

BRIEF REPORTS

REM Latency in Psychotically Depressed Adolescents

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Introduction

Major depression in adults is accompanied by characteristic polysomnographic abnormalities, including shorter latency to rapid eye movement (REM) sleep, less slow wave sleep, impaired sleep continuity, greater REM activity (especially in the first REM period), and elevated REM density compared with nondepressed age-matched controls (Kupfer et al. 1978). Thase et al. (1986) demonstrated that REM latency may be dependent upon depressive subtype. In their study, psychotically depressed subjects had significantly shorter REM latencies than depressed patients without psychotic features. Sleep onset REM periods were frequently seen in their population of psychotically depressed adults.

Studies of REM latency in depressed adolescents have been inconclusive. Goetz et al. (1987) and Appelboom-Fondu et al. (1988) failed to find short REM latency in depressed adolescents compared with normal controls. In contrast, Lahmeyer et al. (1983) and Emslie et al. (1988) found that depressed adolescents had shorter REM latencies than age-matched normal controls. None of these studies compared REM sleep in psychotically and nonpsychotically depressed adolescents. We undertook this preliminary study to examine the relationship between

subtype of depression (psychotic versus nonpsychotic) and REM latency in adolescents. We hypothesized that psychotically depressed adolescents would have shorter REM latencies than depressed adolescents without psychosis.

Methods

The subjects were 12 unipolar depressed adolescents admitted to the University of Michigan Adolescent Psychiatry Unit. Informed consent was obtained. The sample included 5 boys and 7 girls ages 13.4–17.0 years (mean = 15.6 years). Diagnoses were made according to Research Diagnostic Criteria (Spitzer et al. 1973) based on information obtained from a semistructured interview, the Schedule for Affective Disorders and Schizophrenia (Endicott and Spitzer 1978). Depression severity was measured with the Hamilton Rating Scale for Depression (HRSD) (Hamilton 1960) and the Children's Depression Rating Scale—Revised (CDRS-R) (Poznanski et al. 1984). All patients were observed psychotropic medication free in the hospital for 2 weeks prior to the study. Only those who remained depressed throughout the observation period were included in the study. Patients were studied polysomnographically from their own beds for 2 nights using an electroencephalogram (EEG) sleep telemetry system. The studies were performed as described previously (Grunhaus et al. 1988). All polygraph records were scored visually on the basis of 1-min epochs according to Rechtschaffen-Kales et al. (1968) criteria by experienced technicians blind to patient diag-

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Table 1. Sleep Variables in Depressed Adolescents with and Without Psychotic Features (Mean \pm SD)

	Psychotic (n = 5)	Nonpsychotic (n = 7)	<i>p</i> < ^a
Sleep continuity			
Sleep latency	46.8 \pm 17.7	39.2 \pm 14.1	NS
WASO	11.3 \pm 19.2	1.8 \pm 1.8	NS
ALT	4.7 \pm 9.4	1.8 \pm 1.6	NS
Arousals	0.8 \pm 1.0	1.4 \pm 1.5	NS
Sleep efficiency	84.1 \pm 6.6	89.2 \pm 3.4	NS
Sleep stages			
Stage 1 (%)	9.5 \pm 3.1	6.8 \pm 4.4	NS
Stage 2 (%)	51.4 \pm 3.3	50.0 \pm 4.7	NS
Delta sleep (%)	23.3 \pm 4.1	27.1 \pm 9.9	NS
REM (%)	15.9 \pm 2.5	15.4 \pm 4.2	NS
REM variables			
REM latency	70.9 \pm 11.5	122.4 \pm 33.2	0.02
REM activity	57.4 \pm 18.7	58.8 \pm 29.5	NS
Density REM 1	0.7 \pm 0.3	0.6 \pm 0.2	NS
Activity REM 1	6.3 \pm 2.5	8.9 \pm 6.4	NS

^aMann-Whitney U-test.

WASO = wakefulness after sleep onset; ALT = awake last 2 hr.

nosis. Sleep variables were averaged over the 2 nights of the study and analyzed. Data for REM latency (measured from sleep onset to the beginning of the first REM period of at least 3 min duration) were further analyzed to assess the presence of a first night effect between nights and between groups to determine whether the findings were similar when each night was considered separately.

Group comparisons between psychotic and nonpsychotic depressed adolescents were made using a Mann-Whitney U-test. A two-tailed Fisher's exact test was used to examine the distribution of patients into psychotic versus nonpsychotic and short versus long REM latency groups. We used Pearson's product-moment correlation coefficient to study the relationship between REM latency and severity of depression.

Results

Psychotically depressed adolescents did not differ from nonpsychotic patients in terms of age (15.9 \pm 0.8 versus 15.3 \pm 1.5 years; *Z* =

-0.49, NS). As measured by the HRSD, psychotically depressed adolescents were more severely depressed (23.6 \pm 5.0 versus 16.6 \pm 4.3; *Z* = -2.04; *p* < 0.05). The CDRS showed a trend in the same direction (73.6 \pm 13.4 versus 55.7 \pm 12.4; *Z* = -1.87, *p* < 0.07).

