Original Article

Alterations in Polysomnographic (PSG) profile in drug-naïve Parkinson's disease

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Abstract

Objective: We studied the changes in Polysomnographic (PSG) profile in drug-naïve patients of Parkinson's disease (PD) who underwent evaluation with sleep overnight PSG. **Materials and Methods:** This prospective study included 30 with newly diagnosed levodopa-naïve patients with PD, fulfilling the UK-PD society brain bank clinical diagnostic criteria (M:F = 25:5; age: 57.2 ± 10.7 years). The disease severity scales and sleep related questionnaires were administered, and then patients were subjected to overnight PSG. **Results:** The mean duration of illness was 9.7 ± 9.5 months. The mean Hoehn and Yahr stage was 1.8 ± 0.4. The mean Unified Parkinson's Disease Rating Scale (UPDRS) motor score improved from 27.7 ± 9.2 to 17.5 ± 8.9 with sustained usage of levodopa. Nocturnal sleep as assessed by Pittsburgh Sleep Quality Index (PSQI) was impaired in 10 (33.3%) patients (mean PSQI score: 5.1 ± 3.1). Excessive day time somnolence was recorded in three patients with Epworth Sleepiness Scale (ESS) score ≥ 10 (mean ESS score: 4.0 ± 3.4). PSG analysis revealed that poor sleep efficiency of <85% was present in 86.7% of patients (mean: 68.3 ± 21.3%). The latencies to sleep onset (mean: 49.8 ± 67.0 minutes) and stage 2 sleep (36.5 ± 13.1%) were prolonged while slow wave sleep was shortened. Respiration during sleep was significantly impaired in which 43.3% had impaired apnoea hyperpnoea index (AHI) ≥5, mean AHI: 8.3 ± 12.1). Apnoeic episodes were predominantly obstructive (obstructive sleep apnea, OSA index = 2.2 ± 5.1). These patients had periodic leg movement (PLM) disorder (56.7% had PLM index of 5 or more, mean PLMI: 27.53 ± 4 9.05) that resulted in excessive daytime somnolence. **Conclusions:** To conclude, sleep macro-architecture is altered in frequently and variably in levodopa-naïve patients of PD and the alterations are possibly due to disease process per se.

Key Words

Drug-naïve PD, sleep disorders, Parkinson's disease, polysomnography, questionnaire study

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Introduction

Sleep disturbances in Parkinson's disease (PD) have been mentioned since the first descriptions by James Parkinson's Essay on the Shaking Palsy in 1817, but only recently they have become the subject of attention.[1] Sleep abnormalities in PD is possibly due to progressive sleep restructuring.[2] Sleep disorders seen in PD include insomnia, hypersomnia, and parasomnia. Sleep maintenance insomnia results in sleep fragmentation throughout the night. The alerting effect of levodopa contributes to early insomnia and depressive symptoms to late insomnia. Hypersomnia manifests as excessive day time sleepiness (EDS) in PD. A community-based survey showed that nearly two-third of PD patients reported nocturnal sleep disorders and 15.5% experienced EDS. The most common nocturnal sleep disorders reported were frequent awakening (sleep fragmentation) and early awakening. The patients with EDS were more likely to have an advanced stage of disease, be more disabled, been using levodopa for a longer time and showed a higher frequency of cognitive decline compared with the patients without daytime somnolence.[3,4] Parasomnia that occur most frequently in PD is rapid eye movement (REM) sleep behavior disorder (RBD)[5,6] which can sometimes precede the onset of PD by a mean of 3.7 years.[7]

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Sleep macro-architecture have been extensively studied in PD, but most of these are confined to advanced PD. Polysomonography (PSG) based evaluation of sleep in newly diagnosed untreated patients of PD are far and few.[8,9] Hence, We studied the alterations in PSG profile in drug-naïve patients of PD who underwent evaluation with sleep overnight PSG.

Materials and Methods

This prospective, cross-sectional study was carried out in the Department of Neurology at the National Institute of Mental Health and Neurosciences, (NIMHANS), Bangalore from January, 2008Deccember, 2010. Thirty patients with newly diagnosed PD, who have never previously received levodopa, who fulfilled the UK Parkinson's Disease Society Brain Bank Clinical Diagnostic Criteria,[10] and who met the inclusion criteria, were recruited. Response to levodopa was noted in all after the initial evaluation for the study. The study was approved by the institutional ethic committee and written informed consent was obtained from the patients for their participation in the study.

