Title

How To Fix Insomnia: Don’t Get Into Bed

Journal

Proceedings of the UCLA Department of Medicine, 17(1)

Authors

Bierer, Gregory
Mecham, Ian
Wen, Eugenia

Publication Date

2013-05-30

CLINICAL VIGNETTE

How To Fix Insomnia: Don't Get Into Bed

Gregory Bierer, MD, Ian Mecham, MD, Eugenia Wen, MD

Case Report

A 40-year-old female presented with inability to attain adequate sleep. Her history was pertinent for spine surgery 2 years prior to presentation, after which she was treated with opioids and benzodiazepines to facilitate sleep. Initially, pain was a limiting factor for good quality sleep, but after her pain was controlled and eventually subsided, she reported continued insomnia. She continued to experience poor quality sleep after stopping all pain and sleep medications, so she began to use medications in combination to try to find a solution, but this approach has not worked. She has used zolpidem, lorazepam, eszopiclone and a variety of opioids alone or in combination. When taking lorazepam, she described a paradoxical increased level of alertness.

At initial presentation, she reported little to no sleep the prior 3 nights. Typically, she gets into bed between 10 and 11 pm and reports a sleep latency (time to fall asleep) of 1 to 4 hours. She sleeps in a cool, dark, quiet room with no television or radio. She endorses a racing mind and becomes increasingly anxious about her insomnia as the night progresses. Once she does fall asleep, she reports continued awareness of her surroundings and is easily awakened by light or noise. She does not report any specific awakenings from sleep. She wakes daily between 6 to 7:30 am. Apart from an occasional headache, she denies grogginess in the morning and can go about her day without limitation of her activities. Her Epworth Sleepiness Scale is 2 (does not indicate pathologic sleepiness). She does not nap, but states that she "wishes she could". She has no history of snoring, witnessed apneas, abnormal behaviors during sleep, cataplexy, depression or any other psychiatric diagnosis. She currently denies nocturnal awakenings due to pain, nocturia, dyspnea or any other cause except for environmental (noise, light). She reports that the best quality sleep she recalls was during a trip to visit family in the Midwest; she even slept through the night peacefully even though there was a raging thunderstorm that woke up most of the family.

Her past medical history is significant for knee surgery in 1986 complicated by post-operative DVT and PE that was treated with anti-coagulation. She underwent a L4 to S1 laminectomy and partial discectomy in 1992 with another DVT and PE. She was diagnosed with Protein C and Protein S deficiencies and is on full dose chronic anticoagulation. Her last surgery was a right femoroplasty and acetabuloplasty about 1 year prior to presentation.

She has one caffeinated diet soft drink around 5 pm daily but otherwise does not ingest caffeine. She does not drink alcohol, use tobacco or illicit drugs. She is married but she and her husband sleep in separate rooms due to her insomnia. She has not worked since her hip surgery in 2012. She reports a very stressful year with the loss of a family member and an ongoing prolonged legal dispute. She is adopted and unaware of any family history.

On physical examination she has a normal BMI and neck circumference is less than 15 inches. She has a Mallampati class 1 view and no abnormality of the uvula or palate. Her lungs are clear and her heart sounds are normal without murmur. She has no lower extremity edema.

Insomnia Diagnosis

In reviewing her history, she did not have any symptoms or signs indicative of sleep disordered breathing, movement disorder, or an abnormality with her circadian rhythm. Her consistent wake time in the early morning indicates a functioning circadian rhythm. The stimulus to awaken is very closely linked to the rise in body temperature, a process that is aligned by the circadian rhythm. Although she did have a stressful ongoing family situation, she denied depression.

Her chronic insomnia is due to multiple factors. She initially had insomnia after the surgical procedure,

and this was primarily due to pain. The patient did not respond to the medical regimen of a benzodiazepine as expected; therefore she also exhibited insomnia due to a drug, namely a paradoxical reaction to a benzodiazepine. The stressful family situation also may have contributed to difficulty sleeping, and a diagnosis of adjustment insomnia regarding that event is reasonable. The patient then developed learned associations that lead to stress about sleep, and these associations became persistent and amplified her insomnia. This process is known as psychophysiologic insomnia, and this type of insomnia is likely the dominant barrier for her to attain healthy sleep.

