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SPECIAL SECTION

Sleep polysomnography as a predictor of recurrence in children and adolescents with major depressive disorder

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Abstract

Adults with major depressive disorder (MDD) demonstrate certain sleep polysomnographic abnormalities, including sleep continuity disturbances, reduced slow-wave sleep, shortened rapid eye movement (REM) latency, and increased REM density. Findings of sleep EEG studies in depressed children and adolescents have yielded conflicting results, possibly because of methodological variations across the studies. Generally, however, studies have demonstrated that depressed children and adolescents exhibit less sleep continuity and non-REM sleep differences in comparison with control subjects than do adults. Thus, results from adult sleep polysomnography studies cannot necessarily be generalized to children and adolescents. Depressed adults who have reduced REM latency during the symptomatic episode appear more likely to have a relapse once treatment is discontinued than those with normal REM latency. No studies of the relationship between sleep polysomnographic variables and clinical course have been reported in depressed children and adolescents. Data for baseline clinical variables and 3 nights of sleep polysomnography were examined in 113 depressed children ($\leqslant$ 12 yr; $n = 51$) and adolescents ($\geqslant$ 13 yr; $n = 62$) (56 in-patients and 57 outpatients) where data was available on at least 1 yr of naturalistic follow-up. Subjects came from 2 studies of sleep polysomnography in children and adolescents with MDD. Clinical course was assessed using the Kiddie-Longitudinal Interval Follow-Up Evaluation (K-LIFE). This interview was used to define recovery from the index episode of MDD and recurrence, a new episode of meeting full criteria for MDD. Clinically, within 1 yr of initial evaluation 102/113 subjects had recovered from their index episode of depression (minimal or no symptoms for 60 d). Of the 102 subjects who recovered, 36 (35.3%) had a recurrence of MDD. The majority of subjects (55%) who had a recurrence were not on medication at the time of recurrence. Subjects who had a recurrence were more likely to report suicidal thoughts or attempts at baseline compared to those without a recurrence (67 vs. 37%; $F = 8.77$; $p = 0.004$). On baseline sleep polysomnography, subjects with a later recurrence had decreased sleep efficiency and delayed sleep onset (sleep latency $>$ 10 min). Probability of recurrence at 12 months was 0.39 compared to 0.15 in subjects with non-delayed sleep onset ($p = 0.005$). Baseline suicidal ideation and sleep dysregulation on sleep polysomnography predicted recurrence in a large sample of depressed children and adolescents. Depression in children and adolescents is frequently a chronic, recurrent illness. Factors that can predict clinical course are important in increasing our understanding of depression in this age group.

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Introduction

Depression, which is a leading cause of morbidity and mortality in children and adolescents, is estimated to occur in up to 8.3% of young people (Burke et al., 1991; Fleming and Offord, 1990; Kashani et al., 1987a,b; Lewinsohn, 1986, 1993, 1994; Shaffer et al., 1996). It is associated with increased family problems and school failure and, particularly in adolescents, suicide, substance abuse, and truancy, yet it frequently remains undetected and untreated.

Recovery from an index episode of major depression is remarkably consistent across clinical samples, with over 90% of depressed child and adolescent outpatients (Kovacs et al., 1984a; McCauley et al., 1993), child and adolescent in-patients (Emslie et al., 1997a; Strober et al.,

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1993), and non-referred adolescents (Keller et al., 1988) having recovered from an episode of MDD within 1–2 yr. Recurrences (new episodes of depression) are reported in 25–72% of depressed children and adolescents followed for 3–8 yr, with similar rates for in-patients (Emslie et al., 1997a; Garber et al., 1988), outpatients (Goodyer et al., 1991; Kovacs et al., 1984b; McCauley, 1993; Rao et al., 1995), and non-referred community samples (Fleming et al., 1993; McGee and Williams, 1988).

While several factors have been identified as predictors of recurrence in adults, little information about predictive factors of recurrence in children is available. In adults, higher recurrence rates are associated with three or more previous episodes (Keller et al., 1982; Maj et al., 1992), more severe index episodes (Gonzales et al., 1985), presence of psychotic features (Copeland, 1983; Schatzberg and Rothschild, 1992), family and social dysfunction, early age of onset of illness, and double depression (defined as a major depressive disorder superimposed on dysthymic disorder) (Gonzales et al., 1985; Keller et al., 1983). In children, older age, non-Caucasian, psychotic symptoms, and greater symptom severity have been associated with greater recurrence (Emslie et al., 1997a).

