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Case report

# Exacerbation of REM sleep behavior disorder by chocolate ingestion: a case report

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**Abstract**

REM sleep behavior disorder (RBD) is manifest by loss of normal rapid eye movement sleep atonia and the acting out of dreams of often violent content. Both idiopathic and secondary forms of RBD exist. We report on chocolate as a possible new precipitating agent for RBD and comment on a possible mechanism of action in this disorder. 

*Keywords*: REM sleep behavior disorder; Chocolate; Obstructive sleep apnea; Dopamine; Adenosine; Striatum; Caffeine

## 1. Introduction

REM sleep behavior disorder (RBD) manifests as a loss of the usual REM sleep atonia as well as vigorous and potentially injurious behavior during REM sleep [1]. Both idiopathic chronic RBD and secondary chronic RBD have been identified. Common secondary causes include Parkinson's disease, multiple system atrophy and dementia [2]. We report chocolate as a possible new precipitating agent for RBD and comment on some previous work and potential pathophysiology.

## 2. Case report

The patient subsequently described was seen in the Eastern Virginia Medical School/Sentara Norfolk General Hospital. He initially underwent a full sleep disorders history and physical examination. He then underwent a nocturnal polysomnogram including electroencephalogram (EEG) (C3–A2, C4–A1, O1–A2 and O2–A1), electrooculogram (EOG), electromyogram (EMG) (chin, arm and leg), respiratory channels using inductive plethysmography, oxygen saturation, electrocardiogram (ECG) and video monitoring.

This 64-year-old Caucasian male presented with a chief complaint of 'snoring, sleep apnea and nightmares'. He and his wife related classic symptoms of OSAS with loud snoring, witnessed pauses in respiration during sleep and some daytime sleepiness. Additionally, they noted that following a severe motor vehicle accident 10 years prior to his visit (in which there was significant facial trauma but no loss of consciousness) the patient developed violent sleep related turning, thrashing and kicking. These nocturnal events occurred approximately five out of seven nights and were associated with 'hollering'. The patient admitted to nightmares with violent content in which he believed he was protecting himself or preventing intruders from entering the house. In lashing out during these dreams he had previously injured his right hand. Both the patient and his wife agreed that the ingestion of chocolate had repeatedly caused an exacerbation of these events. Cookies containing chocolate, chocolate ice cream and chocolate syrup were all found over the course of 6–8 months to be offenders.

The patient denied medications that lead to RBD. He admitted to one daily cup of coffee, decaffeinated tea only and the rare soft drink. He denied alcohol and though he had a 22-pack year smoking history he had not smoked in 3–4 years. He averaged 6–8 h of sleep daily. He admitted to a history of gastroesophageal reflux disease, hypertension, a previously resected squamous cell carcinoma of the ear and a deviated septum. His family history revealed no evidence of sleep disorders and his review of systems was negative for neurologic symptoms. His physical examination revealed a healthy, pleasant gentleman with a BMI of 33 kg/m$^2$ and no neurologic abnormalities.

