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Predictors of Sleepiness in Epilepsy Patients

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Summary: Sleepiness, a common complaint of epilepsy patients, is frequently attributed to antiepileptic medications. To determine predictors of subjective sleepiness in epilepsy patients, we gave self-administered, validated surveys of sleepiness [Epworth sleepiness scale (our major outcome measure)] and sleep apnea [sleep apnea scale of the sleep disorders questionnaire (SA/SDQ)] to 158 epilepsy patients and 68 neurology patients without epilepsy (controls). An elevated Epworth score (>10) was more likely in epilepsy patients compared to controls after controlling for age and gender ($p < 0.05$). When Epworth scores were adjusted for SA/SDQ scores and restless legs symptoms (RLS), however, epilepsy patients showed only a nonsignificant trend toward elevated Epworth scores compared to controls ($p = 0.08$). SA/SDQ scores ($p < 0.005$) and RLS ($p < 0.007$) were significant predictors of elevated Epworth score in both epilepsy patients and controls. Among the epilepsy patients, the number or type of antiepileptic medication, seizure frequency, epilepsy syndrome (partial vs. generalized), and the presence of sleep-related seizures were not significant predictors ($p > 0.10$) of elevated Epworth score. Before attributing sleepiness in epilepsy patients to antiepileptic medications or uncontrolled seizures, clinicians should consider the possibility of a coexisting sleep disorder. **Key Words:** Epilepsy—Sleep disorders—Daytime sleepiness—Antiepileptic medications—Epworth sleepiness scale—Sleep disorders questionnaire.

Excessive daytime sleepiness (EDS) is common, occurring in 0.5–5.0% of the population (1). Several reports have indicated a high prevalence of sleep disorder symptoms, including EDS, in epilepsy patients (2–4). Although subjectively reported EDS in epilepsy patients is a frequent adverse effect of antiepileptic medication (5), sleep disorders may coexist with epilepsy and contribute to EDS. Diagnosis and treatment of these sleep disorders has important implications in the epilepsy population because treatment not only improves EDS but may also enhance seizure control (6–9).

To determine the relative contributions of symptoms of sleep disorders, antiepileptic medications, and other variables to subjectively reported sleepiness in epilepsy patients, we gave self-administered, previously validated surveys of sleepiness and sleep apnea to 158 epilepsy patients (epilepsy group) and 68 neurology patients without epilepsy (controls). Surveys also assessed restless legs symptoms, antiepileptic and sedative medications, seizure frequency, and other epilepsy-related variables. We tested the hypothesis that symptoms of sleep disorders (e.g. obstructive sleep apnea or restless legs symptoms), the number or type of antiepileptic medications, and seizure frequency would be comparable predictors of subjective sleepiness.

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METHODS

Subjects

This study was approved by the University of Michigan Institutional Review Board, and all subjects gave informed consent to participate. Subjects were screened to determine eligibility for the survey from three sources. The primary source was consecutive epilepsy and nonepilepsy patients seen in the University of Michigan Neurology Clinic between June 1996 and January 1997. To increase the size of the control group, we recruited additional consecutive nonepilepsy patients from those seen in the University of Michigan EMG Laboratory in February 1997. The third source was epilepsy subjects participating in an unrelated electroencephalography-polysomnography study between

TABLE 1. *Epworth sleepiness scale (subjects with moderate or high tendency to fall asleep under specific conditions)*

Activity	Epilepsy group (158 subjects) n (%)	Control group (68 subjects) n (%)	p
1. Sitting and reading	64 (41)	24 (35)	0.46
2. Watching TV	70 (44)	20 (29)	0.04
3. Sitting, inactive in a public place	26 (16)	4 (6)	0.03
4. As a passenger in a car for an hour without a break	51 (32)	19 (28)	0.52
5. Lying down to rest in the afternoon when circumstances permit	103 (65)	37 (54)	0.13
6. Sitting and talking to someone	11 (7)	3 (4)	0.47
7. Sitting quietly after lunch without alcohol	32 (20)	11 (16)	0.47
8. In a car, while stopped for a few minutes in traffic	8 (5)	1 (1)	0.21

