Sleep Medicine 26 (2016) 37–44

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Hypnic jerks are an underestimated sleep motor phenomenon in patients with parkinsonism. A video-polysomnographic and neurophysiological study

Giacomo Chiaro [a], Giovanna Calandra-Buonaura [a,b], Luisa Sambati [a,b], Annagrazia Cecere [b], Caterina Ferri [a], Maria Turchese Caletti [a], Pietro Cortelli [a,b], Federica Provini [a,b,*]

[a] Department of Biomedical and Neuromotor Sciences, University of Bologna, Italy
[b] IRCCS, Institute of Neurological Sciences of Bologna, Bologna, Italy

ARTICLE INFO

Article history:
Received 15 April 2016
Received in revised form 20 July 2016
Accepted 21 July 2016
Available online 23 August 2016

Keywords:
Hypnic jerks
Sleep starts
Myoclonus
Parkinsonism
Sleep fragmentation
Video-polysomnography

ABSTRACT

Introduction: Hypnic jerks (HJs) are sudden contractions of one or more body segments occurring mostly at sleep onset. They are highly sporadic and affect all ages and both sexes with prevalence between 60% and 70% in the general population.
Study objectives: This study describes the frequency and the neurophysiological characteristics of HJs in a population of patients with parkinsonism by means of nocturnal video-polysomnographic recordings.
Methods: This is a prospective cohort study and is reported following the STROBE guidelines. We analyzed the clinical and video-polysomnographic data of the first 66 consecutive patients recruited in the ongoing prospective study "Bologna motor and non-motor Prospective study on Parkinsonism at onset" (BoProPark). Each patient underwent a full neurological workup including a whole-night video-polysomnography. Neurophysiological characteristics including the propagation patterns of the HJs were studied with an extended muscle montage polysomnography.
Results: We recorded a total of 62 HJs in 16 patients out of 66 (24%). Sleep parameters were not statistically different between patients with and without HJs. All HJs were spontaneous and occurred randomly throughout the night. Electromyographic analysis showed that muscle activity arose from different muscles with no prevalence of one over the other and without any ordered propagation. No recurring motor pattern of the jerks was detected.
Discussion and conclusions: Our findings demonstrated that HJs are a frequent, underestimated, sleep-related motor phenomenon in patients with parkinsonism. As they may represent a further cause of sleep disruption and insomnia, HJs should be actively examined. Neurophysiological analysis suggests a subcortical origin of HJs as shown previously for a healthy subject.

1. Introduction

Sleep disorders are common non-motor features of Parkinson's disease (PD) and atypical parkinsonism (AP) that yield a significant impact on patients' sleep and quality of life. Sleep-related motor and behavior disorders such as restless legs syndrome (RLS), periodic limb movements (PLMs), REM sleep behavior disorder (RBD) and NREM-related parasomnias have been studied extensively in both PD and AP [1–5]. On the contrary, little attention has been directed towards hypnic jerks (HJs), a physiological sleep motor phenomenon, which, if intensified, may cause insomnia also in normal subjects [6,7].

HJs are an essentially universal component of the sleep-onset process, although they are often not recalled. They are sudden, brief, non-periodic myoclonic contractions usually involving the whole body or, asynchronically, different and isolated body segments. HJs may be spontaneous or induced by stimuli and sometimes are associated with a peculiar sensory feeling of "shock" or "falling into the void" [8–10]. Various factors such as excessive caffeine or other stimulant intake, intense physical exercise, sleep deprivation, emotional stress and drugs can increase the frequency and severity of HJs [11]. Polygraphically, HJs may be associated with K-complexes, EEG arousals and autonomic activation. The neurophysiological characteristics of HJs have been only recently reported [6,12]. The purpose of this study is to describe the frequency and the neurophysiological characteristics of HJs in a population of patients with PD and AP based on detailed and extensive nocturnal video-polysomnographic (VPSG) recordings.

* Corresponding author. IRCCS, Institute of Neurological Sciences of Bologna and Department of Biomedical and Neuromotor Sciences, University of Bologna, Bellaria Hospital, Via Altura, 3, 40139 Bologna, Italy.
E-mail address: federica.provini@unibo.it (F. Provini).

http://dx.doi.org/10.1016/j.sleep.2016.07.011
1389-9457/

2. Material and methods

2.1. Patients and protocol

This study has a prospective cohort design and is reported following the STROBE guidelines [13]. In the present study, we analyzed the clinical data and the VPSG recordings of the first 66 consecutive patients recruited in the ongoing prospective study "Bologna motor and non-motor Prospective study on Parkinsonism at onset" (BoProPark) (Supplementary material S1).

The BoProPark study aims to prospectively characterize a sample of motor and non-motor features in patients with a progressive neurodegenerative disease starting with parkinsonism (PD; PD with dementia, PDD; multiple system atrophy, MSA; Lewy body dementia, LBD; progressive supranuclear palsy, PSP; corticobasal degeneration, CBD) within three years of disease motor onset and to evaluate their diagnostic and prognostic role in the differential diagnosis of these diseases. The study started in September 2007 and has enrolled a total of 110 consecutive patients referred to our department for a parkinsonism within three years from disease onset. According to the BoProPark protocol each patient undergoes a full neurological examination, quantification of motor impairment by means of part III of Unified Parkinson's disease rating scale [14], objective quantification of motor response to levodopa and evaluation of autonomic, sleep, cognitive and behavioral functions through instrumental procedures, tests and questionnaires. Sleep evaluation also included a whole-night Video-polysomnography (VPSG) (Supplementary methods S2).

