

Sleep Disturbances in Parkinson's Disease

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Abstract

Background: Sleep disturbances are one of the most common of the nonmotor complications of Parkinson's disease (PD), and increase in frequency with advancing disease **Objective:** The aim of this study is to assess the nature of sleep disturbance in PD and to correlate the PSG variables with the clinical findings and several sleep scales **Methods:** Thirty patients with PD and 10 age and sex matched controls were subjected to full neurological examination, clinical scales (UPDRS, H&Y, MMSE, Beck Depression Scale), sleep scales (PDSS & ESS), laboratory tests and polysomnographic assessment. **Results:** Nocturia is the most common symptom (61%) in patients. Patients had significantly lower sleep efficacy and higher no. of awakening and AHI in comparison to controls. Females had significantly better polysomnographic (PSG) results in comparison to males ($p < 0.05$). Patients with bradykinesia had significantly worse PSG results in comparison to those with tremors. Age, bradykinesia and L-Dopa dose is correlated significantly AHI and PSG parameters. Sleep scores is significantly correlated with PLM index ($p < 0.05$). **Conclusion:** These sleep disturbances in PD are common and multifactorial in nature. Some may result from the disease and may be the effects of medications. Early detection and management are important to improve the patient quality of life. (*Egypt J Neurol Psychiat Neurosurg.* 2010; 47(1): 197-205)

Key Words: Parkinson's disease, sleep disturbance, polysomnography, AHI, ESS, PDSS, PLM.

INTRODUCTION

Sleep disturbances in Parkinson's disease (PD) were noted in its original description¹. Recently they have become the subject of increased attention being common, often severe, typically under-recognized and ineffectively treated².

The incidence of sleep complaints ranged from 60-90% in PD patients^{3,4}. These complaints included insomnia, nighttime awakenings, difficulty in changing position, pain, stiffness, nightmares and restless legs symptoms⁵. It was found that even PD patients with recently diagnosed untreated disease, have more sleep disturbances than age-matched controls⁶. Sleep disorders commonly implicated in such complaints are nocturnal motor dysfunctions as nocturnal akathisia and periodic limb movements, sleep apnea, anxiety disorders and REM sleep behavior disorder⁶. Abnormal REM sleep features were found in up to 40 % of PD patients and the prevalence seems to increase with longer disease duration⁷.

The underlying causes for sleep disorders in PD patients are still discussed controversially. Potential causes include the neurodegenerative process of PD, nocturnal bradykinesia and rigidity, psychiatric disorders and circadian rhythm disturbances⁸.

Dopaminergic medications can cause additional sleep problems. High doses of levodopa taken in the evening or at bed time substantially increase nocturnal time awake, decrease REM sleep, and can contribute to nocturnal hallucinations and confusions⁹. In one series of PD patients treated with levodopa, 74% described sleep problems¹⁰. In another study by Factor et al.¹¹, 67% of patients complained of problems initiating sleep and 88% complained from impaired sleep maintenance.

Polysomnography (PSG) can serve to identify some of the potential causes of sleep disorders, such as abnormal motor activity during sleep; sleep related breathing disorders and REM behavior disorder¹². Precise diagnosis of these disorders is important because the significance and treatment of these various disorders are often quite different⁶.

The aim of this study is to conduct a comprehensive research of sleep disturbances in parkinsonian patients. A Search for the possible causes of such disturbances, if present, will be conducted. Furthermore, we aim to correlate the various PSG variables with the clinical findings and several sleep scales and to assess the difference between L-dopa treated patients and non-treated patients.

SUBJECTS AND METHODS

A) Subjects:

This study included 30 patients with idiopathic Parkinson's disease (PD). They were 11 males (36.7%)

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and 19 females (63.3%). Their age ranged from 30 to 71 years with a mean of 58.4 ± 10.95 yr and their body mass index (BMI) ranged from 20-38 with a mean of 24.95 ± 3.52 . A Control group included 10, age and sex matched normal volunteers. They were 4 males (40%) and 6 females (60%). Their age ranged from 32-70 years with a mean of 54.6 ± 8.76 yr and their BMI ranged from 22-28.5 with a mean of 25.1 ± 2.24 . The diagnosis of Idiopathic Parkinson's Disease was based on the UK Parkinson's Disease Society Brain Bank criteria for clinical diagnosis¹³, which include the following three criteria:

- Bradykinesia plus one of the following: rigidity, tremor, or postural instability.
- At least three of the following: rest tremor, progressive symptoms, unilateral onset, early response to levodopa, Levodopa-induced dyskinesia.
- No identifiable cause for the parkinsonism.

