

Somnambulism: clinical aspects and pathophysiological hypotheses

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Somnambulism, or sleepwalking, can give rise to a wide range of adverse consequences and is one of the leading causes of sleep-related injury. Accurate diagnosis is crucial for proper management and imperative in an ever-increasing number of medicolegal cases implicating sleep-related violence. Unfortunately, several widely held views of sleepwalking are characterised by key misconceptions, and some established diagnostic criteria are inconsistent with research findings. The traditional idea of somnambulism as a disorder of arousal might be too restrictive and a comprehensive view should include the idea of simultaneous interplay between states of sleep and wakefulness. Abnormal sleep physiology, state dissociation, and genetic factors might explain the pathophysiology of the disorder.

Introduction

Despite almost five decades of clinical and laboratory investigations, the pathophysiology of somnambulism (or sleepwalking) remains poorly understood. Furthermore, unlike most other sleep disorders, somnambulism is still diagnosed mainly or exclusively on the basis of the patient's clinical history. The widespread belief that sleepwalking is a benign disorder is erroneous because somnambulism can result in various adverse consequences. Although childhood somnambulism is often transitory and harmless, sleepwalking in adulthood has substantial harm potential, including the placing of oneself in dangerous situations (eg, running into walls and furniture, trying to escape imaginary threats, leaving one's house), destruction of property, and serious injuries to the sleeper, bed partner, or others.¹⁻³ Somnambulism is a major cause of reported injurious or violent behaviours after an arousal from sleep,^{4,7} and episodes resulting in injury to the patient or others are more prevalent than is generally recognised.^{3,8,9} A history of aggressive or injurious behaviour during sleep is what leads most adult sleepwalkers to consult a medical specialist.^{1-3,10} The number of legal cases of sleep-related violence is rising.^{4,7,11} Driving of motor vehicles,¹² suspected suicide,¹³ and even homicide¹⁴ or attempted homicide can occur during somnambulism, raising fundamental questions about the medicoforensic implications of these acts and the neurophysiological and cognitive states that characterise patients during such episodes.

Role of somnambulism within sleep

On the basis of a range of physiological measures including electroencephalogram (EEG) activity, eye movement activity, and level of muscle tone, sleep is divided into two very distinct states—rapid eye movement (REM) sleep and non-rapid-eye-movement (NREM) sleep. NREM sleep can be further subdivided into three stages, which the American Academy of Sleep Medicine's revised nomenclature refers to as N1 (sleep onset), N2 (light sleep), and N3 (deep or slow-wave sleep). Table 1 lists the main characteristics of REM and NREM sleep stages, and figure 1 shows the corresponding EEG traces. These sleep states are organised into sleep

cycles that have a characteristic distribution across a typical night (figure 2). The neural structures implicated in these sleep states (ie, the brainstem, anterior and posterior hypothalamus, basal forebrain, ventral tegmental area, thalamus, and cortex), their pathways and interconnections, and the neurotransmitters that generate and regulate these different states are many and have complex interactions.^{15,16}

NREM sleep and REM sleep alternate throughout the night in cycles that last for a mean of around 90 min. However, deep sleep occurs preferentially during the first third of the night, whereas periods of REM sleep are longest during the last third of the night. Somnambulism usually arises from the deepest sleep stage (ie, N3 or slow-wave sleep). Thus, episodes typically take place during the first third of the night when slow-wave sleep is predominant, although episodes can also occur in N2 sleep. Somnambulism is therefore classified as an NREM parasomnia, the category that also includes confusional arousals and sleep terrors. These three parasomnias, which can coexist, have been termed disorders of arousals and might be different phenotypes of the same underlying cause.

Clinical characteristics and epidemiology

Somnambulism is defined as “a series of complex behaviours that are usually initiated during arousals from slow-wave sleep and culminate in walking around with an altered state of consciousness and impaired judgment”.¹⁷ Some somnambulistic behaviours can be mundane and stereotyped, such as gesturing, pointing at a wall, or walking around a room, but others (especially in adults) are surprisingly complex and might necessitate high-level planning and motor control—eg, getting dressed, cooking, playing a musical instrument, driving a car. Episodes can last from a few seconds to more than 30 min. Most behavioural episodes are characterised by misperception and unresponsiveness to external stimuli, mental confusion, perceived threat, and variable retrograde amnesia. The American Academy of Sleep Medicine has established diagnostic criteria for somnambulism in the second International Classification of Sleep Disorders (panel).¹⁷ Sleep-related abnormal sexual behaviours

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	Specific EEG features	Other characteristics	Stage-specific sleep disorders
Wakefulness (eyes closed)	Alpha waves (8–12 Hz)	Alpha rhythm prominent in occipital cortex	..
NREM sleep			
N1 (sleep onset)	Theta waves (4–8 Hz)	Slow, rolling eye movements	Hypnic jerks, hypnagogic hallucinations
N2 (light sleep)	Sleep spindles (11–16 Hz), K-complexes (>0.5 s)	Background of theta rhythm with occasional sleep spindles and K-complexes	Bruxism, nocturnal frontal lobe epilepsy
N3 (slow-wave or deep sleep)	Delta waves (0.5–2 Hz; amplitude >75 µV), slow oscillations (<1 Hz)	Delta waves occupy more than 20% of the sleep period	Somnambulism, sleep terrors, confusional arousals
REM sleep (paradoxical sleep)	Low amplitude, mixed frequencies, saw-tooth theta waves	Rapid eye movements, muscle atonia, desynchronised EEG	REM behaviour disorder, nightmares

EEG=electroencephalogram. NREM=non-rapid-eye-movement. REM=rapid eye movement.

