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Sleep Paralysis: Current Insights and Future Perspective -A Review

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Abstract: *Sleep paralysis (SP) is a temporary sleep-related condition that occurs during the transition between wakefulness and rapid eye movement (REM) sleep. During an episode, the individual is conscious but unable to move or speak, often experiencing vivid hallucinations, a sense of chest pressure, or the feeling of an unseen presence. Although harmless, the condition can cause intense fear and anxiety, leading to disturbed sleep and daytime tiredness. Sleep paralysis has been known for centuries and is often surrounded by cultural or supernatural explanations. However, scientific research shows that SP results from a disruption in normal REM sleep regulation, where the brain awakens while the body remains temporarily paralyzed. It commonly affects students, shift workers, and individuals with stress, irregular sleep schedules, or sleep deprivation. While occasional episodes are considered normal, frequent occurrences may indicate underlying sleep disorders such as narcolepsy. Management focuses on reassurance, maintaining regular sleep patterns, and practicing good sleep hygiene. Recent studies suggest that awareness programs and non-pharmacological interventions can help reduce stigma and improve sleep quality. Continued research is necessary to explore the neurobiological mechanisms and potential therapeutic approaches for this condition in the Indian population.*

Keywords: Sleep paralysis; Rapid eye movement (REM) sleep; Parasomnia; Sleep disorders; Hallucinations

I. INTRODUCTION

Sleep is one of the most essential biological functions that maintains both physical and mental health by allowing the body and brain to restore energy, regulate metabolism, and consolidate memory. The human sleep cycle is divided into non-rapid eye movement (NREM) and rapid eye movement (REM) stages, each serving a unique physiological purpose. Among various sleep disturbances, sleep paralysis (SP) has gained particular scientific and clinical interest due to its unusual presentation in which a person experiences temporary inability to move or speak while remaining conscious [1]. Sleep paralysis occurs when a person partially awakens during the REM stage of sleep but the muscles remain in a paralyzed state. This physiological mechanism, known as muscle atonia, is meant to prevent physical movement during dreams. However, in SP, this atonia continues even after the brain becomes alert, leaving the individual awake yet immobile. The episode may last for several seconds to a few minutes and is frequently accompanied by vivid hallucinations such as sensing a presence in the room, feeling chest pressure, or hearing strange sounds. Although medically harmless, such experiences are often extremely frightening and may cause significant emotional distress [2]. Throughout history, sleep paralysis has been recognized in various forms across cultures and time periods. Ancient European literature described the phenomenon as an “incubus” or a demonic entity pressing on the sleeper’s chest. In Japan, it is referred to as kanashibari, and in African and Caribbean folklore, it is often attributed to witchcraft or spiritual attack. Similarly, in Indian culture, especially in rural areas, many people believe it to be caused by a “chhaya” or evil spirit sitting on the chest during sleep. These cultural beliefs continue to influence how individuals interpret and respond to SP, often leading to fear, anxiety, and reluctance to seek medical advice [3]. Modern medical science, however, explains sleep paralysis as a result of disrupted transitions between REM sleep and wakefulness. During REM sleep, the brainstem, particularly the pons and medulla, inhibits spinal motor neurons through neurotransmitters such as gamma-aminobutyric acid (GABA) and glycine, which induce muscle atonia. When

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the brain awakens before this inhibition ends, the person experiences full consciousness without the ability to move. This dissociation between cortical arousal and muscle control forms the physiological basis of sleep paralysis [4]. Researchers have observed that irregular sleep schedules, sleep deprivation, stress, and anxiety increase the likelihood of SP episodes. It is also linked to other sleep disorders such as narcolepsy, insomnia, and obstructive sleep apnea, suggesting a deeper neurophysiological connection [5]. The prevalence of sleep paralysis varies widely across populations, with global studies reporting that approximately 7–8% of people experience at least one episode during their lifetime. Among students and shift workers, the occurrence is notably higher due to academic pressure, erratic sleep patterns, and lifestyle habits. Indian studies have reported similar findings, highlighting a growing awareness of SP among young adults. A survey conducted among Indian medical and pharmacy students found that around 20% had experienced sleep paralysis, though most initially attributed it to supernatural causes. Such misconceptions point to the urgent need for educational interventions that promote scientific understanding of sleep disorders [6]. Sleep paralysis, while self-limiting, can have significant psychological implications. Individuals who frequently experience episodes often develop anticipatory anxiety, fearing to fall asleep because of the distressing sensations that accompany paralysis. Over time, this leads to disturbed sleep patterns, fatigue, and reduced concentration during the day. Some individuals also report depressive symptoms or panic attacks following recurrent episodes. Therefore, SP not only affects physical rest but also mental well-being, emphasizing the importance of early recognition and counseling [7]. In India, cultural interpretations and limited awareness about sleep disorders make the condition underreported. Many individuals consult faith healers or spiritual practitioners instead of healthcare professionals, delaying proper diagnosis and reassurance. Increasing public awareness about sleep physiology through educational programs and community workshops can significantly reduce stigma. Initiatives led by organizations such as the Indian Psychiatric Society and the Indian Sleep Disorders Association (ISDA) have begun addressing these gaps through seminars, awareness campaigns, and inclusion of sleep health in national mental health programs [8]. Healthcare professionals play a crucial role in identifying and managing sleep paralysis. Pharmacists, physicians, and mental health workers should be trained to differentiate SP from psychiatric or neurological conditions such as schizophrenia, epilepsy, or panic disorder. Early recognition can prevent unnecessary medical interventions and psychological distress. Emphasis should be placed on patient education, reassurance, and sleep hygiene maintenance, including regular sleep schedules, reduced caffeine intake, and minimizing screen time before bed [9]. Ongoing research in neuroscience and sleep medicine in India is helping to better understand the neural pathways involved in SP and its association with emotional stress and lifestyle. With increasing modernization and changing work patterns, sleep disturbances are becoming more common, making it essential to incorporate sleep education into primary healthcare and public awareness campaigns. Understanding the neurophysiological and cultural dimensions of sleep paralysis will not only improve diagnosis and management but also enhance mental well-being across populations [10].

