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Restoration of normal motor control in Parkinson's disease during REM sleep

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Although normal subjects do not move during REM sleep, patients with Parkinson's disease may experience REM sleep behaviour disorder (RBD). The characteristics of the abnormal REM sleep movements in RBD have, however, not been studied. We interviewed one hundred consecutive non-demented patients with Parkinson's disease and their bed partners using a structured questionnaire assessing the presence of RBD. They rated the quality of movements, voice and facial expression during RBD as being better, equal or worse than in awake ON levodopa condition. Night-time sleep and movements were video-monitored during polysomnography in 51 patients to evaluate the presence of bradykinesia, tremor and hypophonia during REM sleep. Fifty-nine patients had clinical RBD with 53/59 bed partners able to evaluate them. All 53 (100%) reported an improvement of at least one component of motor control during RBD. By history, movements were improved in 87% patients (faster, 87%; stronger, 87%; smoother, 51%), speech was better in 77% patients (more intelligible, 77%; louder, 38%; better articulated, 57%) and facial expression was normalized in 47% patients. Thirty-eight per cent of bed partners reported that movements were 'much better', even in the most disabled patients. The video-monitored purposeful movements in REM sleep were also surprisingly fast, ample, coordinated and symmetrical, without obvious sign of parkinsonism. The movements were, however, jerky, violent and often repetitive. While all patients had asymmetrical parkinsonism when awake, most of the time they used the more disabled arm, hand and leg during the RBD ($P = 0.04$). Movements involved six times as often the upper limbs and the face as the lower limbs (OR: 5.9, $P = 0.004$). The percentage of time containing tremor EMG activity decreased with sleep stages from 34.9 ± 15.5% during wakefulness, to 3.6 ± 5.7% during non-REM sleep stages 1–2, 1.4 ± 3.0% during non-REM sleep stages 3–4, and 0.06 ± 0.2% during REM sleep (in this last case, it was subclinical tremor). The restored motor control during REM sleep suggests a transient 'levodopa-like' reestablishment of the basal ganglia loop. Alternatively, parkinsonism may disappear by REM sleep-related disjunction between pyramidal and extrapyramidal systems. We suggest the following model: the movements during the RBD would be generated by the motor cortex and would follow the pyramidal tract bypassing the extrapyramidal system. These movements would eventually be transmitted to lower motor neurons because of brainstem lesions interrupting the pontomedullary pathways which mediate the REM sleep atonia.

Keywords: paradoxic kinesis; Parkinson's disease; REM sleep behaviour disorder; sleep benefit; basal ganglia

Abbreviations: RBD = REM sleep behaviour disorder

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Introduction

Parkinson's disease is the most common serious movement disorder, affecting 1–2% of adults older than 60 years. (Samii *et al.*, 2004) In addition to resting tremor, patients suffer a progressive motor disability, with rare and slow

movements (bradykinesia), low, less fluent and articulate speech with monotony of pitch (hypophonia), and poor facial expression (Hugues *et al.*, 1992). Most symptoms are improved by dopaminergic treatments ('ON' condition), and reappear when withdrawing treatments ('OFF' condition). In contrast with this motor disability, patients with Parkinson's disease may perform movements when normal subjects would not. This is the case during REM sleep behaviour disorders (RBDs), where patients exhibit vigorous complex movements corresponding to enacted dreams (Schenck *et al.*, 1986). As many as half of the patients with Parkinson's disease may indeed kick, laugh, punch or fight invisible enemies during REM sleep, and possibly injure themselves or their bed partner (Comella *et al.*, 1998; Gagnon *et al.*, 2002). In contrast to sleepwalkers, patients with RBD rarely stand up or walk, but can gesticulate to the point of falling out their bed (Scaglione *et al.*, 2005). Sleep monitoring indicates that RBD is caused by the imperfect abolition of muscle tone during REM sleep, probably caused by pontine lesions in the REM sleep atonia system (Sastre and Jouvet, 1979; Lu *et al.*, 2006). No detailed study of the quality of movements during RBD in patients with Parkinson's disease has been performed, however. Interestingly, several spouses sleeping with patients reported that they observed a sharp contrast between the slow, limited movements, and non-intelligible, low voice of their affected spouse when awake, and the fast, vigorous movements with loud voice that the very same patient exhibited during enacted dreams (Arnulf, 2003). From these serendipitous observations, we decided to compare the quality of the movements, speech and face expression in a prospective series of patients with Parkinson's disease when awake and during REM sleep, with the hypothesis that some restored motor control would occur during this stage of sleep. Therefore, we used a face-to-face interview of the patients and their bed partners, and we assessed the motor performances of the patients during sleep with video-polysomnography.