Table 1: Key characteristics of sleep stages

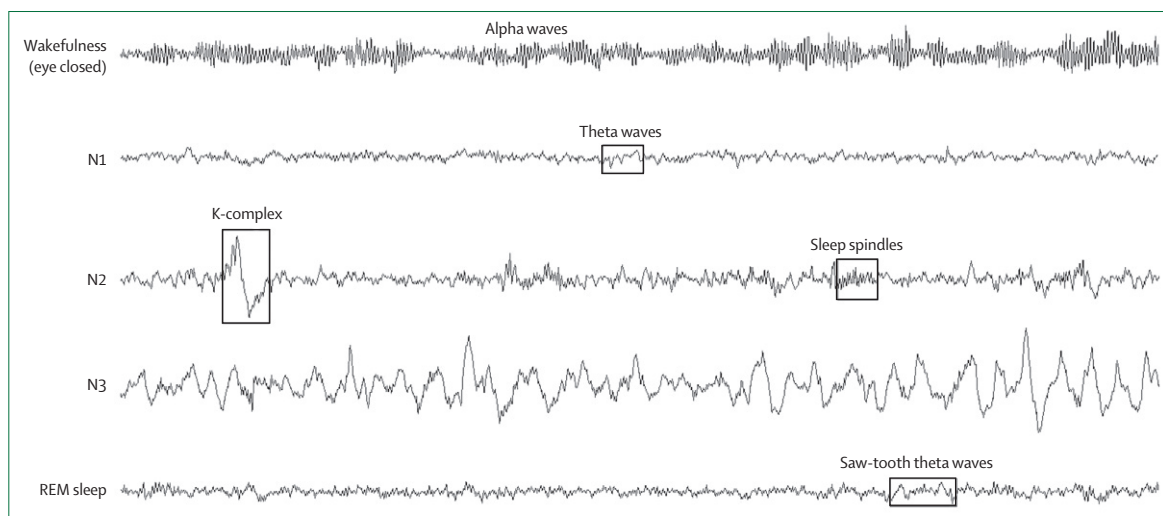


Figure 1: Electroencephalogram tracings corresponding to relaxed wakefulness and different sleep stages in healthy individuals
REM=rapid eye movement.

(so-called *sexsomnia*¹⁸) and sleep-related eating disorder,¹⁹ which are distinct and specialised variants of NREM sleep parasomnias, are not discussed in this Review because they are not classified as *somnambulism* per se.¹⁷

Somnambulism is more common in childhood than in adulthood; most children will have, at least temporarily, one or more of the NREM sleep parasomnias. However, childhood *somnambulism* is typically benign (and non-violent) and usually does not necessitate intervention. The prevalence of *somnambulism* is around 3% in toddlers (age 2–5–4 years),²⁰ and steadily increases to about 11% at 7 and 8 years²¹ and 13.5% at 10 years, before falling to 12.7% at 12 years (data unpublished for ages 10 and 12; figure 3). The prevalence of *somnambulism* rapidly decreases during adolescence^{23,24} to about 2–4% in adulthood.^{9,25,26} Thus, most children outgrow the disorder during adolescence, but *somnambulism* can persist into adulthood in as much as 25% of cases.²⁵ Why *somnambulism* persists into adulthood in some people but not in others is unclear. *Somnambulism* can also arise *de novo* in adults.

No evidence suggests that chronic *somnambulism* during adulthood is associated with the subsequent development of neuropathological disorders (however, longitudinal studies have not been done). This finding contrasts with those for REM sleep behaviour disorder, a parasomnia characterised by loss of muscle atonia and prominent behaviours during REM sleep that generally occurs in patients older than 50 years and is associated with the development of neurodegenerative processes including Parkinson's disease and dementia with Lewy bodies.²⁷

Epidemiological data suggest that roughly 25% of adult sleepwalkers self-report concurrent anxiety or mood disorders.⁹ In early childhood, the occurrence of *somnambulism* might be associated with separation anxiety,²⁰ and anxiety or stress might increase the occurrence of episodes in both children and adults. However, most adults who sleepwalk do not present with a psychiatric or personality disorder,^{3,8} and successful treatment of axis 1 disorders (as defined in the fourth edition of the Diagnostic and Statistical

Manual of Mental Disorders²⁸) usually does not affect sleepwalking frequency.³

Roughly 80% of sleepwalkers have at least one affected family member, and the prevalence of somnambulism is higher in children whose parents have a history of somnambulism than in those whose parents have no such history.²⁹ First-degree relatives of sleepwalkers have a ten-fold increased likelihood of somnambulism compared with the general population.²⁹ A population-based Finnish twin cohort²⁵ showed a concordance rate 1.5 times higher in monozygotic than in dizygotic pairs for childhood somnambulism and 5 times higher in monozygotic than in dizygotic pairs for adult somnambulism. These results suggest that a substantial proportion of the reported familial aggregation could be explained by genetic factors.

