Sleep, **9**(1):250–253

Sleep Apnea in Narcolepsy

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Summary: Polysomnographic and multiple sleep latency studies documented recurrent apneic episodes during NREM sleep stages 1 and 2 and REM sleep in 11 of 16 narcoleptic patients. Episodes were predominantly central, but obstructive and mixed events were also observed. Apneas were accompanied by mild to moderate oxygen desaturation. **Key Words:** Narcolepsy—Apnea—Sleep.

The occurrence of apnea during sleep is well known in association with sleep apnea syndrome (1–4), but the occurrence of apnea in narcolepsy (5–7) is generally less well known. This article emphasizes the documentation of sleep apnea, by appropriate electrophysiological tests, in patients with narcolepsy.

PATIENT POPULATION

Sixteen patients, 15 men and one woman, complaining of daytime hypersomnolence and suspected of having narcolepsy, were referred to the clinical neurophysiology laboratory for evaluation. Table 1 summarizes the pertinent clinical data. Patients' ages ranged from 25 to 65 years (mean 46 years). The diagnosis of narcolepsy syndrome in all 16 patients was based on the characteristic clinical picture (8,9) of uncontrollable sleep attacks during the day, accompanied by some or all of the major associated narcoleptic symptoms, and confirmed by the presence of sleep-onset REM periods (SOREMP) (10,11) at the daytime polygraphic recording. In addition to cataplexy, hypnagogic hallucinations, and sleep paralysis, disturbed nocturnal sleep should be listed as a major associated symptom in narcolepsy syndrome (12). Patients 3, 6, 7, and 11 were mildly obese; patient 3 had high blood pressure. Patients 5 and 14 had episodic lapses of memory and automatic behavior during which they would drive a car for 20 or 30 min without any recollection of the event. Patient 9 had a brother who suffered from narcolepsy. Neurological examination was normal in all patients. None had a history of encephalitis, head trauma, or any other neurological illness. Patients 11 and 14 received *d*-amphetamine; patients 5 and 7 were treated with methylphenidate alone. All patients improved with medication.

Accepted for publication December 1985.
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TABLE 1. *Clinical data in 16 patients with narcolepsy*

Patient	Age (yr)	Sex	Height (in)	Weight (lb)	Duration of hypersomnolence (yr)	Snoring	Cataplexy	Hypnagogic hallucinations	Sleep paralysis	Disturbed night sleep
1	25	M	68	131	5	+	−	+	−	+
2	44	M	74	200	25	+	+	+	−	+
3	46	M	72	240	37	+	+	+	+	+
4	65	M	66	160	35	−	−	−	+	+
5	56	M	67	170	39	+	+	+	+	+
6	33	M	66	195	13	−	+	+	−	−
7	55	M	72	215	25	+	+	+	−	+
8	55	M	72	190	10	+	+	+	+	+
9	43	M	72	190	5	+	+	−	−	−
10	34	M	73.5	187	10	+	+	+	+	+
11	38	M	68	206	19	+	+	−	−	+
12	50	M	68	165	27	−	+	+	+	−
13	50	M	67	165	20	−	+	+	+	+
14	53	M	73	200	20	−	−	−	+	+
15	34	F	68	155	20	−	−	+	−	+
16	64	M	68	165	22	−	+	−	−	+

+, present; −, absent.

METHODS

All patients underwent daytime polysomnographic recordings on 16- or 20-channel electroencephalographic (EEG) equipment (7). Each recording consisted of multiple channels (channels 4–10) of EEGs and electromyograms (EMG), and vertical and horizontal electrooculograms (EOGs); respiratory activities were measured by oral and nasal thermistors, abdominal pneumograph, and intercostal EMG. Oxygen saturation was recorded by an ear oximeter. The voluntary respiratory control system was tested by noting the ability of the patient to stop breathing and to resume breathing after 10 s.

Patients were recorded following a modified format for the multiple sleep latency test (MSLT): Three 20-min recordings were conducted at 2-h intervals (9 a.m., 11 a.m., and 1 p.m.) and a fourth 2-h recording was performed at 3 p.m.

RESULTS

REM sleep occurred 1–15 min after onset of sleep in all patients, and SOREMPs were noted mostly during the first and third recordings of the MSLT.

Eleven of the 16 patients had apneic episodes during stages 1 and 2 NREM sleep and REM sleep (Table 1). Apneas were accompanied by mild to moderate oxygen desaturation. All 11 patients had central apneas. Six patients had purely central apneas; five, both central and obstructive apneas; and four, mixed apneas. In one patient, the mixed apnea began with obstructive apnea and was then followed by central apnea. Table 2 summarizes the physiological data of the 11 patients with sleep apnea. They had mild to moderate syndromes.

The patients were able to control respiration voluntarily, and all snored at night.

DISCUSSION

Unlike the standard MSLT (11) a modified MSLT, which included three 20-min recordings and a fourth 2-h recording was used. The combined polysomnographic and mod-

TABLE 2. *Polysomnographic data in 11 patients with narcolepsy and sleep apnea*

Parameters	Findings[a]
Sleep-onset REM period	Present in all
Apnea index (no. of apneas per hour of sleep)	37.9 ± 7.9
Central apnea (duration in seconds)	16.3 ± 2.2
Obstructive apnea (duration in seconds)	21.3 ± 5.7
Mixed apnea (duration in seconds)	28.0 ± 8.0
Sao_2%	
Highest	93.3 ± 1.5
Lowest	79.1 ± 2.2

[a]Values expressed as means ± SEM.

ified MSLT helped in documenting the presence and the severity of sleep apnea in 11 of 16 narcoleptic patients.