Psychotically depressed adolescents had a significantly shorter REM latency as a group (70.9 \pm 11.5 versus 122.4 \pm 33.2 min; *Z* = -2.36; *p* < 0.02) (Table 1). We found no significant correlation between REM latency and severity of depression as measured by the CDRS-R or HRSD. When the patients were divided into short versus long REM latency groups based on a natural split in the data (Figure 1), all psychotically depressed patients fell into the short REM latency group, a distribution that was statistically significant (Fisher's exact test; *p* < 0.03).

There was no significant difference in REM latency between night 1 and night 2 within either patient group (Mann-Whitney U-test, NS). Analyzing between-group differences by individual nights, REM latency was significantly lower on the second night in psychotically depressed ad-

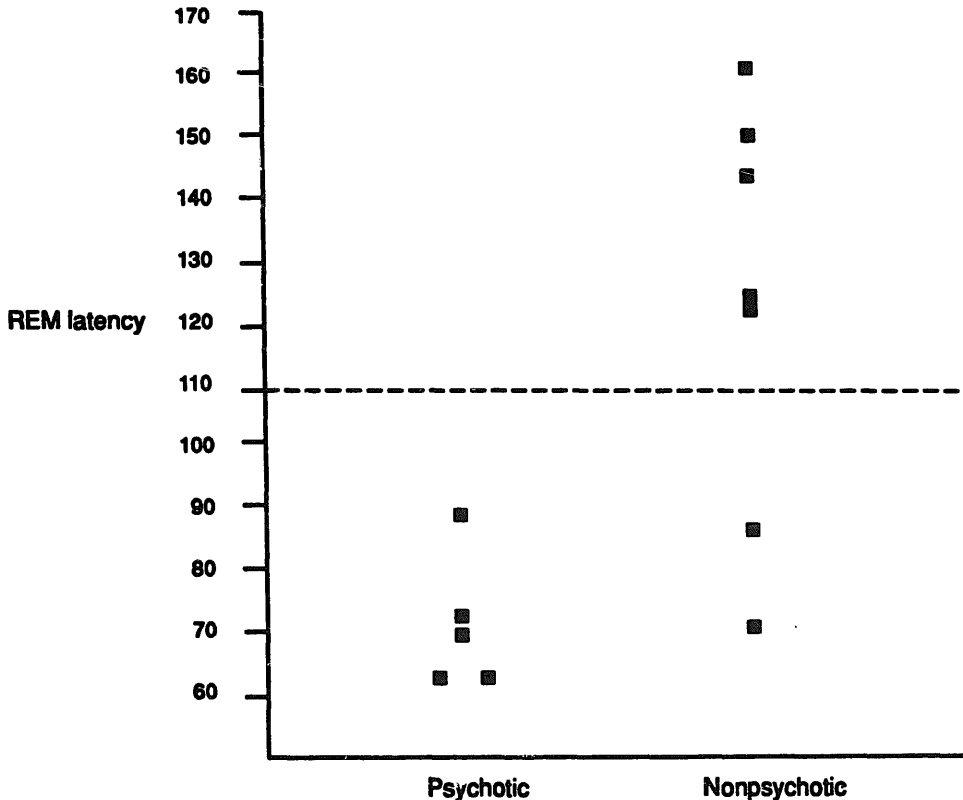


Figure 1. REM sleep latency distribution and depressive subtype.

olescents (72.0 ± 19.7 versus 123.0 ± 40.2 min; $Z = -2.20$; $p < 0.03$). The difference approached significance for the first night (69.8 ± 19.7 versus 121.7 ± 40.9 min; $Z = -1.87$; $p < 0.07$).

Discussion

Our results show that depressed adolescents with psychosis have a shorter REM latency than those without psychosis. We conclude that the development of shortened REM latency in depressed patients is influenced by the subtype of depression. The lack of correlation between severity of depression and REM latency must be viewed with caution due to the small sample size and the discontinuous nature of the REM latency data.

These results may shed some light on the previously mentioned discrepancies between

REM latency findings in depressed adolescents. Studies involving predominantly outpatients have found no REM latency differences between depressed adolescents and normal controls (Goetz et al. 1987; Appelboom-Fondu et al. 1988), whereas those involving inpatients (Emslie et al. 1988) or both inpatients and outpatients (Lahmeyer et al. 1983) have found shortened REM latencies. Inpatient status presumes a level of severity not seen in depressed adolescents treated as outpatients. Depressive subtype may be one mediator of severity. Psychotically depressed adolescents are unlikely to be managed on an outpatient basis.

Several aspects of our study deserve further comment. First, this is a preliminary report and needs to be replicated in a larger sample. Second, we averaged the data across the first and second nights. Whereas the literature points to a clear first night effect of the laboratory on sleep

in normal subjects, including prolongation of REM latency (Agnew et al. 1966), we found no difference between night 1 and night 2 REM latency values in either group of depressed adolescents. This phenomenon was previously described in adults hospitalized for major depression (Ansseau et al. 1985). Third, we did not have a normal control group. The use of a nonpsychotically depressed control group as opposed to normal subjects, however, is a more rigorous test of our hypothesis that psychotic depression is accompanied by shortened REM latency.

Further research is needed to identify factors that may account for the shortened REM latency observed in psychotically depressed adolescents.

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