Common misconceptions

Several widely held views in the medical and neuroscientific communities about somnambulism, including diagnostic considerations, run counter to developments in the specialty. We present three key examples: that sleepwalking has no daytime consequences, that it is characterised by amnesia for the episode, and that it is an automatic behaviour arising in the absence of dream-like mental activity.

Somnambulism has no daytime consequences

Daytime somnolence or impairment of daytime functioning has never been part of the clinical portrayal of somnambulism. Despite the well documented increase in slow-wave sleep fragmentation, little information is available about subjective or objective daytime levels of vigilance. A study³⁰ of ten adult sleepwalkers showed that they had daytime somnolence even after episode-free nights. Notwithstanding a similar proportion of slow-wave sleep, sleepwalkers had significantly lower mean sleep onset latencies (ie, the time needed to transition from wakefulness to sleep) on the multiple sleep latency test (the gold standard in objective assessment of excessive daytime sleepiness) than did matched controls. Seven of these sleepwalkers (and none of the controls) had a mean latency of less than 8 min,³⁰ which is the accepted threshold for clinical somnolence. Similarly, in a retrospective study, Oudiette and colleagues³¹ used the Epworth sleepiness scale to show that 47% of 43 patients with NREM sleep parasomnia had a score greater than 10 (the cutoff for pathological somnolence). These findings are further supported by our results from 71 adult sleepwalkers, which showed that 32 patients (45%) had an Epworth sleepiness score greater than 10, compared with only eight patients (11%) in a group of 71 healthy controls (unpublished). In this cohort, reported somnolence did not seem to correlate with the number of nocturnal awakenings, periodic leg movements in sleep, or higher apnoea-hypopnoea indices.

Taken together, these results suggest that excessive daytime somnolence might be an important characteristic

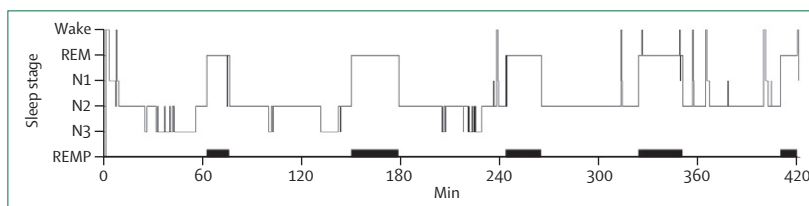


Figure 2: Distribution of different sleep stages across a typical night in healthy individuals
REM=rapid eye movement. REMP=rapid eye movement sleep periods.

Panel: Somnambulism diagnostic criteria of the American Academy of Sleep Medicine—second International Classification of Sleep Disorders

- A Ambulation occurs during sleep
- B Persistence of sleep, a changed state of consciousness, or impaired judgment during ambulation shown by at least one of the following:
 - Difficulty in arousal of the person
 - Mental confusion when awakened from an episode
 - Amnesia (complete or partial) for the episode
 - Routine behaviours that occur at inappropriate times
 - Inappropriate or nonsensical behaviours
 - Dangerous or potentially dangerous behaviours
- C The disturbance is not better explained by another sleep, medical, neurological, or mental disorder; drug use; or substance use disorder

Adapted from the second International Classification of Sleep Disorders.¹⁷

of somnambulism. Transcranial magnetic stimulation and brain imaging have been used to show daytime functioning anomalies in sleepwalkers, thereby supporting the idea that clinical considerations should not be limited to patients' sleep.^{32,33}

Somnambulism is characterised by episodic amnesia

Because somnambulism is usually diagnosed exclusively on the basis of clinical history, the validity and reliability of the diagnostic criteria are of prime importance. A study³³ of the reliability of the diagnosis of various parasomnias based on criteria from the second International Classification of Sleep Disorders showed that sleepwalking only had a "fair" interobserver reliability because of disagreement about the "amnesia for the episode" criterion—which was also included in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders.²⁸ However, work based on 94 patients referred to our sleep clinic for chronic sleepwalking (unpublished data presented at the fourth Meeting of the World Association of Sleep Medicine) suggests that a substantial proportion of adult sleepwalkers recall specific elements of their episodes, at least occasionally. Upon awakening, 80% of patients remembered sleep mentation during somnambulistic episodes. Additionally, 61% reported remembering specific behaviours that they displayed during episodes. Similarly, perceptual elements from the