Excluded from the study patients with (1) features atypical for idiopathic parkinsonism such as pyramidal manifestations, cerebellar signs or prominent autonomic dysfunction, history of toxic exposure, head injury, encephalitis or cerebrovascular disease and depression, (2) patients with a body mass index (BMI) >30, (3) Mini Mental State Examination (MMSE) < 24, (4) Presence of depression, (5) Patients on medications influencing sleep such as benzodiazepines or barbiturates.

Patients were divided according to their antiparkinsonian medications into two subgroups; subgroup I (15 patients) were untreated and subgroup II (15 patients) were on L-Dopa treatment.

B) Methods:

All patients underwent thorough clinical evaluation and neurological examination. The following batteries of assessments were also carried out:

- 1- **The Unified Parkinson Disease Rating scale (UPDRS)** the scale ranges from 0 to 199 points; 199 represents the worst (total) disability while 0 represents no disability.¹⁴
- 2- **The modified Hoehn and Yahr scale** It assesses the patient's stage (from stage 0: no signs of Parkinson's disease, to stage 5: wheelchair bound or bedridden unless aided)¹⁵.
- 3- **Parkinson's Disease Sleep Scale (PDSS):** It is used for evaluation of sleep disturbances in Parkinson's disease. It quantifies the various aspects of nocturnal sleep problems using a visual analogue scale addressing 15 commonly reported symptoms associated with sleep disturbance in Parkinson's disease. Individual item score below 6 or a total score below 90 are considered abnormal¹⁶.

- 4- **The Epworth Sleepiness Scale:** It is used to quantify the level of daytime sleepiness¹⁷. A score of 10 or more is considered sleepy.
- 5- **Mini Mental State Examination (MMSE):** It was used to exclude patients with dementia¹⁸.
- 6- **Beck Depression Questionnaire:** It was used to exclude patients with depression¹⁹.
- 7- **Laboratory tests:** Fasting and 2 hours post prandial blood sugar level, liver and kidney function tests were carried out to exclude diabetic patients or patients with liver or kidneys diseases.
- 8- **Neuroradiological tests:** Computerized topography (CT) and/or Magnetic Resonance Imaging (MRI) of the brain were carried out to exclude other causes of parkinsonism.
- 9- **Polysomnography:** A full night polysomnography recording was carried out using **Shwarzer Epos 32 GmpH amplifier (medical diagnostic equipment polysomnogram, Shwarzer, Germany) and Somnologica software.** During the PSG the following electrophysiological data are recorded:
 - Continuous EEG from four channels: C3/A2, C4/A1, O1/A2 and O2/A1. Locations were designated according to the International 10-20 System.
 - Continuous electro-oculogram (EOG) using left outer canthus, (LOC) and right outer canthus (ROC) electrodes referenced to the contralateral mastoid.
 - Continuous submental surface electromyogram (EMG).
 - Continuous EMG of the right and left tibialis anterior muscles.
 - To measure respiratory effort, two elastic belts containing stretch-sensitive piezo crystals were placed around the thorax and abdomen.
 - Airflow was measured using a cannula connected to a transducer to provide a qualitative measure of the patient's airflow.
 - A peripheral blood-oxygen (SaO2) probe was placed on the patient's finger to measure oxygen saturation.
 - Two ECG electrodes on the chest.
 - A snoring sensor consisting of a minute microphone was placed on the anterior superior portion of the neck.

The polysomnography was scored visually according to the standardized Rechtschaffen and Kales²⁰ criteria. The following sleep variables were scored for each subject:

1. **Total sleep time (TST).**
2. **Sleep Efficacy** ([TST/Time in bed] X 100).