All procedures are performed at baseline (T0), after 16 months (T1) and at a five-year follow-up (T2). Diagnoses for each patient are carried out based on data available at T1 according to the international diagnostic criteria for PD [15], PDD [16,17], MSA [18], DLB [19], PSP [20], and CBD [21]. Patients not fulfilling such criteria are diagnosed as unspecified (AP). In the present study we described the VPSG data collected at baseline (T0) of the first 66 patients, for which the second evaluation (T1) was available in December 2015.

The study was approved by the local ethics committee of Bologna (AUSL of Bologna). Patients gave their written informed consent to participate in the study and to publish the data.

2.2. VPSG study

All patients underwent a whole-night VPSG recording at T0, which included electroencephalogram (EEG) (C3-A2, O2-A1, Cz-A1), right and left electrooculogram (EOG), surface electromyogram (EMG) from submental, intercostalis, right and left tibialis anterior and extensor carpi radialis muscles, tracheal microphone, oro-nasal airflow, thoracic and abdominal respirogram, electrocardiogram (ECG), and oxyhemoglobin saturation (SaO_2) by means of finger oxymeter [22]. During the three days preceding VPSG recording, patients were asked to refrain from excessive caffeine intake and smoking.

Out of all 66 VPSGs, we detected 62 HJs in 16 patients. During the follow-up of these patients, we had the opportunity to repeat seven VPSG with a full muscle montage, in order to study the HJs motor propagation pattern. This montage included EEG, bilateral EOG, surface EMG of mylohyoideus and, bilaterally, of orbicularis oculi masseter, deltoid, wrist extensor, rectus abdominis, paraspinalis, rectus femoris, tibialis anterior, and ECG. Sleep stages, arousals, PLMs, and apnea/hypopnea events were scored according to the American Academy of Sleep Medicine criteria [23].

The HJs motor pattern was analyzed in detail, considering how frequently the EMG activity started in a particular muscle and the time delay between the first activated muscle and each of the other muscles, in order to determine whether an ordered pattern of propagation existed.

2.3. Statistical analysis

Demographic, clinical, and VPSG data between the 16 patients with HJs (HJs group) and the 50 patients without HJs (non-HJs group) at first evaluation were compared by means of the Mann–Whitney test for non-parametric measures.

Sleep parameters of the two VPSG of the patients with HJs that repeated VPSG with a full muscle montage and presented HJs during the second recording were also compared by means of Wilcoxon test for non-parametric dependent measures. Statistical significance was set at 0.05. Bonferroni correction was also applied to verify statistical significance after correction for multiple comparisons.

3. Results

We recruited a total of 66 patients, 25 females and 41 males. We recorded a total of 62 HJs in 16 patients out of 66 (24%) (HJs group).

In the HJs group, ten patients were diagnosed with PD, one with MSA, one with PSP and four with undefined AP. In the non-HJs group (50 patients), 40 patients were diagnosed with PD, two with CBD, one with MSA, one with PSP and six with undefined AP.

There were no significant differences concerning demographic and clinical data between the two groups (Table 1). In the HJs group,

Table 1
Clinical data of the HJs group and non-HJs group at T0. Data are expressed as median (25th–75th percentile).

	HJs group (16pts)	Non-HJs group (50pts)	*p*-value
Age at onset (years)	61.5 (53.5–64)	59 (52–67)	0.994
Age at study (years)	63 (55.5–66)	60.5 (54–69)	0.976
BMI	26.8 (25.40–30.37)	26.53 (23.63–30.12)	0.675
Disease duration (months)	24 (15–30)	18 (12–24)	0.120
UPDRS	17.5 (14–22)	15 (10–24)	0.776
LED (mg)	0 (0–126)	0 (0–0)	0.194

BMI = body mass index; UPDRS = Unified Parkinson's Disease Rating Scale; LED = levodopa equivalent dose.

Table 2
VPSG findings of the HJs group and the non-HJs group at T0. Data are expressed as median (25th–75th percentile).

	HJs group (16pts)	Non-HJs group (50pts)	*p*-value
TST (min)*	224 (121.25–280.25)	262.25 (227.5–310)	0.047
WASO (min) (n.v. <30)*	169.25 (117.25–224.5)	111 (83.5–149)	0.020
SE (%) (n.v. >85)*	55.93 (34.05–64.9)	66.77 (58.48–75.13)	0.016
Sleep latency (min) (n.v. < 30)	18 (9.75–29.25)	12 (7.5–24.5)	0.185
REM latency (min) (n.v. 60–90)*	199.5 (69–291)	85.25 (56.5–186.5)	0.042
Stage N1 (% of TST) (n.v. 3–8)	7.45 (3.3–10.17)	4.88 (3.07–7.86)	0.113
Stage N2 (% of TST) (n.v. 45–55)	46.10 (37.85–50.23)	46.08 (37.27–54.39)	0.482
Stage N3 (% of TST) (n.v. 15–23)	29.6 (22.07–33.82)	28.47 (21.39–34.59)	1
Stage REM (% of TST) (n.v. 20–25)	17.81 (12.92–23.55)	18.16 (11.19–24.60)	0.770
AHI (n.v. < 10)	0.92 (0–4.78)	0.25 (0–1.87)	0.354
Arousal Index (n.v. < 10)*	8.80 (4.7–13.51)	13.50 (9.63–17.1)	0.034
PLM Index	21.46 (13.83–97.43)	16.53 (5.4–33.44)	0.120
PLMS Index (n.v. < 15)	26.12 (11.63–96.13)	13.14 (3.12–36.58)	0.084

TST = total sleep time; WASO = wake after sleep onset; SE = sleep efficiency; NREM = non-rapid eye movement; REM = rapid eye movement; AHI = apnea/hypopnea index; PLM = periodic limb movements; PLMS = periodic limb movements during sleep.
Uncorrected results. After Bonferroni correction no statistical significance was detected between the two groups.