

EEG alpha power and alpha power asymmetry in sleep and wakefulness

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Abstract

Asymmetry of waking electroencephalography (EEG) alpha power in frontal regions has been correlated with waking emotional reactivity and the emotional content of dream reports. Little is known regarding alpha asymmetry during sleep. The present study was performed to compare alpha power and alpha power asymmetry in various brain regions across states of sleep and wakefulness. Waking and sleep EEG were recorded in a group of patients undergoing polysomnographic evaluation for possible sleep disorders. Alpha EEG asymmetry in frontal and temporal regions was significantly correlated in waking versus sleep, particularly during rapid eye movement (REM) sleep. These results suggest that patterns of frontal alpha asymmetry are stable across sleep and waking and may be related to emotional reactivity during dreaming. During sleep, alpha power was highest during slow-wave sleep and lowest during REM sleep. Implications of these data for understanding the functional significance of alpha power during waking and sleeping are considered.

Descriptors: Electroencephalography, Alpha, Sleep, REM sleep, Human

Individual differences in alpha band (8–12 Hz) electroencephalography (EEG) power asymmetry at anterior scalp regions predict important features of emotional reactivity (Davidson, 1992, 1995; Davidson & Tomarken, 1989). In adults, these asymmetries appear to be traitlike characteristics: they are stable over time and exhibit excellent internal consistency and reliability (Tomarken, Davidson, Wheeler, & Kinney, 1992).

In general, increased alpha band power activity during waking is thought to reflect decreased activity of the corresponding region of the underlying cerebral cortex (Shagass, 1972), though precise localization must be avoided. Studies measuring alpha power during cognitive tasks have shown that alpha power is decreased in regions thought to be associated with the task. For example, during verbal tasks alpha is suppressed in left posterior regions, whereas during visuospatial tasks the opposite pattern of asymmetric alpha power has been reported (Davidson, Chapman, Chapman, & Henriques, 1990). Suppression of alpha rhythms has also been related

to increased glucose metabolic activity in the thalamus (Larson et al., 1997).

Subjects at the extremes of the distribution in baseline frontal alpha asymmetry differ in their dispositional affect. Those individuals with low left-to-right frontal alpha band power (presumably representing relative activation of left frontal areas) report more dispositional positive affect and less dispositional negative affect (Davidson, 1995; Davidson & Tomarken, 1989). Such people also display more behavioral activation compared with behavioral inhibition than do subjects at the opposite extreme (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). It has also been shown that subjects with relatively less right frontal alpha power (presumably relatively greater activation of the right hemisphere) respond more negatively to laboratory challenges designed to provoke negative affect (Wheeler, Davidson, & Tomarken, 1993). Individual differences in frontal alpha asymmetry are present by 10 months of age in human infants and predict reactivity of the infants to emotional challenges such as a brief episode of maternal separation (Davidson & Fox, 1989). Similar associations between frontal alpha asymmetry and behavior have also been found in rhesus monkeys (Davidson, Kalin, & Shelton, 1993).

In short, individual differences in waking EEG frontal alpha asymmetry are present early in life, stable over time, similar in humans and rhesus monkeys, and predict important features of emotional reactivity. The relationship between these measured alpha power asymmetries and emotional reactivity is presumably a consequence of the asymmetry of frontal activation reflected by alpha band power measurements.

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In the research performed to date comparing frontal activation patterns with emotional reactivity, measures of brain electrical activity have been acquired in awake subjects. Although several investigators have examined effects of sleep on lateralization of EEG activation patterns during sleep, rarely have sleeping and waking EEG been compared. Measurements were usually obtained from central rather than frontal regions, and alpha band power has generally not been assessed (Armitage, Hoffmann, Loewy, & Moffitt, 1989; Armitage, Roffwarg, & Rush, 1993; Goldstein, Stoltzfus, & Gardocki, 1972). It has recently been shown, however, that individual differences in frontal activation asymmetry while awake predicted the emotional content of dream reports from a subsequent sleep episode (Donzella, Davidson, Stickgold, & Hobson, 1994). As expected, subjects with greater waking right frontal activation reported dreams with more negative affect than subjects who had more waking left frontal activation. Based on these data, we expected that the asymmetric frontal activity seen in the waking EEG would also be present during sleep, and particularly during rapid eye movement (REM) sleep, when most dreaming mentation occurs.