Recurrence of MDD is difficult to predict from clinical features at initial assessment. Attempts have been made to determine if the sleep polysomnogram predicts the clinical course of MDD. Four sleep polysomnographic features are consistently found in adults with MDD: (1) sleep continuity disturbances (e.g. prolonged sleep latency and decreased sleep efficiency); (2) decrease in slow wave sleep [stages 3 and 4 non-rapid eye movement (NREM) sleep]; (3) shortening of time from sleep onset to appearance of first rapid eye movement (REM) (i.e. REM latency); and (4) increase in REM activity (e.g. increased REM time and REM density) (Kupfer and Foster, 1972; Kupfer et al., 1985; Rush et al., 1982, 1986).

Depressed adults with shortened REM latency have been reported to be more likely to respond to antidepressant treatment vs. placebo and more likely to relapse if medication is discontinued (Giles et al., 1987; Rush et al., 1989). There have also been some suggestions that REM sleep abnormalities may occur prior to the development of depression in at-risk samples (Giles et al., 1998).

In children and adolescents with MDD, sleep polysomnographic abnormalities are less consistently reported. However, depressed children and adolescent in-patients have been shown to demonstrate sleep continuity disturbances, an increase in REM pressure (shortened REM latency and increased REM sleep percentage), but not disturbances in slow wave sleep (Dahl et al., 1990; Emslie et al., 1990, 1994; Kutcher et al., 1991; Lahmeyer et al., 1983). The results in outpatients have been generally negative (Goetz et al., 1987; Puig-Antich et al., 1982). Perhaps in-patient units provide a stable sleep/wake cycle prior to the sleep study, thereby decreasing the inter-individual variance. While outpatients as a group show less sleep architecture differences compared to normal controls, some subgroups of outpatients clearly have decreased REM latency and increased sleep latency (Dahl et al., 1991).

No data are available on sleep polysomnographic variables as predictors of clinical course in depressed children and adolescents. This study examines whether sleep polysomnographic variables assessed during the index episode of MDD in two samples of children and adolescents predicted recurrence of depression during a 1-yr naturalistic follow-up.

Method

Subjects

All eligible subjects were identified from two studies of sleep polysomnography in depressed children and adolescents (in-patients, $n = 70$; outpatients, $n = 80$) conducted between 1984 and 1995. In addition, subjects had to have data of at least 1 yr of naturalistic follow-up. The sleep polysomnographic data from the in-patients have been published previously (Emslie et al., 1990, 1994). For the in-patients, the follow-up data were collected retrospectively 1–5 yr after the initial sleep study. These follow-up results have been published previously (Emslie et al., 1997a), with data available on 56 out of 70 in-patients. No baseline demographic or clinical differences were found between those followed and those not followed for at least 1 yr, with the exception of socio-economic status (SES). Subjects who were followed for at least 1 yr had a higher SES than those who dropped out after the sleep evaluation (3.0 ± 1.0 vs. 2.3 ± 1.0; $p = 0.022$). The depression severity of the two groups was similar. The outpatient sample was from our double-blind, placebo-controlled study of fluoxetine (Emslie et al., 1997b) who were followed naturalistically for 1 yr (Emslie et al., 1998). Of the 80 outpatients with sleep polysomnography, 57 had 1-yr follow-up data. No baseline demographic or clinical differences were found between those who had 1-yr follow-up data and those who did not. The sample, therefore, consisted of 113 children ($n = 51$) and adolescents ($n = 62$) aged 8–18, with MDD (DSM-III-R), with both baseline sleep polysomnographic assessment and 1 yr of naturalistic clinical follow-up data.

Diagnosis

Initial diagnoses, including comorbid conditions, were

similar for both groups (Emslie et al., 1990, 1994, 1997b). In summary, the study and informed consent were approved by the Institutional Review Board at the University of Texas Southwestern Medical Center of Dallas. After written informed consent was obtained from parents and informed assent was obtained from patients, they were interviewed separately by a trained, experienced research assistant using a structured interview, the Diagnostic Interview for Children and Adolescents (DICA; Herjanic and Reich, 1982; Welner et al., 1987).