Material and methods

Patients

From January to May 2005, 100 consecutive patients and their bed partners with Parkinson's disease followed in the neurology department of the University Hospital of Saint Antoine were recruited to take part in the study. Patients met the criteria for definite idiopathic Parkinson's disease (Hugues *et al.*, 1992), and had no significant cognitive impairment, with a score in the Mini-Mental State Examination (MMSE) above 24 (Folstein *et al.*, 1975). They slept most of the night with a person able to observe their nocturnal behaviour during sleep. All participants (patients and co-sleepers) gave written informed consent for the protocol, which was approved of by the local ethics committee. There was at the same time a separate consent for the video-polysomnography and 51 patients accepted to be enrolled for this test. These 51 patients did not differ for age, sex, disease course and motor disability from the 49 non-monitored patients, but had more frequent clinical RBD (70% versus 49%, $P = 0.03$), which suggests they were more motivated for undergoing a full sleep monitoring.

Clinical evaluation

Data about demographic characteristics, medical history, Parkinson's disease course and treatment (with particular attention to the use of psychoactive drugs) were collected during a face-to-face interview. We calculated the total daily levodopa equivalent dose using previously reported formulae (Hobson *et al.*, 2002). We assessed the motor disability of the patients using the Unified Parkinson's Disease Rating Scale motor examination, UPDRS-III (Fahn *et al.*, 1987) in patients showing the optimal effect of the antiparkinsonian treatment levodopa ('ON' condition), and determined the side most affected by parkinsonism. We also calculated the 'axial score' [sum of items 22 (neck) and items 27–30 subscores] and the 'limbs score' [sum of items 20, 22 (except neck) and 23–26 subscores] in the UPDRS-III. We administrated the MMSE, the Frontal Assessment Battery (Dubois *et al.*, 2000), and checked for the presence of hallucinations.

In addition, patients were interviewed about their sleep habits during the current year using a structured questionnaire adapted from an RBD questionnaire (Comella *et al.*, 1998). The patients and their bed partners were separately interviewed about RBD in presence of each other. Clinical RBD was definite when the bed partner reported significant, purposeful limb or body movements (as if patients were acting out their dreams) and when these movements were associated with a dream recall when the patient was awakened. The structured questionnaire also assessed the quality of movement during RBD. We asked the bed partner to compare the movements of the patient during RBD versus wakefulness (speed, smoothness and strength), facial expression and quality of speech (volume of the voice, articulation and intelligibility). They had to score each item as 'much better than awake in ON condition', 'better than in ON condition', 'similar to the ON condition', 'similar to the OFF condition', 'worse than the OFF condition' or 'do not know'.

Sleep monitoring

Sleep and nocturnal movements were monitored during a single night after at least 10 h of antiparkinsonian treatment withdrawal, in order to increase the specificity when detecting normalized movements while in OFF condition. Also, the drugs causing or aggravating RBD (antidepressants, venlafaxine, selective serotonin reuptake inhibitors, etc.) were withdrawn for a period >5 half-lifes. Patients came to the sleep laboratory in the afternoon, and were instructed not to take their levodopa and dopamine agonists after noon, so that they were in OFF condition during the night. Fifty-one patients accepted to undergo a video-polysomnography. The monitoring included Fp1-Cz, O2-Cz, C3-A2 electroencephalography, right and left electro-oculogram, nasal pressure through a cannula, tracheal sounds through a microphone, thoracic and abdominal belts to assess respiratory efforts, electrocardiography, pulse oximetry, EEG-synchronized infrared video-monitoring and ambiance microphone. In addition, we monitored the EMG of the levator menti, sternomastoid, carpi radialis (especially for monitoring tremor) and tibialis anterior muscles. The sleep stages, arousals, alpha rhythm on EEG, respiratory events, periodic leg movements and muscle activities were scored through visual inspection according to standard criteria and definitions previously reported (Arnulf *et al.*, 2005).

