Chapter 6
Sleepwalking in Children and Adolescents

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Introduction

Sleepwalking or somnambulism is a non-rapid eye movement (NREM) parasomnia characterized by apparently purposive complex motor behavior during N3 sleep. It is of interest to clinicians as a differential diagnosis of partial-onset seizures and other sleep disorders and to neuroscientists as a model that may provide insight into the regulation of N3 sleep. It is also of importance to evolutionary biologists, as it is not observed in nonhuman primates and is regarded as an acquired behavioral response associated with humanoid differentiation [1].

Sleepwalking is reported to have a prevalence of 2–14 % in children [2–5] and 1.6–2.4 % in adults [6] in mixed clinic and community based samples. Some of these patients outgrow their affliction if the onset is before 10 years of age [7]; however, up to 25 % have been documented to continue sleepwalking in adulthood [8].

Etiological Associations

The exact etiology of sleepwalking is not delineated. However, there are several known associations that may partially contribute to causation.

Genetic Factors

Evidence for a genetic basis of sleepwalking comes from multiple different sources including epidemiologic and cytogenetic studies. A small study ($n = 37$) including

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children whose one or both parents had sleepwalked during childhood found that such children are more likely to manifest sleepwalking by 8 years of age as compared with controls [9]. A large population-based Finnish study ($n = 11,220$) with 1,045 monozygotic and 1,899 dizygotic twins found that for childhood-onset sleepwalking, the concordance rate for probands was 0.55 for monozygotic and 0.35 for dizygotic pairs [8]. For adult-onset sleepwalkers, the corresponding values were 0.32 and 0.06, respectively. The authors calculated that the proportion of total phenotypic variance attributable to genetic influence was 66 % in males and 57 % in females in childhood-onset sleepwalking and 80 % in males and 36 % in females in adult-onset sleepwalking [8]. In the human leukocyte antigen (HLA) studies, 35 % of sleepwalkers were found to be positive for HLA-DQB1-050 as compared with 13 % controls (odds ratio 3.5, 95 % confidence interval 1.4–8.7) in a study including 60 sleepwalkers with matched controls [10]. Excess transmission was noted for DQB1-05 and DQB1-04 alleles in familial cases [10]. Recently, autosomal dominant inheritance with reduced penetrance was reported in family members with sleepwalking for a genetic locus at chromosome 20q12-q13.12 [11]. Hence, it is hypothesized that although genetic factors may not be sufficient for expression of sleepwalking phenotype by themselves, they likely have a predisposing influence.

Acquired Factors

Most of the acquired factors are believed to cause partial arousals or poor quality sleep, which precipitate sleepwalking episodes.

Other Sleep Disorders Sleep-disordered breathing has been observed in up to 58 % of children with sleepwalking, with about half of them also having a family history of respiratory problems. Successful treatment of disordered breathing was further noted to alleviate sleepwalking in all patients in this series [12]. In another study, comorbid obstructive sleep apnea (OSA) or upper airway resistance syndrome (UARS) was noted in all sleepwalkers [13]. On the other end, 54 % of children with OSA reported sleepwalking on a questionnaire [14]. It has been proposed that abnormal respiratory events trigger frequent arousals contributing to sleepwalking behavior [15]. Restless legs syndrome (RLS) and periodic limb movement syndrome (PLMS) has also been reported to precipitate parasomnia [12].

Sleep Deprivation Sleep deprivation has been tried as an activation procedure to produce sleepwalking in controlled circumstances, possibly because it is known to increase slow-wave sleep [16]. In a study ($n = 10$), 38 h of sleep deprivation was noted to increase frequency and complexity of sleepwalking episodes and the number of arousals from slow-wave sleep [17]. However, another study ($n = 10$) found no increase in sleepwalking with sleep deprivation, although complex stereotypical sleep behaviors were noted to increase and were considered by authors as preliminary stage of sleepwalking [16]. Forced arousal produced by auditory stimulation in slow-wave sleep during first and second NREM–rapid eye movement (REM) cycles, during

a recovery following 25 h of sleep deprivation, was shown to precipitate greater somnambulistic episodes as compared with normal sleep [18].