June and December 1996 to characterize interictal epileptiform activity in relation to sleep depth. This source was selected because all these subjects had polysomnography performed routinely as part of a screen for the study, and the polysomnographic results could be correlated with survey findings. All patients were aged 18–65 years and had a history of recurrent unprovoked seizures consistent with International League Against Epilepsy criteria (10). Those referred primarily for a sleep disorder were excluded, as were those with pseudoseizures, cognitive, neurodegenerative, brainstem, spinal cord, upper airway, or pulmonary disorders, neoplasm, or psychosis. Subjects were given the survey to complete while waiting for their appointments. Greater than 90% of subjects consented to participate, resulting in a sample size of 158 epilepsy and 68 control subjects. The most common neurological conditions in the control subjects included headaches, peripheral neuropathy, vertigo, dystonia, tic disorder, facial pain, back pain, and lumbar radiculopathy.

Content of survey

Two published and previously validated self-administered questionnaires were used: the Epworth sleepiness scale (ESS) and the sleep apnea scale of the sleep disorders questionnaire (SA/SDQ). The ESS, our outcome measure, is an eight-item scale that measures a subject's general level of subjective daytime sleepiness (11). The ESS has high test-retest reliability ($r = 0.82$) and a high level of internal consistency (Cronbach's alpha = 0.88) (12). ESS scores increase with the severity of obstructive sleep apnea syndrome (OSAS) (13). Subjects are asked how likely they are to doze off or fall asleep in specific situations (never, slight, moderate, high; 0–3), as listed in Table 1. Total scores range from 0 to 24. In our analysis, a binary outcome measure (ESS $\leq$ 10 vs. ESS $\geq$ 10) was chosen because it was more clinically interpretable in assessing predictors of subjective sleepiness than raw ESS scores. An ESS score greater than 10 was used as a cutoff for excessive daytime sleepiness on the basis of our clinical experience and as suggested by the published literature. For example, in Johns (11), ESS scores greater than 10 were two standard deviations from the mean. In addition, in the study of Johns (11), all 27 patients with either narcolepsy or idiopathic hypersomnia and 12 of 13 patients with severe OSAS had ESS scores greater than 10.

The second validated measure, the SA/SDQ (14), is a 12-item survey that provides a measure of sleep apnea. The SDQ has high test-retest reliability ($r = 0.84$) and a high level of internal consistency (Cronbach's alpha = 0.855). Items are listed in Table 2. Total scores range from 0 to 60. Douglass et al. (14) using receiver operating characteristic curves, has suggested cutoff points for apnea of 36 for males and 32 for females, although these cutoffs await confirmation in larger scale studies. Because only a small number of subjects in our study reached these cutoffs, SDQ scores were analyzed as continuous variables rather than dichotomous variables.

TABLE 2. *Sleep apnea scale of the sleep disorders questionnaire (SA/SDQ)*

1. I am told I snore loudly and bother others.
2. I am told I stop breathing ("hold my breath") in sleep.
3. I awake suddenly gasping for breath, unable to breathe.
4. I sweat a great deal at night.
5. I have high blood pressure (or once had it).
6. I have a problem with my nose blocking up when I am trying to sleep (allergies, infections).
7. My snoring or my breathing problem is much worse if I sleep on my back.
8. My snoring or my breathing problem is much worse if I fall asleep right after drinking alcohol.
9. What is your current weight? (5 categories)
10. How many years were you a smoker? (5 categories)
11. How old are you now? (5 categories)
12. Body mass index (calculated from weight in kilograms/height in meters squared). (5 categories)

TABLE 3. *Comparison of predictors of sleepiness examined in the epilepsy and control subjects*

	Epilepsy group (158 subjects)	Control group (68 subjects)	p
Age (mean years ± SD)	43.1 ± 10.8	36.4 ± 11.3	0.0001
Women	93 (59%)	44 (65%)	0.41
SA/SDQ score (mean ± SD)	24.1 ± 6.8	23.2 ± 7.0	0.33
RLS[a]	55 (35%)	20 (29%)	0.43
Sedative medication	12 (7.6%)	15 (22%)	0.002
Hours of sleep (mean ± SD)	8.1 ± 1.8	8.4 ± 1.9	0.20

SA/SDQ, sleep apnea scale of the sleep disorders questionnaire; RLS, restless legs symptoms.
[a] Restless legs symptoms occasionally, often, or almost always.

