Behavioral Sleep Medicine, 13:349–358, 2015

ISSN: 1540-2002 print/1540-2010 online
DOI: 10.1080/15402002.2014.898304

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A Case of Successful Use of Hypnosis in the Treatment of Parasomnia Overlap Disorder

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A young male patient was successfully treated for parasomnia overlap disorder (POD) using hypnosis. In 2006, this 16-year-old patient underwent a clinical evaluation for episodes of sleep talking, sleepwalking, and dream enactment. This initial assessment was followed by polysomnographic evaluation, a brain MRI, and three sessions of treatment using hypnosis. From the beginning, until the last contact in December 2011, benefits from the hypnotic suggestions were noted and documented.

Parasomnia overlap disorder (POD) is a sleep disorder characterized by the association of rapid eye movement (REM) sleep parasomnia with a non-REM (NREM) sleep parasomnia in the same patient. A search of the medical literature revealed very few cases of POD and only one that was treated with hypnosis.

We now report a successful treatment of POD during a period of 5.5 years (May 2006 to December 2011) using a combination of hypnotic induction, home practice with an audiotape, and temporary low-dose clonazepam. Clonazepam and hypnotherapy were used in combination for approximately 2 years. The patient underwent three hypnotherapy sessions. He then regularly practiced hypnotherapy on his own with a taped hypnotherapy session throughout most of that period. The benefits of hypnotherapy were noted initially and benefits continued to increase throughout the treatment period.

CASE PRESENTATION

The patient was a right-handed male, 16.75 years of age. He was seen at the Sleep Center for evaluation of "sleep talking, and sleep running with screaming." The patient first developed

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sleepwalking with dream enactment in August of 2005, near his 16th birthday. He dreamt a lizard was in his bed, and he got out of bed and ran. On another night in August, he thought that someone was chasing him, and he got out of bed and ran. In January of 2006, he dreamt that he was late for school, got out of bed, and went to take a shower. On April 2, 2006, he dreamt that thieves took over the school and he had to stop them; they drew a gun on him and began fighting, so he ran from them. His mother tried to stop him and he began fighting with her. He fell over some exercise equipment, which caused minor musculoskeletal trauma. He had at least seven more episodes of dream enactment prior to his initial evaluation on May 22, 2006. The patient had a history of sleep talking approximately three nights a week, usually occurring approximately two to three hours after going to sleep. There were no other reports of childhood parasomnia.

The patient typically went to bed between 10:00 p.m. and 11:00 p.m. and fell asleep quickly. He slept throughout the night except for the episodes mentioned above. He would get out of bed at 6:00 a.m., feeling tired. He did not nap during the day. He denied excessive daytime somnolence; he scored 2 on the Epworth Sleepiness Scale. The patient slept in a queen-sized bed in the supine position in his own room.

His mother related that when he had the episodes he "barreled out of the room" and presented a danger to himself and to her. There was no history of snoring. He would occasionally awaken in the morning with a headache. He denied awakening with a dry mouth. There was no restless leg symptomatology, nor were there reports of any repetitive limb movements. There was no history of nocturnal enuresis, gastroesophageal reflux disease, or deterioration of memory. There had been no cataplexy, sleep paralysis, or hypnagogic hallucinations. He had a history of occasional night sweats. The patient had a possible allergy to Ceclor, which caused hives. He was taking no medication on a regular basis.

Past medical history revealed no history of severe infections. He had an adenoidectomy in 2003 and wisdom teeth removed in 2005. At age 10 he was hit in the right frontal area with a metal bat, without loss of consciousness. There was no history of nasal fracture. He denied use of cigarettes, alcohol, or illicit drugs. He drank a minimal amount of caffeine.

Review of systems was negative for anemia, diabetes mellitus type II, thyroid dysfunction, and cardiac, pulmonary, or renal dysfunction. There had been no syncope or seizure-like activity. There was no history of hypertension. He denied headaches and depression, and he scored a zero on the Beck Depression Inventory.

The patient was born full term at 8 pounds 13 ounces. The pregnancy, labor, and delivery were uncomplicated. Developmental milestones were normal. At the time of initial evaluation he was 16 and getting A's in school.

The family history at initial evaluation revealed the patient's mother was 49, with a history of insomnia. The patient's father was 53. He had two half-brothers; one on the maternal side had several minor episodes of sleepwalking. The patient's paternal grandfather had a history of sleepwalking. There was no family history of brain tumor, cerebral aneurysm, neurodegenerative disease, neuromuscular disease, or convulsive disorder. Social history revealed his parents were divorced and he lived with his mother.