Treatment

Treatment for insomnia includes pharmacologic and non-pharmacologic approaches, of which the focus will be the latter. Psychological and behavioral therapies are recommended as first line therapy for chronic primary insomnia and insomnia secondary to comorbidities[1]. The non-pharmacologic therapies include sleep hygiene, relaxation therapy, stimulus control, sleep restriction, and cognitive behavioral therapy. We will discuss sleep restriction therapy in greater detail as this was used for our patient.

Sleep hygiene involves ensuring that the sleep environment as well as the patient's behavior is conducive for sleep. Relaxation therapy is a modality that is often employed, using strategies such as progressive muscle relaxation to facilitate a relaxed body and mind. Stimulus control is an approach in which the patient is advised to only use the bed (even the bedroom) for sleep and minimize any other activities (reading, etc) in that space. Cognitive behavioral therapy involves sessions with a therapist designed to modulate the patient's attitudes and beliefs to foster a healthy approach to sleep[1].

Sleep restriction therapy utilizes the natural propensity for sleep that increases with time awake. This therapy purposefully restricts time in bed to maximize the drive to sleep. The basic principle that the longer one is awake, the greater the natural sleep drive, is used in this therapy to attempt to consolidate sleep. Patients with chronic insomnia, particularly of the psychophysiologic type, may also spend excessive time in bed or have unrealistic expectations of normal total sleep time. This excessive time in bed may lead to "tossing and turning" and perpetuate anxiety about sleep. In sleep restriction therapy, a typical approach is to approximate the patient's estimated total sleep time (TST) based on the history or sleep log, and then restrict the total time in bed (TIB) to equal the TST or TST plus 30 minutes. Usually practitioners will not prescribe less than 5 hours of TIB as a minimum. The patient is then requested to set a regular wake time and not deviate from that time, and most importantly, not to nap. Every 3 – 7 days, the patient can increase the TIB by 15 to 30 minutes until he or she achieves the target sleep time[2]. Patients should maintain sleep efficiency between 80 and 90%. Sleep efficiency (SE) is calculated by the patient's sleep time divided by the total time in bed. This is measured during a sleep study, but for outpatients this is a subjective measure.

The 2006 update to the Practice Parameters for the Psychological and Behavioral Treatment of Insomnia by the American Academy of Sleep Medicine regard sleep restriction as an "effective and recommended therapy in the treatment of chronic insomnia"[1]. Sleep restriction therapy has been shown to be effective as a single modality therapy in a few trials. The initial description in 1987 studied 49 patients with sleep onset and sleep maintenance insomnia, but 14 of those dropped out. Eight were discouraged and had difficulty adhering to the therapy, four did not complete the study for other reasons, and 2 had such improvement in the first few days that they did not want to complete the 8-week protocol. The 35 patients studied had a mean age of 46 years with insomnia for 15 years on average. Patients already using a hypnotic medication continued using it during the trial. This trial had patients limit the TIB to their perceived total sleep time with a minimum of 4.5 hours in bed at the initiation of the protocol. A mean value of subjective sleep efficiency (SE) was calculated over 5 nights of sleep and decisions were made based on the value. If the SE was greater than 90%, then the subject increased TIB by 15 minutes. If the SE was less than 85% for a 10-night average, then TIB was decreased to the mean sleep time of the prior 5 days. If the SE was between 85% and 90%, then no change was made. In these 35 patients, sleep latency decreased from 48 minutes to 19 minutes and total sleep time increased from 320 minutes to 343 minutes with sleep efficiency improving from 67% to 87%. Wake time after sleep decreased from 159 minutes to 50 minutes. On a questionnaire, 71% of patients felt that their insomnia improved. These improvements were maintained at a 36-week follow up interval[3]. Another trial evaluated chronic insomniacs with a mean age of 69 years and compared sleep restriction therapy (n=10) to relaxation therapy (n=12) in an 8-week trial. During active treatment, both groups had reduced sleep latency and wake time after sleep onset, but at a 3-month follow-up, the sleep restriction patients

maintained the improvement whereas the relaxation group did not. Total sleep time increased in both groups, but the magnitude of the improvement in the sleep restriction group was approximately twice that of the relaxation group[4]. A recent investigation utilized sleep restriction therapy with basic sleep hygiene education as a method to establish healthy sleep patterns and withdraw patients from routine hypnotic use. The study found significant improvements in sleep duration and reductions in hypnotic use that persisted at 6 and 12-month intervals, after the 8 week intervention[2]. This study adds to the evidence that sleep restriction alone can be an effective modality. Clinicians may often combine sleep restriction therapy with stimulus control therapy. The addition of stimulus control, using the bed only for sleep, further assists in dislodging negative learned associations that are often operative in psychophysiologic insomnia[5].