3. **Percentage of total sleep time in each stage** (S1, SII, SIII, SIV & REM).
4. **Number of awakenings.**
5. **Apnea/Hypopnea Index (AHI).** Apneas were defined as cessation of the airflow lasting more than 10 seconds. Hypopneas were defined as airflow reduction of more than 30% lasting than 10 seconds and associated with an oxygen desaturation of at least 4% with or without an arousal²¹. AHI between 0-5/h is normal; 5.1-15 is mild, 15.1-30/hr moderate and more than 30/hr is severe.
6. **Periodic Limb Movement (PLM) Index:** PLMs were scored according to the revised International Classification of Sleep Disorders²².

Statistical Analysis

All data are presented as mean and standard deviation. Descriptive statistics were used for parametric data and for categorical data. Two-tailed Student's t-tests were used for all parametric data when comparing between two groups⁽²³⁾. Correlations between the various scales, polysomnographic results and clinical variables were done using Pearson correlation coefficient. Results are presented with p-values. A *P-value* <0.05 was considered statistically significant. All tests were performed using SPSS 10 for windows. Graphical presentations have been performed using MS Excel

RESULTS

I. Clinical results:

The duration of illness ranged from 2 months to 10 years with mean of 2.27 ±3.17 yr. according to the predominant manifestation; 18 (60%) had tremors mainly and 12 (40%) had mainly bradykinesia and rigidity. Eleven patients (36.7%) had sleep complaint while 19 (63.3%) had no sleep complaint. Fifteen patients (50%) were untreated (group I) and 15 (50%) were on L-Dopa treatment (group II). The dose of L-dopa in Group II ranged from 250mg-750mg/day with a mean of 412.5±144.95mg/day and the duration of treatment ranged from 1month-10 years with a mean of 3±3.64yr.

II. Clinical scales:

- 1) **Unified Parkinson Disease Rating scale (UPDRS):** it ranged from 15-85 with a mean of 48.7 ±18.7.
- 2) **The modified Hoehn and Yahr scale (H&Y):** it ranged from 2-4.5 with a mean of 2.7 ±0.75.
- 3) **The Epworth Sleepiness Scale (ESS):** A score >10 was considered abnormal, accordingly 8 patients (26.7%) showed abnormal results. The

comparison between patients and controls in the range Mean±SD is shown in Table (1).

- 4) **The Parkinson's Disease Sleep Scale (PDSS):** A score less than 90 was considered abnormal, accordingly 19 (63.3%) of patients had abnormal results. The comparison between patients and controls in the range Mean±SD is shown in Table (1).
- 5) **Type of sleep complaint:** the percentage of different types of sleep complaint among 19 patients with abnormal PDSS is shown in Figure (1).

III. Results of polysomnography:

- 1) **Sleep efficacy:** The difference between the patients and controls was statistically significant ($p < 0.05$), as shown in Table (2).
- 2) **Number of awakenings:** In spite that patients had more frequent awakenings in comparison to the controls, the difference between both groups was not statistically significant ($p > 0.05$) as shown in Table (2).
- 3) **Arousal index (AI):** Although the AI was higher in the parkinsonian patients, their results didn't reach a statistically significant value in comparison to controls, as shown in Table (2).
- 4) **Periodic Leg Movement Index:** three of our patients (10%) had pathological PLM i.e. PLM index > 5/hr, while none of the control subjects did. However, no statistically significant difference was found in the mean values between the two groups, as shown in Table (2).
- 5) **Apnea/Hypopnea Index (AHI):** 20 patient (66.7%) had normal values, 1(3.3%) had mild A/H, 8(26.7%) had moderate degree and 1(3.3%) had severe A/H. The AH index was considerably higher in the PD patients in comparison to controls and the difference was statistically significant ($p = 0.01$), as shown in Table (2)
- 6) **Sleep Stages:** The percent for each sleep stage of total sleep time in patients and control group is shown in Table (2).