Additional questions pertained to age, gender, medications, the presence and frequency of restless legs symptoms, and the average number of hours of sleep obtained each night. Restless legs symptoms (RLS) were assessed by the item, "How often do you have restless legs (crawling or aching feelings and inability to keep your legs still) when trying to get to sleep?", with responses of never, seldom, occasionally, often, or almost always. Sedative medications, including tricyclic antidepressants (amitryptiline, nortryptiline), narcotics, muscle relaxants, antihistamines, and neuroleptics (haloperidol, prolixin), were noted and incorporated into the analysis. Within the epilepsy group, survey questions assessed the number and type of antiepileptic medications, the epilepsy syndrome, the history of epilepsy surgery, the presence of sleep-related seizures, and whether the subject was seizure-free and, if not, the number of seizures per month. Epilepsy syndromes and medications were confirmed by reviewing each subject's chart.

Polysomnography

Polysomnography was performed on 27 epilepsy subjects, 20 of whom were participating in the University of Michigan Clinical Research Center neurophysiological study. The other seven subjects were referred for sleep studies by their physicians for clinical indications. Polysomnography was performed either in the University of Michigan Sleep Disorders Laboratory or in the Clinical Research Center. Overnight recordings were performed on 21-channel polygraphs or computerized electroencephalogram (EEG) systems (Telefactor, West Conshohocken, PA) and EEG, electrooculogram, submental electromyogram (EMG), nasal-oral airflow, respiratory effort, pulse oximetry, and anterior tibialis EMG were recorded. In our laboratory, an apnea is defined by a decrease in airflow or effort to 20% or less of baseline for 10 or more seconds. A hypopnea is defined by any decrease in airflow or effort that is accompanied by either EEG signs of arousal [defined by American Sleep Disorders Association criteria (15)] or a 4% or greater decrease in oxygen saturation. The respiratory disturbance index (RDI) is calculated by dividing the number of apneas and hypopneas by the total number of hours asleep. In our laboratory, periodic leg movements are defined by movements in the anterior tibialis channel of 0.5–5.0 seconds in duration, in trains of at least three movements with intermovement intervals of 4 to 120 seconds.

Data analysis

For all statistical tests, the level of significance was set at $\alpha = 0.05$. All statistical tests were performed using the SAS statistical analysis package (SAS Institute Inc., Cary, NC). Crude ESS scores and responses to individual items on the ESS scale for the epilepsy and control groups were compared using chi-square tests (Table 1). Major predictor variables between the epilepsy and control groups were compared using chi-square and two-tailed Student's t tests (Table 3). Internal consistency of the ESS and SA/SDQ survey items was assessed by the Cronbach's alpha statistical procedure. Logistic regression models were constructed to compare adjusted ESS scores for the epilepsy and control groups and to calculate odds ratios for predictors of elevated ESS score (>10) in the combined groups and in the epilepsy subgroup. Continuous variables included age, hours of sleep on a weekday night, and SA/SDQ scores. Categorical variables included gender, sedative medications, and RLS (binary variable for never or seldom vs. occasionally, often, or almost always). In the epilepsy group, additional variables included the epilepsy syndrome (partial vs. generalized), diurnal variation of seizures (binary variable for seizures during sleep or during sleep and wakefulness vs. not during sleep), history of epilepsy surgery, whether the subject was seizure-free (continued seizures vs. seizure-free for 1 year or more), the number of seizures in the last month, and the number and type of antiepileptic medications. Because only three subjects were not on antiepileptic medications, a binary variable was used for the number of medications (none or monotherapy vs. polytherapy). The relationship between SA/SDQ score and RDI (>5 vs. ≤5) was assessed by a two-tailed student's t test.