Following the initial screening, patients and parents were then independently evaluated by two psychiatrists with extensive experience in early onset mood disorders. In these interviews with parents and patients (separately), the DICA was reviewed, and the clinician completed the Brief Psychiatric Rating Scale for Children (BPRS-C; Overall and Pfefferbaum, 1982), an instrument to assess presence of a variety of psychiatric symptoms and conditions, and Clinician Bellevue Index of Depression (BID; Petti, 1985), which assesses severity of depression. The initial structured interview and the clinician assessments were completed over 2 wk. Final diagnoses were confirmed in a consensus conference utilizing the data from the 2 interviews. In addition, during the consensus conference, clinical symptoms, severity, family psychiatric history, and course of illness were reviewed. Suicidal behaviour was coded on a 5-point scale (1 = none, 2 = morbid thoughts/death wishes, 3 = suicidal thoughts, 4 = suicidal plans, 5 = suicidal attempts), and was based on all available clinical information provided during the evaluations (e.g. clinician, parent, and child reports). Eligible patients then underwent 3 nights of polysomnographic (PSG) evaluation.

Polysomnographic procedures

A minimum 14-d, medication-free, alcohol-free, and caffeine-free period was required prior to the PSG evaluation. No medication of any type except acetaminophen was allowed during this period. For the week prior to sleep study, alcohol use was proscribed and caffeine was restricted to one 10-oz. beverage or other consumable containing caffeine before 23:00 hours. All subjects completed a sleep history questionnaire and a detailed sleep log describing the 5 nights before the sleep study. During these 5 nights and continuing throughout the sleep evaluation, set bedtimes and rise times were established for each subject (at least 7 h per night) to optimize sleep sufficiency (i.e. bedtime at 22:00 hours with rise time at 06:00 hours would be adhered to for this period). If the sleep log for any subject revealed an average variation in bedtime and rise times exceeding 1 h during the pre-laboratory schedule, the subject was excluded from the study.

The PSG recordings, which used EEG electro-oculogram (EOG) and submental-electromyogram (EMG) lead placements, were carried out on three consecutive nights in the Sleep Study Unit of the Department of Psychiatry, University of Texas Southwestern Medical Center of Dallas. The procedures have been described in detail elsewhere (Armitage et al., 1994). The electrode montage included electroencephalograms (EEG) recorded from C3 and C4 and right and left EOGs with contralateral mastoid reference, and a bipolar chin-cheek EMG. Tibialis leads, chest and abdomen respiration bands and a nasal–oral thermistor were also used on night 1 only to rule out unreported or unknown independent sleep disorder. Periodic limb movements, apnoea or hypopnoea events of $\geqslant 10$ per hour excluded participants from further study. This occurred in one patient. Additionally, one patient with narcolepsy was removed from the study.

Sleep stages were scored in half-minute epochs in strict accordance with standard sleep scoring criteria (Rechtschaffen and Kales, 1968) by sleep technicians trained to better than 90% agreement on an epoch-by-epoch basis. Sleep technicians were blind to the clinical status of the subjects, as normal control subjects were being slept concurrently for additional data. A number of sleep architectural measures were derived from the stage-score data. The onset of persistent sleep (sleep latency) was defined as the time of the initial half-minute of sleep leading the first 10 consecutive minutes of any non-REM (NREM) sleep recording, including stage 1 sleep, containing no more than 2 min of awake or movement time, *or* the time of the first REM-sleep epoch, whichever is sooner. REM latency was equal to the number of minutes, including waking, from persistent sleep onset to the first appearance of stage REM. REM density, an index of phasic eye movement activity in REM sleep, was scored on a 0- to 4-point scale (0 = no eye movements, 4 = eye movements in $> 75\%$ of epoch) for each epoch and averaged per minute of REM. The total sleep period (TSP) was defined as the minutes of sleep time from persistent sleep onset to the morning awakening. Sleep efficiency was defined as the TSP divided by the total time in bed from lights out to lights on (TIB). In addition, the amount of each REM and NREM sleep stage and awake and movement times were computed as a percentage of TSP.