Based on his history, he had spells with characteristics of presumed REM Sleep Behavior Disorder (RBD). He also had evidence of sleepwalking, an NREM parasomnia, with no memory for the episodes. A polysomnogram (PSG) was indicated to confirm lack of REM atonia. The PSG performed on June 27, 2006, in an American Academy of Sleep Medicine (AASM)

accredited sleep center following AASM guidelines, showed a sleep latency of 25 minutes, REM latency of 73.5 minutes, and sleep efficiency of 87%. Sleep architecture revealed that he spent 18% in slow wave sleep (SWS) and 28% in stage REM. The RDI was 4.02 and the sleep disturbance index was 19.3. The patient had no episodes of snoring. Oxyhemoglobin saturation nadir was 91%. There were no periodic limb movements (PLMs). He had notable intermittent loss of REM atonia on chin EMG across all four REM sleep episodes, but had no vocalization or abnormal movement. Based on PSG and patient history, he had evidence of both REM behavior disorder (RBD) and somnambulism, an NREM parasomnia.

He was seen in follow-up on July 10, 2006, and reported no intervening dream enactment. He did have several episodes of sleepwalking occurring one to three hours after falling asleep when he got out of bed. His mother saw him and asked him what he was doing and he answered with bizarre speech and returned to bed. There was no memory for the episodes, in contrast to episodes of dream enactment. The results of the PSG were discussed. His neurological examination and neurological history were unremarkable. Potential etiologic factors for RBD were discussed. It was recommended that a brain MRI scan be obtained. During the visit the patient underwent a hypnosis session with visual imagery induction. (In visual imagery induction the patient is assisted in visualizing a scene of his choice prior to induction. The hypnotherapist uses this image in inducing the trance). Posthypnotic suggestions were given for improved sleep and for awakening whenever his feet would touch the floor. The session was held with the patient's feet on a stool. An audiotape of the hypnosis session was made and he was asked to practice with it once or twice a day.

He returned for follow-up on July 17, 2006. He had not been able to practice hypnotherapy since the tape had malfunctioned. He reported two episodes of dream enactment. On one occasion when he and his mother were out of town in a motel, he dreamt there was something on his pillow. He "hopped" out of bed and "hopped" onto his mother's bed. On another occasion he dreamt there was something in his bed and hopped out of bed. There was no evidence of definite additional sleepwalking. Another hypnotherapy session occurred utilizing visual imagery induction with posthypnotic suggestions for improved sleep, and the suggestion that whenever his feet would touch the floor he would immediately awaken and would go back to sleep as soon as he wanted to. There were also suggestions for ego strengthening such as: "You have great inner strength to accomplish whatever you wish to." The session was again carried out with the patient's feet on a stool. An audiotape was made for him to use to practice once or twice a day.

On July 20, 2006, the patient's mother was called to discuss the results of the MRI scan. The MRI showed a possible old lacunar infarct in the left basal ganglia. Further evaluation by a neurologist was recommended. The patient reported having an episode where he hopped out of bed and awoke as soon as he stood up. He was utilizing his hypnotherapy tape on a regular basis.

On October 12, 2006, his mother called, reporting that he had made significant improvement but had recent episodes of hopping out of bed and running. It was recommended that we proceed with an additional hypnotherapy tape with additional posthypnotic suggestions. She did not wish to start him on medication at that point. They did not follow through with an additional session.

On December 7, 2006, his mother called relating that he was to go abroad the following summer and she wanted the episodes completely controlled. Approximately once a week he

was getting out of bed, but did not run and would talk with no awareness for the event. Several times per week he would stay in bed and have episodes of sleep talking without recall. He was placed on a trial of clonazepam 0.5 mg at bedtime.

On January 11, 2007, his mother called, relating that he had been practicing his hypnotherapy tape and using 0.5 mg clonazepam at bedtime. Rare episodes were reported: on one occasion he stood by the bedside but did not walk, and on another occasion he urinated on the wall. He had no memory of either episode. His clonazepam was increased to 0.75 mg.

He was seen at the Sleep Center on December 17, 2007. There had been no episodes of sleepwalking or dream enactment over the past semester while at college. He typically was going to bed between 12:30 a.m. and 1:00 a.m., falling asleep relatively quickly. He was getting up at 10:00 a.m., feeling refreshed. His clonazepam was continued and the dose was increased to 1 mg at bedtime.