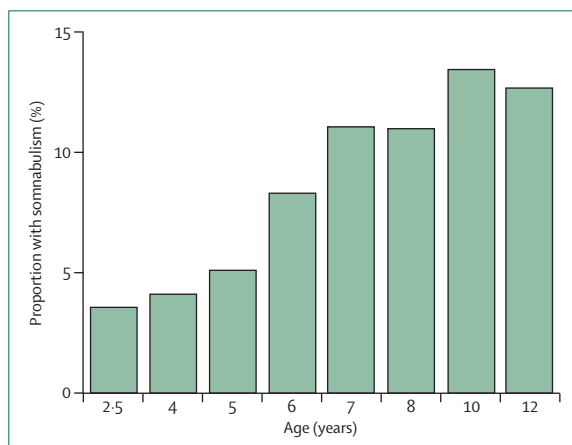


Figure 3: Prevalence of somnambulism in children aged 2.5–12 years in a prospective cohort of 1400 children
Adapted from data from the Quebec Longitudinal Study of Child Development (Quebec Institute of Statistics). Only data for children aged 2.5–8 years have been published.²²

sleepers' actual environment during somnambulistic episodes were recalled upon awakening by 75% of patients. 75% of sleepwalkers reported that emotions including fear, anger, frustration, and helplessness were often or always experienced during their episodes.³⁵ These data add to descriptive reports^{36–38} showing that many patients can and do recall at least portions of episodes upon awakening, and thus suggest that complete amnesia of the event is not standard for adult sleepwalkers. In children, somnambulism might be more likely to consist of automatic behaviours and complete amnesia might be more common, possibly because of higher arousal thresholds.³⁹

Somnambulism is an automatic behaviour arising in the absence of dreamlike mental activity

That dreamlike mentation is not confined to REM sleep only,^{40,41} but occurs in NREM sleep (including slow-wave sleep) also, is now well established. Previously, complex mental contents were thought not to be implicated in behaviour during somnambulistic episodes, but a growing body of evidence shows the contrary. In addition to well documented cases,^{36–38} empirical evidence suggests that sleep mentation is not only frequently part of the main experience of somnambulism, but also can modulate motor behaviour during an episode. Oudiette and colleagues³¹ showed that 27 of 38 patients (71%) recalled short, unpleasant, dreamlike mentations associated with sleepwalking episodes. Furthermore, the mentation reported by patients was congruent with recorded nocturnal behaviour, suggesting that sleepwalking might be the acting out of dreamlike mentations. Sleep laboratory investigations of adult sleepwalkers suggest that, when available, patients' phenomenological experiences are broadly consistent with behaviours recorded during episodes.⁴² However, although

sleepwalkers are aware of their immediate physical environment during an episode and can interact with other people nearby, such findings are not reported in normal dreaming or in dream-enactment behaviours in patients with REM-sleep behaviour disorder.⁴³ Additionally, sleepwalkers' eyes are usually open throughout episodes, thereby allowing navigation, whereas REM and NREM dreaming occur in a virtual, offline environment with very restricted awareness of the actual physical environment.

Various findings show that somnambulistic behaviours are construed by many patients as being motivated by an intrinsic sense of urgency or underlying logic (although judgment is often impaired) that accounts for actions during actual episodes. This evidence raises important questions about the role that sleep-related mentations have in how somnambulistic episodes are experienced and unfold.

Diagnosis and clinical management

Both nocturnal frontal lobe epilepsy and REM-sleep behaviour disorder can cause complex and sometimes violent behaviours during sleep that can be confused with somnambulism (table 2). Guidelines²² and the frontal lobe epilepsy and parasomnias scale⁴⁴ have been suggested to help with differential diagnosis. Challenging cases might warrant thorough polysomnographic assessments with an extended EEG montage and continuous audiovisual recording. Disorders that are known to increase pressure for deep sleep or arousals during sleep, or to induce confusional states should be considered in clinical management of somnambulism. Factors that increase pressure for deep sleep include intense exercising in the evening, fever, and lack of sleep; disorders that produce repeated arousals during sleep include sleep apnoea and periodic leg movements during sleep (figure 4)

Situations that intensify pressure for slow-wave sleep (eg, sleep deprivation) might precipitate disorders of arousal in predisposed individuals. Thus, clinicians should emphasise the importance of getting sufficient sleep and avoidance of irregular sleep schedules to patients with somnambulism. Similarly, most causes of increased arousals from sleep (eg, environmental disturbances, stress) and the presence of concomitant sleep disorders capable of causing recurrent microarousals are precipitating factors. Thus, clinicians should ensure that breathing problems and movement disorders during sleep, if present, are treated, which should alleviate and control the parasomnia.

Disorders that ease dissociation or induce confusional states can trigger somnambulism. Sleepwalking has been noted in patients with psychiatric disorders⁴⁵ and those given various psychotropics, including sedatives, hypnotics, antidepressants, neuroleptics, lithium, stimulants, and antihistamines.^{5,45,46} Possibly, these disorders and drugs ease regional dissociation and lead to somnambulism through modulation of states of sleep and alertness.