Records were scored in strict accordance with accepted sleep scoring conventions by sleep technicians blind to the clinical status of the subjects. The nightly REM latencies were consensus-scored by all laboratory technicians (4–6) whose overall sleep-scoring reliability exceeded 90% for all stages.

Follow-up evaluations

The clinical follow-up of subjects has been previously described (Emslie et al., 1997a, 1998). In summary, during the follow-up evaluations, patients and parents were interviewed using the Kiddie-Longitudinal Interval Follow-Up Evaluation (K-LIFE), a modification of the LIFE (Keller et al., 1987). During the interview, course of depressive symptoms were assessed during the 6-month intervals. Additionally, comorbid diagnosis and treatment were assessed. For in-patients the K-LIFE was completed once from 1 to 5 yr after the initial evaluation. In addition to the interview, medical records were also used (see Emslie et al., 1997a for a full description). Outpatients were interviewed twice, at 6 months and at 1 yr following initial assessment (see Emslie et al., 1998 for a full description).

For both groups, the severity of MDD during the follow-up period was coded using the criteria in the K-LIFE (6 = severe, 5 = definite criteria, 4 = marked symptoms, 3 = partial remission, 2 = residual symptoms, 1 = usual self). Changes in status, i.e. change in MDD rating, improvement or development of other disorders or treatment, were coded by dates. When this change was approximately identified, then the midpoint of that time was used as the change point, e.g. patient meeting criteria for MDD again in mid-September was coded 15 September. Episode length, time to recovery, etc., were all coded in days, but are reported in weeks.

For describing the course of depressive symptoms during the follow up, we used the terms proposed by Frank et al. (1991). The level of symptom rating was the MDD criteria from the K-LIFE. *Recovery* was defined as an asymptomatic period of at least 60 d (MDD K-LIFE rating of 1 or 2). *Recurrence* was defined as an episode of depression after recovery. A subsequent *episode* of depression was defined as an MDD K-LIFE rating of 5 or greater for 14 d. As proposed by Frank et al. (1991), these terms were assessed independent of treatment.

Statistical analyses

All data were analysed utilizing a Statistical Analysis Software™ (SAS Institute Inc.) statistical program. Comparison of the various sleep and clinical parameters of the subjects were done primarily using analysis of variance (ANOVA). χ^2 analyses compared discontinuous variables across groups. In addition, multivariate analysis of variance (MANOVA) were completed to determine interaction effects, and a survival curve was performed to determine the probability of recurrence based on sleep latency. Since the two separate samples (in-patient and outpatient) had different lengths of clinical follow-up, we made the 2 samples comparable by using only the first year follow-up data for in-patients.

Results

Clinically, within 1 yr 102 out of 113 subjects had recovered from their index episode of MDD (i.e. no or minimal symptoms for 2 months). Of the 102 who recovered, 36 (35.3%) had a recurrence (i.e. a new major depressive episode as evidenced by a score of 5 or 6 on the LIFE for $\geqslant$ 2 wk).

In comparing those who recovered without a recurrence ($n = 66$) and those who recovered but who had a recurrence ($n = 36$), no significant differences were found in age (12.2 ± 2.4 vs. 12.8 ± 2.3 yr), gender (43.9% female vs. 50% female), SES (2.5 ± 1.1 vs. 2.4 ± 1.1), and ethnic grouping (92.4 vs. 80.6% Caucasian). Similar proportions of in- and outpatients were present in each group (66 vs. 63% without a recurrence; 34 vs. 37% with a recurrence, respectively).

Table 1 shows the clinical characteristics at initial evaluation for subjects with and without recurrences. No differences were found for age of onset of illness, length of illness, duration of current episode, number of previous episodes or severity of illness as measured by CGI severity, BID and BPRS-C depressive items. Those with recurrences were more likely to report suicidal plans or attempts at initial evaluation than those without recurrences (67 vs. 37%, $F = 8.77$, $p = 0.004$). Current comorbid diagnoses also did not differ between the groups, with both groups having similar numbers, types, and severity (BPRS-C total score) of comorbid diagnoses. Anxiety disorders tended to be less common in those with recurrences (27.8 vs. 47%, $F = 3.63$, $p = 0.059$), but the BPRS-C anxiety scores were not different (9.6 ± 2.7 vs 9.4 ± 2.5 ns).