The primary purpose of the present study was to examine the relationship between EEG alpha asymmetry across waking and sleep states. We hypothesized that measures of alpha power asymmetry obtained from the midfrontal scalp region (the primary dependent measure in the studies referred to above) during waking and various sleep stages would be substantially correlated, and that correlations would be greatest between the stages with the greatest cortical activation, waking and REM sleep. In addition, we compared asymmetry of alpha power measures during waking and the different sleep stages to determine whether there were any consistent shifts in the degree of alpha asymmetry with sleep state.

Another goal of the study was to compare regional alpha EEG power across waking and sleep states. During sleep, elevated overall alpha band power has been reported in patients with disorders such as chronic pain (Wittig, Zorick, Blumer, Heilbronn, & Roth, 1982), fibromyalgia (Moldofsky, Scarisbrick, England, & Smythe, 1975) and depression (Hauri & Hawkins, 1973). Because these patients usually have fragmented sleep, and complain of nonrestorative sleep and daytime fatigue, it has been assumed that the high alpha activity during sleep is an indication of arousal and, by inference, cortical activation. It is also possible that alpha EEG power within sleep reflects decreased cortical activation as it does during waking; the increase in alpha power in patients with unsatisfying sleep could indicate inactivation in response to poor sleep, rather than activation that causes the low quality sleep. We therefore hypothesized that relative alpha power across sleep states might correlate negatively with the relative degree of brain activation during those states as previously established by other measures, such as cerebral blood flow and metabolic rate, which are increased in waking and REM sleep and lowest during slow-wave sleep (SWS) (Buchsbbaum et al., 1989; Madsen et al., 1991; Madsen & Vorstrup, 1991; Sakai, Meyer, Karacan, Derman, & Yamamoto, 1980).

Methods

Subjects

Subjects were recruited from a pool of incoming patients referred to the University of Wisconsin Comprehensive Sleep Disorders Center for a clinical nighttime polysomnographic (NPSG) evaluation of possible sleep apnea. Subjects were right-handed as assessed by the Chapman Handedness Inventory (Chapman &

Chapman, 1987). All patients had clinical histories of excessive daytime sleepiness and snoring. Sleep recordings were obtained for 17 subjects. For 4 subjects, there was not enough SWS for analysis; therefore, only 13 subjects were used for analyses of SWS. The mean age of the 17 subjects (13 men, 4 women) used in the statistical analyses was 49.35 ± 14 years. Polysomnographic studies resulted in the diagnosis of sleep apnea in 7 patients (apnea-hypopnea indices ranging from 9.5 to 63 events per hour of sleep), 3 patients with upper airway resistance syndrome, and 7 patients without evidence of sleep-related breathing disorder. One subject with sleep apnea showed evidence of severe periodic leg movement disorder. One subject was known to have bipolar disorder and was being treated with venlafaxine and sodium valproate at the time of the study. Two other patients were taking amitriptyline at bedtime, and one was being treated with sertraline. No other psychotropic drug use was reported.

Procedures

Subjects arrived at the Sleep Disorders Center at approximately 8:45 p.m., and informed consent was obtained in accordance with procedures approved by the Institutional Review Board. Subjects completed the Chapman Handedness Inventory (Chapman & Chapman, 1987) and the state version of the Positive and Negative Affect Schedule (PANAS-NOW; Watson, Clark, & Tellegen, 1988). The EEG recording electrodes were then applied and waking EEG was recorded while the subject was reclined and resting but still awake. Eight 1-min trials of waking EEG were recorded, four with the subject's eyes open (A), four with eyes closed (B). The trials were run in an ABBA BAAB, or BAAB ABBA design, with the order randomized across the subjects. EEG was recorded throughout the entire night until the subject awoke in the morning. Upon waking, the subjects completed a second PANAS-NOW.

