his balance and in addition developed mild rigidity affecting his limbs with no other signs of Parkinsonism. A new VPSG revealed severe PLMs with index of 141.5, mild OSA (AHI 11.5), respiratory related arousals (9/h) and violent behaviour during REM sleep (Fig. 2). In addition to ropinirole the patient received melatonin 3 mg/day resulting in mild improvement of his RBD.

3. Video sequences

Video 1: Abnormal behaviour during REM sleep. The patient punches, kicks and finally falls out of bed.
Video 2: An almost rhythmic, 1–2 Hz oscillation of the posterior soft palate characteristic of symptomatic palatal tremor. However, some of the movements are clearly myoclonic in nature and the term palatal myoclonus probably better describes the movement disorder in this case.

4. Discussion

Palatal tremor (syn. palatal myoclonus) is a rare movement disorder subdivided into essential (EPT) and symptomatic (SPT) forms [1]. EPT is a syndrome of unknown aetiology in which there is typically isolated rhythmic movements of the anterior soft palate often associated with ear clicks which can be heard by the patient [1]. SPT is characterized by rhythmic oscillation of the posterior soft palate accompanied by other signs and symptoms mainly of brainstem origin. Definable aetiologies for SPT most commonly include monophasic structural lesions such as stroke, trauma, demyelination and posterior fossa tumors [2]. However, up to 17% of all SPT cases emerge in the spectrum of a neurodegenerative process [2]. In these cases there are prominent signs and symptoms of cerebellar and/or brainstem dysfunction, and they form a heterogeneous syndrome, the so called "syndrome of progressive ataxia and palatal tremor" that is considered by some authors a distinct neurodegenerative disorder [2,3]. In all reported cases of PAPT there is no information regarding sleep related disorders. In the case we present, the appearance of idiopathic RBD 2 years prior to PAPT emergence turns the diagnosis towards a synoucleinopathy [4]. The combination of idiopathic RBD, followed by cerebellar and extrapyramidal dysfunction, can be encountered in MSA. The MRI of the brain was negative both times but was performed early in the course of the disease. The VPSG both times reveals REM sleep without atonia accompanied by aggressive behaviour, PLMs and mild OSA – a combination that is prevalent in MSA [5]. However, the absence of autonomic dysfunction after 6-year follow-up is against the diagnosis of MSA.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.sleep.2009.04.007.

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Concurrent presentation of palatal myoclonus and sleep apnea: A polysomnographic assessment

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1. Introduction

This case discusses the possible relationship of palatal myoclonus (PM) demonstrated in a patient who presented to our sleep clinic for evaluation of sleep-disordered breathing.

2. Case presentation

A 69-year-old woman with a 10-year history of idiopathic PM was referred to our sleep center for evaluation of difficulty maintaining sleep. At her initial sleep consultation, she reported snoring and daytime hypersomnia, often experiencing difficulty breathing and loud snoring during sleep.

She initially presented to her neurologist with a history of auditory ear clicks and involuntary movements in her neck and throat. Her current medications included fexofenadine, amlodipine and atenolol, and her physical exam was significant for bilateral rhythmic movements of the mentalis muscles and prominent rhythmic palatal movements (approximately 90/min). The rest of her examination was normal, including normal phonation and tongue movements. A brain MRI did not show evidence of stroke, masses or hypertrophy of the inferior olives. She was subsequently diagnosed with idiopathic PM. She was tried on clonazepam, lioresal, divalproex sodium and lamotrigine without substantial improvement.

A polysomnogram was recommended based on her history of snoring, hypersomnolence, and post-menopausal status which revealed a sleep latency of 67 min with decreased slow wave and REM sleep and decreased sleep efficiency (59%). The apnea–hypopnea index (AHI) was 17 events/h, with a REM AHI of 49 events/h. Almost all of the respiratory events were obstructive. The average non-supine oxygen saturation was 96.5% and 96.4% in the supine position. The minimum SpO_2 was 88% and was 97.1% during NREM sleep and 96.8% during REM sleep. The PM persisted during NREM sleep but was more variable in frequency and amplitude during REM sleep. During arousals in REM sleep (usually related to respiratory events), the PM amplitude and frequency increased to ranges demonstrated in the lighter stages of sleep. She opted for CPAP therapy and a recommended pressure of 10 cm H_2O was initiated. The patient reported difficulty tolerating CPAP and discontinued it. There was no documented improvement of the PM during CPAP treatment.