Table 2 describes the clinical course of the two groups. Time to recovery was not different for the groups. The majority of both groups recovered on medication. Interestingly, recurrence occurred on antidepressants in 16 of 36 (44.4%) of patients. As expected, the recurrence group spent more time in a MDD episode from beginning of treatment (MDD rating of 5 to 6, 23 vs. 11%, $F = 23.33$, $p < 0.0001$). Also, the recurrence group spent less time well (MDD rating of 1 or 2, 59.6 vs. 76.0%, $F = 16.81$, $p = 0.0001$). Because of the naturalistic nature of the treatment, it is not possible to examine specifically the impact of medication, though both groups were on medication for an equivalent amount of time during the 12-month period (59.8 vs. 52.0%). In all the patients who had a recurrence, the average time to recurrence was 27 ± 13.4 wk from beginning recovery and 38 ± 13.4 wk from initial evaluation.

Table 1. Clinical characteristics

	No recurrence ($n = 66$) Mean (S.D.)	Recurrence ($n = 36$) Mean (S.D.)
Age of onset	11.9 (2.5)	12.5 (2.3)
Length of illness (months)	18.9 (23.5)	21.6 (23.1)
Duration of episode (wk)	17.8 (15.9)	18.3 (17.4)
Number of episodes	1.7 (0.9)	1.8 (0.9)
1	50.0% (33)	44.4% (16)
2	30.3% (20)	39.9% (14)
3+	19.7% (13)	16.7 (0.9)
CGI severity	4.8 (0.8) ($n = 42$)	4.9 (0.8) ($n = 26$)
BID	71.3 (17.1)	74.8 (9.5)
BPRS depressive items (range 0–21)	12.7 (2.5)	13.1 (2.7)
Suicidal behaviour	2.9 (1.4)	3.7 (1.3)**
None	20.0% (13)	8.3% (3)
Morbid/wishes/thoughts	43.1% (28)	25.0% (9)
Plans/attempts	36.9% (24)	66.7% (24)**
No. current comorbid diagnoses	1.3 (1.1)	1.3 (1.0)
0	25.8% (17)	25.0% (9)
1	34.9% (23)	27.8% (10)
2	19.7% (13)	36.1% (13)
3+	19.7% (13)	11.1% (4)
Current comorbid diagnoses		
None	25.8% (17)	25.0% (9)
Dysthymia	36.4% (24)	36.1% (13)
Behaviour	37.9% (25)	33.3% (12)
ADHD	21.2% (14)	22.2% (8)
Anxiety	47.0% (31)	27.8% (10)
BPRS-C total score	49.7 (11.3)	47.7 (15.5)
BPRS anxiety items (range 0–21)	9.6 (2.7)	9.4 (2.5)

* $p \leqslant 0.05$; ** $p \leqslant 0.01$; *** $p \leqslant 0.001$.

Table 3 examines sleep architectural variables for those with and without recurrences. All variables are from the means of nights 2 and 3 unless otherwise stated. Those with recurrences within 1 yr had a longer sleep latency, approaching significance ($F = 3.90$, $p = 0.051$), and lower sleep efficiency ($F = 9.5$, $p = 0.003$) than those without recurrences. Neither REM nor NREM sleep stages differed between the groups, including REM latency.

Of the 66 without recurrence during the 1-yr observation period, 15 later went on to have a recurrence (inpatients only). Data are not available for outpatients past the first year. If we exclude these 15 subjects ($n = 51$) from the group without recurrence, then sleep latency was significantly shorter in the 'non-recurrence group' (14.2 ± 10.7 vs. 22.7 ± 16.1, respectively; $F = 8.77$, $p = 0.004$).