IV. Comparisons:

- 1) **Comparison between treated and untreated patients:** No statistically significant difference was detected between group I and group II in either the sleep scales or the polysomnographic results ($p > 0.05$).
- 2) **Comparison between patients with tremors and bradykinesia:** in sleep scales and polysomnographic results is shown in Table (3).
- 3) **Comparison between males and females:** is shown in Table (3).

IV. Correlation of polysomnographic results:

The correlation between polysomnographic results and clinical data (age, tremors and bradykinesia grade and L-Dopa dose) is shown in Table (4). No statistically significant correlation was found between the duration of illness and polysomnographic results. The correlation between polysomnographic results and the clinical and sleep scales (UPDRS, H&Y, PDSS and ESS) is shown in Table (5).

V. Correlation of clinical scores:

There is a statistically significant negative correlation between bradykinesia severity and PDSS scores ($p=0.001$). A statistically significant positive correlation was found between the duration of illness and ESS ($p=0.025$). Also, there was a statistically significant negative correlation between the total score of UPDRS and those of the PDSS ($p=0.002$). No significant relation was found between the dose of L-Dopa and the sleep scales ($p>0.05$).

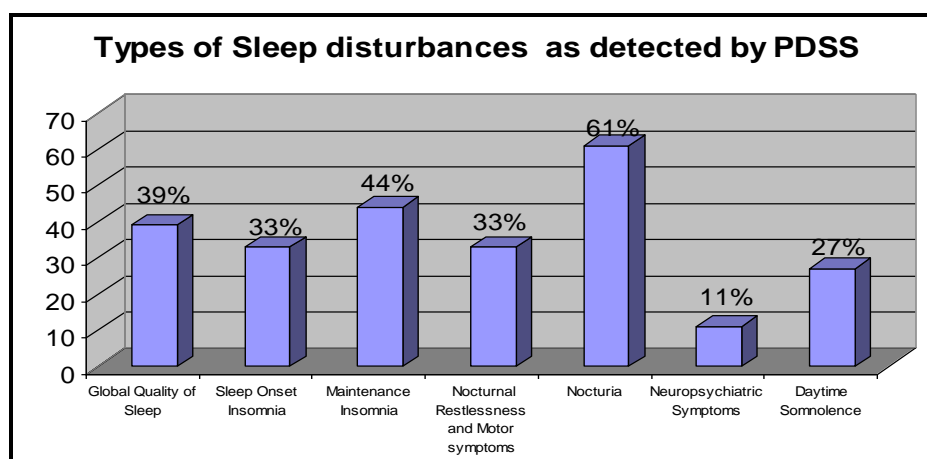


Figure 1. The type of sleep complaints among 19 patients with abnormal PDSS.

Table 1. The range and Mean \pm SD of ESS and PDSS of patients (30) and controls (10).

variable	Patients (30)		Controls (10)		P-value
	Range	Mean \pm SD	Range	Mean \pm SD	
ESS	2-22	7.0 \pm 4.75	0-5	2.0 \pm 1.22	0.003**
PDSS	43-124	83.6 \pm 36.33	135-150	144.8 \pm 5.68	0.000**

ESS=Epworth sleepiness scale PDSS= Parkinson's Disease Sleep Scale *Highly significant

Table 2. The results of polysomnography in patients and control subjects.

Polysomnographic findings	Patients (30)		Controls (10)		P-value
	Range	Mean \pm SD	Range	Mean \pm SD	
Sleep efficacy	34.8-80.5	55.8 \pm 15.4	26.8-92	64.1 \pm 12.5	0.049*
No of awakening	12-28	17.2 \pm 4.1	7-20	13.3 \pm 3.9	0.023*
Arousal index	2.6-28.7	9.99 \pm 7.3	1.6-20.1	6.86 \pm 6.3	0.301
PLM index	0.7-10.7	1.0 \pm 2.6	0.8-1.7	0.25 \pm 0.56	0.370
AHI	0.5-32.4	14.1 \pm 10.7	0.0-21.7	6.36 \pm 8.44	0.010**
Sleep stage%	6.12-20.4	13.26 \pm 14.79	5.82-29.5	17.67 \pm 15.42	0.473
% of TST in S1	37.7-53.6	45.64 \pm 16.46	39.1-55.3	47.2 \pm 10.53	0.799
% of TST in S2	6.49-26.6	16.58 \pm 20.95	4.92-15.1	10.05 \pm 6.66	0.370
% of TST in S3	9.29-23.3	16.3 \pm 13.15	9.01-18.1	13.55 \pm 4.31	0.620
% of TST in S4	11.8-20.6	16.21 \pm 8.77	8.99-19.7	14.35 \pm 6.4	0.590
% of TST in REM					