Electrophysiological Recording and Quantification

EEG was recorded using Rochester Electro-Medical silver-silver chloride electrodes applied according to the International 10-20 System (Jasper, 1958). EEG was recorded at 13 scalp sites (F3/4, F7/8, T3/4, T5/6, P3/4, O1/O2, and CZ), all referenced to the mastoid of the left ear (A1). The right ear (A2) was also recorded for rederivation of a linked ears reference offline. Chin electromyogram (EMG) and two channels of electrooculograms (EOG) were also recorded: horizontal (HEOG) from the external canthi of each eye; and vertical (VEOG) from the supra- to suborbit of the right eye. Clinical polysomnographic assessment included monitoring of the following additional variables: combined nasal/oral airflow, chest and abdominal plethysmography, electrocardiogram, pulse oximetry, intercostal EMG, anterior tibialis EMG, and microphone and infrared video recording. All EEG electrode impedances were less than 10,000 Ω . EOG electrode impedances were less than 20,000 Ω . The EEG and EOG were amplified with a Melville Multi-Channel amplifier (Melville Diagnostics, Ottawa, Ontario) with a bandpass of 1/100 Hz and an internal 60-Hz digital notch filter. The signals were digitized on-line at 200 Hz by an Analogic MS-DAS-12 A/D board attached to an IBM-compatible PC, using the Sandman (Melville Diagnostics, Ottawa, Ontario) collection and analysis system version 2.

Sleep was visually staged in 30-s epochs according to Rechtschaffen and Kales (1968) criteria; 120 s of artifact-free data were then selected from Stage II, SWS, and REM sleep. EEG data from all conditions except SWS sleep were corrected for EOG artifact using the method described by Gasser, Ziegler, and Gattaz (1992). SWS EEG was excluded from EOG correction as there are

virtually no eye movements during SWS, hence large voltages in the EOG channel are much more likely to represent contamination of the EOG by the EEG rather than the reverse. Following the EOG correction procedure, the EEG was inspected and edited to remove artifact due to gross muscle activity and movement. When artifact occurred in a given channel, corresponding data from all channels were removed. Artifact-free chunks of data were extracted through a Hamming window, which reduces spurious power estimates at the beginning and end of each chunk. A Fast Hartley Transform (FHT; Bracewell, 1984) was applied to all extracted artifact-free chunks of data that were 1.024 s in duration, with chunks overlapping 50%. Data were rereferenced offline to the average reference. Power density ($\mu\text{V}^2/\text{Hz}$) was computed for each trial for the alpha (8–13 Hz) band by summing power values across each 1-Hz bin within the alpha band and dividing by the number of bins. For the eight trials of waking, weighted means within each condition (eyes open or eyes closed) were computed by averaging the power density across all trials within each condition, weighted by the number of chunks of artifact-free data. Finally, all power density values were log transformed to normalize the distribution of the data. Alpha asymmetry scores (log right – log left power density) were computed for each of the six pairs of homologous sites.

The time of night at which the analyzed data occurred across stages of sleep was similar (and not significantly different). The median number of hours from lights out to the start of REM data analyzed was 2.62 hr as opposed to 2.11 hr for Stage II and 1.77 hr for SWS.

Data Analysis

Average alpha power values by sleep stage were calculated for the sites for which data were available for the group of 13 subjects who displayed SWS. The sites T3/4 and P3/4 were omitted from this analysis because data was not available from these sites for 4 subjects due to technical problems during the recordings.

To determine whether patterns of EEG alpha activity changed across sleeping and waking conditions, a series of repeated-measures analyses of variance (ANOVAs) were run. All repeated-measures significance levels were Huynh–Feldt corrected (Huynh & Feldt, 1970). A three-way repeated-measures ANOVA was run (Region \times Hemisphere \times Condition). The Condition factor consisted of five levels: eyes open, eyes closed, REM, Stage II, and SWS sleep. If any cell was missing data, that subject's data were not used in the ANOVA, leaving data from 13 subjects for these computations. A follow-up two-way ANOVA was run to examine the Condition \times Hemisphere interaction for each region. Another two-way ANOVA was run to compare this same interaction for each region, but the conditions were collapsed into two levels: wake and sleep.

Pearson r values were also computed comparing waking eyes-open and eyes-closed alpha asymmetry with alpha asymmetry during the three sleep stages.

Results

Correlations Between Sleep and Waking EEG Asymmetry

Because measures of emotional reactivity have been correlated with asymmetry of frontal alpha band power during waking, we first compared alpha band power asymmetry from various scalp locations across waking, Stage II sleep, REM sleep, and SWS. Pearson r values were calculated for the correlations between eyes-open and eyes-closed waking alpha asymmetry and alpha asym-

metry during sleep for frontal (F3/4 and F7/8), temporal (T3/4 and T5/6), parietal (P3/4), and occipital (O1/2) regions (Table 1). The correlations for asymmetry were most robust for comparison between waking and REM sleep, with significant correlations at F3/4, T3/4, T5/6, and O1/2 for both eyes-open and eyes-closed waking conditions. Significant correlations between eyes-closed waking and nonrapid eye movement (NREM) sleep were also found, particularly in the midfrontal region (F3/4) and temporal regions. Figure 1A depicts scatterplots of the correlations between eyes-open midfrontal (F3/4) alpha power and Stage II, SWS, and REM alpha power and Figure 1B presents the correlations for the same region for the eyes-closed condition.

