ELSEVIER

Sleep Medicine 2 (2001) 353–354

SLEEP MEDICINE

www.elsevier.com/locate/sleep

Journal search and commentary

Article reviewed: Improvement of sleep architecture in Parkinson's Disease with subthalamic nucleus stimulation[☆]

Diego Garcia-Borreguero

Sleep Disorders Unit, Department of Neurology, Fundación Jiménez Díaz, Avenue Reyes Católicos 2, 28040 Madrid, Spain

Objective

To determine the degree of improvement of sleep architecture in ten patients with Parkinson's Disease (PD) undergoing high-frequency subthalamic nucleus stimulation.

Methods

The study included 11 patients suffering from severe akinetic idiopathic PD and treated successfully with continuous bilateral high-frequency stimulation of the subthalamic nucleus (STN). All patients had suffered from insomnia before surgery and were asked to undergo consecutively for two nights polysomnographic studies. Following a randomized order of treatment conditions, patients were assigned to undergo a sleep study with and without STN stimulation. On both nights, patients were treated simultaneously with their usual medication, namely levodopa or levodopa and dopamine agonists. Last dosages of medication were taken at 20:00 h and on the sleep studies without treatment, the stimulator was turned off at 19:00 h.

Results

All but two patients completed the study. These two patients became anxious and asked for the stimulator to be turned off at 13:00 h. UPDRS scores in the morning were 66% lower after treatment than without treatment. On the off-treatment night, patients reported painful akinesia (6), dystonia (5), and nocturia (2). None of these complaints were present on the treatment night.

Treatment with STN stimulation increased total sleep time (by 47%) and sleep efficiency (by 36%), and decreased wakefulness after sleep onset (by 51 min). No changes were seen in fragmentation indexes, number of REM-NREM cycles, or apnea-hypopnea index. A mild increase in PLM-index was observed in three patients. Phasic REM sleep activity along with behavioural symptoms of REM Sleep Behaviour Disorder also increased in patients during stimulation.

Conclusion

The authors conclude that night-time motor disability and sleep architecture improved following treatment. STN stimulation alleviated dystonia but did not alter the number of awakenings. Dystonia followed awakenings but did not cause them. Thus dystonia increased the length of arousals, thereby disturbing

[☆] Arnulf I, Beijani BP, Garma L, Bonnet AM, Houeto JL, Damier P, Derenne JP, Agid Y. (Neurology 2000;55:1732–1734).

PII: S1389-9457(01)00102-2

sleep architecture. The authors suggest that sleep fragmentation in PD results mainly from motor disability at night.

Discussion

The pallido–subthalamic pathway exerts a powerful control over the basal ganglia and its hyperactivity has been implicated in the movement disorders observed in Parkinson's disease. Previous studies have shown that high frequency bilateral stimulation of the STN improves daytime PD symptoms and thereby reduces the dose requirements for dopaminergic medication. Unlike the effects of levodopa, which are relatively short-lasting, the effects of STN stimulation are continuous throughout the 24 h. Thus, sleep studies during simultaneous STN stimulation can be helpful to examine the degree to which sleep abnormalities are caused by motor symptoms.

The study showed an overall improvement of night-time motor dysfunction during STN stimulation. As a result, motor improvement was associated with an increase in total sleep time (notably, stage 2 sleep) and a decrease in WASO. The number of awakenings did not decrease, although their mean duration was far shorter in the treated condition. Episodes of dystonia always took place during wakefulness. Since the duration of the episodes of wakefulness were shorter during stimulation, the author hypothesizes that dystonia does not cause arousal, but rather prevents the subject from falling back to sleep once awake.

The effects of STN stimulation on sleep itself without changes in motor function are not known so it is unclear the extent to which the sleep improvement is secondary to less disruption from the motor symptoms or a primary effect of the stimulation itself. Furthermore, the study does not include a group of healthy, untreated subjects as a comparison group for normal sleep parameters. Since STN stimulation exerts its main effect over motor symptoms, other factors contributing to sleep fragmentation cannot be excluded. So, for example, the frequency (rather than the length) of arousals and awakenings could be due to the disease process per se, as STN stimulation does not reduce either. Thus, sleep fragmentation could result from several processes, and motor disturbance accounts only for part of it.

It needs to be noted that periodic limb movements of sleep (PLMS) do not improve as a result of STN stimulation, supporting an involvement of dopaminergic pathways other than the nigro-striatal circuitry, as other studies have also suggested. Interestingly, phasic activity increases as a result of STN stimulation, a finding that resembles the effects of levodopa. In this regard the increase in REM behaviour disorder with stimulation deserves some attention as a possible significant adverse effect of the treatment.

In summary, STN stimulation opens new, non-pharmacologic, perspectives to explore sleep disturbance in PD.