PLM= Periodic Leg Movement AHI= Apnea/Hypopnea Index *Significant **Highly significant
TST=Total Sleep Time, S1=Stage I, S2=Stage II, S3=Stage III, S4=Stage IV.

Table 3. Results of sleep scales and polysomnography in males and females and in patients with tremors and bradykinesia.

Variable	Males	Females	p-value	Tremors	bradykinesia	P-value
	Mean± SD	Mean± SD		Mean± SD	Mean± SD	
PDSS	79.0 ±30.8	86.07 ± 38.7	0.559	80.0 ± 40.4	89.0 ± 28.09	0.444
ESS	8.857 ± 5.9	6.0± 3.6	0.066	6.3± 3.54	8.0± 6.0	0.277
Sleep efficacy	48.86 ±10.9	59.1± 16.0	0.053*	60.31 ±16.2	49.76 ±11.6	0.033*
No. of awakenings	23.16± 9.8	15.0 ±5.7	0.003**	15.63± 5.0	20.25± 10.6	0.042*
% of TST in S1	24.53 ±21.1	8.06± 5.5	0.001**	7.62± 5.84	21.01± 19.1	0.004**
% of TST in S2	49.28± 14.2	43.96± 17.1	0.356	41.79± 14.6	50.95 ±17.2	0.086
% of TST in S3+S4	12.33 ±10.1	34.00 ±14.2.	0.005**	34.40± 13.4	15.0 ±12.85	0.012*
% of TST in REM	13.86 ±6.55	17.38 ±9.43	0.256	18.73± 8.8	13.06±7.5	0.049*
Arousal index	12.86 ±9.33	8.66± 5.72	0.095	8.8± 6.23	11.62 ±8.29	0.239
PLM index	1.62± 3.85	0.68± 1.58	0.270	0.74 ±1.63	1.42 ±3.62	0.423
AHI	26.82± 17.0	8.38 ±10.7	0.001**	9.45±11.63	20.17±17.94	0.049*

ESS=Epworth sleepiness scale PDSS= Parkinson's Disease Sleep Scale PLM= Periodic Leg Movement.
 AHI= Apnea/Hypopnea Index TST=Total Sleep Time, S1=Stage I, S2=Stage II, S3=Stage III, S4=Stage IV.
 *Significant **Highly significant

Table 4. The correlation between polysomnography and clinical data.

Variable	Age		Tremors		Bradykinesia		L-Dopa dose	
	r	p	r	p	r	p	r	p
Sleep efficacy	-0.001	0.997	0.290	0.077	-0.257	0.119	0.661	0.003**
No. of awakenings	0.338	0.038*	-0.247	0.073	0.175	0.294	-0.161	0.523
% of TST in S1	0.056	0.740	0.089	0.594	0.385	0.017*	-0.573	0.013*
% of TST in S2	0.348	0.032*	0.258	0.118	0.188	0.259	0.256	0.305
% of TST in S3+S4	-0.509	0.003**	-0.165	0.322	-0.537	0.001**	0.855	0.000**
% of TST in REM	-0.014	0.937	-0.243	0.154	0.375	0.024*	-0.566	0.014*
Arousal index	0.008	0.600	-0.049	0.373	-0.006	0.971	-0.322	0.192
PLM index	-0.047	0.772	0.221	0.170	-0.004	0.982	0.818	0.000**
AHI	0.373	0.035*	0.151	0.408	0.415	0.018*	0.659	0.003**

*Significant **Highly significant

Table 5. The correlation between polysomnography and the clinical and sleep scales.