The magnitude of the expected correlation between the Negative Affect scale of the PANAS (-0.47) with waking asymmetry (Tomarken, Davidson, Wheeler, & Doss, 1992) is too small to be detected with the sample size used in this study. It is not surprising, therefore, that none of the correlations reached significance. Nonetheless, there was a negative correlation of the Negative Affect scale with both eyes open (-0.275) and eyes closed (-0.23) frontal alpha asymmetry.

Alpha Power During Sleep and Wake by Site

Alpha power values for each site by sleep stage are presented in Figure 2. Although the correlations reported above demonstrated concordance between waking and sleeping alpha power asymmetry, there were systematic variations in alpha power across sleep and waking states. As expected, alpha power increased during eyes-closed compared with eyes-open waking conditions, particularly in the posterior scalp regions. At all sites recorded, alpha power was lowest during REM sleep. In frontal regions, NREM alpha power was comparable to waking alpha power, whereas in more posterior regions, alpha power was highest during the eyes-closed waking condition.

Alpha Band ANOVAs

To determine whether any of these observed patterns were significant, a three-way Region \times Hemisphere \times Condition ANOVA was

Table 1. Right–Left Alpha Asymmetry: Correlations Between Sleep Stages and Waking EEG

Waking	Sleep stages		
	REM	Stage II	SWS
Eyes open			
F4-F3	.62**	.44	.49
F8-F7	.14	.27	.21
T4-T3	.63**	.77*	.43
T6-T5	.61**	.33	.53
P4-P3	.39	.18	.06
O2-O1	.51*	.20	.11
Eyes closed			
F4-F3	.73***	.53*	.69**
F8-F7	.19	.32	.17
T4-T3	.68**	.69**	.39
T6-T5	.74***	.52*	.64*
P4-P3	.45	.65**	.34
O2-O1	.67**	-.06	.39

Note: EEG = electroencephalogram; REM = rapid eye movement; SWS = slow-wave sleep.

