

## Somnolence and Periodic Limb Movements Due to Treatment with Baclofen

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### Abstract

**Background and Objective:** Excessive sleepiness is a common reason for a referral to sleep medicine clinics. The clinical picture and context usually suggest the underlying cause; however, all possibilities should be considered and only after sleep studies are done, one can determine the likely cause of the patient's symptoms.

**Case Report:** We presented the case of a man who was referred for evaluation of hypersomnolence along with snoring and possible apnea during sleep. Obstructive sleep apnea (OSA) was suspected, so home sleep apnea test (HSAT) and later polysomnography (PSG) were performed and both ruled out this possibility. The PSG showed frequent periodic limb movements (PLMs), some associated with arousal; abnormal sleep structure suggested the effect of medication. The patient had been taking baclofen for musculoskeletal pain and it was concluded that baclofen was the cause of the patient's somnolence and PLMs. The patient's clinical presentation was compounded by his history of post-traumatic stress disorder (PTSD) and gastroesophageal reflux which probably caused increased arousals and the subjective feeling of poor sleep quality.

**Conclusion:** Treatment with medications that have sedative effects should be considered in all patients presenting with excessive daytime sleepiness even if the initial clinical picture suggests another possible cause. Baclofen can cause PLMs, sedation, changes in N3 stage, and reduction in rapid eye movement (REM) sleep.

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**Keywords:** Baclofen; Disorders of excessive somnolence; Nocturnal myoclonus syndrome

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### Introduction

Hypersomnolence is a common complaint in sleep medicine clinics. It is frequently due to sleep-disordered breathing (SDB), central hypersomnia, circadian rhythm disorders as well as many other sleep disorders. Sometimes a patient has multiple possible explanations and it may be difficult to determine initially which one is the main cause of the patient's complaint and all need to be addressed to result in significant clinical improvement. In the case discussed below, we present a case of a man with excessive daytime sleepiness and highlight how multiple factors might have contributed to his

complaint and how considering these factors helped explain the findings of his polysomnography (PSG), all in the context of his clinical history.

### Case Report

A 56-year-old man presented with snoring, nocturnal choking, non-refreshing sleep, and daytime somnolence. He denied morning headaches, sleep paralysis, cataplexy, and discomfort in his legs or an urge to move them in the evening. He would typically sleep from 11:00 PM to 5:30 AM on weekdays and weekends, and his sleep schedule did not change in many years.

He had history of degenerative joint disease of the lumbosacral spine and knees, right biceps pain due to tendinitis, gastroesophageal reflux, hypogonadism, erectile dysfunction (ED), and post-

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traumatic stress disorder (PTSD) for which he was not taking his prescribed treatment.

His medications included: baclofen 10 mg orally 3 times a day, omeprazole 40 mg twice a day, tadalafil 20 mg when needed, and testosterone 200 mg intramuscularly (IM) every 2 weeks.

His Epworth Sleepiness Scale (ESS) score was 15, body mass index (BMI) was 27 kg/m<sup>2</sup>, and upper airway was Mallampati class II. His vital signs were normal and general medical examination was unremarkable except for musculoskeletal tenderness in the right shoulder. Laboratory evaluation showed normal renal function tests, iron studies, and vitamin and hormonal levels.

Prior to presenting to sleep medicine service, he had a home sleep apnea test (HSAT) that was reported as normal. A PSG was performed and showed the findings in table 1:

**Table 1.** Polysomnographic (PSG) findings

Parameter	Value
Total sleep time (minute)	352.0
Sleep efficiency (%)	88.0
Waking after sleep onset (minute)	44.1
TAI (/hour)	66.5
SOL (minute)	4.0
REM sleep latency (minute)	386.0
N1 stage (%)	40.5
N2 stage (%)	56.0
N3 stage (%)	1.1
REM stage (%)	2.4
AHI (/hour)	1.7
Minimum oxygen saturation (%)	89.0
PLMI (/hour)	91.2
PLM-arousal index (/hour)	43.5

TAI: Total arousal index; SOL: Sleep onset latency; REM: Rapid eye movement; AHI: Apnea-hypopnea index; PLMI: Periodic limb movement index; PLM: Periodic limb movement

Hypnogram of his sleep study showed reduction in rapid eye movement (REM) sleep and stage N3, frequent periodic limb movements (PLMs), frequent arousals, and absence of sleep

apnea (Figure 1).

Figure 2 shows PLMs, some of them are associated with arousal.

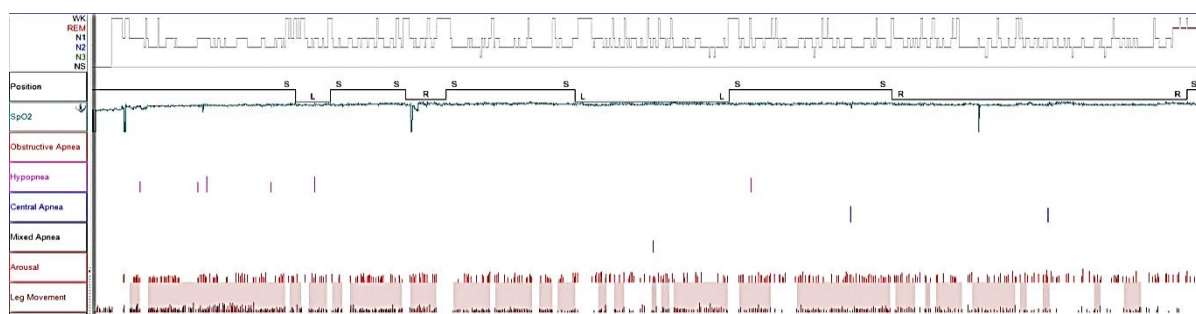
## Discussion

Although the patient's history is suggestive of obstructive sleep apnea (OSA), his HSAT and PSG clearly ruled out this possibility. Snoring is not unusual in patients who do not have sleep apnea; these patients have primary snoring. The patient's reported nocturnal choking could be due to acid reflux or exaggerated reaction to rare apneas in a patient with hyperarousal related to PTSD.

His PSG shows a significant number of PLMs and given that a significant number of them is associated with arousal, it is possible that his somnolence and disrupted sleep are due to periodic limb movement disorder (PLMD). However, it is also possible that both hypersomnolence and increased PLMs are due to treatment with baclofen. It has been reported that baclofen can increase PLMs (1) and it is known to cause sleepiness. The patient was questioned about the onset of his hypersomnolence and history suggested that it started after baclofen was prescribed for him.

The patient's PSG data show increased stage N1, minimal stage N3 and REM sleep, and increased REM latency. These findings raise the possibility of the effect of baclofen which can explain some but not all of these findings. It was reported that baclofen could decrease REM stage sleep especially with high doses, increase stage N3, increase total sleep time, and decrease sleep latency. It can also increase the number of PLMs and decrease arousals induced by the leg movements (1-4).

It has also been reported that baclofen can produce a minor reduction of oxygen saturation during sleep (2) and can also induce central sleep apnea (CSA) (5).



**Figure 1.** Hypnogram summarizing polysomnographic (PSG) findings



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