RESULTS

Univariate and bivariate analyses

One hundred fifty-eight epilepsy and 68 control subjects participated in the survey. Forty-five (28%) epilepsy and 12 (18%) control subjects had elevated ESS scores (>10). This unadjusted analysis showed a nonsignificant trend toward epilepsy patients having elevated ESS scores ($p = 0.08$). Table 1 displays the number and percentage of epilepsy and control subjects who reported a moderate or high tendency to fall asleep under specific conditions.

Seven men (4%) and nine women (6%) in the epilepsy group and two men (3%) and three women (4%) in the control group met the SDQ cutoff scores for sleep apnea (36 for men and 32 for women) (14). Loud disruptive snoring was reported usually or always in 27 (17%) of the epilepsy subjects, and 8 (12%) of the control subjects, with witnessed apnea reported usually or always in 6 (4%) of the epilepsy subjects and 3 (4%) of the control subjects.

Internal consistency was high for the ESS in both the epilepsy (Cronbach's alpha = 0.81) and control subjects (Cronbach's alpha = 0.84) (12). Internal consistency for the SDQ was good in both groups (Cronbach's alpha = 0.72 for epilepsy patients and 0.70 for control patients), although lower than reported previously in the literature (14).

Table 3 compares age, gender, SA/SDQ scores, RLS (occasionally, often, or almost always), the presence of sedative medications, and the hours of sleep on a weekday night in the epilepsy and control patients. The two groups were comparable on all variables except age and sedative medications. The epilepsy subjects were significantly older than the control subjects, and the controls were significantly more likely to be taking sedative medications. Table 4 shows the distribution of variables for the epilepsy subjects. As the data illustrate, the majority of subjects continued to have seizures, and all but three subjects were on antiepileptic medications.

TABLE 4. *Additional predictors of sleepiness examined in the 158 epilepsy subjects*

Predictor	n (%)
Epilepsy syndrome	
Partial, 2° generalized	108 (68)
Generalized	50 (32)
Seizures during sleep	70 (44)
History of epilepsy surgery	28 (18)
Seizure-free for 1 year or longer	39 (25)
Seizures/month	
None	63 (40)
1–5	55 (35)
>10	40 (25)
Number of medications	
None	3 (2)
1	92 (58)
2	52 (33)
>3	11 (7)
Antiepileptic medication[a]	
Carbamazepine	73 (46)
Phenytoin	61 (39)
Valproate	29 (18)
Gabapentin	27 (17)
Lamotrigine	25 (16)
Phenobarbital	11 (7)
Clonazapam	6 (4)
Primidone	4 (2.5)

[a] Total percent exceeds 100 because 63 patients were on more than one medication.

Multivariable analysis

Logistic regression models were constructed to examine the effects of age, gender, SA/SDQ scores, RLS, and epilepsy on elevated ESS score (ESS > 10). Examination of the data suggested that SDQ scores were linear but that age was nonlinear. Specifically, subjects younger than 30 or older than 45 years were more likely to have elevated ESS scores than subjects aged 30–45 years, a result consistent with previous work showing elevated multiple sleep latency test (MSLT) scores in subjects aged 30–48 years compared to younger and older normal volunteers (16). This nonlinear pattern for age also occurred in the epilepsy and control groups when these groups were analyzed separately. Therefore, three age groups were created and categorical variables were constructed for age categories 30–45 and older than 45 years, with younger than 30 years as a reference group.