	Somnambulism	Nocturnal frontal lobe epilepsy	REM-sleep behaviour disorder
Age at onset	Usually childhood	Variable	>50 years
Family history	69–90% of patients	Less than 40% of patients	Rare
Time of night	First third of the night	Anytime	Last half of the night
Sleep stage	Slow-wave sleep	N2 sleep	REM sleep
Event duration*	1–30 min	A few seconds to 3 min	1–10 min
Events per week *	0–3	≥7	1–7
Behavioural manifestations	Simple to complex movements (ambulation), might be goal directed, eyes are open	Highly stereotyped (eg, abnormal posturing) and purposeless, eyes can be open or closed	Typically gross motor movements (eg, flailing of limbs), related to dream content, eyes are closed
Can get out of bed	Yes	No (patient remains prone or supine)	Yes
Can leave bedroom	Yes	No	Rarely
Interaction with immediate environment	Might respond to external stimulation or verbal questions, and navigate in familiar environments	No	Poor or incidental
Spontaneous full awakening after the event	Infrequent	Rare	Frequent
Recall of the events	Variable	No	Vivid dream recall
Mental state after the event when awakened	Confused and disoriented	Usually fully awake	Fully awake and functional
Arousal threshold	High	Not applicable	Low
Triggers	Sleep deprivation, noise, stress, obstructive sleep apnoea, periodic leg movements during sleep	Often none	Alcohol withdrawal, selective serotonin reuptake inhibitors, tricyclic antidepressants
Autonomic activation	Low to moderate	Strong	None
Polysomnographic findings	Frequent awakenings and microarousals in slow-wave sleep, hypersynchronous delta waves	Often normal, epileptiform in about 10% of patients	Absence of muscle atonia or excessive phasic electromyographic activity in REM sleep
Potential for injury or violence	Yes	Yes	Yes

REM=rapid eye movement. *These values are estimates based on means reported in published work and should be interpreted with caution because frequency and duration of episodes vary greatly between and within patients.

Table 2: Key clinical features of somnambulism, nocturnal frontal lobe epilepsy, and REM-sleep behaviour disorder

Irrespective of underlying disorders, precautions should be taken to ensure a safe sleep environment. When the parasomnia continues to cause distress or poses a threat, three main treatment options are available: hypnosis, scheduled awakenings, and drugs. However, as emphasised in a 2009 review,⁴⁷ no adequately powered, controlled, clinical trials for the treatment of somnambulism have been done. Hypnosis (including self-hypnosis) is effective in both children and adults with chronic somnambulism.^{48,49} In children, the preferred treatment is anticipatory or scheduled awakening⁵⁰—a behavioural technique whereby parents awaken their child nightly about 15 min before the typical time of occurrence of an episode for about a month.

Drugs should be prescribed only when behaviours are potentially hazardous or extremely disruptive to bed partners or other household members. Benzodiazepines, particularly clonazepam and diazepam, have been used.^{51,52} These drugs decrease arousals and anxiety and suppress slow-wave sleep, but do not always adequately control sleepwalking.⁸ Even when pharmacotherapy is chosen, treatment should always include instructions

about regular sleep routine and good sleeping habits, avoidance of sleep deprivation, and stress management.

Theoretical frameworks for understanding somnambulism

Somnambulism is usually classified as a disorder of arousal;¹⁷ however, several clinical and experimental findings suggest that somnambulism could be due to a dysfunction in slow-wave sleep regulation (figure 4). We review these complementary views and the neurophysiological findings that support each conceptual framework.

Somnambulism as a disorder of slow-wave sleep

Two lines of evidence favour the notion of a dysfunction in slow-wave sleep as the primary underlying cause for somnambulism—namely, the presence of intrinsic abnormalities in slow-wave sleep and the atypical response that sleepwalkers have to sleep deprivation.

A robust feature of the otherwise preserved sleep architecture in sleepwalkers relative to healthy controls is the absence of NREM sleep continuity, which is shown

