The Potential for Somnambulism Associated with Gabapentin
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Citation
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Abstract
Somnambulism, or sleepwalking, is a parasomnia occurring during Non-Rapid Eye Movement (NREM) sleep. It is defined as complex behaviors during slow-wave (or deep) sleep, with the person often having no recollection of these events upon waking. It is associated with several medications, including sedative-hypnotics, antipsychotics, antidepressants, lithium, stimulants, antihistamines, and anticonvulsants. Other risk factors include a history of sleepwalking in a first-degree relative, sleep-disordered breathing, restless leg syndrome, separation anxiety, or events that increase deep sleep, such as evening exercise, sleep deprivation, or fever. Benzodiazepines are recommended for treating somnambulism, despite a lack of controlled trials. Gabapentin is often prescribed for seizures, neuropathic pain, or anxiety. Although there are no reports of somnambulism associated with gabapentin, it is hypothesized that gabapentin may cause somnambulism, given its effect on sleep architecture and anecdotal reports.

INTRODUCTION
Somnambulism, or sleepwalking, is a parasomnia occurring during Non-Rapid Eye Movement (NREM) sleep. It is defined as complex behaviors during slow-wave (or deep) sleep, with the person often having no recollection of these events upon waking. It is associated with several medications, including sedative-hypnotics, antipsychotics, antidepressants, lithium, stimulants, antihistamines, and anticonvulsants. Other risk factors include a history of sleepwalking in a first-degree relative, sleep-disordered breathing, restless leg syndrome, separation anxiety, or events that increase deep sleep, such as evening exercise, sleep deprivation, or fever. Benzodiazepines are recommended for treating somnambulism, despite a lack of controlled trials. Gabapentin is often prescribed for seizures, neuropathic pain, or anxiety. Although there are no reports of somnambulism associated with gabapentin, it is hypothesized that gabapentin may cause somnambulism, given its effect on sleep architecture and anecdotal reports.

Gabapentin and benzodiazepines differ in their effects on sleep architecture. Benzodiazepines decrease slow-wave sleep, the time during which somnambulism occurs; hence they have demonstrated efficacy and are recommended to treat somnambulism. In contrast, gabapentin increases slow-wave sleep and decreases REM sleep. Similarly, the sedative-hypnotic zolpidem increases slow-wave sleep and suppresses REM sleep, and has been associated with somnambulism. An Internet survey of more than 37,000 individuals found that 0.2% of them reported “moderate to severe” somnambulism which they attributed to gabapentin. However, results are confounded by two factors. First, nearly 60% of those reporting somnambulism were taking amitriptyline, which has been associated with somnambulism. Furthermore, there is no information regarding whether patients discontinued, or discontinued then restarted gabapentin, and experienced changes in somnambulism. Therefore, it is impossible to determine whether gabapentin caused somnambulism from this online report.

HYPOTHESIS TESTING
An open-label prospective trial of adequate duration (3 months minimum) of adult men and women could assess whether users experience sleepwalking from gabapentin. Participants would preferably have no history of gabapentin use or seizures, as certain seizure disorders are associated with sleepwalking-like behaviors. Participants’ consent to
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and use of video recording, or the presence of another person, could confirm the reported somnambulism. Gabapentin would be prescribed at the beginning of the study and titrated to efficacy. Because somnambulism has been associated with other medications, starting, stopping, or adjusting doses of other medications would be minimized and preferably avoided. In addition to participants’ gender and age, the gabapentin dosage, frequency of administration, and time of administration are factors that may contribute to somnambulism and would be evaluated in statistical analysis.

If somnambulism occurs, the affected participants would stop gabapentin and assess for resolution of sleepwalking. If sleepwalking resolves, a retrait of gabapentin and subsequent return of somnambulism would implicate gabapentin as the cause. However, if somnambulism persists in the absence of gabapentin, then this would suggest that gabapentin is not the cause. Throughout the study, investigators should periodically monitor for adverse effects of gabapentin, such as sedation, cognitive impairment, mood changes, or weight gain, and discontinue participation if these effects are severe or intolerable.

CONCLUSION

Gabapentin displays opposite effects on sleep architecture from benzodiazepines, which are used to treat somnambulism. Gabapentin shares similar effects on sleep architecture with zolpidem, which is known to cause somnambulism. In addition to effects on sleep architecture, anecdotal reports raise questions regarding the possibility of somnambulism with gabapentin. The proposed study above could answer whether sleepwalking is associated with gabapentin. The potential exists for somnambulism with gabapentin, and monitoring by prescribers and patients is recommended.

References

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