Sleep Cycle and Its Relation to Sleep Paralysis

Sleep is a naturally recurring physiological state essential for maintaining brain function, energy conservation, and overall health. It is governed by the complex interplay of neural, hormonal, and circadian mechanisms that regulate periods of wakefulness and rest. The human sleep cycle is broadly divided into two types: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. Each full cycle lasts about 90 to 110 minutes and repeats four to six times during a typical night's sleep. The transition through these stages is critical for restoring both physical and mental functions, while disturbances in the pattern can lead to various parasomnias, including sleep paralysis [11].

During NREM sleep, which occupies about 75–80% of total sleep time, the body undergoes gradual relaxation. It is further classified into three stages—N1, N2, and N3. Stage N1 represents the lightest sleep, where muscle tone decreases, eye movements slow down, and brain activity begins to shift from alpha to theta waves. In stage N2, the body's physiological processes slow further; heart rate and temperature decrease, and sleep spindles appear in the EEG pattern. The deepest stage, N3 (slow-wave sleep), is dominated by delta waves and is essential for tissue repair,

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immune system strengthening, and memory consolidation [12]. These stages of NREM sleep are restorative but lack significant dreaming activity.

The REM sleep phase, which constitutes around 20–25% of total sleep, is distinct because of its intense brain activity and vivid dreaming. During this stage, the brain exhibits electrical activity similar to wakefulness, but the skeletal muscles remain largely paralyzed—a state known as REM atonia. This atonia prevents individuals from physically acting out their dreams, thereby protecting the sleeper from injury. REM sleep is regulated by brainstem structures, particularly the pons and medulla, which release inhibitory neurotransmitters such as GABA (gamma-aminobutyric acid) and glycine that suppress motor neuron activity in the spinal cord [13].

Sleep paralysis occurs when this REM atonia persists while the individual regains consciousness. Normally, the transition from REM sleep to wakefulness involves simultaneous cessation of dreaming and restoration of voluntary muscle movement. However, in sleep paralysis, cortical arousal (awareness) occurs before muscle control returns, leading to a dissociated state where the person is mentally awake but physically immobile. This mismatch between the brain's wakefulness and the body's paralysis forms the core mechanism of sleep paralysis [14].

Neurophysiological studies have shown that during SP episodes, the reticular activating system (RAS), responsible for maintaining wakefulness, becomes active prematurely, while the subcoeruleus region of the brainstem, which maintains muscle atonia, remains engaged. This asynchronous activation results in partial REM intrusion into wakefulness. Consequently, individuals may also experience REM-related features such as vivid dream imagery, auditory or visual hallucinations, and sensations of chest pressure or suffocation. These experiences are often interpreted as supernatural or threatening due to their realistic nature [15].

The sleep–wake cycle is tightly controlled by the circadian rhythm, an internal biological clock regulated by the suprachiasmatic nucleus (SCN) in the hypothalamus. Factors such as irregular sleep schedules, night shifts, stress, and exposure to artificial light can disrupt this rhythm, leading to fragmented sleep architecture and increasing the likelihood of REM– wake overlap. Students, healthcare workers, and individuals with shift-based occupations in India often report higher incidences of sleep paralysis due to disturbed circadian patterns and inadequate sleep duration [16].

The relation between sleep deprivation and SP has been well-documented. Prolonged wakefulness or irregular sleep timing increases REM rebound—the compensatory increase in REM sleep following deprivation. During REM rebound, the boundaries between sleep stages become unstable, making transitions to wakefulness abrupt and incomplete. This instability significantly raises the risk of SP episodes. Similarly, individuals suffering from narcolepsy, a neurological disorder characterized by excessive daytime sleepiness and sudden REM onset, are particularly prone to recurrent sleep paralysis [17].

Stress and emotional disturbances further influence the sleep cycle by altering the secretion of hormones such as cortisol and melatonin. Elevated cortisol levels during night-time interfere with the normal onset of sleep and reduce deep NREM stages. Melatonin, secreted by the pineal gland, plays a crucial role in synchronizing circadian rhythm with light–dark cycles. In Indian urban populations exposed to extended screen time and late-night study or work routines, melatonin suppression has been linked to increased frequency of SP and other sleep disturbances [18].