Study details

The demographic and phenotypic details including age at onset of illness, duration of illness, presenting symptoms and signs, other relevant past and family history were enquired and a detailed neurological examination was done and the same recorded. The PD severity was assessed using modified Hoehn and Yahr staging (H and Y); Schwab and England activities of daily living (S and E ADL) scale; and Unified Parkinson's disease rating scale (UPDRS). Anxiety, depression and fatigue were screened by administering Hamilton anxiety rating Scale (HARS); Hamilton depression rating scale (HDRS); and Fatigue severity scale (FSS) and Parkinson's disease fatigue scale (PDFS). Sleep was assessed using following sleep questionnaire instruments: Epworth Sleepiness Scale (ESS) and Pittsburgh Sleep Quality Index (PSQI), and NIMHANS comprehensive sleep disorder questionnaire (NCSDQ).[11]

Overnight PSG recording

Patients consenting for the study were subjected to overnight sleep study during which various parameters like Electroencephalography (EEG), Electrooculography (EOG), Electromyography (EMG), Electrocardiography (ECG), oxygen saturations, respiratory effort, airflow, body position were monitored. The participants underwent a full overnight PSG from 10 pm to 6 am. PSG included an EEG with 4 channels used to score sleep, an EMG on the chin and right anterior tibialis, an EOG, to record eye movements, an ECG, a nasal airflow sensor, chest and abdomen belts, a pulse oximeter, a snore microphone and body sensors. Total sleep time (TST), sleep efficiency, sleep latency, duration of individual sleep stages, arousals, isolated and periodic movements, snore and respiratory events were scored. Sleep macroarchitecture analysis was carried out. The indices for the obstructive apnea, central apnea, mixed apnea, hypopnea, apnea-hypopnea, periodic limb movement (PLM), arousal, snore related arousals, average SaO2 in sleep (%), desaturation and average heart rate in sleep were calculated. The scoring of sleep was done according to the rules and technical specification set forward by American Academy of Sleep Medicine (AASM) Manual for the scoring of sleep and associated events. The PSG technologists were on continuous duty during the PSG recording. Subsequently, the PSG was evaluated page by page by the investigators (SPJ,SS) with special emphasis on any marked/reported events.

Statistical analysis

Statistical analysis was done using the Statistical Package for Social Sciences (SPSS). Categorical and continuous data were examined for frequencies, characteristics and distribution. Continuous data was further explored to check the normality. Normally distributed parametric data was expressed as mean ± SD. Categorical data was compared using chi-square test. Pearson's and Spearman's correlation coefficients were used where appropriate. A P-value ≤ 0.05 was deemed significant.

Result

Thirty patients (M:F = 23:7; mean age: 57.2 ± 10.7 years) were evaluated with demographic data, clinical phenotype, sleep assessment with PSG. The phenotypic characteristics of the cohort are provided in [Table 1]. The mean UPDRS motor score of this cohort was 27.7 ± 9.2 at baseline. Responsiveness to levodopa was assessed and the following UPDRS motor score was obtained in OFF state 31.1 ± 9.6 and ON state 17.5 ± 8.9, respectively.

Disturbed sleep was noted in 18 patients: Difficulty falling asleep in 12, frequent awakening after falling asleep in17 and early morning arousal in 8, increased sleep in 2, abnormal behaviour during sleep in 2 and feeling of crawling, aching of legs with an inability to keep the legs still while falling asleep in 5 of the patients. Nocturnal sleep as assessed by PSQI was impaired in 10 (33.3%) patients. The mean PSQI score was 5.1 ± 3.1. Four patients with ESS score ≥8 while 3 had ≥10. The mean ESS for the study was 4.0 ± 3.4. On correlating the phenotype with the ESS and PSQI scores, patients with higher score on Schwab and England Activities of Daily living scale had more frequent disturbed nocturnal sleep (P = 0.02) and daytime sleepiness (P = 0.01). Patients with higher scores on depression scales had greater disturbance of nocturnal sleep PSQI scores (P = 0.001). None of the other parameters assessed showed significant correlation.