Drugs Several different drugs have been observed to produce new-onset sleepwalking behavior as well as reactivation of dormant sleepwalking in patients with past or family history of this disorder. It has been proposed that the possible pathogenesis may be due to increased slow-wave sleep [19]. Most of the evidence for drug-induced sleepwalking exists for lithium and zolpidem. In one of the earliest reports on sleepwalking, 9 % of psychiatric patients on lithium and neuroleptic comedication were found to have sleepwalking behavior [20]. Since then, the association between lithium therapy and sleepwalking has been reported often, and also in patients on other psychotropic medications [21, 22]. Reactivation of childhood sleepwalking in up to 27 % of patients with lithium-induced parasomnia has also been reported [22, 23]. Similarly, there have been isolated reports of both new-onset and reactivation sleepwalking with the use of zolpidem [24–26]. At least in some of these patients, there were other possible factors that could have contributed to precipitation of sleepwalking, e.g., previous brain injury [26] and concomitant use of valproate [27]. However, in one patient, sleepwalking disappeared after discontinuing zolpidem [24]. Additionally, there has been a report of a patient developing sleepwalking after initiation of bupropion that is a centrally acting dopaminergic stimulant [28].

Hyperthyroidism A case series with eight patients of new-onset sleepwalking coinciding with onset of thyrotoxicosis has been reported [29]. In this report, sleepwalking disappeared in all patients as they attained a euthyroid state and relapsed in two whose hyperthyroidism became poorly controlled because of lack of compliance with treatment. Interestingly, the authors hypothesized that the fatigue associated with thyrotoxicosis may contribute to onset of sleepwalking [29].

Other Associations Several other factors have been associated with onset or relapse of sleepwalking in isolated reports. Other reported possible risk factors include herpes simplex encephalitis, alcohol abuse, and even a trial of continuous positive airway pressure, where two episodes of sleepwalking were seen during a period of delta rebound [30–32].

Clinical Features, Polysomnography (PSG), and Video-Electroencephalogram (EEG)

Sleepwalking represents episodes of partial arousal occurring out of slow-wave sleep. These are characterized by ambulatory behaviors during altered state of consciousness and impaired judgment. Episodes may begin with sitting up in bed with eyes open. Complex behavior of change in body position, turning, playing with sheets, and eventually getting out of the bed may occur. Behaviors such as walking around the house, going to kitchen or bathroom, and going downstairs are associated. Complex behaviors such as cooking, cleaning, eating, and even driving have been reported.

These semipurposeful movements are performed with some dexterity. Typically, the child is calm, but if awakened, agitation and confusion can occur along with prolongation of the episode. Child often returns to bed and has no recollection of the event in the morning. Due to altered awareness and ambulation, there is risk of injury from falling on stairs, jumping out of windows, going out of house, and running into closed doors. Inappropriate behaviors such as urinating in unusual places and violence are also seen especially in men [33–36]. In an adult, atypical presentation with multiple episodes in a night is reported [37].

Some of the clinical features of sleepwalking have been best observed during PSG. Sleepwalking episodes occur most frequently during first 3 h of sleep, when N3 sleep is most abundant. The episodes can last for 1–30 min [38]. PSG findings consistently reported in sleepwalkers include reduced sleep efficiency and decreased N2 sleep, but increased N3 sleep [39]. Although duration of N3 sleep is increased, it has an abnormal architecture and is characterized by increased fragmentation and frequent arousals [39, 40].

During the few minutes preceding sleepwalking-related arousal, increased slow-wave activity has been reported as compared with baseline [39, 41]. Specifically, a study found greater slow-wave activity 2 min before a sleepwalking episode as compared with other samples obtained 10 min before the same episode and at the actual arousal [39]. This finding has been confirmed with analysis of power spectrum, with increased delta power being noted to be temporally related to arousal resulting in sleepwalking. At the same time, there was decreased physiologic slow-wave activity or delta power during other periods of N2–N3 sleep [41]. These suggest the inability to sustain slow-wave sleep. These features were more prominent during first or initial few sleep cycles [40]. During the actual sleepwalking episode, diffuse alpha frequency activity has been reported to be superimposed on delta rhythm, thought to be reflecting incomplete arousal [42, 43]. Diffuse rhythmic delta activity and, less frequently, alpha and beta activity are also reported as postarousal EEG activities [43]. For some time, the hypersynchronous delta activity was considered as a hallmark of sleepwalking, but it is disputed as a non-specific marker probably reflecting arousal activity [44].