The next contact was on August 3, 2009, when he was reevaluated at the Sleep Center. He had been having episodes approximately once per month where he would get out of bed and awaken as soon as his feet touched the floor. Sleep talking had been present. There was no evidence of dream enactment. He had discontinued clonazepam because of excessive daytime somnolence. He was going to bed around 10:30 p.m. Over the past six months he developed increasingly worse sleep initiation insomnia, taking several hours to fall asleep, and started taking zolpidem 5 mg at bedtime. He was getting up at 8:00 a.m., feeling relatively refreshed. A hypnotherapy session was held with his feet on a stool with visual imagery induction and posthypnotic suggestions for improved sleep, ego strengthening, and a reminder that whenever his feet touched the floor he would immediately awaken. Behavioral techniques for improved sleep were discussed. He was to continue zolpidem 5 mg at bedtime as needed.

On January 4, 2010, he returned to the Sleep Center and reported that approximately twice a month he would dream there was something in his bed that caused him to get out of bed. However, as soon as his feet touched the floor he would be immediately awake and then would go back to sleep. He continued to experience both sleep initiation and sleep maintenance insomnia. Zolpidem 5 mg was beneficial to some degree. Behavioral techniques for improving sleep were again reviewed. He was placed on zolpidem ER 12.5 mg at bedtime. Zolpidem does not have any reported efficacy in improving parasomnias. Its effect on parasomnias is quite the opposite. There are numerous reports in the literature of zolpidem causing sleepwalking. His episodes continue to be controlled in spite of his use of zolipidem.

The hypnosis modified his behavioral response to the arousal, that is, the control of the walking, but did not control all of the arousals.

A phone conference was held in December 2011. Episodes were occurring every 4 to 6 weeks when he would get out of bed, but he would awaken as soon as his feet touched the floor. He was subsequently lost to follow-up.

DISCUSSION AND REVIEW OF LITERATURE

Hypnosis is a dynamic process of focused attention with specific neural changes occurring throughout induction and posthypnotic suggestions. Individual hypnotizability varies widely and has a probable genetic component. Hypnotic induction and deepening techniques vary.

At the beginning of the hypnotherapy session, the therapist (WCK) described the hypnotic process to the patient and together they chose some effective visual images for use during the session. In a quiet room, in this case with the subject's feet on a stool, the therapist directed the subject to fix his eyes on an object over his shoulder and to breathe deeply; the therapist then began the chosen visual imagery. In some cases, deepening techniques such as walking down a staircase or other visual images may be employed. Posthypnotic suggestions in this case included ego strengthening, suggestions for improved sleep, and the suggestion that whenever his feet touch the floor he would immediately awaken and then go back to sleep whenever he wanted to. The session was reviewed with the subject to determine any changes he would like to make. A second session was taped so the patient could listen to it once or twice a day.

NREM parasomnias, including sleepwalking, typically occur during stages of NREM sleep, most frequently stage N3, whereas RBD occurs in REM sleep with loss of inhibition of alpha motoneurons. The detailed underlying pathology is unclear. There appears to be a genetic basis for both but no definite gene loci have been identified. Various Central Nervous System (CNS) pathologies as well as medication have been associated with RBD (Schenck & Howell, 2013).

A review of the medical literature revealed very few cases of POD. Prior to 1997, only seven cases of potential POD were reported (Blanco & Garay, 1995; Bokey, 1993; Kushida, Clerk, Kirsch, Hotson, & Guilleminault, 1995). Recently Schenck and Howell (2013) reclassified POD with the number of confirmed cases at 139 to that point. Hypnosis has been previously reported to successfully treat sleepwalking (Hauri, Silber, & Boeve, 2007; Hurwitz, Mahowald, Schenck, Schluter, & Bundlie, 1991; Kohler, 2011; Reid, Ahmed, & Lavie, 1981). However, there are no detailed reports on the use of hypnosis to treat RBD.

Howell, Arneson, and Schenck (2011) reported the successful use of a bed alarm with a calming message in 4 RBD patients with sleep-related injury (SRI) who were refractory to medication. They felt that the low arousal threshold of REM sleep, its rapid transition to wakefulness, and the capacity for complex auditory processing would make this intervention plausible. Conversely, during NREM sleep there is diminished response to eternal stimuli. It is conceivable that hypnosis through internal stimulation would be able to facilitate arousal in both the REM and NREM parasomnia.