Video and EMG movement analysis

We determined the presence or absence of RBD in this group of patients with Parkinson's disease. They were defined as the presence of complex motor behaviours (talking, laughing, yelling, reaching, gesturing, punching, sitting and kicking) in the video–audio recording during REM sleep (American Academy of Sleep Medicine, 2005). Alternatively, if no motor behaviour was observed in the video, we requested the presence of REM sleep without atonia, and history of clinical RBD (American Academy of Sleep Medicine, 2005). Movements on video were detected by a single scorer (V.C.D.C). Collected movements were then rated in a single session by a group of movement disorders specialists by consensus (M.V., E.A., I.A., V.C.D.C., E.R.).

We characterized the pattern of these movements. We assessed the number of movement episodes, the nature of movements (flexion, kicking, etc.), the location in the body parts (face, neck, trunk, arm, hand, leg), the left or right side (compared with the most affected side by Parkinson's disease when awake) and the dynamics of movements (speed, strength, amplitude and smoothness). The characteristics of movements during REM were compared with those observed during nocturnal awakenings and when they woke up in the next morning, before the first intake of antiparkinsonian treatment. Moreover, they were compared with the quality of movements that patients were able to perform in the ON condition during daytime.

We performed an EMG movement analysis during bedtime. During REM sleep, we analysed the duration, location and pattern of distribution (orbicularis oculi, levator menti, sternomastoid, extensor radialis and tibialis anterior muscles) and propagation of the muscle twitches in order to characterize their origin (Caviness and Brown, 2004). We also paid attention to tremor, defined as a rhythmic (4–6 Hz) activity on chin and limb EMG muscles, during wakefulness, and during all stages, including REM sleep. We studied the percentage of time with tremor across stages. In order to obtain the maximum of information on tremor characteristics during sleep, we focused in patients with patent rest tremor when awake, that we arbitrarily defined as more than 10% time awake with tremor on EMG.

Statistical analysis

Statistics were performed using analyses of variance for comparison of continuous measures between the two groups. Adjustment for age was performed using analysis of covariance. Proportions were compared using the χ^2 test and adjustment for age was performed using logistic regression. Results are reported as mean ± SD, unless otherwise stated.

Results

Of the 100 patients, 60 had RBD according to the interview (Fig. 1). Among the 51 patients who underwent the sleep monitoring, 36 had RBD according to the interview and 35/36 were confirmed after sleep monitoring (one with somnambulism instead of RBD). In addition, 6 patients who did not report RBD at the interview had complex movements during REM sleep. Eventually, 41 patients met the international criteria of RBD diagnosis (American Academy of Sleep Medicine, 2005).

Patients had developed their RBD before (22%), at the same time (23%) or after (55%) the onset of Parkinson's disease. When the RBD preceded the onset of Parkinson's disease it had occurred from a few months to 4 years before. RBD occurred less than once a month in 40% patients, between once a week and once a month in 27% patients and more than once a week in 33% patients. RBD were more frequent in the second (64%) than in the first (36%) part of the night. Only 11% patients had also RBD during naps. The episodes of RBD were mostly brief, with 37% of them usually lasting a few seconds, 29% 1–2 min, 28% between 2 and 30 min, and 6% >30 min. Dreams during RBD referred to fighting or running/fleeing in 54% patients. Forty-six per cent of patients had injured themselves and 34% had injured their co-sleepers during sleep. The injuries were mostly contusions but one patient had an open wound when falling from bed and required stitches. Only 8% patients had taken measures to protect themselves or their co-sleepers, mostly by sleeping in twin beds.