Cyclic alternating patterns (CAP) are phenomena characterized by sequences of transient EEG events (phase A) with relatively stable background activity (phase B), with a periodicity of less than 1 min. Phase A is a marker of cortical activity and arousal, whereas phase B represents rebound deactivation [45]. A higher CAP rate has been documented in sleepwalkers [13, 46]. In one study, increase in CAP rates was similar to that in patients with UARS [13]. Hence, there is increased instability in NREM sleep in sleep walkers. Along with PSG and video-EEG, assessment of daytime sleepiness, sleep time, sleep quality, OSA, RLS, and PLMS, should also be performed.

Coexistent Conditions

Psychological Conditions

Children with sleepwalking were noted to have higher separation anxiety and high hyperactivity–inattention score [47]. Family or personal history of panic disorder has been reported in sleepwalkers [48]. In a study, 85 % of the teenagers with sleepwalking or sleep terrors had a psychiatric comorbidity. These included panic disorder, overanxious disorder, alcohol or cigarette use, suicidal ideation, and simple phobia. Increased fatigue, dysmenorrhea, and low mood in patients were also observed [49]. A study based on Rorschach tests noted inhibited aggression in sleepwalkers, which was hypothesized by the authors as a mature defense mechanism against anxiety [2]. In another study, experience of a recent stressful event along with higher prevalence of mood and anxiety disorders were noted in adult sleepwalkers [6]. In adults, aggression, anxiety, hysteria, panic disorder and phobias, and profiles similar to post-traumatic stress disorder have been reported with sleepwalking [19, 50, 51]. Patients with later age of onset and persistent sleepwalking in adulthood display active, outward behavioral patterns, which suggest difficulties in handling aggression [7]. However, in another study in adults, prevalence of Axis I psychiatric disorders was not increased in sleepwalkers [52]. It is unclear whether sleepwalking and coexistent psychopathology stem from common neurochemical disturbance or whether at least some of the psychomorbidity is just an epiphenomenon partially due to social stigmatization.

Other Sleep Disorders

Sleepwalking is known to be associated with other parasomnias. In a study exploring hereditary aspects of sleepwalking, it was observed that 80 % of sleepwalking pedigrees and 96 % of night terror pedigrees included other individuals, besides proband, who were affected by sleepwalking, night terrors, or both [53]. It was proposed that sleepwalking and night terrors probably share a common genetic predisposition [19, 53]. Among persistent sleepwalkers at 6 years of age, 92 % had somniloquy and 41 % had night terrors [47].

Excessive daytime sleepiness has been reported both subjectively and on multiple sleep latency test (MSLT). Increased automobile accidents are also reported that may be due to excessive daytime sleepiness [54]. Children with parasomnia also have bedtime resistance, sleep onset delay, inappropriate sleep duration, and increased night waking [55].

Neurological Diseases

Two unrelated entities, migraine and Tourette syndrome, have been reported in childhood-onset sleepwalking. In a study looking at this association, 19 % of children with Tourette syndrome had sleepwalking [56]. The authors proposed that probably both the conditions share a common neurochemical basis and result from disturbed brainstem serotonin metabolism [56].

There are several reports of association between migraine headaches and sleepwalking. In one study, up to 30 % of children with migraine had sleepwalking and 67 % of childhood-onset sleepwalkers suffered from migraine [57]. Another subsequent study also found high frequency of sleepwalking in migraine patients, although not to this degree. In patients with childhood-onset sleepwalking, migraine episodes usually had prominent visual symptoms at onset and occipital localization of headache [58].

Differential Diagnosis

Partial-Onset Seizures

The major consideration in any paroxysmal disorder is to rule out an epileptic basis for the episodes. Both hypermotor behaviors observed in autosomal dominant nocturnal frontal lobe epilepsy and complex automatisms of some temporal lobe seizures can mimic sleepwalking episodes [59]. However, there are certain clinical and EEG features that can help in differentiating sleepwalking from seizures. Usually, sleepwalking episodes occur during first few cycles of N3 sleep as compared with seizures, which often occur during drowsiness of N2 sleep anytime during the night. Vocalizations including screaming and distal upper extremity automatisms are also more indicative of a seizure episode. By definition, sleepwalking is an NREM parasomnia and does not result in clinical arousal, whereas awakening and postictal confusion are fairly common in seizures. Frontal lobe seizures are brief, occur multiple times in night, occur mostly out of N2, and have highly stereotypic semiology [60–62]. Frontal Lobe Epilepsy and Parasomnias (FLEP) scale was validated in studies for differential diagnoses [63, 64].