Assessment with overnight PSG

The PSG parameters are provided in [Table 2]. There was definite reduction in sleep efficiency. There was delay in sleep

Table 1: Phenotypic characteristics of the cohort (n = 30)

Parameters	Mean ± SD (range)
Gender (M:F)	23:7
Age (years)	57.2±10.7
Mean duration of PD (months) (range)	9.7±9.5 (3-48)
Mean UPDRS (motor) (range)	27.7±9.2 (11-45)
Mean Hoehn andYahr stage (range)	1.8±0.4 (1-2.5)
Mean BMI (Kg/ sq. m)	21.7±3.3 (16.8-29.3)
Mean Neck size (cm)	36.2±2.1 (29-39.5)
Mean Schwab and England activities of daily living (range)	77.9±10.1 (50-90)
Mean PSQI (range)	5.1±3.1 (2-14)
Mean ESS (range)	4±3.4 (1-15)
Mean HAMA (range)	5.6±2.7 (1-12)
Mean HDRS (range)	7.0±3.2 (2-16)

*Pearson's correlation coefficient, UPDRS = Unified parkinson's disease rating scale, S and E ADL = Schwab and england activities of daily living, PSQI = Pittsburgh sleep quality index, ESS = Epworth sleepiness scale, HAMA = Hamilton anxiety rating scale, HDRS = Hamilton depression rating scale, FSS = Fatigue severity scale, PDFS = Parkinson's fatigue scale

onset, that is, increased sleep onset and stage 2 (N2) latencies. There was slight reduction is stage 2/N2 and stage 3 + 4/N3 sleep duration. The REM latency was also increased to a mean of 166.5 ± 101.7 minutes.

Patients had respiratory events during sleep, their apnea-hypopnea index (AHI) was 8.3 ± 12.1 (0-56.6) and that associated with desaturation was 9.1 ± 27.9. The number of obstructive apnea events/ indices exceeded the central apnea events/ indices. Thirteen out of 30 patients had an abnormal AHI of ≥5.

They had isolated events or those occurring periodically, that is, PLM. Patients in this cohort had a mean periodic leg movement (PLM) index of 27.5 ± 49.05, with 17 of 30 patients having an abnormal PLM index of >5. The PLM index associated with arousal was 2.09 ± 3.3 and that associated with apnea was 0.48 ± 1.1. There was no feature of REM sleep behavior disorder (RBD or REM without atonia during the overnight PSG recording.

The mean arousal index was 24.3 ± 28.6. The arousal associated with respiratory events was 1.9 ± 4.1, and the snore arousal was 0.8 ± 1.4. Arousal events either isolated or associated with other events were more common in non-rapir eye movement (NREM) sleep except snore arousal but none were statistically significant.

The patients had maintained an average saturation of 95.4 ± 2.2 during the course of the sleep. The mean desaturation events per hour were 15.97 ± 20.92. The mean duration of the desaturation was 33.2 ± 38.2 seconds. The greatest percentage desaturation was 6.2 ± 3.1. The average heart rate of patients during sleep was 67.2 ± 9.2. The heart rate during NREM sleep was slightly less than that during REM sleep but without significance. The total number of tachycardia and bradycardia were 8.6 ± 26.4 and 5.4 ± 29.2 respectively [Figure 1].

Discussion

Sleep disturbances in PD are variably attributed to disease progression and to dopaminergic treatment. Evaluation of patients with PD before starting treatment and dividing them into two groups: a) levodopa vs. b) placebo would be ideal to study the role of disease vs. levodopa in sleep disorder. However, would be inappropriate to withhold medication. Hence, the present study was initiated to characterize the PSG sleep profile in newly diagnosed levodopa-naïve patients with PD.

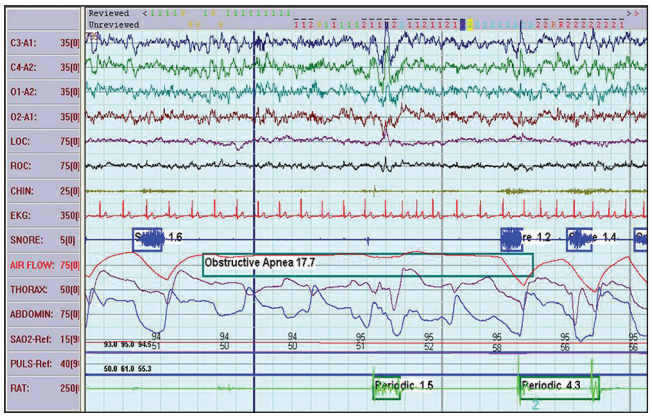

Figure 1: Obstructive apnea, the above is a 30 sec epoch showing a drop in the peak thermo sensor excursion by ≥90% of baseline with the duration of the event lasting at least 10 sec as the apnea is associated with continued or increased inspiratory effort throughout the entire period of absent airflow. Periodic leg movement (PLM) is also depicted

Table 2: Sleep macrostructure and events during PSG study of patients with Idiopathic Parkinson's disease (*n* = 30)