Several polysomnographic studies have demonstrated that individuals experiencing sleep paralysis often show disrupted sleep architecture, with shortened NREM stages and longer REM latency. They also exhibit higher proportions of micro-arousals—brief awakenings that fragment the continuity of sleep. These physiological irregularities emphasize that SP is not merely a psychological event but a neurophysiological condition arising from instability within the REM control system [19].

In summary, sleep paralysis represents a complex interaction between REM sleep mechanisms, brainstem neurochemistry, and circadian regulation. Any factor that destabilizes these interactions—such as stress, sleep deprivation, irregular routines, or neurological disorders can precipitate the phenomenon. Understanding the normal sleep cycle and its regulation is therefore essential to comprehend why SP occurs and how it can be managed. Maintaining consistent sleep schedules, improving sleep hygiene, and reducing night-time stress are practical

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preventive measures. Recognizing these biological links also helps dispel myths and encourages scientific approaches toward treatment and awareness in the Indian population [20].

Pathophysiology of Sleep Paralysis

Sleep paralysis (SP) is a distinct parasomnia arising from an incomplete transition between rapid eye movement (REM) sleep and wakefulness. During normal REM sleep, the body experiences muscle atonia — a physiological paralysis that prevents individuals from acting out their dreams. This atonia results from inhibitory neurotransmitters such as gammaaminobutyric acid (GABA) and glycine, which suppress spinal motor neuron activity. When consciousness returns before these inhibitory signals wear off, the result is a state of wakefulness with persistent muscle paralysis, the hallmark of SP [21].

Neurophysiologically, SP originates from a dysregulation of the brainstem reticular formation, subcoeruleus nucleus, and magnocellular reticular neurons, which are responsible for inducing REM atonia. In normal REM sleep, these neurons are highly active, releasing inhibitory neurotransmitters to ensure motor immobility. When the reticular activating system (RAS) triggers cortical arousal prematurely, wakefulness and REM atonia coexist. This mismatch between cortical and spinal activation creates the sensation of being awake but unable to move [22].

Evidence from electroencephalographic (EEG) and polysomnographic studies indicates that SP represents a hybrid state combining features of both REM and waking consciousness. EEG recordings during episodes often show low-amplitude, mixed-frequency patterns resembling wakefulness, while electromyography (EMG) confirms persistent muscle atonia typical of REM sleep. This mixed state explains why individuals remain conscious of their surroundings yet immobile. The experience usually lasts for seconds to minutes and terminates spontaneously when motor inhibition dissipates [23].

The frightening sensations reported during SP, such as visual or auditory hallucinations and chest pressure, are explained by the abnormal activation of limbic and temporoparietal brain regions. The amygdala, which mediates fear and threat perception, becomes hyperactive during partial awakening, resulting in intense emotional arousal. Simultaneously, dysfunction of the temporo-parietal junction (TPJ) and vestibular nuclei may produce out-ofbody or floating sensations. These phenomena, while sometimes interpreted as supernatural, have a clear neurobiological basis [24].

Biochemically, sleep paralysis involves disturbances in neurotransmitters that regulate sleep stages — notably acetylcholine, serotonin, and norepinephrine. During REM sleep, acetylcholine activity is dominant, while monoaminergic neurons (serotonin and norepinephrine) are largely inhibited. When these monoaminergic systems reactivate too slowly upon awakening, REM-related atonia can persist into wakefulness. This delayed reactivation explains the continuation of paralysis and the concurrent dreamlike hallucinations [25].

A deficiency of orexin (hypocretin), a neuropeptide that promotes wakefulness, has also been implicated in recurrent SP and narcolepsy. Orexin neurons located in the lateral hypothalamus stabilize transitions between sleep and wake states. Their dysfunction may cause sudden intrusions of REM phenomena into waking consciousness, manifesting as sleep paralysis [26].

Psychological and emotional factors play a crucial role in predisposing individuals to SP. High levels of stress, anxiety, and irregular sleep schedules disrupt normal REM regulation and promote sleep fragmentation. Cortisol surges and hyperactivity of the limbic system heighten vulnerability to partial awakenings from REM sleep. Patients with post-traumatic stress disorder (PTSD) and generalized anxiety disorder (GAD) exhibit a higher frequency of SP due to exaggerated sympathetic responses during sleep [27].

The autonomic nervou” system (ANS) shows abnormal activation patterns during SP. Sympathetic overactivity results in tachycardia, sweating, and chest tightness, while voluntary movement remains inhibited. The diaphragm continues functioning, but intercostal and accessory muscles remain paralyzed, causing sensations of suffocation or “pressure on the chest.” This illusion of breathlessness is physiological and not lifethreatening [28].

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Genetic factors also appear to influence susceptibility to SP. Studies have reported associations between certain polymorphisms in circadian clock genes (CLOCK, PER2, PER3) and serotonin receptor genes (5-HT2A). These genetic variations may affect REM sleep timing and stability. However, Indian population-based genetic data remain limited, and further studies are necessary to clarify regional patterns [29].