Variable	UPDRS		H&Y		PDSS		ESS	
	r	p	r	p	r	p	r	p
Sleep efficacy	-0.181	0.276	-0.026	0.878	0.071	0.672	0.033	0.844
No. of awakenings	0.088	0.600	-0.080	0.634	0.164	0.325	-0.080	0.629
% of TST in S1	0.304	0.063	-0.012	0.945	-0.010	0.922	-0.100	0.536
% of TST in S2	0.068	0.683	0.099	0.552	0.182	0.275	-0.050	0.742
% of TST in S3+S4	-0.468	0.007**	-0.315	0.079	0.113	0.540	0.082	0.657
% of TST in REM	0.007	0.969	-0.142	0.407	-0.290	0.084	0.152	0.377
Arousal index	0.296	0.071	0.198	0.234	0.052	0.757	-0.250	0.122
PLM index	0.119	0.464	0.040	0.808	-0.360	0.022*	0.829	0.000**
AHI	0.037	0.839	0.030	0.899	0.082	0.654	0.158	0.388

TST=Total Sleep Time, S1=Stage I, S2=Stage II, S3=Stage III, S4=Stage IV. REM=rapid eye movement
 PLM= Periodic Leg Movement AHI= Apnea/Hypopnea Index *Significant **Highly significant.

DISCUSSION

Patients with Parkinson's disease commonly have sleep disturbances that significantly alter quality of life but are often under-recognized²⁴. It may be due the disease itself with its underlying immobility, the impact of dopaminergic medication or to a concomitant depression²⁵. The percent of PD patients with sleep complaint in our work was 36.7% (as assessed by the single sleep related question in UPDRS). A higher percentage was found by other authors (60-90%)^{4,5}. This difference may be because we excluded patients with depression and dementia, both factors have high contribution to sleep disturbance in the PD patients.

Nocturia (recurrent awakenings for urination) was the most frequent cause of sleep disturbance in our patients (61%). This is in accordance with several previous studies^{26,27}. This was attributed to incomplete bladder emptying, which probably reflects a high incidence of autonomic dysfunction in PD or it may occur as the dose of L-dopa medication wears off, and therefore a change to a longer-acting form of medication at night may be required²⁸. Sleep Maintenance Insomnia was found in (44%) of our patients followed by sleep onset insomnia (33%), nocturnal restlessness and motor symptoms (33%), daytime somnolence (27%), and neuropsychiatric symptoms (distressing dreams and hallucinations) (11%). These results were in accordance to Singer et al.⁵. Many factors have been implicated as: Nocturnal motor dysfunctions (nocturnal akathisia and PLM), sleep apnea, anxiety disorders, RBD and the effects of medications⁶.

In this work 27% of patients complained of excessive daytime somnolence (EDS) (which is not detected in any control subject). These results were similar to the study of Tandberg and coworkers²⁹ and they suggested that mild daytime sleepiness may be a result of normal aging, whereas more severe EDS can be explained by the neuropathologic changes of PD.

Sleep efficacy, number of awakenings, and AH index were significantly worse in our patients in comparison to controls ($p < 0.05$) and despite that the arousal index and PLM index were higher in patients however the difference was not statistically significant. This goes with previous studies stating that sleep fragmentation is the most common sleep disorder reported by PD patients^{4,30}. On the other hand, Hans et al.³¹, found no significant differences in the conventional sleep parameters between patients with PD and a control group, with the exception of a tendency to a more shallow sleep PD patients.

Pathological PLM (PLM index > 5 /hr) were found in 3 patients (10%) with indices of 5 to 10.5. The same result was found by Arnulf et al.³² and

Wetter et al.³⁰, who reported that PLM affects 15% of patients with PD and they had increased PLMD compared to patients with MSA and healthy controls. They also stated that PLM index is significantly higher in newly diagnosed PD patients, suggesting that it is the neurodegenerative process that cause of PLM⁽³³⁾. Movements in limbs in PD may be favored by the presence of an "off" nocturnal phenomenon as a result of more severe basal ganglia neuronal degeneration³⁴. However, it was argued that the prevalence of PLM in the general population > 65 years and in PD patients is similar³⁵.