The patient in our case exhibited classical findings of RBD with dream enactment along with lack of REM atonia on the PSG as well as episodes of sleepwalking, a non-REM parasomnia for which he had no memory. Hypnosis as the initial treatment improved his symptoms, which continued to improve after starting clonazepam. After clonazepam was discontinued, his symptoms continued to improve and respond to the posthypnotic suggestion, "Whenever your feet touch the floor you'll be immediately awake" (see Figure 1 for the patient's time line).

Schenck, Boyd, and Mahowald (1997) reported 33 PSG-confirmed cases of parasomnia overlap disorder (POD) involving sleepwalking (SW), sleep terrors (ST), and RBD. The report was based on a review and evaluation of cases diagnosed at a clinical center during an 8-year period (1988–1996); these cases comprised approximately 21% of all RBD cases and 28% of all SW/ST cases seen at the center during that time period. Patients underwent clinical and PSG evaluations. The clinical evaluations included patient interviews and the completion of a structured questionnaire. Mean age of these 33 cases was 34 ± 14 years; mean age of their parasomnia onset was 15 ± 16 years (range 1 to 66 years), and 70% ($n = 23$) were males. According to the summary, "45% ($n = 15$) had previously received psychological or

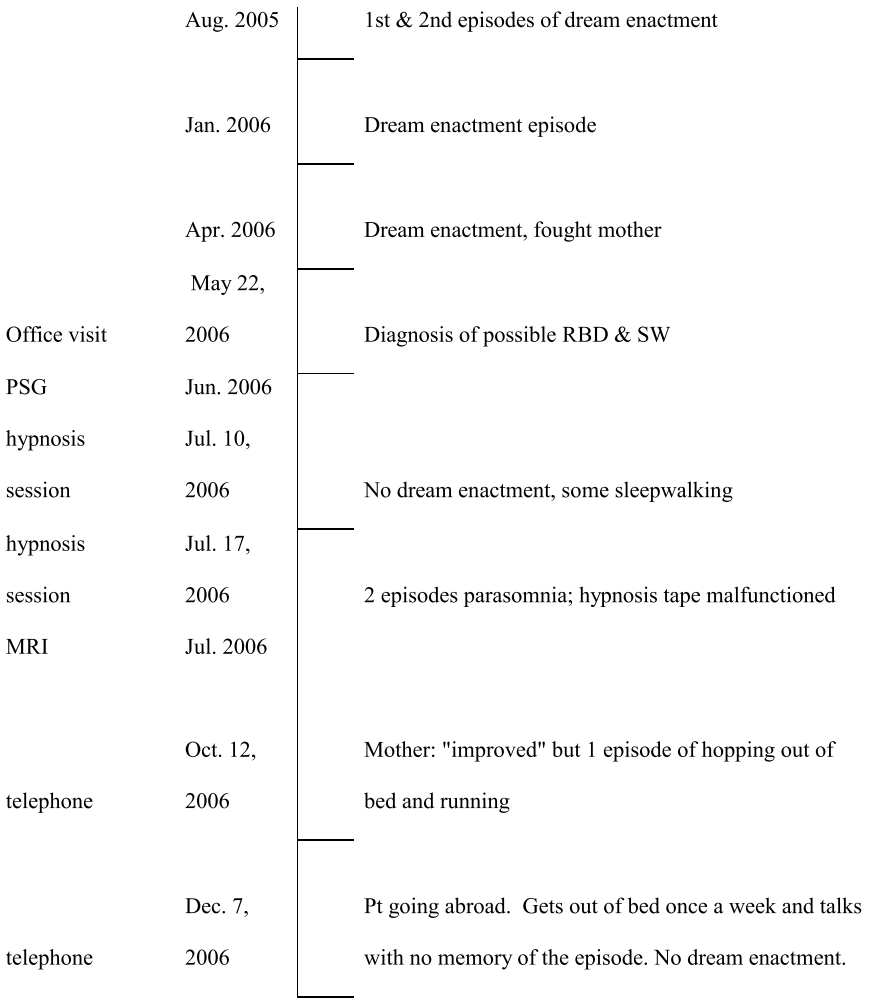

FIGURE 1 Timeline of events. (*continued*)

psychiatric therapy for their parasomnia without benefit." Treatment outcomes were available for 20 patients: "90% ($n = 18$) had substantial parasomnia control with bedtime clonazepam ($n = 13$), alprazolam and/or carbamazepine ($n = 4$), or self hypnosis ($n = 1$)." The authors concluded that POD is a treatable condition that can be "understood within the context of current knowledge on parasomnias and motor control/dyscontrol during sleep."