An overnight video-EEG is often helpful to exclude seizures and is indicated for this purpose in a patient being thought to have sleepwalking. Previously, there have been reports of abnormal interictal epileptiform activity in the EEG records of sleepwalkers [65, 66]. However, it probably represents a chance association, as up to 8 % of apparently normal subjects can have such activity [67]. Even in patients thought of as having sleepwalking, observation of evolving ictal patterns in EEG has led to the correct diagnosis [68]. In this context, “episodic nocturnal wandering” is a poorly characterized phenomenon, which probably has an epileptic basis. At least in one patient, the episode of nocturnal wandering was associated with ictal activity

originating from right temporal lobe with spread to cingulate gyrus, on prolonged video-EEG monitoring with intracranial electrodes [68, 69].

REM Behavior Disorder (RBD)

This disorder is sometimes confused with sleepwalking. However, it usually involves more violent motor activity in older men, said to represent acting out of their dreams, as compared with sleepwalking, which is more commonly seen in children and adolescents and rarely has a hypermotor character. However, RBD in association with narcolepsy may be seen in younger patients. Other differentiating feature is little autonomic activation seen in RBD as compared with marked activation in sleepwalkers. Also, memory of vivid dream leading to the motor behavior is present in RBD. There is typically a complete arousal following the event [34]. On PSG, intermittent decrease/absence in REM atonia or increase in phasic movements is seen. These features help distinguish the conditions. Parasomnia overlap disorder should also be considered if sleepwalking and RBD occur in the same individual [33].

Management

Nonpharmacologic Measures

Hypnosis

Since as late as 1990's, there has been much enthusiasm for hypnosis as a treatment for sleepwalking. In an earlier series, 4/6 patients reported complete remission with the use of hypnosis [70]. In another report by the same group, sustained improvement up to 1 year was observed in patients with sleepwalking but otherwise free of concurrent psychiatric disorders, with 6 brief sessions of hypnosis [71]. In a later adult study ($n = 27$), 74 % reported improvement with self-hypnosis practiced at home [72].

Anticipatory Awakening There are a few reports of successful treatment of sleepwalking with anticipatory awakening. An 8-year-old boy was woken up just before the anticipated time of sleepwalking episode for five consecutive nights. This was reported to later eliminate the sleepwalking behavior altogether [73]. In another family with 3 sleepwalking siblings, anticipatory awakening was reported to be curative [74].

Safety Measures

Adaptations and modifications of sleep environment are usually recommended to ensure safety of the patient. These may include removal of potentially dangerous

objects from the bedroom, clearing the floor obstructions, keeping windows and sometimes even doors closed, and allowing the patient to sleep on the ground floor, on a mattress placed directly on floor or in a sleeping bag [38]. Maintaining good sleep hygiene with regular schedule, avoidance of alcohol, stress management, and discontinuing or changing medications with adverse influence on N3 sleep architecture may sometimes be sufficient to relieve sleepwalking [38, 75]. Sleepwalkers should not be awakened but guided back to bed and allowed to fall back to sleep. Social stigmatization should be avoided at all costs and individual and family counseling may be helpful in this regard.

Drug Therapy

Occasional episodes of sleepwalking are usually managed conservatively. However, recurrent episodes with a risk of injury to self or others are usually an indication for drug treatment. There are no evidence-based recommendations for choice of drug (s), dose, or duration of treatment.

Benzodiazepines

Although there is a lack of well-designed controlled studies for any pharmacological therapy in sleepwalking, most of the anecdotal evidence supports benzodiazepines. A small double-blind, crossover, placebo-controlled study found daily bedtime 10 mg diazepam to alleviate sleepwalking in adult patients [76]. Four out of five patients reported satisfactory benefit and continued diazepam for 9 months with no serious adverse events or development of tolerance [76]. This report was consistent with uncontrolled case reports of efficacy of diazepam in adults with sleepwalking [77]. In children, efficacy of diazepam has been documented in isolated cases, with some concern for relapse on discontinuation. However, prompt recovery on resumption of treatment and lack of recurrence with 6 weeks therapy and slow taper over 4 weeks has also been documented [78].