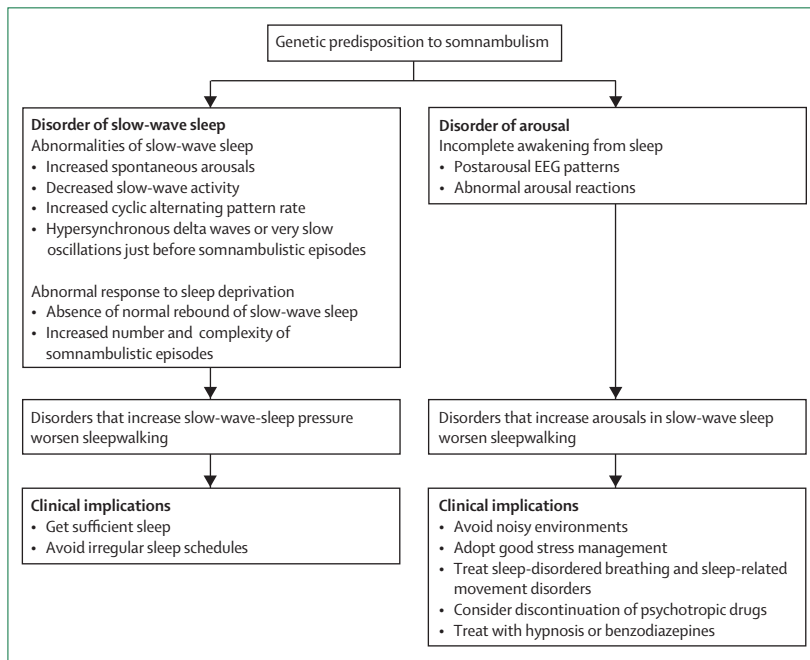


Figure 4: Somnambulism as a disorder of arousal or slow-wave sleep

by increased spontaneous awakenings and EEG recorded arousals outside slow-wave sleep, even on nights without episodes.^{53–55} This finding is especially noteworthy because the number of awakenings in the other sleep stages does not increase.⁵³

Sleepwalkers also have disturbances in sleep intensity as measured quantitatively by slow-wave activity (spectral power in the delta band frequency). Specifically, their sleep is characterised by an overall decrease in slow-wave activity during the first sleep cycles^{53,56} and a different timecourse of slow-wave activity decay throughout the night.⁵⁴ These results suggest that sleepwalkers' frequent awakenings from deep sleep interfere with the normal build-up of slow-wave activity, especially during the first two sleep cycles when most awakenings from deep sleep occur in sleepwalkers. Consistent with findings suggesting a disturbance in consolidation of slow-wave sleep, recurring electrocortical events marked by abrupt changes in EEG frequency or amplitude are noted in sleepwalkers during NREM sleep. These periodic sequences of transient EEG activity have been formally investigated as part of the cyclic alternating pattern rate,^{57–59} an endogenous rhythm thought to be a physiological marker of NREM sleep instability. An increased cyclic alternating pattern rate has been recorded both in adult and in child sleepwalkers^{58,59} even on nights without episodes. That this abnormal transient EEG activity might lead to recurrent fragmentation of slow-wave sleep and contribute to the occurrence of NREM sleep parasomnia has been suggested.^{57,60}

Hypersynchronous delta waves, which are usually defined as several continuous high-voltage ($\geq 150 \mu\text{V}$) delta waves occurring during deep sleep, were probably

the first EEG marker described in relation to somnambulism.⁶¹ Irrespective of behavioural episodes, sleepwalkers have significantly more hypersynchronous delta waves during NREM sleep than do controls.⁶² Episode onset, however, does not seem to be preceded by a gradual accumulation of hypersynchronous delta waves,⁶² but rather by an abrupt change in high-amplitude slow oscillations ($< 1 \text{ Hz}$) in the 20 s immediately preceding the episodes.⁶³ These processes may show cortical reactions to brain activation.

In healthy sleepers, sleep deprivation produces a rebound of slow-wave sleep and generates a consolidated (ie, fewer awakenings) NREM sleep as a result of heightened sleep homeostasis pressure (ie, a physiological need for sleep to restore the body's equilibrium between sleep and wakefulness).⁶⁴ This physiological response is not noted in sleepwalkers, and sleep deprivation actually results in more awakenings from slow-wave sleep during recovery sleep (ie, sleep occurring immediately after sleep deprivation) than are noted during baseline sleep (ie, normal nocturnal sleep without deprivation). This uncharacteristic response to sleep deprivation seems to be restricted to slow-wave sleep; awakenings from N2 and REM sleep are reduced.⁶⁵

More importantly, 25–38 h of sleep deprivation increases the number of somnambulistic events recorded in the laboratory by a factor of 2.5 to 5 compared with baseline.^{42,65,66} Sleepwalkers' responses to sleep deprivation differ so greatly from those of healthy sleepers that they are highly sensitive and specific for the diagnosis of adult somnambulism.^{42,65,66} That none of the healthy controls in these studies had nocturnal behavioural manifestations shows that sleep deprivation does not lead to sleepwalking, but rather that sleep deprivation increases the probability of somnambulistic behaviours in predisposed individuals.

Sleep deprivation also substantially increases the complexity of somnambulistic events recorded during recovery sleep.^{65,66} Somnambulistic episodes are not only more complex but also often more agitated, with forced arousals out of recovery slow-wave sleep.⁴² A possible explanation for these findings is that other subcortical regions might be recruited after sleep deprivation. Two functional MRI studies^{67,68} showed that sleep deprivation increased activation of the amygdala resulting from the presentation of negative visual stimuli and significantly strengthened amygdala connectivity with autonomic activating centres of the brainstem. This activation was accompanied by a decrease in connectivity with the prefrontal cortex—a top-down cognitive regulator of emotions.^{67,68}

Somnambulism as a disorder of arousal

Somnambulism was originally described as a disorder of arousal⁶⁹ because of the autonomic and motor arousals that propel patients towards incomplete wakefulness. Three postarousal EEG patterns have been described that

characterise most slow-wave-sleep arousals⁷⁰ and somnambulistic events⁷¹ in adults with somnambulism or sleep terrors. The same EEG patterns are present in somnambulistic events during N2 sleep.⁷¹ Delta activity (indicative of sleep-related processes) is recorded in almost half of all episodes during slow-wave sleep and about 20% of those during N2 sleep.⁷¹ Taken together, these findings suggest that sleepwalkers are caught between NREM sleep and full EEG arousal and are thus neither fully awake (as translated clinically by the seeming absence of conscious awareness or insight) nor fully asleep (as shown behaviourally by the capacity to interact with others and navigate the immediate environment) during episodes.