One of the main limitations of sleep restriction therapy is that the patient must be motivated to carry out the plan. Discipline is required to not only limit the bed time, but also to avoid naps and maintain limited time in bed when one develops sleepiness. Needless to say, many patients with insomnia are already frustrated at being tired during the day, and the plan to restrict time in bed may, at first, sound counter-intuitive. This may lead to an inability to adhere to the schedule, as the perception of barriers to the implementation of a sleep restriction protocol has been associated with worse efficacy[6].

Another aspect of the therapy to consider is the possible adverse effects associated with this approach. A study designed to explore such side effects found more than 50% of the 18 patients in the trial had an adverse effect. The most common side effects were fatigue, extreme sleepiness, reduced energy, and headache during the treatment period. They did note that those experiencing these difficulties were actually responders to the therapy[7].

Treatment Implementation

Our patient was very motivated to try a new approach. When using hypnotics, she had not had any success and actually experiencing worsening symptoms. Since she was dedicated to fixing her insomnia and did not have any other concomitant sleep disorders, sleep restriction therapy was prescribed. The patient had a target wake time of 7:00 am. She agreed to not go to sleep until 2:00 am for 3 consecutive nights, then increase her opportunity for sleep by 15 to 30 minutes every 3 nights to a target sleep time of 11:00 pm. She agreed not to take naps during the day.

On her first follow up visit, the patient reported that she was initially in disbelief that the program would work. She felt it was ironic to complain of insomnia and be told to sleep less. Nevertheless, she adhered to the program and the patient reported excellent results. She did not go to bed until 2:00 am for the first 3 nights. She reported feeling progressively tired on each subsequent day, but did not take a nap. She then went to sleep at 1:30 am on night 4. By the fifth night, she was sufficiently sleepy that she went to bed just after 11:00 pm and slept until 7:00 am. She maintained this schedule of sleeping from 11:00 or 11:30 until 7:00 am for the next 3 weeks. She felt much better and refreshed after her sleep.

We discussed that insomnia may occur again, and that a rare night of poor sleep is not cause for alarm. The sleep restriction therapy could be used again if she were to develop a similar type of insomnia for more than a week, and the patient reported feeling confident that she could utilize this approach.

REFERENCES

1. **Morgenthaler T, Kramer M, Alessi C, Friedman L, Boehlecke B, Brown T, Coleman J, Kapur V, Lee-Chiong T, Owens J, Pancer J, Swick T**; American Academy of Sleep Medicine. Practice parameters for the psychological and behavioral treatment of insomnia: an update. An american academy of sleep medicine report. *Sleep*. 2006 Nov;29(11):1415-9. PubMed PMID: 17162987.
2. **Taylor DJ, Schmidt-Nowara W, Jessop CA, Ahearn J**. Sleep restriction therapy and hypnotic withdrawal versus sleep hygiene education in hypnotic using patients with insomnia. *J Clin Sleep Med*. 2010 Apr 15;6(2):169-75. PubMed PMID: 20411695; PubMed Central PMCID: PMC2854705.
3. **Spielman AJ, Saskin P, Thorpy MJ**. Treatment of chronic insomnia by restriction of time in bed. *Sleep*. 1987 Feb;10(1):45-56. PubMed PMID: 3563247.
4. **Friedman L, Bliwise DL, Yesavage JA, Salom SR**. A preliminary study comparing sleep restriction and relaxation treatments for insomnia in older adults. *J Gerontol*. 1991 Jan;46(1):P1-8. PubMed PMID: 1986039.
5. **Toth CM**. The anxious sleeper. Initiate sleep-restriction and stimulus-control techniques. *J Clin Sleep Med*. 2010 Aug 15;6(4):403-4. PubMed PMID: 20726291; PubMed Central PMCID: PMC2919673.
6. **Vincent N, Lewycky S, Finnegan H**. Barriers to engagement in sleep restriction and stimulus control in chronic insomnia. *J Consult Clin Psychol*. 2008 Oct;76(5):820-8. doi: 10.1037/0022-006X.76.5.820. PubMed PMID: 18837599.

7. **Kyle SD, Morgan K, Spiegelhalder K, Espie CA**. No pain, no gain: an exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia. *Sleep Med*. 2011 Sep;12(8):735-47. doi: 10.1016/j.sleep.2011.03.016. Epub 2011 Sep 9. PubMed PMID: 21907616.

Submitted on May 30, 2013