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Nocturnal Sleep of Narcoleptic Patients: Revisited

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Summary: Narcoleptic patients were compared to idiopathic hypersomniac patients and REM hypersomnia patients with regard to nocturnal sleep disruption. Results showed specificity of the narcoleptic sleep pattern and a possible correlation between REM fragmentation and cataplexy. Patients with and without periodic movements in sleep (PMS) were compared and no difference was found in their nocturnal sleep pattern, suggesting that PMS is not the major determinant of sleep disruption previously described in narcoleptic patients. Finally, nocturnal sleep disruption was treated with γ-hydroxybutyrate (GHB) and results further indicate a possible link between REM fragmentation and cataplexy. **Key Words:** Narcolepsy—Cataplexy—Periodic movements in sleep—γ-Hydroxybutyrate.

Ten years ago, at the First International Symposium on Narcolepsy, it was already known that narcoleptics had severe sleep disruption (1). Among other characteristics they have an increased number of awakenings, increased waketime after sleep onset, and increased time spent in stage 1 NREM sleep. They also have more specific REM sleep disruptions, such as sleep onset REM periods (SOREMPs), REM sleep fragmentation, and intermediate stages of sleep, i.e., where only some features of REM sleep are present at the same time (1–3). More recently, some authors have questioned the specificity of nocturnal sleep disruption in narcolepsy (4).

Specificity of narcoleptic sleep pattern

In a previous study (5), three groups of hypersomnia patients were studied, namely, narcoleptic patients, NREM hypersomnia patients corresponding in most cases to idiopathic central nervous system hypersomnolence of the Association of Sleep Disorders Centers classification, and REM hypersomnia patients. This last group was characterized by excessive daytime sleepiness (EDS) and sleep attacks without cataplexy but with SOREMPs during the multiple sleep latency test (MSLT).

NREM hypersomnia patients had a totally different sleep pattern from that of narcoleptic patients. They slept longer, had less awakenings and waketime after sleep onset, and a normal sleep efficiency. In addition they did not show the REM sleep abnormalities noted in narcoleptic patients. REM hypersomnia patients, however, had a sleep pattern similar to that of narcoleptic patients but without REM fragmentation. Consequently, these patients

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were clinically different from narcoleptic patients because of the absence of cataplexy and polygraphically by the absence of REM sleep fragmentation. We hypothesized at this time that they were probably narcoleptic patients at an early stage of their illness. Six of these patients were followed regularly in our laboratory over the past 5 years and 5 of them eventually developed cataplexy. In addition, all of them were found to have the HLA-Dr2 antigen, supporting the original hypothesis that they were probably narcoleptic.

In summary, these results suggest that nocturnal sleep disturbance found in narcoleptic patients is quite specific, and that a correlation may exist between some aspect of nocturnal sleep pattern and diurnal symptomatology, namely REM sleep fragmentation and cataplexy.

Periodic movements in sleep and narcolepsy

One of the major recent findings in this area is the discovery that a large number of narcoleptic patients have periodic movements in sleep (PMS) (4). With others, we found that ~50% of patients with narcolepsy have PMS of various severity and that narcoleptic patients with PMS are significantly older than those without PMS (mean age difference = 10.6 years, $p < 0.05$). However, when these groups of narcoleptic patients with and without PMS were matched for age, no difference was found in their sleep pattern except for increased number of shifts from stage 2 to stage 1 or waking in the PMS group (6). On the basis of these observations we concluded that PMS are not, in our opinion, the main factor responsible for nocturnal sleep disruption in narcolepsy.

Treatment of sleep disturbance with γ-hydroxybutyrate

Over the past 6 years, 36 narcoleptic patients were treated with γ-hydroxybutyrate (GHB) in our laboratory; 10 patients were investigated recently on a standard protocol. In this study, patients were recorded for 2 nights and 5 naps (MSLT) before and after 1 month of treatment. All patients were given a single dose of 2.25 g GHB at bedtime for the second polygraphic recording session.

Effect on diurnal symptoms. Thirty patients (83%) had a marked improvement of their cataplexy with GHB, and 14 (39%) reported subjective improvement of their EDS. No side effects were reported by narcoleptic patients treated with GHB, except in the case of 3 patients (8%). One of these patients experienced severe hypnagogic hallucinations for 3 consecutive nights, another reported sleep paralysis of long duration at bedtime, and the last one presented severe restless legs syndrome at night and PMS during the first part of the night. In all 3 cases, GHB was discontinued.

In the 10 patients recently studied, all reported a marked improvement of their cataplexy and this symptom was completely suppressed in 7 patients. Four patients reported improvement of EDS although GHB had no effect on sleep latencies measured during the MSLT.

Effect on nocturnal sleep. GHB produced a marked improvement of nocturnal sleep disturbance in narcoleptic patients. Seven of 10 patients complained of nocturnal sleep disruption prior to the onset of treatment and sleep improved subjectively in all of them. Results of polygraphic recordings will be published elsewhere but the main changes are listed in Table 1. These results are similar to those previously reported by Broughton and Mamelak (7). Of special interest are the changes in REM sleep variables with GHB. This medication facilitates and consolidates REM sleep in narcoleptics with more SOREMPs, less REM fragmentation, and increased REM efficiency. As mentioned previously, cataplexy during the day is correlated with REM fragmentation during nocturnal sleep recording. Results obtained with GHB further support this hypothesis, since suppression of REM fragmentation with GHB is associated with the improvement of cataplexy.

TABLE 1. *Effect of GHB on nocturnal sleep*[a]

	GHB (2.25 g)[a]
Changes in sleep	Decreased REM latency
	Increased number of SOREMPs
	Increased REM efficiency
	Decreased REM fragmentation
	First third of the night
	Decreased waketime and stage 1 NREM sleep %
	Increased stages 3 and 4 NREM sleep %
	Increased stage REM %

[a]Single dose at bedtime.

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