Other findings support the notion of somnambulism as a disorder of arousal. Arousals from slow-wave sleep, whether occurring spontaneously, triggered by external stimuli, or produced by other sleep disorders, can induce sleepwalking episodes in predisposed individuals. Several studies,⁷²⁻⁷⁴ including a population-based cohort study of preadolescent children,⁷³ have shown an association between somnambulism and both obstructive sleep apnoea and upper airway resistance syndrome. Treatment of sleep-disordered breathing might result in the disappearance of somnambulism through restoration of, or an increase in, sleep consolidation.

Experimentally triggered arousals by auditory stimulation during slow-wave sleep induce episodes in sleepwalkers during normal sleep and even more frequently during recovery sleep. In a study by Pilon and coworkers,⁴² the combined effects of sleep deprivation and auditory stimulation induced somnambulistic episodes in all ten sleepwalkers tested but in none of the control population. Furthermore, the mean intensity of stimuli that induced somnambulistic episodes during slow-wave sleep (roughly 50 dB) was similar to the mean intensity that produced full awakenings both in sleepwalkers and in controls. In another more extensive study,⁷⁵ auditory arousal thresholds in sleepwalkers did not differ significantly from those in controls for either slow-wave sleep or N2 sleep. However, the mean proportion of auditory stimulations that induced arousal responses during slow-wave sleep was significantly higher in sleepwalkers than in controls.

These findings show that sleepwalkers are neither more easy nor more difficult to awaken from deep sleep than are controls, but rather that sleepwalkers have abnormal arousal reactions. A study⁷¹ corroborated that 50% of postarousal EEG signals in sleepwalkers contained clear evidence of delta activity, which could explain the mental confusion after awakenings from slow-wave sleep and point to changes in cortical reactivity.

Somnambulism as a phenotypical expression of simultaneous states of sleep and wakefulness

Irrespective of the two notional frameworks discussed already, somnambulism needs to be considered in view of

new models and findings about the interplay between the states of wakefulness, REM sleep, and NREM sleep.^{76,77} Although human sleep has traditionally been thought of as a global process occurring uniformly in the whole brain, increasing evidence shows that sleep—or functional correlates of sleep—might be controlled by local events. Surface EEG studies^{78,79} showed that sleep depth does not occur simultaneously throughout the brain and that the frequency-specific topographical differences are distributed along the anteroposterior axis. Data obtained via intracerebral electrodes showed that EEG patterns related to sleep and wakefulness can coexist simultaneously in different brain regions. During an episode of sleepwalking in an epileptic patient, Terzaghi and colleagues⁸⁰ recorded an EEG pattern of wakefulness in the motor and central cingulate cortices and concomitant increased delta bursts (indicative of sleep) in the frontal and parietal dorsolateral associative cortices, suggesting that awakening of the motor and cingulate cortices is in apparent conflict with the simultaneously persistent sleep state of the associative cortical regions. The cingulate and motor cortices could cause the complex motor behaviours, and the degree of activation of frontoparietal associative cortices could explain the various degrees of awareness of the environment and mentation that accompany the awakening.

Nobili and colleagues⁸¹ used a similar depth EEG electrode strategy and noted frequent, short-lasting, local activations of the motor cortex, which were characterised by an abrupt interruption of the slow-wave pattern and a high-frequency EEG pattern, suggesting the coexistence of sleep and wakefulness. These activations in the motor cortex were paralleled by a concomitant increase in slow waves in the dorsolateral prefrontal cortex. Brain imaging by SPECT during a sleepwalking episode⁸² showed, on one hand, a deactivation of frontoparietal associative cortices (typical of sleep), and, on the other hand, an activation of the posterior cingulate and anterior cerebellum networks and no deactivation of the thalamus—characteristic of the emotionally driven behaviour of wakefulness.