Also in our study the influence of age on the sleep stages was strongly significant, as we found that the older the patients the more the tendency to shallow sleep on the expense of deep sleep. Also, age showed a significant positive correlation with the number of awakenings and the A/H index ($p < 0.05$). This is in agreement with previous reports which stated that older adults experience an increase in the number of sleep disruptions⁽³⁶⁾ and an increased incidence of Sleep Disordered Breathing³⁷ and PLM³⁸. A statistically significant correlation was found between bradykinesia severity and sleep disturbance in our patients, both, subjectively (detected by significant negative correlation with the PDSS) and objectively (detected by a significant negative correlation with Total Sleep Time in Stages III & IV).

The present study also showed that there was a significant positive correlation between the duration of illness and the degree of sleepiness (assessed by ESS). This was in agreement with the results of Tan et al.³⁹, who found that a longer duration of the disease and a higher dose of L-dopa were the only significant predictors of Sleep Attacks (SA) in patients with PD who also had much higher ESS scores. On the contrary, our results showed no relation between L-dopa dose and sleepiness ($p > 0.05$). Similar result was found by Arnulf et al.³², who found no correlation between sleepiness and dopamine agonist. They stated that absence of dose-related sleepiness is a strong argument against an effect of these drugs on the mechanisms of sleepiness and suggested that individual characteristics of the patients predominate.

On the other hand, a significant positive correlation was found between the dose of L-dopa and sleep efficacy and % TST in S3+S4, indicating a better quality of sleep with higher doses of L-dopa. These results disagree with Young et al.,⁴⁰ who suggested that drug effects played a greater role in sleep disruption in PD patients than the severity of the disease. A possible explanation for this finding is that medications may treat symptoms during the night and thus improve sleep in some PD patients. In addition, the waning of medication can cause nocturnal akinesia with difficulty turning over in bed and inability to shift positions during sleep⁴¹. Furthermore, in mildly to

moderately affected patients with PD, L-Dopa may cause sleep disruption by their effects on sleep regulation, while in more severely affected patients, the beneficial effects of these drugs on nocturnal disabilities that cause sleep disruption in PD prevail⁴². Also L-dopa dose is significantly correlated with % TST in REM. in our patients. This is in concordance with Hogl et al.⁴³. Several conditions have been suggested as causes of the reduction of REM% such as frequent awakenings due to motor deficits, levodopa, dopaminergic agonists causing hallucinations, selegiline, benzodiazepines, amitriptyline and the first-night effect of the sleep study⁴².

The AHI was significantly correlated with the dose of L-dopa in our work, similar results were obtained by Rice et al.⁴⁴, who found a striking change in respiratory rate after administration of L-dopa, with the emergence of irregular tachypnea alternating with brief periods of apnea, in a pattern consistent with a central origin. The temporal relationship of the respiratory disturbance to the administration of L-dopa suggested a peak-dose drug effect.

We found a significant negative correlation between the UPDRS and the PDSS and %TST in Stages III & IV indicating a strong link between the clinical disability of the patient and his severity of sleep complaint. and indicating a considerable decrease in the time spent in the deep sleep and a more tendency towards a light fragmented sleep with increasing disability. These results are in agreement with Happe et al.²⁵. These findings may suggest that the pathologic process itself is a significant factor in causing disordered sleep in PD. In addition to striatal and mesencephalic dopaminergic depletion, serotonergic neurons of the dorsal raphe, noradrenergic neurons of the locus coeruleus, and cholinergic neurons of the pedunculopontine nucleus are also affected. Each of these neuronal populations is involved in control of the sleep-wake cycle⁴⁵. A significant negative correlation was found between PLM index and PDSS and positive correlation between PLM index and ESS. Thus we can postulate that PLM in PD is another cause of sleep fragmentation in addition to being a potential cause of abnormal DTS⁴⁶. On the contrary, Arnulf et al.³² showed no correlation between PLM and sleepiness.