Sleep latency was divided into 'short' ($\leqslant 10$ min) vs. 'long' (> 10 min). Nine out of 36 (25%) subjects who had a recurrence had a sleep latency of $\leqslant 10$ min as opposed to 31/66 (47%) with no recurrence ($p = 0.05$). Figure 1 shows the survival curves to recurrence for the 2 groups based on short or long sleep latency. Group 1 includes subjects who had a sleep latency $\leqslant 10$ min; group 2 includes subjects with sleep latency > 10 min. By 12 months, the probability of recurrence is 0.39 for subjects with sleep latency > 10 min compared with only 0.15 for those with sleep latency $\leqslant 10$ min (2-tail Fisher's exact test; $p = 0.005$).

Finally, a multivariate discriminant analysis was con-

Table 2. Clinical course (recovery/recurrence)

	No recurrence ($n = 66$) Mean (S.D.)	Recurrence ($n = 36$) Mean (S.D.)
Time to recovery (wk)	11.9 (10.8)	10.9 (9.7)
Time to recurrence from recovery (wk)	na	27.2 (13.4)
Time to recurrence from start of study (wk)	na	38.2 (13.4)
Episode length[a] (wk)	29.7 (19.4)	29.2 (19.1)
Percent time well[b]	76.0% (19.4)	59.6% (19.3)***
Percent time depressed[c]	11.0% (10.1)	23.3% (15.6)***
Percent time on medication	59.8% (38.3)	52.0% (38.3)
Recovered while taking medication	75.8%	77.8%
Recurred while taking medication	na	44.4%

[a] Episode length: length of episode at initial evaluation plus time to recovery.
[b] Percent time well: MDD rating of 1 or 2 on K-LIFE.
[c] Percent time depressed: MDD rating of 5 or 6 on K-LIFE.
* $p \leqslant 0.05$; ** $p \leqslant 0.01$; *** $p \leqslant 0.001$.

Table 3. Sleep polysomnographic characteristics of subjects with or without recurrence (means of nights 2 and 3)

	No recurrence ($n = 66$) Mean (S.D.)	Recurrence ($n = 36$) Mean (S.D.)
Sleep continuity variables		
Sleep latency (min)	16.4 (15.0)	22.7 (16.1)
Total sleep period (min)	510.6 (40.0)	512.5 (42.1)
Sleep efficiency (%)	95.8 (3.1)	93.6 (4.0)**
Total sleep time (min)	496.4 (39.5)	495.5 (40.3)
Sleep stage variables		
Stage 1 (%)	10.2 (6.1)	10.1 (4.4)
Stage 2 (%)	46.9 (8.7)	47.5 (7.5)
Stage 3 (%)	10.8 (5.8)	10.2 (4.9)
Stage 4 (%)	9.7 (6.0)	9.2 (5.6)
Delta (stages 3 and 4) (%)	20.5 (9.4)	19.5 (8.4)
Awake movement (%)	2.8 (1.6)	3.3 (1.5)
REM variables		
REM (%)	19.4 (5.4)	19.4 (3.7)
REM density (min)	2.6 (0.6)	2.4 (0.5)
REM latency (min)	92.1 (36.1)	89.8 (33.7)
Shortest REM latency[a] (min)	73.7 (26.7)	70.6 (19.4)

[a] Shortest REM latency: shortest REM latency of any of the 3 nights of recording.
* $p \leqslant 0.05$; ** $p \leqslant 0.01$; *** $p \leqslant 0.001$.

ducted to determine whether four predictors – suicidal behaviour, sleep efficiency, sleep latency (continuous), and sleep latency disturbance (yes/no based on 10-min cut-off) – could predict recurrence during the 1-yr follow-up. The overall Wilks' lambda was significant [$\Lambda = 0.88$, χ^2 (2, $n = 99$) = 11.9, $p = 0.003$], indicating that two predictors (suicide and negative sleep efficiency) differentiated between the recurrence and non-recurrence groups (11.7% of the variance accounted for). Based on the coefficients used, positive suicidal behaviour (plans or attempts) at baseline and negative sleep efficiency demonstrated the strongest relationship with the discriminant function, while sleep latency (continuous variable) and poor sleep latency (dichotomous variable) were not