Alves and colleagues (1999) published a case of a 27-year-old with sexual behavior during sleep that they felt was a case of probable POD. He had a history of sleepwalking since age 9 and developed disruptive violent nocturnal behavior with dream enactment at age 20 with injurious behavior to himself, his wife, and their infant. He was also amnestic to sexual activity

		Clonazepam 0.5 mg started
telephone	Jan. 11, 2007	Mother: "Rare episodes of sleepwalking/no dream enactment." Hypnosis tape used regularly. Clonazepam increased to 0.75 mg
office visit	Dec. 17, 2007	Pt. reports "everything better." Clonazepam 1 mg
		Clonazepam stopped.
hypnosis session	Aug. 3, 2009	Pt. reports approximately 1 episode per month when he hops out of bed while asleep and wakes when his feet hit the floor. Pt reports sleep initiation insomnia and has has been taking zolpidem 5 mg.
office visit	Jan. 4, 2010	Approximately twice a month pt dreams something is in the bed; as soon as his feet touch the floor, he immediately goes back to sleep. He is experiencing sleep maintenance insomnia. Zolpidem ER 12.5 mg started.

FIGURE 1 (*Continued*).

telephone	Dec. 2011	Pt reports he is doing well. Dream enactment episodes occur every four to six weeks but he awakens when his feet touch the floor.

FIGURE 1 (*Continued*).

occurring during sleep. Remarkable clinical improvement was noted using clonazepam 2 mg at bedtime.

Bonakis, Howard, Ebrahim, Merritt, and Williams (2009) retrospectively evaluated 91 patients with RBD. They divided the patients by age into two groups: under age 50 and 50 years or older. The younger group consisted of 23 male and 16 female patients; 20 had idiopathic RBD and 19 had secondary RBD. Among the patients with idiopathic RBD, 13 had complex behavior, including episodes of sleep talking and sleepwalking. In the older group, 39 were males and 13 were females; 33 had a diagnosis of idiopathic RBD, and 2 of the 33 also experienced behavior compatible with sleepwalking. They concluded that there was a very strong association between RBD and NREM parasomnias, with the coexistence of RBD with non-REM parasomnias not uncommon in cases of idiopathic RBD affecting patients below 50 years of age.

In our case study, the significance of the possible MRI finding of an old lacunar infarct in the left basal ganglia is unclear. RBD has been associated with significant CNS lesions (Schenck & Howell, 2013). Limousin and colleagues (2009) reported on a case of a brainstem inflammatory lesion in the pontine tegmentum causing RBD and sleepwalking. They hypothesized that a unilateral lesion by itself was sufficient to cause both the RBD and sleepwalking. They suggested that unilateral lesions of the REM sleep atonia system are sufficient to enhance/release the axial and bilateral limb muscle tone during REM sleep and also to trigger sleepwalking. Melatonin at a dose of 9 mg per day improved the symptoms, but clonazepam did not result in any improvement.

Cicolin and colleagues (2011) reported on two patients with sexual behaviors during sleep (SBS) associated with POD as documented by PSG: Video-PSG documentation of sexual behavior was reported in one of the two cases, a 60-year-old female with an episode of masturbation occurring during slow wave sleep (SWS) preceded by hypersynchronous delta pattern. The EEG pattern showed the persistence of SWS with increasing alpha activity. According to the abstract, "when awoken by technicians, the patient was not aware of her sexual behavior and did not report any dreams." The second patient was a 41-year-old male and this POD case was documented by PSG. According to the abstract, "This is an unprecedented report of SBS in patients with PSG-confirmed POD and of SBS documented during video-PSG."

Dumitrascu, Schenck, Applebee, and Attarian (2013) reported five individuals with POD where the arousal, NREM parasomnia, was a more prominent feature than the RBD component. After analyzing their cases and case reports in the literature, they concluded that POD is most

likely a separate condition and not a subtype of RBD. Currently, the International Classification of Sleep Disorders (2nd ed., 2005) identifies POD as a variant of RBD rather than a separate disorder.

CONCLUSION

This case study describes the successful use of hypnosis in the treatment of both components of this patient's parasomnia overlap disorder (POD), the dream enactment (REM) as well as the sleepwalking (NREM) aspects. Hypnosis is a potentially important nonpharmacologic tool for effectively treating POD. This case report should stimulate further research into the efficacy of hypnosis and hypnotic suggestions for treatment of potentially dangerous RBD as well as for NREM parasomnias that occur with no recall of the episode.

ACKNOWLEDGMENTS

The authors wish to thank Dr. Carlos H. Schenck, MD, for his encouragement, for his review of the manuscript, and for his valuable suggestions.

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