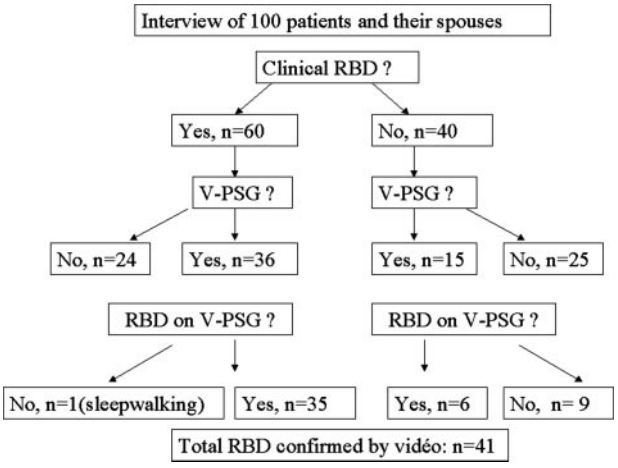

Fig. 1 Diagram of patient flow.

The clinical characteristics of patients with and without clinical RBD were not different except for a longer disease duration and more frequent restless legs syndrome in patients with RBD (Table 1). Similarly, there were no differences between groups for sleep measures, except for higher REM sleep percentages and higher percentages of limb muscle activity during REM sleep in patients with RBD (Table 2).

Fifty-three (53/59) bed partners were able to compare the quality of movements, speech and facial expression of their co-sleeper during RBD and awake. The six other bed partners said that they were asleep or that the room was too dark to evaluate these aspects. All 53 (100%) reported an improvement either of the movements, speech or facial expression. The quantification of the motor response compared to the ON and OFF conditions is shown Fig. 2. The movements were improved in 87% of patients, including increased speed (87%), strength (87%) or smoothness (51%). Speech was improved in 77% patients. It was more intelligible (38%), better articulated (57%) or with a louder volume (77%). Facial expression was normalized in 42% patients during

Table 1 Demographic and clinical characteristics of patients with Parkinson's disease with and without RBD

	With RBD	Without RBD	*P*
Number	65	35	
Age (years)	65 (9)	61 (13)	0.124
Sex (% men)	69	60	0.479
Body mass index (kg/m^2)	25 (4)	24 (3)	0.186
Disease course (years)	8 (5)	6 (3)	0.017
Motor disability when treated (UPDRS-III/108)	21 (12)	18 (11)	0.256
Axial score (/20)	3.9 (3.0)	3.1 (3.0)	0.186
Limbs score (/72)	13.6 (7.8)	11.7 (6.5)	0.203
Use of dopamine agonists (%)	53	63	0.512
Levodopa-equivalent dose (mg/day)	666 (428)	624 (306)	0.606
Use of benzodiazepine (% patients)	23	23	0.826
Use of selective serotonin reuptake inhibitors (% patients)	29	17	0.276
Sleep benefit (% patients)	45	35	0.430
Hallucinations (% patients)	24	14	0.341
Mini-Mental State Examination score (/30)	28 (2)	28 (2)	0.416
Frontal assessment battery score (/18)	16 (2)	16 (2)	0.827
Beck Depression Inventory (depression if $\geqslant$13)	11 (7)	12 (7)	0.377
Epworth Sleepiness Scale score (/24)	9 (5)	9 (4)	0.849
% Sleepy patients (score >10)	34	40	0.694
Restless legs syndrome (%)	29	9	0.020

Data are mean (SD). UPDRS = Unified Parkinson's Disease Rating Scale.

Table 2 Sleep measures in patients with Parkinson's disease with and without RBD

Sleep measures	With RBD	Without RBD	*P*
Number	41	10	
Clinical RBD (number)	35	1	
Occipital alpha waking rhythm (Hz)	8.7 (1.4)	9.6 (1.5)	0.093
Night-time sleep			
Total sleep period (min)	514 (97)	473 (67)	0.212
Total sleep time (min)	360 (105)	291 (100)	0.063
Sleep efficiency	69.1 (17.4)	61.6 (22.2)	0.249
Latency to (min)			0.369
Sleep onset	47 (41)	64 (83)	0.581
REM sleep	141 (108)	165 (127)	0.110
Sleep duration (% total sleep time)			
Stage 1	4.8 (5.4)	3.5 (3.2)	0.307
Stage 2	57.6 (13.3)	63.4 (14.6)	0.527
Stages 3–4	17.9 (9.7)	19.4 (9.1)	0.241
REM sleep	19.7 (9.9)	14.3 (11)	0.045
Sleep fragmentation (no./per hour)			
Arousals	14.4 (10.2)	28.6 (11.3)	0.241
Periodic leg movements	11.4 (17.4)	13.2 (15.5)	0.429
Apnoea–hypopnoea	6.4 (10.4)	0.8 (1.2)	0.093
REM sleep without atonia (% total REM sleep)			
Chin muscle activity	25.1 (24.0)	0.2 (0.4)	<0.0001
Limb muscle activity	5.9 (6.5)	0.0 (0.1)	<0.0001

Data are mean (SD).