In a logistic regression model of all 226 subjects, adjusted for age group and gender, epilepsy subjects were more likely to have elevated ESS scores (odds ratio = 2.2, $p < 0.05$), and subjects older than 45 years were more likely to have elevated ESS scores (odds ratio = 2.5, $p < 0.04$). However, when RLS and SA/SDQ score were included in the model along with age group, gender, and epilepsy, significant predictors of elevated ESS score included SA/SDQ score (odds ratio for a 10-point increase in SA/SDQ score = 2.2, $p < 0.005$) and RLS (odds ratio = 2.6, $p < 0.007$), whereas presence of epilepsy conferred only a nonsignificant trend (odds ratio = 2.0, $p = 0.08$) and age group was not significant ($p > 0.10$). Including the variables of sedative medications or the number of hours of sleep in the analysis did not change the results significantly.

In the subgroup of 158 epilepsy subjects, logistic regression models were constructed to assess the following variables as predictors of elevated ESS score: 1) SA/SDQ score, 2) RLS, 3) number of antiepileptic

medications, 4) specific types of antiepileptic medications, 5) diurnal variation of seizures, 6) history of epilepsy surgery, 7) seizure-free for greater than 1 year, 8) number of seizures in the last month, 9) sedative medications, 10) hours of sleep. The only significant predictors were SA/SDQ score and RLS. In a logistic regression model adjusting for age, gender, and sedative medications, significant predictors of an elevated ESS score included SA/SDQ score (odds ratio for a 10-point increase in SA/SDQ score = 1.9, $p < 0.05$), RLS (odds ratio = 3.4, $p = 0.002$), and age group 30–45 years (odds ratio = 0.33, $p = 0.03$) but not the number or type of antiepileptic medication (odds ratio = 1.3, $p = 0.53$) or the presence of sedative medications (odds ratio = 0.5, $p = 0.32$).

Polysomnography

Of 27 epilepsy subjects undergoing sleep studies, 11 subjects had RDI > 5. RDI values were between 11 and 30 for two subjects and were greater than 50 for two subjects. SA/SDQ scores were significantly elevated in the subjects with RDI > 5 (29.5 ± 5.2, mean ± standard deviation) compared to those with RDI ≤ 5 (21.9 ± 5.5, $p < 0.002$). Four of the 27 patients had more than 10 periodic limb movements (PLMs) per hour with arousals. Three of these four subjects reported having RLS symptoms often or almost always; the fourth subject reported never having RLS symptoms. Of 14 subjects reporting RLS symptoms occasionally, often, or almost always, six had PLMs recorded.

DISCUSSION

In this study, we found that 1) subjective EDS is common in both epilepsy subjects and controls, and 2) symptoms of treatable sleep disorders are associated with subjective sleepiness in both epilepsy and control subjects. Although epilepsy subjects were more likely to have elevated ESS scores compared to controls after adjusting for age and gender, the statistical significance of this finding diminished when the additional variables of RLS and SA/SDQ score were incorporated into the logistic regression model. Within the subgroup of epilepsy subjects, symptoms supporting the presence of treatable sleep disorders (e.g. RLS and SA/SDQ scores) were stronger predictors of sleepiness than seizure frequency or the number or type of antiepileptic medications.

This study is unique in that we examined the relative contributions of symptoms of sleep disorders, antiepileptic medications, seizure control, and other variables to a validated measure of sleepiness. The use of logistic regression models permitted us to adjust for several covariates in our model, including age and gender, and to assess the importance of sleep disorder symptoms relative to other epilepsy variables. Previous studies (2–4,17) have examined subjective sleepiness in epilepsy patients, although none have used logistic regression models to determine predictors of sleepiness, and only two (4,17), in abstract form, included a validated scale to measure sleepiness. Hoeppner et al. (2) inquired about "awakening fatigue" in a self-reported sleep questionnaire given to 30 adult epileptic subjects. The majority of epileptic subjects described feeling mildly tired or very tired upon awakening. Vaughn et al. (4) reported complaints of EDS in 61% of 154 epilepsy patients given the ESS. Foldvary et al. (17) compared ESS scores in 17 consecutive patients admitted to an epilepsy monitoring unit and 26 subjects with obstructive sleep apnea (OSA) having apnea-hypopnea indices of 20 or greater on overnight polysomnography. They found that epilepsy subjects had significantly lower mean ESS scores than subjects with OSA. However, the incidence of specific sleep disorders, including OSA, in the epilepsy subjects was not assessed by questionnaire data or polysomnography. Frost et al. (3) found that sleepiness was reported more frequently by epilepsy subjects than by nonepileptic controls of comparable age and gender, although a validated scale was not used. In the Frost et al. (3) study, the epilepsy subjects and controls did not differ in their reports of snoring, apnea, or leg symptoms. In our study, after controlling for age, gender, RLS, and SA/SDQ score, epilepsy patients were only somewhat more likely than other neurology patients to have elevated ESS scores, presumably because coexisting sleep disorders were prevalent in both patient groups.