The two sets of brain regions that are incongruent during somnambulism—ie, the motor and cingulate cortices, and the medial prefrontal and lateral parietal cortices—have been associated with the so-called task-positive (brain regions activated during cognitively demanding tasks) and default mode (cortical regions activated during the brain's resting state) networks,⁸³ respectively. A dysfunctional interplay between these two networks is implicated in other pathological disorders including schizophrenia,⁸⁴ Alzheimer's disease,⁸⁵ and depression.⁸⁶

Collectively, these findings support the idea that sleep and wakefulness are not mutually exclusive—an increasingly accepted view termed local sleep.⁸⁷ They also suggest that somnambulism and other parasomnias might result from an imbalance between the two

behavioural states.⁷⁷ Thus, disorder of arousal might be too notionally restrictive to fully account for the pathophysiology of somnambulism. A broad and unifying view might implicate the simultaneous activation of localised cortical and subcortical networks that have roles in sleep and wakefulness.⁸⁸

Future research

Three promising research directions could help to elucidate the pathophysiology of somnambulism. First, PET neuroimaging can detect subtle changes in cerebral blood flow and metabolism throughout the human sleep–wake cycle⁸⁹ and specific measures—eg, neural correlates of delta activity during NREM sleep.^{90,91} However, few neuroimaging studies have been done in sleep-disordered patients, and the only neuroimaging study of somnambulism is the single case report by Bassetti and colleagues.⁸² Variations in regional cerebral blood flow during NREM sleep in sleepwalkers are unexplored, but could further understanding of NREM sleep parasomnias.

Second, the general daytime functioning of sleepwalkers should be investigated to record the nature and extent of impairments. In addition to findings suggesting excessive daytime somnolence in some patients, two studies^{32,33} support the view that adult sleepwalkers evince functional abnormalities during wakefulness. A transcranial magnetic stimulation study⁷² of sleepwalkers showed hypoexcitability of some cortical GABAergic and cholinergic inhibitory circuits during wakefulness, and a high-resolution SPECT study³³ done during wakefulness in sleepwalkers showed hypoperfusions in the frontopolar cortex, superior and middle frontal gyri, superior and inferior temporal gyri, and angular gyrus, and additional hypoperfusions of limbic structures (hippocampus). Changes in limbic structures might be associated with disturbances in emotional regulation in sleepwalking patients when submitted to sleep deprivation.

Finally, despite several reports of familial aggregation, very few molecular studies to identify genes that predispose to somnambulism have been done. Licis and coworkers⁹² did a genome-wide study in a single family of 22 members. They assumed an autosomal dominant

model with reduced penetrance, and established significant linkage at chromosome 20q12–q13.12. The candidate interval included the adenosine deaminase gene, which has been associated with slow-wave sleep.⁹³ Unfortunately, sequencing revealed no coding mutation within the gene. Lecendreux and colleagues⁹⁴ described an association between familial sleepwalkers and the HLA DQB1*05 and DQB1*04 alleles. However, the functional importance of this finding is unclear. None of these findings has been replicated so far.

An alternative approach for the identification of genes that affect complex traits is association analysis of candidate genes.⁹⁵ Genes^{94,96,97} implicated in sleep homeostasis, sleep depth, or slow-wave generation could be attractive candidates. To that effect, a twin study⁹⁸ showed substantial genetic overlap between parasomnias and dyssomnias, supporting the notion of somnambulism as a disorder of regulation of slow-wave sleep and the association between sleepwalking and excessive sleepiness.

Conclusions

Although a comprehensive understanding of the clinical, neurobiological, and genetic factors associated with chronic somnambulism remains elusive, much progress has been made in clarification of the key relations between waking and sleep-related processes in this disorder. However, some misconceptions about somnambulism have hindered refinements in clinical assessment and definition. The validation and use of a polysomnographically based diagnosis for somnambulism, such as a sleep-deprivation protocol, would be useful in uncertain diagnoses. But in the context of medicolegal cases of sleep-related violence,^{5,6} whether even a polysomnographically diagnosed sleepwalker was having a somnambulistic episode during a remote forensic event cannot be ascertained. Similarly, because neurophysiological markers of sleepwalking can also be detected in controls, they cannot be used to provide direct evidence in the courtroom. Well designed clinical trials for the treatment of chronic somnambulism are virtually non-existent.⁴⁷ Greater efforts are needed to establish treatment efficacy for somnambulism, which should be thought of, at least in most adults, as a disorder with a high potential for serious injury and both night-time and daytime sequelae.

Contributors

All authors contributed equally to the search of medical literature, structuring and writing of the review, and critically reviewing all versions of the paper.

Conflicts of interest

JM has received grants from the Canadian Institute of Health Research and the Government of Canada Senior Research Chair in Sleep Medicine (PI), research grants or support, or both, from Merck and GlaxoSmithKline, and honoraria for speaking engagements from Valeant and Otsuka, and has served as an advisor for Sanofi-Aventis, Servier, Merck, Jazz, Valeant, and Impax. All other authors declare that they have no conflicts of interest.

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Search strategy and selection criteria

We searched PubMed with the search terms “sleepwalking” and “somnambulism” for papers published between Jan 1, 1979, and Sept 30, 2012. Additional references (abstracts) and book chapters that were cited in relevant reports were also used. We included some papers about topics other than somnambulism to strengthen hypotheses about underlying mechanisms. Most papers included were published in English, but non-English articles with English abstracts were included when relevant.

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