The findings of sleep apnea in a large number of narcoleptic patients are in contrast to those obtained by Kales et al. (13). These authors noted sleep apnea in only one out of 50 narcoleptic patients. Our findings are in agreement, however, with those of Guilleminault et al. (2,5). In our patients, sleep apnea was predominantly central, but obstructive and mixed apneas were also noted. In 1972 Guilleminault et al. (5) reported central sleep apnea in two patients with narcolepsy and later extended this observation and found sleep apnea in a large number of patients with narcolepsy (2). Laffont et al. (6) noted sleep apnea in five patients with narcolepsy. One of their patients had central, one had obstructive, and three had both central and obstructive apneas. Sleep apneas may have aggravated our patients' disability by worsening already disrupted nocturnal sleep and exacerbating excessive daytime somnolence. It is well known that in primary sleep apnea syndrome, apnea is the single most important factor leading to excessive daytime somnolence (1,4).

Neither obesity nor advancing age were important factors in the genesis of sleep apnea among our narcoleptic patients. While obesity can play an important role in the pathogenesis of obesity-hypoventilation and sleep apnea synrome (1,4,14), only four of our patients were mildly obese, and this factor did not appear to be of primary importance. Advancing age can have deleterious effect on the respiratory control mechanism during sleep (15), but most of our patients with narcolepsy and sleep apnea were <50 years of age Therefore, age did not seem to contribute toward sleep apnea.

Based on our study we suggest that patients with narcolepsy have a high incidence of sleep-related respiratory dysrhythmia, which appears to be less intense than that noted in patients with primary sleep apnea syndrome. Furthermore, apnea is predominantly central in narcolepsy but predominantly obstructive or mixed in type in sleep apnea syndrome. It is important to diagnose sleep apnea in narcolepsy syndrome for appropriate evaluation and treatment.

The presence of apnea in NREM sleep implied a dysfunction of the automatic respiratory control system (16). On the other hand, periodic apnea in REM sleep suggested a dysfunction, possibly of the voluntary respiratory control system (16). However, the ability of the patients to control their breathing voluntarily indicated that the voluntary respiratory control system was grossly intact. It should be noted that at present there is no satisfactory test available to study more accurately the voluntary respiratory control system. The exact cause of narcolepsy is unknown, but it is thought that there is a disturbance of sleep-waking systems in this disorder (12). Therefore, parallel impairment of central control of breathing,

as manifested by respiratory dysrhythmia during sleep and sleep-waking systems in narcolepsy syndrome, suggests a neural dysfunction in an area where respiratory and sleep-waking systems are closely interrelated, such as the nucleus tractus solitarius and ponto-medullary reticular formation (17).

REFERENCES

1. Guilleminault C, Tilkian A, Dement WC. The sleep apnea syndrome. *Ann Rev Med* 1976;27:465–84.
2. Guilleminault C, Van Den Hoed J, Mitler MM. Clinical overview of the sleep apnea syndrome. In: Guilleminault C, Dement WC, eds. *Sleep apnea syndromes*. New York: Alan R. Liss, Inc., 1978:1–12.
3. Cherniack NS. Respiratory dysrhythmias in sleep. *N Engl J Med* 1981;305,325,330.
4. Chokroverty S, Sharp JT. Primary sleep apnea syndrome. *J Neurol Neurosurg Psychiatry* 1981;44:970–82.
5. Guilleminault C, Eldridge F, Dement WC. Insomnia, narcolepsy and sleep apneas. *Bull Eur Physiopathol Respir* 1972;8:1127–38.
6. Laffont F, Autret A, Minz M, Beillevaire T, Cathala HP, Castaigne P. Sleep respiratory arrhythmias in control subjects, narcoleptics and non-cataplectic hypersomniacs. *Electroencephalogr Clin Neurophysiol* 1978;44:697–705.
7. Chokroverty S. Phasic tongue movements in human rapid eye movement sleep. *Neurology* 1980;30:665–8.
8. Zarcone V. Narcolepsy. *N Engl J Med* 1973;288:1156–66.
9. Guilleminault C, Dement WC, Passouant P, eds. *Narcolepsy. Advances in sleep research*. New York: Spectrum, 1976.
10. Rechtschaffen A, Wolpert EA, Dement WC, Mitchell SA, Fisher C. Nocturnal sleep of narcoleptics. *Electroencephalogr Clin Neurophysiol* 1963;15:599–609.
11. Mitler MM, Van Den Hoed J, Carskadon MA, et al. REM sleep episodes during the multiple sleep latency test in narcoleptic patients. *Electroencephalogr Clin Neurophysiol* 1979;46:479–81.
12. Larkin KT. The neurobiology of the narcoleptic syndrome. *Int J Neurosci* 1984;25:1–17.
13. Kales A, Cadieux RJ, Soldatos CR, et al. Narcolepsy-cataplexy: I. Clinical and electrophysiologic characteristics. *Arch Neurol* 1982;39:164–8.
14. Sharp JT, Barrocas M, Chokroverty S. The cardiorespiratory effects of obesity. *Clin Chest Med*, 1980;1:103;–18.
15. Miles LE, Dement WC. Sleep and aging. *Sleep* 1980;3:119–220.
16. Phillipson EA. Control of breathing during sleep. *Am Rev Resp Dis* 1978;118:909–39.
17. Berger AJ, Mitchell RA, Severinghaus JW. Regulation of respiration. *N Engl J Med* 1977;297:92–7, 138–43, 194–201.