Overall, the pathophysiology of sleep paralysis can be summarized as a multifactorial interaction among brainstem inhibition, neurotransmitter imbalance, and psychological stress. The persistence of REM atonia during awakening, combined with heightened limbic activity and autonomic arousal, produces the hallmark symptoms of paralysis, fear, and hallucination. Although alarming, the condition is physiologically benign, and awareness of its neurobiological basis can help reduce anxiety and cultural misinterpretation surrounding it [30].

Epidemiology of Sleep Paralysis

Sleep paralysis (SP) is a relatively common yet underreported parasomnia occurring across diverse populations and age groups. It can occur as isolated sleep paralysis (ISP) in healthy individuals or as part of narcolepsy. Epidemiological studies have revealed wide variation in its prevalence, influenced by cultural, genetic, and lifestyle factors. Globally, lifetime prevalence rates range from 7% to 40% depending on population type, with higher occurrence among students and individuals with irregular sleep patterns [31].

The prevalence of SP is notably higher in younger adults, particularly between the ages of 18 to 30 years, a group more prone to sleep deprivation, stress, and irregular sleeping schedules. In India, community-based studies have reported prevalence ranging from 8% to

15% in college and medical students. The Indian Sleep Disorders Association (ISDA) attributes these findings to academic pressure, nighttime gadget use, and poor sleep hygiene, which disturb REM cycles and predispose individuals to SP episodes [32].

Gender distribution of SP varies slightly between studies. Some evidence suggests that females experience SP more frequently, possibly due to greater vulnerability to anxiety and depression, while other reports show no significant gender difference. A study conducted in southern India found that SP was more prevalent among female students, particularly those with coexisting insomnia or stress-related disorders [33].

Sleep paralysis also shows strong associations with ethnic and cultural backgrounds. Studies indicate that individuals of Asian and African descent report higher rates of SP compared to Western populations. Cultural beliefs often influence how people interpret their experiences — in India, SP is sometimes described as “pressure by an unseen force” or associated with supernatural explanations. Such beliefs can increase fear and stress, worsening recurrence rates. Public awareness and education about the physiological basis of SP are therefore essential to reduce stigma [34].Several risk factors contribute to the epidemiology of SP. These include irregular sleep–wake cycles, shift work, sleep deprivation, jet lag, psychiatric conditions (especially anxiety and PTSD), and the use of stimulants or antidepressants. A cross-sectional study among Indian medical students found a significant correlation between high stress levels, poor sleep quality, and increased SP frequency. The same study emphasized that adopting regular sleep schedules and relaxation practices reduced episode recurrence [35].

Although SP is benign, its recurrence can cause substantial psychological distress. Epidemiological data suggest that up to 30% of individuals who experience SP develop anxiety or avoidance behavior related to sleep. Therefore, understanding its prevalence and risk factors is critical for prevention and early intervention. Regular screening in sleep clinics and universities can help identify at-risk populations and promote better sleep hygiene [36].

Clinical Features of Sleep Paralysis

Sleep paralysis (SP) presents as a transient state of complete muscle immobility occurring at the boundaries between sleep and wakefulness. It is characterized by the individual’s full consciousness combined with an inability to move or speak, often accompanied by fear, chest pressure, and vivid hallucinations. These episodes occur either while falling asleep (hypnagogic SP) or during awakening (hypnopompic SP). The affected person perceives their environment

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accurately but finds themselves frozen, unable to call out or move. Although the condition is benign, its symptoms can be extremely distressing to the individual [37].

The hallmark symptom of sleep paralysis is temporary paralysis of voluntary muscles, known as REM atonia. Normally, during REM (Rapid Eye Movement) sleep, inhibitory neurons in the brainstem release neurotransmitters such as GABA and glycine to suppress skeletal muscle activity. This mechanism prevents the body from physically enacting dreams. However, in SP, a mismatch occurs — the brain awakens before the inhibitory mechanism ends, leading to conscious awareness while the body remains paralyzed. Individuals can move only their eyes and may experience a heavy pressure sensation in the chest due to partial inhibition of respiratory muscles [38].

During the episode, the person often feels an intense sense of fear or dread, even without any external threat. This is attributed to the overactivation of the amygdala, the brain's center for emotional processing. The combination of fear, paralysis, and helplessness creates an experience that feels both realistic and terrifying. Some individuals also report palpitations, shortness of breath, and sweating once the episode resolves. The duration usually ranges from a few seconds to two minutes, but the perception of time is often distorted, making it feel much longer [39].

A striking clinical feature of SP is the presence of vivid hallucinations. These hallucinations are sensory experiences that blend dream content with wakeful perception. They may involve seeing shadowy figures, hearing voices, or feeling the presence of an unseen entity. Hallucinations in SP are broadly classified into three categories:

Intruder hallucinations – sensations of a threatening figure in the room, accompanied by auditory or visual stimuli such as footsteps, whispers, or dark silhouettes.

Incubus hallucinations – feelings of chest pressure, suffocation, or the sensation of an entity sitting on the chest.

Vestibular–motor hallucinations – out-of-body sensations, floating, spinning, or experiencing self-motion.

These hallucinations result from overlap between REM sleep and wakefulness, where dream imagery persists while conscious perception returns. Activation of the temporal and parietal cortices causes distortion in body orientation and spatial awareness, while increased limbic activity amplifies emotional intensity [39, 40].