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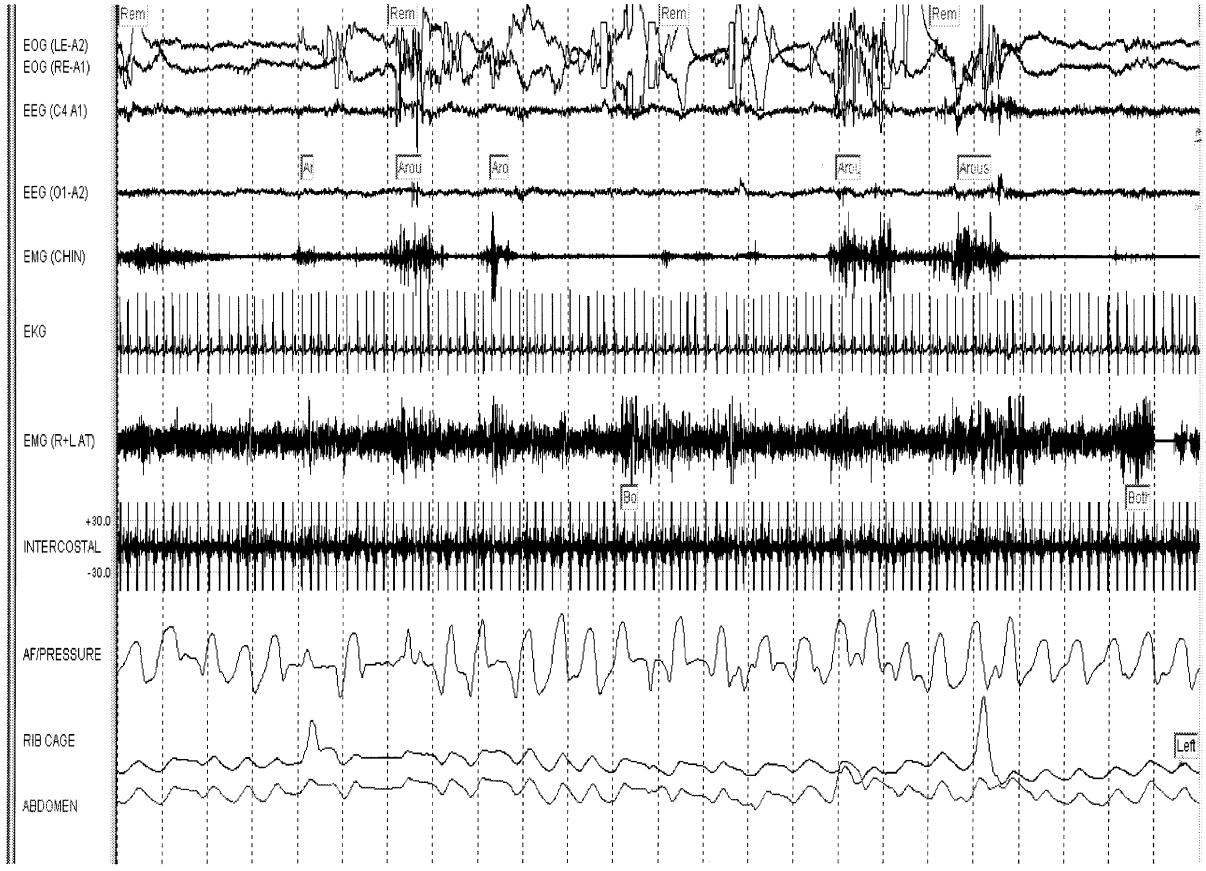


Fig. 1. The 120-s sweep demonstrating variably increased chin tone activity during REM sleep (each increment is 5 s).

The initial impression was of both the obstructive sleep apnea syndrome and RBD.

The patient's nocturnal polysomnogram revealed an overall respiratory disturbance index (RDI) of 14. His REM RDI was 21. The low oxygen saturation was 90%. He had increased muscle activity in REM sleep but no overt behaviors. The diagnosis was of OSAS and RBD. Subsequently, the patient was treated with CPAP for his OSAS. It is noteworthy that the CPAP titration study on this patient also showed PLMS with persistence into REM sleep and increased chin muscle activity in REM sleep (see Fig. 1). Clonazepam 0.5 mg has dramatically improved his RBD symptomatology.

## 3. Discussion

Our patient demonstrated typical symptoms and polysomnographic features of RBD with a history suggesting an interaction between chocolate ingestion and exacerbation of RBD. Two reports support a possible relationship. A large telephone survey on violent behavior in sleep suggested a link between heavy caffeine ingestion and nocturnal violent behaviors [3]. Another report linked caffeine and RBD in a patient with prolific coffee intake [4]. Cessation of caffeine improved the patient's symptoms, which began to recur on resumption of caffeine intake. Our patient differs from their account in that his intake was of chocolate and his intake was more modest.

Caffeine has long been known to alter sleep and has been reported to lead not only to REM sleep suppression, but also to REM rebound [5]. Caffeine exerts its effects through the inhibition of the putative sleep substance, the purine adenosine [6,7]. In addition to soporific effects, adenosine affects locomotor behavior. In mice, activation of the adenosine subtype receptor A2A likely leads to diminished locomotion [8] and A2A inhibition leads to the stimulation of locomotion [9].

Recent evidence reveals diminished dopaminergic activity in the striatum of RBD patients. Use of IPT-SPECT techniques demonstrated a reduction in dopamine transporters in the striatum of RBD patients [10]. Albin et al utilized PET technology to demonstrate diminished dopaminergic activity in the striatum [11]. Data do link both adenosine and dopamine striatal receptors suggesting that caffeine may well exert its effects on locomotion both through adenosine and dopamine [12]. We therefore postulate that our patient's RBD was worsened by chocolate due to antagonism of the adenosine A2A striatal receptors in the setting of perturbed dopaminergic activity. The adenosine A2A receptor has

come under increasing interest as a therapeutic portal for Parkinson's disease [13,14]. Parkinson's disease follows RBD in some 38% of RBD patients [15]. Perhaps adenosine deserves further scrutiny for import in RBD.

Two final points deserve emphasis. It is tantalizing to implicate the patient's motor vehicle accident and attendant head trauma as a precipitant for his RBD. The authors are not aware of literature showing such a link. Finally, this patient was afflicted with mild sleep apnea, which can cause abnormal sleep-related behaviors. His classic symptoms of RBD and his polysomnographic findings consistent with RBD argue that he had RBD exacerbated by chocolate.

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