Parameter	Duration Mean ± SD (Range)	Percentage
Sleep efficiency (%)	–	68.3±21.3 (19.8-88.3)
Latency to sleep onset (min)	49.85±67.04 (0-348)	–
Duration of Stage 1 (min)	64.33±41.07 (8-172)	14.44±7.62 (4.1-33.3)
Latency to Stage 1/N1 (min)	53.02±67.34 (0-348.5)	–
Duration of Stage 2/N2 (min)	157.8±71.57 (21-310.5)	36.57±13.07 (8.2-61)
Duration of Stage 3and4/N3 (min)	20.8±20.6	5.08±4.9 (1.5-17.5)
Duration of REM Stage (min)	47.18±29.51 (3-114.5)	11.2±6.44 (1.3-26.3)
Latency to REM Stage (min)	166.48±101.68 (8– 351.5)	
Obstructive apnea Index**	2.23±5.1 (0-24)*	4.53±19.37 (0-104)
Central apnea Index	1.33±2.57 (0-13.4)	0.8±2.35 (0-12)
Mixed apnea Index	0.48±0.91 (0-4.2)	1±2.77 (0-11)
Hypopnea Index	4.23±5.59 (0-26.2)	2.77±7.28 (0-37)
Apnea Hypopnea Index	8.27±12.07 (0-56.6)	9.1±27.96 (0-135)
Periodic movement Index	27.53±49.05 (0-236.8)	Periodic movement Index
Arousal Index	24.3±28.58 (0.2-134.9)	Arousal Index
Snore Arousals Index	0.83±1.45 (0-5.4)	Snore Arousals Index
Average SaO2 in sleep (%)	95.41±2.20 (92-100)	Average SaO2 in sleep (%)
Desaturation Index	15.97±20.92 (0-71.6)	Desaturation Index
Average heart rate in sleep	67.16±9.17 (51-91)	Average heart rate in sleep

PSG = Polysomnographic, REM = Rapid eye movement

Sleep stages

In healthy adult population time spent in stage one sleep increases and Stage 3 and 4 sleep decreases with age. The duration of REM sleep and its latency also tend to decrease with advancing age.[12] Previous PSG studies in advanced PD patients have shown that sleep is fragmented, with increased periods of wakefulness, increased sleep latency and reduced sleep efficiency. It was also observed that stages of light sleep increased compared to slow wave sleep and REM sleep in these patients.[1,13,14] Sleep efficiency of <85% is considered poor sleeper.[15] The patients of early drug-naïve PD in the present study also had reduced sleep efficiency with mean values (86.7%) corresponding to previous studies in similar group (69.2 ± 13.1%), and 72.1 ± 17.0%.[8,9] The present study, had reduced total sleep time with patients spending 58.0% of the time in bed awake. The patient slept onset close to an hour after the lights were put out (49.8 ± 67.0 minutes). Half of them had sleep onset latency of >30 min. The other investigators also observed prolongation of sleep latency to 30.7 ± 19.1 and 32.6 ± 21.5.[8,9] These values are significantly prolonged when compared to studies in normal elderly adults in the community.[16] A comparative table with other similar studies is provided [Table 3].

Sleep disordered breathing (SDB) in patients with drug-naïve PD

Sleep disordered breathing has been observed in PD.[17] In the present study, 43.3% of patients had AHI ≥5. The mean AHI score and the number of patients suffering from SDB was less in patients with drug-naïve/early PD compared to that in advanced PD on treatment indicting a milder SDB in the former.[17]

In PD, the most common subtype of apnea observed is the obstructive, 90% of total apnea.[18] The most common subtype of SDB observed in our study of drug-naïve early PD was of obstructive variety, OSA was 69.3% and CSA was 30.7% of total apnoea events. In a previous study in drug-naïve patients the average number of obstructive apnea reported was 23.7 ± 6.4 compared to 17.0 ± 10.4 central sleep apnea.[8]

Periodic leg movements in sleep in drug-naïve PD

Periodic leg movements in sleep (PLMS) is a nocturnal non-motor symptom which impairs sleep in advanced PD. PSG carried out previously in a few drug-naïve PD patients who complained of significant sleep disturbance had documented PLMS.[19] The present study has observed a PLM index of 27.53 ± 49.05 among early drug-naïve PD patients. A PLM index of ≥ 5 was noted in 56.7% of patients. Other studies in drug-naïve PD have shown high PLMI of 22.02 ± 36 and 55.4 ± 34.7.[8,9] PLM cause arousal and contribute to the disturbed nocturnal sleep in patients with PD. The PLM-arousal Index in the present study was 2.1 ± 3.3, compared to 7.0 ± 5.4 and 45.7 ± 27.9.[8,9]