The emotional aftermath of SP is also significant. Many individuals experience panic attacks or anxiety after repeated episodes. Persistent SP can lead to chronic sleep avoidance, where individuals deliberately delay sleep due to fear of recurrence. This in turn increases sleep deprivation, which paradoxically triggers more episodes, creating a vicious cycle. In Indian studies, university students who experienced frequent SP episodes reported poor sleep quality, excessive daytime sleepiness, and depressive symptoms compared to unaffected peers [41].

SP episodes are commonly reported when sleeping in the supine position (on the back). The supine posture is believed to facilitate airway resistance and promote REM-related muscle atonia, making individuals more vulnerable. Additionally, stress, irregular sleep patterns, and fatigue increase the frequency of episodes. Those who work night shifts or have disrupted circadian rhythms are particularly prone to recurrent SP. The episodes may occur singly or in clusters over a few nights, followed by long symptom-free intervals [41].

In Indian cultural contexts, SP is often interpreted in supernatural or spiritual terms. Individuals may attribute their experiences to ghosts, witches, or evil spirits — commonly referred to as "bhoot dab gaya" or "churail baithi thi" in rural areas. These cultural interpretations are deeply rooted in folklore and religious belief systems, which can intensify fear and delay medical consultation. Health professionals should recognize these interpretations while explaining the physiological basis of SP to patients, as reassurance and education can greatly reduce anxiety [42].

From a clinical standpoint, diagnosis of sleep paralysis is primarily based on the patient's description of symptoms. A typical episode involves preserved consciousness, muscle atonia, and hallucinations occurring during sleep–wake transitions. Polysomnography is used in research or complex cases to differentiate SP from other sleep disorders such as narcolepsy, obstructive sleep apnea, or nocturnal panic attacks. Unlike these conditions, SP does not cause oxygen desaturation or significant motor activity during episodes. A detailed sleep history, including sleep duration, stress levels, posture, and medication use, helps confirm the diagnosis [38].

Recurrent sleep paralysis is medically classified as Recurrent Isolated Sleep Paralysis (RISP) when it occurs in the absence of narcolepsy or other neurological conditions. Management includes reassurance, regulation of sleep cycles,

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avoidance of stimulants, and psychological counseling when anxiety is prominent. In India, the Indian Sleep Disorders Association (ISDA) recommends creating public awareness programs in colleges and medical institutions to dispel myths about SP and promote healthy sleep practices [42].

In summary, the clinical features of sleep paralysis are multifaceted, involving neurological, psychological, and cultural dimensions. The essential symptoms—transient paralysis, chest pressure, fear, and hallucinations—reflect a partial overlap between REM sleep and wakefulness. Understanding these features allows clinicians and pharmacists to distinguish SP from other disorders and provide both physiological and emotional reassurance to affected individuals [40, 42].

Risk Factors and Predisposing Conditions of Sleep Paralysis

Sleep paralysis (SP) is a multifactorial condition influenced by a combination of physiological, psychological, and environmental factors. Although the underlying mechanism is primarily neurophysiological, several external and internal conditions can increase an individual's susceptibility to SP. Identifying these risk factors is crucial for prevention, early intervention, and patient counseling. Research conducted globally and in India highlights that lifestyle habits, sleep quality, mental health, and genetic predisposition all contribute to the occurrence of this parasomnia [43].

1. Sleep Deprivation and Irregular Sleep Patterns

One of the most well-established risk factors for SP is sleep deprivation. Inadequate or fragmented sleep disrupts the normal cycling between REM and non-REM stages, leading to incomplete transitions between sleep and wakefulness. When REM sleep intrudes into waking consciousness, it produces the characteristic symptoms of SP. Individuals who sleep fewer than six hours per night or have irregular bedtimes are more prone to recurrent episodes. A study conducted among Indian university students showed that late-night study schedules, excessive screen time, and irregular sleeping hours significantly increased the risk of SP [44].

Shift work and jet lag are additional contributors. People engaged in night duties or those who frequently travel across time zones experience disturbances in their circadian rhythm, which controls the sleep–wake cycle. This disruption alters REM onset timing and increases REM fragmentation, creating favorable conditions for SP. Maintaining consistent sleep timing, even on weekends, has been shown to reduce episodes in susceptible individuals [45].

2.Psychological Stress and Emotional Disturbances

Emotional stress plays a major role in the onset of sleep paralysis. Psychological conditions such as anxiety, depression, and post-traumatic stress disorder (PTSD) are strongly associated with higher SP frequency. Stress leads to increased secretion of cortisol and sympathetic activation, which disrupts normal REM regulation. Moreover, anxious individuals often experience hypervigilance and fragmented sleep, both of which heighten the risk of REM–wake overlap. Indian studies have documented that students under academic or emotional stress during examination periods report a higher frequency of SP episodes compared to stress-free intervals [46].

Individuals with a history of panic attacks or generalized anxiety disorder often perceive SP episodes with greater emotional intensity. The amygdala's hyperactivation during partial arousal amplifies fear, creating a self-perpetuating cycle of stress and recurrence. Chronic stress also alters neurotransmitter levels (notably serotonin and norepinephrine), further destabilizing REM regulation. Effective management of stress through relaxation techniques, meditation, and counseling has been shown to significantly reduce SP occurrence [47].