RBD. Thirty-eight per cent bed partners reported this improvement as much better. Demonstrative examples of patients able to squat, sing and slap during RBD (video), while unable to do it awake, are shown in Table 3. When the subgroup of the most disabled patients (UPDRS-III $\geqslant$ 30, n = 14) was considered, 86% had improved movements (increased speed, 79%; increased strength, 86%; or better smoothness, 50%), 100% spoke better (clearer intelligibility, 36%; better articulation, 57%; or louder volume, 100%) and 45% had normal facial expression during RBD. In addition, the term 'much better than awake' was chosen by 50% co-sleepers versus 35% co-sleepers of less disabled patients (UPDRS < 30, n = 41, P = 0.478).

In the video monitoring, two-thirds of patients with RBD had simple and complex movements, and spoke or shouted during REM sleep, whatever the sleep cycle. All of them remained in bed during RBD. For example, a patient gesticulated while speaking then repeatedly pointed his index

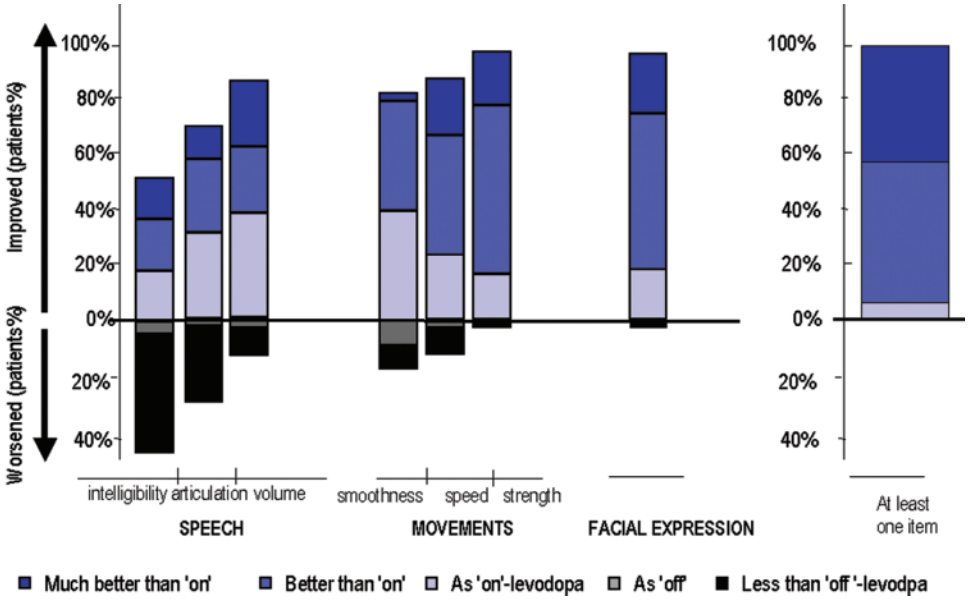

Fig. 2 Changes in motor control during REM sleep in Parkinson's disease (n = 53).