REM sleep behaviour disorder (RBD) in drug-naïve PD

Patients of PD have loss of atonia during the REM sleep and this can be associated with acting out of dreams with violent movements of the limbs during sleep resulting in injury to self and to partner, which is termed REM behaviour disorder (RBD). RBD occurs in a third of advanced PD patients. RBD was observed in 33% of 33 PD patients with a mean disease duration of 7.7 ± 5.8 years and mean H and Y stage of 2.2 ± 0.8,[20] and 30% of 10 PD patients with mean disease duration of 5.5 ± 4.2 years and mean H and Y stage of 2.2 ± 0.6.[8] Previous studies in drug-naïve early PD patients observed and incidence of RBD of 6.6% (one in 15 patients) similar to our observation of 3.3% (one in 30 patients).[9]

Arousals in patients with drug-naïve idiopathic PD

The significance of arousal indices is that it is a direct measure of the sleep disturbance in patients with PD. The arousal could be associated with respiratory events, snore or periodic leg movements. The total arousal index was 24.3 ± 28.6, arousal associated with PLMS was 2.3 ± 3.6 in the present study. Previous study in untreated early PD reported number of awakening as 11.1 ± 2.8 and PLM associated arousal index as 7.0 ± 5.2.[9]

Oxygen saturation and desaturation indices in patients with drug-naïve PD

The mean desaturation index was 15.9 ± 20.9 events per hour. The mean duration of desaturation was 33.2 ± 38.2 sec, out of

Table 3: Comparison of sleep parameters in various studies on drug-naïve PD

Parameters	Wetter *et al*., 2000 (Ref 8)	Kaynak *et al*., 2005 (Ref 9)	Present study, 2012
Study subjects	10 drug-naïve PD	15 drug-naïve PD	30 drug-naïve PD
Age (years)	65.2±5.6	65.4±10.4	57.2±10.6
Duration of illness	66.0±4.2 months	8±3.1 months	13.8±5.4 months
UPDRS (motor)	20.4±6.3	28.2±12.4	28.6±9.02
Mean H and Y* stage	2.2±0.6	2.2±0.8	1.8±0.4
Sleep efficiency (%)	72.1±17.0	69.2±13.1	68.3±21.3
Sleep onset latency (min)	32.6±21.5	30.7±19.1	49.8±67.0
REM latency (min)	126.7±78.8	92±74.5	166.4±101.6
Stage 1/N1 (%SPT)	9.3±2.6	3.6±1	14.4±7.6
Stage 2/N2 (% SPT)	41.3±13.6	42±11.6	36.5±13.1
SWS/N3 (% SPT)	2.8±3.1	16.5±7.5	4.9±4.9
REM (% SPT)	11.5±4.9	13.4±4.1	11.2±6.4
PLMS index	68.3±46.7	20.2±36	27.5±49.1

PD = Parkinson's disease, REM = Rapid eye movement, UPDRS = Unified parkinson's disease rating scale, PLMS = Periodic leg movements in sleep, SWS = Slow wave sleep, SPT = Sleep period time

which the longest desaturation was 321.1 sec and the greatest desaturation was 6.2 ± 3.2% form baseline. Patients with PD have episodes of upper respiratory tract obstruction during sleep manifesting as OSA. Desaturation events can also be explained by the same mechanism.

Heart rate and its variability in patients with drug-naïve idiopathic PD

The average heart rate of the patients with PD during sleep was 67.2 ± 9.2, and during the awake state was 73.2 ± 9.2. During the overnight PSG record 5 patients were noted to have tachycardia and 2 patients were noted to have bradycardia. Previous studies on drug-naïve early and advanced PD had documented presence of dysautonomia with or without orthostatic hypotension.[8,9] Pathologic and clinical studies of autonomic pathways have expanded the concept of PD from a movement disorder to a multi-level widespread neurodegenerative process with non-motor features spanning several organ systems.[21]

Comparison with matched healthy controls would have provided better information regarding the true prevalence of sleep alterations in PD. During PSG, patients are exposed to a new environment and may have "first night effect" on sleep parameters studied. The sample size was relatively small and hence limits the conclusions from this data. The present study included drug-naïve patients of PD, a cohort that is very difficult to obtain. The second challenge was in scheduling and performing PSG as early as possible once the diagnosis is established. Presence of abnormalities in sleep parameters in a significant proportion of levodopa-naïve patients of PD suggested that these are due to PD itself.

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