3.Sleep Posture and Physical Conditions

Sleep position has a significant impact on the likelihood of SP episodes. Studies consistently report that SP is more common in individuals who sleep supine (on their back). In this posture, gravity can cause the tongue and soft tissues to fall backward, increasing airway resistance and affecting breathing patterns. This may trigger brief awakenings during REM sleep while atonia persists, resulting in the classical paralysis and chest pressure sensation. A study among Indian medical students found that 72% of reported SP episodes occurred in the supine position, suggesting a strong link between body posture and episode onset [48].

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Other physical factors such as obesity, respiratory disorders, and sleep apnea also predispose individuals to SP. Obesity narrows the upper airway, leading to disturbed breathing and frequent arousals during REM sleep. Similarly, people suffering from asthma, sinus congestion, or chronic fatigue syndrome may have more disrupted sleep architecture, which increases the probability of REM intrusion into wakefulness.

4.Substance Use and Medication Effects

Certain drugs and lifestyle habits can influence the development of SP by altering neurotransmitter balance or REM sleep patterns. Stimulants such as caffeine, nicotine, and amphetamines, as well as alcohol or sedative withdrawal, can cause REM rebound, resulting in abnormal transitions between sleep states. Similarly, medications that affect serotonin and norepinephrine — including antidepressants, beta-blockers, and corticosteroids — may either suppress or exaggerate REM phases depending on dosage and individual sensitivity
[49].

In the Indian context, consumption of energy drinks, late-night caffeine use, and smartphone exposure before bedtime have emerged as modern risk factors. Blue light from screens suppresses melatonin secretion, delaying sleep onset and reducing sleep quality. Counseling individuals to avoid caffeine after evening hours and maintain digital hygiene before bedtime can markedly reduce SP episodes [49].

5.Genetic and Familial Predisposition

Genetic factors are believed to account for approximately 30–40% of SP vulnerability. Twin studies and family-based analyses suggest that genes regulating circadian rhythm and REM sleep — such as PER2, PER3, and CLOCK — may influence susceptibility. People with a family history of SP or narcolepsy are more likely to experience recurrent episodes. In addition, heritable traits such as high emotional reactivity, neuroticism, or anxiety-prone personality types may indirectly contribute by increasing physiological arousal during sleep. Indian data on genetic contribution remain limited but emerging studies point toward a familial tendency among siblings reporting similar sleep phenomena [50].

6.Coexisting Sleep and Medical Disorders

SP frequently coexists with other sleep-related conditions such as insomnia, narcolepsy, obstructive sleep apnea, and restless leg syndrome. Among these, narcolepsy shows the strongest association due to shared mechanisms involving REM intrusion. Individuals with chronic insomnia exhibit reduced sleep efficiency and shortened REM latency, predisposing them to isolated or recurrent SP. Additionally, disorders such as migraine, epilepsy, and autonomic dysfunction have been associated with higher prevalence of SP due to altered neurophysiological control during sleep–wake transitions [50].

In summary, sleep paralysis is influenced by an interplay of biological, psychological, and environmental risk factors. Lack of adequate sleep, stress, irregular schedules, certain medications, and sleeping posture are major contributors. Awareness and modification of these risk factors, along with stress management and consistent sleep hygiene, can drastically reduce episode frequency. In India, where cultural interpretations often delay medical attention, public education and proper counseling play a key role in prevention and reassurance [46, 49].

Diagnosis of Sleep Paralysis

The diagnosis of sleep paralysis (SP) primarily depends on clinical evaluation, as there are no specific laboratory or imaging tests that confirm the condition directly. The main diagnostic aim is to differentiate SP from other sleep-related disorders such as narcolepsy, epilepsy, or panic attacks. In most cases, SP can be recognized by its characteristic pattern of temporary paralysis upon awakening or at sleep onset, where the patient remains conscious but unable to move [51].

In India, cases of SP are often underdiagnosed because patients may interpret the experience through spiritual or supernatural beliefs. Therefore, taking a comprehensive clinical history that includes both medical and cultural aspects is essential for proper diagnosis. Physicians are encouraged to explore the timing, frequency, and emotional response associated with the episodes to identify patterns suggestive of recurrent isolated SP [52].

1. Clinical Evaluation and History Taking

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A detailed history of sleep behavior is the foundation for diagnosis. Clinicians should ask the patient about sleep-wake timings, sleep quality, and presence of stress or anxiety, as these can precipitate SP episodes. The defining feature is consciousness during paralysis, accompanied by symptoms such as fear, chest pressure, or visual and auditory hallucinations.

The clinician should also inquire whether episodes occur at sleep onset (hypnagogic SP) or upon awakening (hypnopompic SP), since this helps in classifying the type of paralysis and excluding narcolepsy [53].

In Indian studies, it has been reported that a significant number of cases are missed due to limited awareness among healthcare professionals, leading to misclassification under psychiatric or neurological disorders. Recognizing SP as a benign but distressing REM sleep phenomenon prevents unnecessary medical interventions [53].

2. Diagnostic Criteria

The International Classification of Sleep Disorders, Third Edition (ICSD-3) specifies the diagnostic features of Recurrent Isolated Sleep Paralysis (RISP).