Table 3 Examples of improved motor control during RBD observed by the co-sleeper (each line is a different patient)

Behaviour observed by co-sleeper	Dream content	Awake
Squatting on the bed, waving his arms as if flying, shouting 'pin pon' (the two-tone sound of a siren) with a duck's voice	I am a police-duck, flying after a pigeon-thief	Unable to squat, bradykinesia, hypophonia
Sitting on the bed, singing 'Le plus beau de tous les tangos du monde' (a famous popular song of the past) with a strong and sonorous voice, a wide smile on his face	I am dreaming that I am singing as I used to before PD in my bathroom	Unable to sing, poor facial expression
Sitting on the bed rowing without paddles, shouting 'Help, caimans!', getting hold of a heavy oak bedside table and throwing it across the room	I am on a canoe, attacked by caimans, trying to make them flee	Unable to carry the heavy bed-side table, impaired coordinated movements, hypophonia
Extending arms and legs and giving blows all over	I am flying lying on my back with the feet in front and I am braking with my feet	Bradykinesia
Declaiming political speeches with a loud voice	I am rehearsing a speech for the town council	Hypophonia, monotony of pitch
Fighting with an invisible foil, with great agility and shouting 'Manon, Charlemagne!' (an old-fashioned war cry)	I am a knight fighting with a foil to save my endangered lady-love	Bradykinesia, no rapid alternative movements, loss of agility

finger with anger then kicked. Another held something in his hand seeming to protect himself. The next morning, he reported he had a nightmare where he was attacked in the street and had to protect himself using his cell phone as a weapon. In all patients, the movements were surprisingly fast, without parkinsonism. Movements were realised with the same strength, amplitude and speed as what could be observed in healthy, awake subjects. Despite the fact that the movements were in the normal range, their aspect was jerky, violent and often duplicated. While all patients had asymmetrical parkinsonism when awake, they used more often the most disabled arm, hand and leg during RBD (P = 0.04). Movements involved six times more often the upper limbs and the face than the lower limbs (OR: 5.9, P = 0.004).

In contrast, the movements performed during the nocturnal and morning awakenings were slow, with reduced amplitude. There were movements oriented to nocturnal needs, such as turning in the bed, scratching the nose, reaching the alarm, taking a basin to urinate, replacing the bed linen or the clothes, and drinking water. Clinical tremor was observed in two-thirds of the patients when awake. Since movements were often jerky, we checked for possible brief intrusions of normal REM sleep muscle atonia during the movements. This was not the case, as for example chin muscle tone could remain elevated during transient decrease of hand muscle tone. In addition, in EMG analysis, the jerks did not have a focal location, or a pattern of distribution and propagation of the muscles twitches, allowing to characterize

their origin as can be done for myoclonus (Caviness and Brown, 2004). The percentage of time containing tremor EMG activity in the levator menti, extensor carpi radialis or tibialis anterior muscles decreased with sleep stages from 34.9 ± 15.5% during wakefulness, to 3.6 ± 5.7% during non-REM sleep stages 1–2, 1.4 ± 3.0% during non-REM sleep stages 3–4, and 0.06 ± 0.2% during REM sleep (in this last case, this was subclinical tremor).

Discussion

There is a transient restored motor control during RBD in patients with Parkinson's disease. Indeed, all patients improve (and sometimes 'much better') their movements, speech or facial expression during REM sleep, compared with movements performed awake and during arousals. This improvement is confirmed both by bed partners and by direct observation of night-time video in a large group of patients. REM sleep movements are faster, smoother, coordinated and become symmetrical while asymmetrical (due to parkinsonism) awake. Patients move their disabled side more often. This improvement is particularly remarkable if one realizes patients are off levodopa for 12–20 h. There is no bradykinesia, or clinical tremor as observed in untreated patients with Parkinson's disease. In addition the improvement is also observed in the most severely disabled patients. This is the first time, to our knowledge, that a restored motor control during REM sleep is objectified in patients with Parkinson's disease.

There are other circumstances where patients with Parkinson's disease experience a spontaneous, transient motor improvement. This includes the phenomenon of paradoxic kinesis first described by Souques in 1921 (Souques, 1921). A demonstrative example was a bedridden patient suddenly able to stand up and flee his house in a fire. The mechanism of paradoxic kinesis is still unknown, but may be associated with pacing, life-threatening conditions and strong emotions. Of interest, the dreams in REM sleep are also frequently emotionally charged (McNamara *et al.*, 2005), and amygdala (an important part of the emotionally influenced memory system) activity is higher during REM sleep than during wakefulness (Braun *et al.*, 1998). One could then hypothesize that restored motor control during REM sleep is a paradoxic kinesis caused by dream emotions. Most patients indeed improved their movements while dreaming they were fighting for survival against imaginary aggressors, a frequent theme of RBD (Fantini *et al.*, 2005). This is unlikely, however, as movements were similarly improved when patients were acting out dreams with no or little emotional content including singing under the shower.