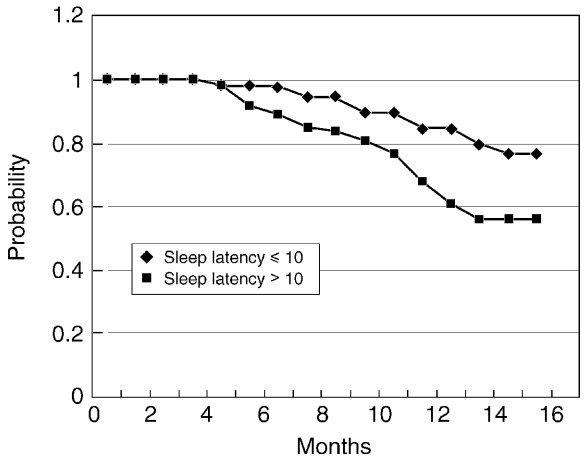

Figure 1. Survival curve based on short vs. long sleep latency.

significant predictors. The discriminant analysis correctly predicted 68% of the cases (best prediction was for the non-recurrence at 86.4%).

Conclusion

Most (90%) children and adolescents with MDD (both in-patients and outpatients) recovered from the index episode, with an average time to recovery of approx. 2.5 months. However 36% had a new episode of MDD within 1 yr of the initial assessment. While severity of illness and comorbid conditions were not different between the two groups, suicidal ideation at baseline was significantly higher in the recurrence group.

Contrary to our expectations, no differences in REM sleep measures in the two groups were found. However, sleep continuity variables, specifically sleep efficiency and sleep latency, were more disrupted in those with recurrences. Subjects with more prolonged sleep latency had a shorter time to recurrence and a greater likelihood of recurrence.

While REM sleep measures were not different between these two groups, we have previously published that REM latency is shorter in depressed child and adolescent in-patients than normal controls (Emslie et al., 1990, 1994). In fact, 40% of the sample of in-patients had a REM latency less than 65 min, which is considered short in adult samples. The sleep continuity disturbances suggest that subjects who are likely to have a recurrence are experiencing greater disturbances in sleep regulation at baseline despite similar levels of symptom severity. Of interest is a recent report by Rao et al. (1997) suggesting that sleep PSG variables in depressed adolescents are trait-like (i.e. unchanged between an episode and remission). Similar findings have been reported in adults (Kupfer et al., 1990). Collectively, these data are provocative in light of the findings that persistent sleep disturbance and insomnia in particular, increase the risk for relapse and recurrence in adults with depression (Fawcett et al., 1990; Ford and Kamerow, 1989; Wingard and Berkman, 1983).

Sleep latency is of further interest as a possible outcome predictor, given the ease of measuring sleep latency in subjects' own homes using actography data (Sadeh et al., 1994). Whether this would give a similar result as a sleep laboratory measurement is not known. Similarly, the relationship between clinically reported initial insomnia and sleep laboratory measured sleep latency bears investigation. It is also of note that Dahl (1996) has suggested that difficulties in initiating sleep and subsequent prolonged sleep latency are more stable characteristics of sleep disturbances in early onset depression than REM latency or REM density. The results from the present study support this view and point to the potential clinical utility of this measure in predicting risk of recurrence.

Nevertheless, this report has several limitations. The non-recurrence group clearly contains subjects who will go on to have a recurrence if followed long enough. Data were available beyond 1 yr on some but not all in-patients and none of the outpatients. The result of having later recurrers in the non-recurrence group is to minimize possible differences between the groups. These results are therefore more relevant in terms of time to recurrence. Another limitation is the naturalistic nature of the clinical data. First of all, interviews were scheduled at 6-month intervals, which may be too far apart to provide sufficient accuracy of treatment. Also, it is not possible to control for the effects of treatment (e.g. in the literature on depressed adults, REM latency predicted recurrence upon medication discontinuation) (Giles et al., 1987; Rush et al., 1989). Finally, larger sample may allow for other covariates to be taken into account when using sleep to predict recurrence, such as gender, age, family or psychiatric history. However, this paper reports on a large sample of carefully diagnosed depressed children and adolescents who had sleep polysomnography and systematic clinical follow-up, and clearly demonstrates that further research into sleep dysregulation in this population is warranted. The next step may be to assess micro-architecture, given its ability to differentiate between patients and controls (Armitage et al., 2000).

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