Several case reports have documented beneficial effect of clonazepam in both children and adults with primary and neuroleptic-associated sleepwalking [79–82]. In a study extending over 12 years, 170 adults with disruptive sleep disorders, including 69 with injurious sleepwalking or sleep terrors, were treated for more than 6 months with daily benzodiazepines, including 136 patients who received clonazepam [83]. Overall, 146 patients (86 %) achieved complete or substantial control of their sleep disorders [83]. Clonazepam has longer duration of action and relatively better adverse effect profile as compared with diazepam [84]. Hence, it is the most commonly prescribed medication for sleepwalking in both children and adults [75].

Recently, isolated reports of benefit with other benzodiazepines in sleepwalking have also appeared, including triazolam and flurazepam [79, 85].

As discussed earlier, sleepwalking is associated with increased N3 sleep, and traditionally, it has been believed that benzodiazepines have their beneficial effect in

arousal parasomnias by decreasing N3 sleep. However, clonazepam has been shown to increase N3 sleep [86]. Hence, the efficacy of benzodiazepines may be related to better sleep consolidation by reducing fragmentation of N3 sleep and decreasing reactivity to triggering arousals [75].

Tricyclic Antidepressants

Imipramine has been documented to achieve effective control of sleepwalking in children receiving 10–50 mg at bedtime for 8 weeks [87]. Adults with coexisting night terrors also experienced remission with 50–300 mg given for 8 months to 1 year [48, 88]. Although there was recurrence on discontinuation of imipramine in adults, efficacy was restored on restarting the medication [88]. Two patients unresponsive to diazepam have also been documented to have remission with imipramine [48, 88]. The mechanism of action of imipramine in sleepwalking is not known but is believed to be related to increased norepinephrine and 5-hydroxytryptamine levels [89].

Selective serotonin reuptake inhibitors (SSRI) Paroxetine 20–40 mg once in the morning was used in a patient whose sleepwalking was already in remission with years of clonazepam use. After starting paroxetine, clonazepam could be successfully tapered and stopped with continued remission [90]. It is arguable if the disorder was itself active at that time or not. Although the exact mechanism of action of SSRI in sleepwalking is not known, they are known to have some efficacy in sleep-disordered breathing. This effect is mediated by mildly increased tongue muscle contractile tone due to noradrenergic influence [75]. Decreased sleep fragmentation resulting as a secondary gain contributes to the efficacy in sleepwalking.

Treatment of Associated Disorders

As discussed earlier, sleepwalking is known to exist as an epiphenomenon in several other disorders, including OSA, UARS, RLS, and PLMD. Treatment of these disorders is crucial and usually leads to resolution of sleepwalking [75]. Psychological evaluation and treatment should be considered in cases with suspicion of comorbid psychiatric conditions. Anxiety should also be addressed.

Legal Considerations

Actual or Attempted Homicide

There have been several reports where somnambulism was cited as a defense in cases of homicide or violent assault [91–94]. It has also been pointed out that sleepwalking

is usually accepted as a successful defense in homicidal cases [95]. Most of the law courts in different countries require the diagnosis of sleepwalking to have been established before the criminal event, in addition to other supporting evidence in the form of personal or family history of psychopathology [19, 94, 95]. From a clinical perspective, it is almost impossible to unequivocally say whether the episode in question was a sleepwalking phenomenon even in a known case of somnambulism.

Sexual Crimes

Several different types of sexual behaviors have been reported in sleepwalkers, often with legal implications. These behaviors have included indecent exposure, sleep sex with a lawful partner, sexual misconduct, and alleged rape [80, 96–101]. Usually, these behaviors have been reported to coexist with violent motor activity, nonsense vocalizations, and amnesia for the event [101, 102]. In this context, after a PSG confirmed case, Schenck and Mahowald coined a legal term, parasomnia with continuing danger as a noninsane automatism [98]. At least in one report, sleepwalking was used as a successful defense against three counts of rape [101].