Some patients with Parkinson's disease may also experience fluent mobility upon awakening from night sleep prior to any drug intake, a phenomenon called 'sleep benefit' (Currie *et al.*, 1997; Hogl *et al.*, 1998). We do not think, however, that restored motor control is an equivalent to sleep benefit. First, we observed a major qualitative difference between the rapid, ample movements performed during REM sleep and the slow, disabled movements performed during arousals from sleep (including from REM sleep) and during the next morning before the first levodopa intake. In addition, since 55% patients with restored motor control during REM sleep reported no sleep benefit, movements cannot only be improved by the mechanism at play during sleep benefit. One of the possible mechanisms of sleep benefit is a restoration of dopamine stores during sleep. If this is true, the longer the patients sleep, the better the sleep benefit. In our patients, however, restored motor control could be observed during the first REM sleep episode, after only one to two hours asleep. All in all, we do not think that the mechanism of sleep benefit or paradoxic kinesis can explain restored motor control during REM sleep.

The source of movements of enacted dreams during REM sleep is unknown. Some authors have proposed they could be archaic movements, determined by central pattern generators in the mesencephalon, pons and spinal cord, subserving innate motor behaviours essential for survival such as feeding, locomotion and reproduction (Tassinari *et al.*, 2005). The movements during REM sleep were, however, elaborated, complex, non-stereotyped, with learned speeches (e.g. political speech, lectures) and songs in our series, suggesting they result from the same cortical mechanisms as awake complex activities, rather than from primary automatisms. The high proportion of face and arm movements (involving the tongue, lips, arm and hand, which have a large cortical representation) during REM sleep are further evidence for cortical origin.

If the movements during REM sleep have not the characteristics of parkinsonism, they are, however, not totally normal. Even when purposeful and patterned, they are jerky, broken, rough and too fast, resembling Chaplin's movies. One hypothesis for the broken aspect of movements could be transient brief intrusions of REM sleep muscle atonia in the course of the movement. However, EMG recordings did not support this possibility, since chin muscle tone was not completely abolished between jerks. The observation that parkinsonism disappears during REM sleep suggests that the basal ganglia loop for motor control is transitoryily restored, possibly by REM sleep-mediated inhibition of overactive nuclei (e.g. the subthalamic nucleus). To confirm this hypothesis, deep brain recordings of the activity of the basal ganglia during different sleep stages are needed. In animal models, whether healthy or parkinsonian, the activity of the subthalamic nucleus is on the contrary further increased during REM sleep (Urbain *et al.*, 2000; Chouvet *et al.*, 2003), suggesting no beneficial effect of REM sleep on the functioning of this output nucleus. The alleviation of parkinsonism suggests that the normal functioning of the basal ganglia loop is restored, or that the upper motor neurons (e.g. supplementary motor area) are no more submitted to the deleterious influence of

pathological basal ganglia. Loss of synaptic functional connections in the voluntary motor system has been recently reported at the transition between wakefulness and non-REM sleep in healthy subjects (Massimini *et al.*, 2005). Interestingly, the broken, jerky, rough aspect of REM sleep movements suggests they are unfiltered. They could result from the expression of the primary motor cortex, relieved from the filtering, smoothing control of the basal ganglia. Therefore, the extrapyramidal pathway would be bypassed. To support this hypothesis, one should gather information about the quality of movements during established sleep in non-parkinsonian conditions. Normal subjects cannot be studied for this purpose, as they do not move during sleep but only during arousals. The observation of similarly jerky, broken, rough movements during RBD in subjects without parkinsonism (i.e. idiopathic RBD, Iranzo *et al.*, 2005) RBD in cerebellar diseases would support this model of disjunction between pyramidal and extrapyramidal systems.

We suggest the following model: the movements during the RBD would be generated by the motor cortex and would follow the pyramidal tract bypassing the extrapyramidal system. These movements would eventually be transmitted to the lower motor neuron because of brainstem lesions interrupting the pontomedullary pathways which mediate the REM sleep atonia.

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