In this study, younger (aged 18–29) and older (aged 46–65) subjects were more likely to have elevated ESS scores than subjects 30–45 years old. This finding remained consistent throughout the analyses. In a study relating MSLT to age, Carskadon and Dement (16) found that the percentage of MSLTs showing a mean sleep latency less than 10 was 35, 20, and 66% in groups of subjects aged 17–25, 30–48, and 62–77 years, respectively. Although we used ESS scores rather than MSLTs to quantify sleepiness, and although our age groups differed slightly from this previous investigation, our results are consistent with those of Carskadon and Dement (16). Levine et al. (18) reported a shorter mean sleep latency in younger subjects (aged 18–29) compared to older subjects (aged 30–80) and suggested sleep restriction as a possible etiology. We could not directly assess the possibility of sleep restriction in our study. To explore the possibility that reported hours of sleep might explain age differences in elevated ESS score, we adjusted for the reported number of hours of sleep per night in our analysis of

age group as a predictor of elevated ESS score. Even after adjustment for hours of sleep, our results showed the same consistent pattern, with subjects aged 30–45 years being least likely to have an elevated ESS score.

This study has several limitations. First, polysomnography was not available for the majority of subjects to confirm the presence of OSA. In those subjects having polysomnography, however, we found a significant difference in SDQ scores between subjects with RDI > 5 and those with RDI ≤ 5. The SA/SDQ studies also used polysomnography to determine sensitivity and specificity for its scales (14). Second, in this study, RLS may have been overestimated, as we asked only one question related to restless legs symptoms. We did not follow-up on the questionnaire responses to determine if subjects met the minimal criteria defined by the International Restless Legs Syndrome Study Group, which include 1) desire to move the extremities, often associated with paresthesias/dysesthesias; 2) motor restlessness; 3) worsening of symptoms at rest with at least temporary relief by activity, and 4) worsening of symptoms in the evening or night (19). Polysomnographic data supported a relationship between PLMs and RLS, in that PLMs were present in six of 14 subjects reporting RLS, and RLS was present in three of four subjects with more than 10 PLMs per hour with arousals. Polysomnographic data, however, have limited sensitivity and specificity in diagnosing RLS, as PLMs are absent in approximately 20% of patients with idiopathic RLS and may occur independently of RLS. Finally, follow-up studies are needed to relate ESS scores to polysomnography and to detailed evaluation for sleep disorders. In addition, MSLT data may be useful in follow-up studies, as ESS scores correlate only modestly (20,21) or not at all (22) with MSLT scores and these tests provide complementary information.

Recognizing and treating sleep disorders in epilepsy patients has important implications not only for improving the quality of life of this patient population but also for seizure control. Several case series have reported an improvement in seizure control when OSA was treated (6–9). A likely mechanism may be improvement in sleep fragmentation and deprivation resulting from treatment. Patients may also tolerate higher doses of existing medications or additional medications needed to control seizures once coexisting causes of sleepiness are treated.

In summary, our data support the premise that symptoms of treatable sleep disorders are stronger predictors of subjective sleepiness than the number or type of antiepileptic medications or the frequency of seizures. Before attributing sleepiness in epilepsy patients to antiepileptic medications, clinicians should consider the possibility of an underlying sleep disorder.

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