The diagnostic criteria include:

One or more episodes of inability to move voluntary muscles during sleep onset or awakening.

The episode lasts a few seconds to several minutes.

The person is conscious and aware during the episode.

The event causes distress, fear, or sleep disruption.

The symptoms are not explained by another mental or medical condition [54].

These criteria ensure that SP is clearly distinguished from narcolepsy, REM behavior disorder, and panic attacks.

3.Polysomnography (Sleep Study)

In complex or recurrent cases, polysomnography (PSG) may be used to confirm the diagnosis and rule out other sleep disorders. PSG records brain waves (EEG), muscle activity (EMG), and eye movements (EOG) throughout the night.

During an SP episode, PSG typically shows REM-related muscle atonia persisting into wakefulness, indicating an overlap between REM and conscious states [55].

Polysomnographic studies conducted in Indian tertiary hospitals have demonstrated that patients with recurrent SP show shortened REM latency and fragmented sleep, reflecting poor REM stability. This tool also helps rule out nocturnal seizures or obstructive sleep apnea, which may mimic SP [55].

4. Differential Diagnosis

SP must be differentiated from several other sleep or neurological conditions.

Narcolepsy presents with SP but also includes cataplexy and daytime sleepiness.

Epileptic seizures involve loss of consciousness and postictal confusion, unlike SP.

Nocturnal panic attacks cause sudden awakening with fear but without paralysis.

Night terrors and sleepwalking occur during non-REM sleep and lack awareness during episodes [52].

Thus, a careful history and, when needed, PSG are critical for confirming the diagnosis. Early recognition also reduces anxiety and improves patient education.

5.Psychological Evaluation

Patients who develop fear of sleeping or anxiety after SP episodes may undergo psychological screening using instruments such as the Pittsburgh Sleep Quality Index (PSQI) and Beck Anxiety Inventory. These tools assess the emotional burden of SP and help plan counseling or behavioral therapy. Educating patients that SP is a harmless physiological event helps in reducing distress and restoring normal sleep confidence [53].

Management and Further Considerations of Sleep Paralysis

The management of sleep paralysis (SP) focuses on reducing the frequency and intensity of episodes, addressing underlying psychological factors, and promoting healthy sleep hygiene. Since SP is usually benign and self-limiting, pharmacological treatment is rarely required. Instead, emphasis is placed on patient education, behavioral therapy, and management of comorbid conditions such as anxiety, depression, or narcolepsy [56].

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In India, awareness about SP remains limited, and many patients first seek help from traditional healers or spiritual practitioners. Thus, education and reassurance play a crucial role in treatment, helping patients understand that the experience is caused by a temporary physiological overlap between wakefulness and REM sleep rather than supernatural forces
[57].

1.Patient Education and Reassurance

The most important step in managing SP is educating patients about the benign nature of the condition. Clinicians should explain that SP occurs when REM-related muscle atonia persists briefly into wakefulness. Patients should be reassured that the inability to move or speak is temporary and does not indicate paralysis or brain dysfunction.

Educational counseling can significantly reduce fear and recurrence rates. Studies from Indian sleep clinics have shown that when patients are informed about the scientific basis of SP, the anxiety associated with episodes decreases markedly, and sleep quality improves [57].

2.Sleep Hygiene and Behavioral Interventions

Maintaining proper sleep hygiene is a cornerstone of SP management. This includes:

Maintaining a consistent sleep schedule, with adequate 7–8 hours of sleep per night.

Avoiding irregular bedtime and daytime napping.

Reducing caffeine and alcohol intake, especially near bedtime.

Limiting screen exposure before sleep.

Sleeping in a comfortable and dark environment to enhance REM stability.

Behavioral methods such as cognitive behavioral therapy for insomnia (CBT-I) have shown effectiveness in reducing SP frequency. CBT-I helps correct maladaptive beliefs about sleep and reduces anxiety that often precedes SP episodes [58].

Regular exercise, meditation, and relaxation practices (such as yoga and deep-breathing techniques) can also help stabilize sleep patterns and reduce emotional stress, which are known triggers for SP. In Indian studies, participants who practiced yoga regularly reported fewer SP episodes and lower anxiety levels [59].

3.Management of Psychological and Medical Comorbidities

SP is frequently associated with anxiety, depression, post-traumatic stress disorder (PTSD), and sleep deprivation. Addressing these comorbidities is vital for long-term relief. Psychotherapy, mindfulness training, and stress reduction programs are beneficial adjuncts.

In individuals where SP is secondary to narcolepsy, treatment with REM-suppressing medications such as selective serotonin reuptake inhibitors (SSRIs) or tricyclic antidepressants (e.g., clomipramine) may be considered. These drugs suppress REM sleep, thereby reducing the occurrence of SP episodes [60]. However, pharmacological therapy should be reserved for patients with severe or recurrent SP unresponsive to behavioral interventions.

4.Coping Strategies During an Episode

Patients should be trained to manage episodes effectively when they occur. Keeping eyes closed, attempting slow rhythmic breathing, and focusing on small voluntary movements such as moving fingers or toes can help terminate the episode. Recognizing that the event is temporary and harmless reduces panic and helps restore full mobility faster [56].

