of SP [2, 3]. Algorithms like Sleep Smart integrate IoT data streams with machine learning to recognize sleep fragmentation trends, circadian mismatch, and external factors that may precipitate SP [3].

c. Early Detection of Neuroendocrine and Hormonal Trigger

Biosensors can also support the study of hormonal variations (cortisol, melatonin, estrogen, progesterone) that influence sleep quality and REM control [4–6]. Continuous tracking of these bioindicators, combined with wearable sleep observation, may explain why women are particularly vulnerable to SP during menstrual, perimenopausal, or postpartum phases [4, 6]. Coupling neuroendocrine biosensing with sleep architecture observation provides a comprehensive model for predicting SP beginning.

d. Predicting Neurological Disorders and chronic risk

SP has been associated with REM is out of control, narcolepsy, and circadian rhythm disorders [8, 9, 13]. Modern biosensor-driven home polysomnography is increasingly applied not only for diagnosis but also for identifying long-term neurological risks linked to recurrent SP episodes [11]. By analyzing connectivity alterations and REM fragmentation, biosensors can detect trends suggestive of susceptibility to disorders such as narcolepsy or depression [11, 15].

e. Individualized care and protective measures

The integration of wearable biosensors, IoT platforms, and AI analytics allows for personalized response cycles, that inform users about behaviors contributing to SP—such as irregular bedtimes, late-night light exposure, and stress [10, 12, 16]. Biosensor data can be used to develop protective measures: enhancing sleep hygiene, adjusting circadian phase with light exposure, and observation stress indicators that exacerbate SP risk [14, 16].

f. Upcoming pathways

With continued advancement in remote biosensing technologies, SP management may shift toward anticipatory approaches. Future AI-driven biosensor platforms could not only predict SP episodes but also interrupt in immediate through haptic or auditory cues to interrupt REM intrusion. This holds potential for revolutionizing SP management from a responsive to a predictive and preventive model [1, 3, 11].

XI. LIMITATIONS AND FUTURE DIRECTIONS

Although biosensor-based strategies hold significant potential in the detection and management of sleep paralysis (SP), several obstacles persist:

- Signal Accuracy and Consistency – Data from biosensors may suffer from distortion, noise, or irregularities caused by body movements, incorrect placement, or environmental factors (Restivo, 2023 [12]; Krishnan, 2020 [16]).
- Affordability and Availability – High costs of advanced biosensors and IoT-enabled sleep monitoring devices restrict their accessibility for widespread clinical and domestic use ([2]).
- Patient Adherence – Continuous monitoring demands regular usage, yet bulky or uncomfortable devices often discourage long-term compliance ([10]).
- Complexity in Data Integration – Merging neuroendocrine, neurological, behavioral, and environmental datasets into a single predictive model is still a challenging task ([13][8]).
- Data Security and Ethical Concerns – Persistent surveillance of physiological and neurological parameters raises issues related to privacy, data ownership, and responsible usage ([14]).

Future Directions:To overcome these limitations, future studies and technological innovations should emphasize:

- Enhanced Signal Processing with Artificial Intelligence – Advanced machine learning algorithms can refine data interpretation by minimizing noise and improving diagnostic precision ([2][3]).
- Low-Cost and Scalable Technologies – Upcoming wearable biosensors are anticipated to become more economical, compact, and suitable for at-home applications ([13]).
- Ergonomic and Comfortable Wearables – The development of lightweight, flexible, and skin-friendly devices will improve comfort, thereby encouraging consistent use ([10]).
- Integrated Monitoring Ecosystems – Cloud-based platforms that combine physiological, hormonal, and behavioral signals will enable more comprehensive prediction and management of SP ([8] [12]).
- Secure and Ethical Data Management – Strong governance frameworks, encryption standards, and privacy-preserving mechanisms are essential for maintaining trust and safeguarding user information ([14]).

XII. CONCLUSION

Sleep paralysis (SP) illustrates the complex relationship between brain mechanisms, endocrine balance, biological clocks, and environmental influences in shaping human sleep. While SP itself is harmless, its persistent nature and association with perceptual disturbances can lead to considerable psychological distress, necessitating a deeper medical and scientific understanding. Initial methods such as overnight sleep studies have contributed valuable insights into sleep structure and sleep disturbances but remain narrow in scope by cost, availability, and environment.

Emerging advances in body-worn monitors, artificial intelligence (AI), and IoT-enabled platforms represent a transformative changes in sleep research and clinical practice. These technologies provide instantaneous , non-invasive, and continuous monitoring of physiological signals, creating opportunities for timely identification of REM–wake disruption events and personalized therapies for SP. By merging brain science, endocrinology, biomedical engineering, and data science, future work can translate mechanistic insights into practical, patient-centered solutions. Ultimately, bridging the gap between conventional sleep research and technology-enables innovations approaches offers the potential to not only optimize SP detection and treatment but also reshape broader sleep medicine into a more precise, accessible, and personalized field.

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