3. Discussion

PM is a rare movement disorder defined as brief, rhythmic involuntary movements of the soft palate usually at a frequency of 1.5–3.0 Hz [1]. Symptomatic PM typically persists during sleep, but the frequency and amplitude may vary based on sleep stage [2]. It has been proposed that PM may represent a primitive unmasked reflex since PM resembles the rhythmic gill movements of fish. This explanation is not only interesting, but may provide some insight as to why PM persists during sleep [3].

Our patient demonstrated PM during wakefulness and all stages of sleep with a reduced frequency and amplitude during REM sleep. PM amplitude and frequency during arousals from REM

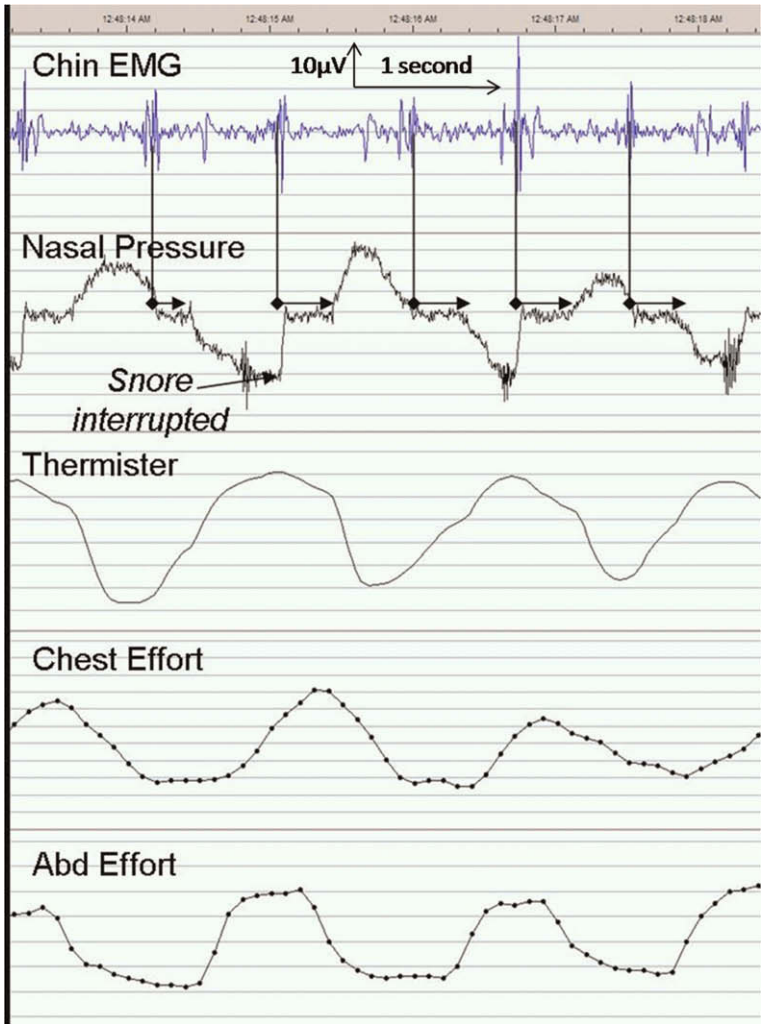

Fig. 1. This is approximately five seconds of the patient's polysomnography featuring the breathing channels. The patient is in REM sleep and oxygen saturation (not shown) is 93%. The inflections on the chin EMG show the palatal myoclonus; this shows how the patient is snoring on the nasal pressure signal (high frequency inflections) and that when there is a myoclonus burst, the snore is abbreviated. Chin EMG, genioglossus electromyography; nasal pressure, airflow as measured by nasal cannula pressure transducer; thermistor, airflow as measured by nasal–oral thermistor; chest effort; respiratory effort in chest as measured by respiratory inductive plethsmography; Abd effort, respiratory effort in abdomen as measured by respiratory inductive plethsmography.