Some patients benefit from sleep position modification, as SP tends to occur more frequently in the supine (lying on back) posture. Sleeping on the side can significantly reduce recurrence rates [58].

5.Cultural and Psychosocial Considerations

In India and many Asian cultures, SP has been traditionally associated with supernatural interpretations—such as “pressure from a spirit” or “ghost attack.” These beliefs can increase distress and delay medical consultation. Therefore, it is essential for healthcare providers to approach such patients with cultural sensitivity, combining scientific explanation with respect for personal beliefs [57].

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Public health initiatives that include community awareness programs and sleep education workshops can further help in destigmatizing SP. Incorporating SP education into mental health and sleep hygiene campaigns in schools and colleges may promote early recognition and management among young adults [59].

Research Directions and Gaps in Sleep Paralysis

Although significant progress has been made in understanding sleep paralysis (SP), major research gaps still limit the development of effective diagnostic and treatment strategies. Current findings have highlighted the role of REM sleep dysregulation, psychological factors, and cultural influences, but many aspects remain unclear. Future studies must aim to establish a clearer connection between these variables and enhance awareness, especially in developing countries like India [61].

Epidemiological and Cultural Gaps

Epidemiological data on SP from non-Western and Indian populations remain scarce. Most studies are hospital-based and focus on small sample sizes, which do not represent the general population. There is an urgent need for community-level research to determine true prevalence, associated factors, and differences across regions and occupations. Moreover, in India, SP is often interpreted through cultural or supernatural beliefs, which delays medical attention. Understanding these sociocultural aspects through anthropological and psychological research can help develop culturally sensitive awareness programs [62].

Neurobiological and Psychological Research Needs

The neurobiological mechanisms underlying SP—especially the interaction of neurotransmitters such as serotonin, acetylcholine, and dopamine—are not yet fully established. While studies confirm that REM atonia persists abnormally into wakefulness, the exact neural pathways remain uncertain. Advanced imaging tools like fMRI and EEGbased connectivity mapping could help identify specific regions involved in REM dysregulation. Indian research in this field is still minimal due to limited access to sleep laboratories and specialized neuroimaging equipment [63].

SP is also closely linked to stress, anxiety, and poor sleep hygiene, yet few longitudinal studies explore how these factors contribute to recurrence. Understanding psychological vulnerability—particularly in students, shift workers, and individuals with trauma—may help in designing targeted prevention programs [64].

Intervention and Methodological Gaps

Therapeutic research on SP remains in its early stages. Most recommendations rely on behavioral interventions, yoga, and psychoeducation, with little evidence from controlled clinical trials. In India, yoga-based interventions and CBT (Cognitive Behavioral Therapy) show promise, but further validation through randomized studies is essential.

Moreover, the absence of standardized diagnostic tools limits consistency across studies. Developing validated questionnaires and incorporating polysomnography criteria into Indian sleep clinics can improve diagnostic accuracy and enable cross-cultural comparison of findings [65].

In summary, addressing these research gaps—especially through multicentric, culturally informed, and technology-assisted studies—will deepen understanding of SP and pave the way for better management strategies tailored to diverse populations.

II. CONCLUSION

Sleep paralysis is a transient yet distressing phenomenon that reflects an incomplete transition between rapid eye movement (REM) sleep and wakefulness. During this state, the individual becomes conscious but remains unable to move or speak, often accompanied by vivid hallucinations and a strong sense of fear. What was once regarded as a supernatural or spiritual occurrence is now understood as a neurophysiological event linked to temporary disturbances in sleep regulation. Over recent decades, growing research has highlighted that factors such as irregular sleep schedules, emotional stress, anxiety, and sleep deprivation play a crucial role in triggering episodes of sleep paralysis. Although it can affect anyone, the condition is seen more frequently among students, shift workers, and individuals under psychological strain. By improving awareness and promoting healthy sleep habits, many of these risk factors can be effectively minimized.

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The physiological basis of sleep paralysis lies in the persistence of REM-related muscle atonia into wakefulness. This disconnect between brain arousal and body paralysis creates a vivid and frightening experience. The sensory perceptions often reported—such as pressure on the chest or a feeling of presence—are understood to be extensions of dream imagery carried into consciousness. Despite this understanding, the condition remains underrecognized in clinical practice, especially in developing countries, due to social stigma and cultural interpretations that associate it with paranormal events.

Effective management focuses primarily on non-pharmacological interventions such as regular sleep routines, stress management, and cognitive behavioral therapy. In India, traditional practices like yoga, meditation, and relaxation techniques have shown beneficial effects in reducing both the frequency and emotional impact of sleep paralysis episodes. Education and reassurance remain central to care, as understanding the benign nature of the condition greatly alleviates fear and anxiety in affected individuals.

In summary, sleep paralysis is a multifactorial and manageable sleep disorder that bridges the fields of neurology, psychology, and cultural study. Continued research, particularly in the Indian context, is essential to close existing gaps in epidemiological data, awareness, and treatment approaches. With sustained public education and interdisciplinary research, sleep paralysis can move from a misunderstood experience to a well-recognized and effectively managed aspect of sleep health.

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