Practical Points

- Sleepwalking is common in children and adolescents.
- The events occur out of slow-wave sleep and are associated with ambulatory behaviors.
- Genetic factors play an important role in predisposing individuals.
- OSA, RLS, and PLMS have been reported as precipitating factors.
- Separation anxiety is associated with sleepwalking in children.
- Differentiation from REM behavior disorder and nocturnal seizures is important, which can be achieved by PSG and video EEG.
- Low-dose clonazepam and safety are important measures in management.

Case Example

A 9-year-old boy was referred to the sleep clinic for evaluation of nighttime events, which started at the age of 5–6 years. He was found walking, sometimes going to the kitchen, and eating by his grandmother. He had also urinated on the kitchen floor or sink on several occasions. He would sometimes grab food and go to bed, and was found with a chicken-bone in mouth once. He had no recollection of the events the next day. He also had trouble falling asleep and staying asleep.

He usually went to bed at 8.30–9.00 pm on weeknights and weekends. He took clonidine 0.2 mg at 8.00 pm, which helped him fall asleep within next 20–30 min. He

usually woke up at 6.00 am on weekdays and 5.00 am on weekends, usually without difficulty and with the help of other family members. He was waking up four times a night for about 15–60 min, and could not return to sleep without help.

He was born at 37 weeks' gestation by spontaneous vaginal delivery. His mother was a smoker and had gestational diabetes and placental abruption. He was born healthy and was discharged after regular stay at the newborn nursery. History of nocturnal events was not present in the family; however, extended history of father's family was not available.

He had a history of bipolar disorder, oppositional defiant disorder, depression, and attention deficit hyperactivity disorder (ADHD). He was on aripiprazole, imipramine, sertraline, trazodone, and methylphenidate for these disorders. His physical examination was normal.

He was sent for a PSG study with extended EEG along with arm and leg electromyography (EMG) monitoring. Typical events were captured on the study. During two events, he screamed loudly. During the third event, he got out of the bed, walked around the room, and went back to sleep. Figure 6.1 shows EEG and EMG patterns during the third event. The PSG study did not show OSA or PLMD.

The patient was diagnosed with sleepwalking and sleep-related eating disorder. He was started on clonazepam 0.5 mg and was motivated to maintain strict sleep hygiene. Also, trazodone was stopped. On last follow-up, his events had reduced in frequency; however, they continue to occur.

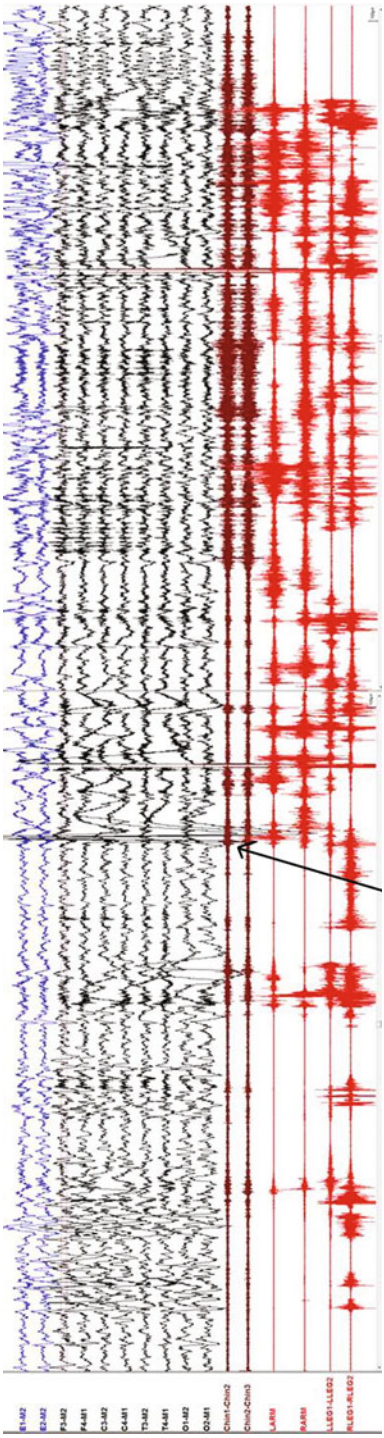

Fig. 6.1 EEG showing the sleepwalking event in the patient (the event onset is shown by the *arrow*)

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