Our PSG measurement of sleep quality actually shows better preserved sleep architecture in females than males who had significantly higher scores of awakenings, Arousal Index and AHI. The PLM Index was more and their Sleep Efficacy was significantly less than the females. Our study also showed that males complained from EDS or a higher incidence of dozing when assessed by ESS. These results are in agreement with the results of Happe et al.²⁵ and Svenja et al.⁴⁷, who stated that mainly male patients snored and had long pauses between breaths while asleep and

they suffered from more trouble staying awake during the day than the females. This impact of gender on sleep has also been shown in an elderly healthy population, indicating that this is not just a disease-dependent phenomenon⁴⁷.

Patients presented by bradykinesia had a significantly lower Sleep efficacy and %TST in S3+S4, REM and a significantly higher %TST in S1, and AHI and number of awakenings than patients presented by tremors indicating a tendency to shallow sleep on the expense of deep sleep. This can be attributed to immobility with an inability to turn around in bed and stiffness⁴². Arnulf et al.³² suggested that the upper airway dysfunction could cause obstructive apneas in PD patients, possibly because of nocturnal akinesia of upper airway muscles.

In this study there was no statistically significant difference between treated and untreated patients, suggesting that sleep disturbances in PD patients is related to its pathological process than drug related factors. This was in disagreement with Nausieda et al.¹⁰, who found sleep complaints increased in prevalence with longer periods of levodopa therapy and sleep abnormalities tended to increase in severity with continued treatment. However this contradiction may be related to a methodological issue, as in the previous study they assessed sleep disturbances depending on a questionnaire while we used the PSG in our study.

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الملخص العربي

اضطرابات النوم في مرض باركنسون

اضطرابات النوم في مرض باركنسون (الشلل الرعاش) تم ذكرها منذ بدايات وصف المرض، ولقد ازداد مؤخرًا الاهتمام بها نظرًا لخطورة تأثيرها على المريض وتباين الدراسات القديمة والحديثة في تحديد أنواع هذه الاضطرابات وأسبابه. لذا فقد استهدف البحث دراسة الأنواع المختلفة من اضطرابات النوم وعلاقتها بشدة المرض ومدته وجرعة الدواء المحنوي على الدوبامين. ولقد أجريت هذه الدراسة على ثلاثين مريضًا بداء باركنسون ومجموعة ضابطة من عشرة أصحاء من عمر وجنس متقارب من المرضى، ولقد أجريت لهم جميعًا الفحوصات والأبحاث التالية: (1) فحص إكلينيكي شامل للجهاز العصبي. (2) تصني ف لدرجة الإعاقة باستخدام مقياس مرض باركنسون الموحد ومقياس هوهني ويار. (3) دراسة درجة اضطراب النوم باستخدام: مقياس مرض باركنسون للنوم، مقياس ابورث للنعاس، فحص تخطيط النوم المتعدد.

ولقد تلخصت النتائج فيما يلي:

1. أظهرت الدراسة أن 30.6% من مرضى باركنسون الذين لا يعانون من الاكتئاب أو تدهور في الوظائف المعرفية، لهم شكاوى متعلقة بالنوم.
2. اضطرابات النوم كانت أشد في مرضى باركنسون الذي يغلب عليهم بطء الحركة عن الذين يغلب عليهم الارتعاش.
3. لم يوجد فرق واضح بين المرضى الذين ليسوا على علاج والذين هم على علاج الدوبامين من حيث الشكاوى المتعلقة بالنوم و معالم تخطيط النوم المتعدد.
4. أظهرت الدراسة أن اضطرابات النوم في مرضى باركنسون تتأثر بمدة المرض وشدته وجرعة الدواء.
5. أظهرت الدراسة أن هناك زيادة في نسبة الاضطرابات التنفسية أثناء النوم في مجموعة مرضى باركنسون مقارنة بالمجموعة الضابطة و كانت هذه الزيادة ذات دلالة احصائية.
6. لم يوجد الفرق بين مرضى باركنسون ذوي الشكاوى المتعلقة بالنوم والمرضى عديمي الشكاوى من حيث معالم تخطيط النوم المتعدد.

ولقد استنتج من هذه الدراسة: أن اضطرابات النوم في مرض باركنسون من الصفات الهامة لهذا المرض ولها عدة عوامل منها طبيعة المرض، جرعة الدواء والاضطراب الحركي المصاحب.