*** $p < .005$, ** $p < .01$, * $p < .05$, two tailed.

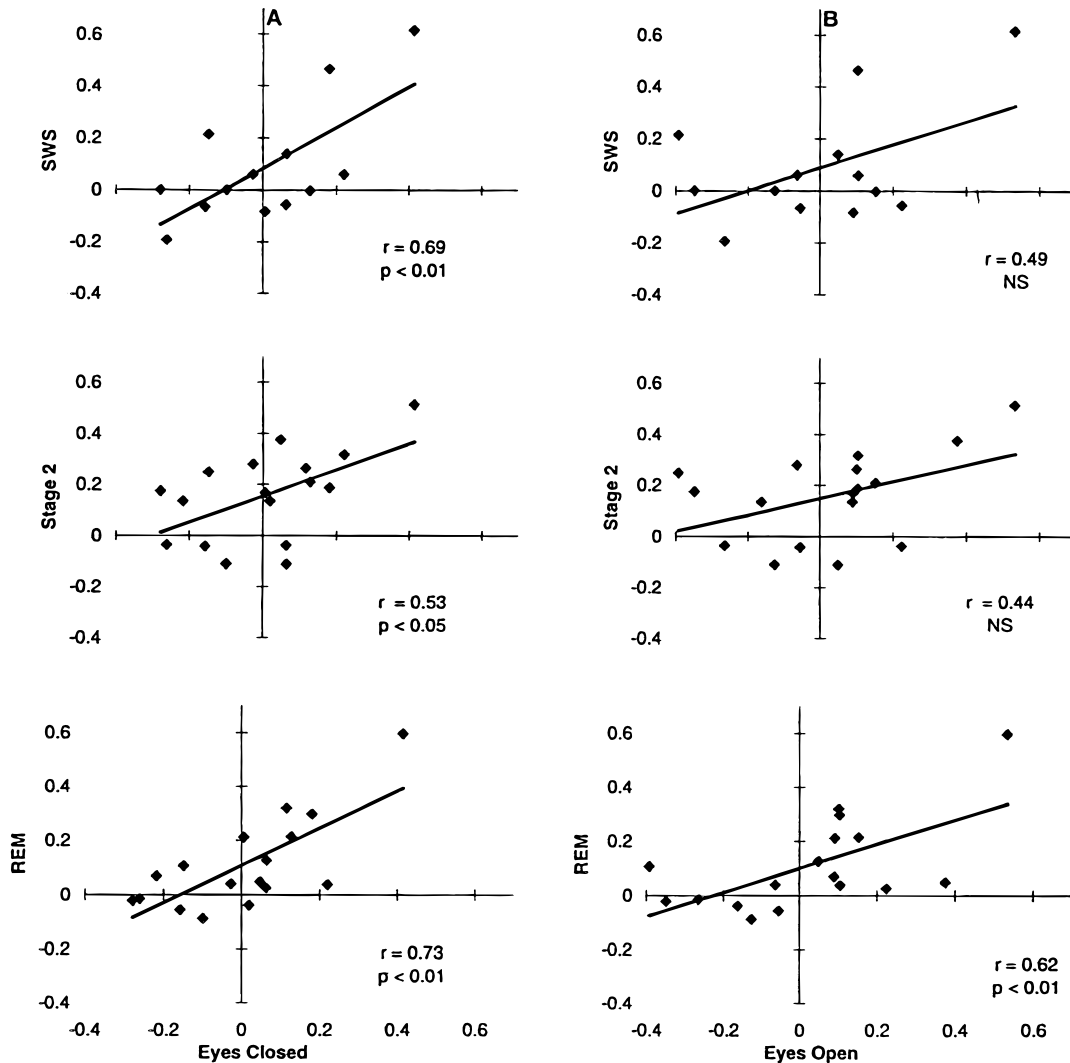


Figure 1. Scatter plots of the relation between alpha band power asymmetry at F3/4 for: (A) each stage of sleep plotted against eyes closed waking condition; and (B) each stage of sleep plotted against eyes open waking condition. All axes represent $\log(\alpha \text{ power [F4]}) - \log(\alpha \text{ power [F3]})$.

performed (Table 2), which revealed significant main effects for Condition, $F(4,48) = 7.77, p = .005$, H-F $\epsilon = 0.4164$, and Region, $F(3,36) = 3.56, p < .02$, H-F $\epsilon = 0.9984$. The three two-way interactions were all significant: Condition \times Region, $F(12,144) = 14.47, p < .001$, H-F $\epsilon = 0.4520$, Condition \times Hemisphere, $F(4,32) = 4.36, p < .01$, H-F $\epsilon = 0.5636$, and Region \times Hemisphere, $F(3,36) = 9.73, p < .001$, H-F $\epsilon = 0.8836$. The three-way Condition \times Region \times Hemisphere, however, was not significant, $F(12,144) = 0.96$. These results suggest main effects for Condition (e.g., eyes open, eyes closed, REM sleep, Stage II sleep, SWS) and scalp Region on alpha power. Although the three-way interaction was not significant, follow-up two-way Condition \times Hemisphere ANOVAs for each Region were computed to specify with more precision where the interaction was present. These analyses revealed that the midfrontal and lateral frontal regions had significant Condition \times Hemisphere interactions: for midfrontal (F3/4), $F(4,48) = 3.07, p < .05$, H-F $\epsilon = 0.6841$; for lateral frontal (F7/8), $F(4,48) = 3.55, p < .04$, H-F $\epsilon = 0.5593$. Significant interactions were not present for any other region (see Figure 2).

These differences appeared to be due largely to greater right than left frontal alpha power during the three sleep conditions as compared with the two waking conditions.

Discussion

The results of this study demonstrate strong positive correlations between waking alpha band power asymmetry in frontal and temporal regions and alpha asymmetry in those same regions during sleep. This report is the first of an association between waking and sleeping measures of anterior activation asymmetry. These associations were strongest when waking asymmetry was measured with the subjects' eyes closed. REM sleep alpha asymmetry was more highly correlated with both waking measures than were the other sleep states. Correlations between REM sleep and waking alpha asymmetry were noted in a number of sites, including frontal, temporal and occipital, but not lateral frontal or parietal regions.

Alpha power asymmetry in frontal regions during waking predicts emotional reactivity during waking (reviewed above) and the

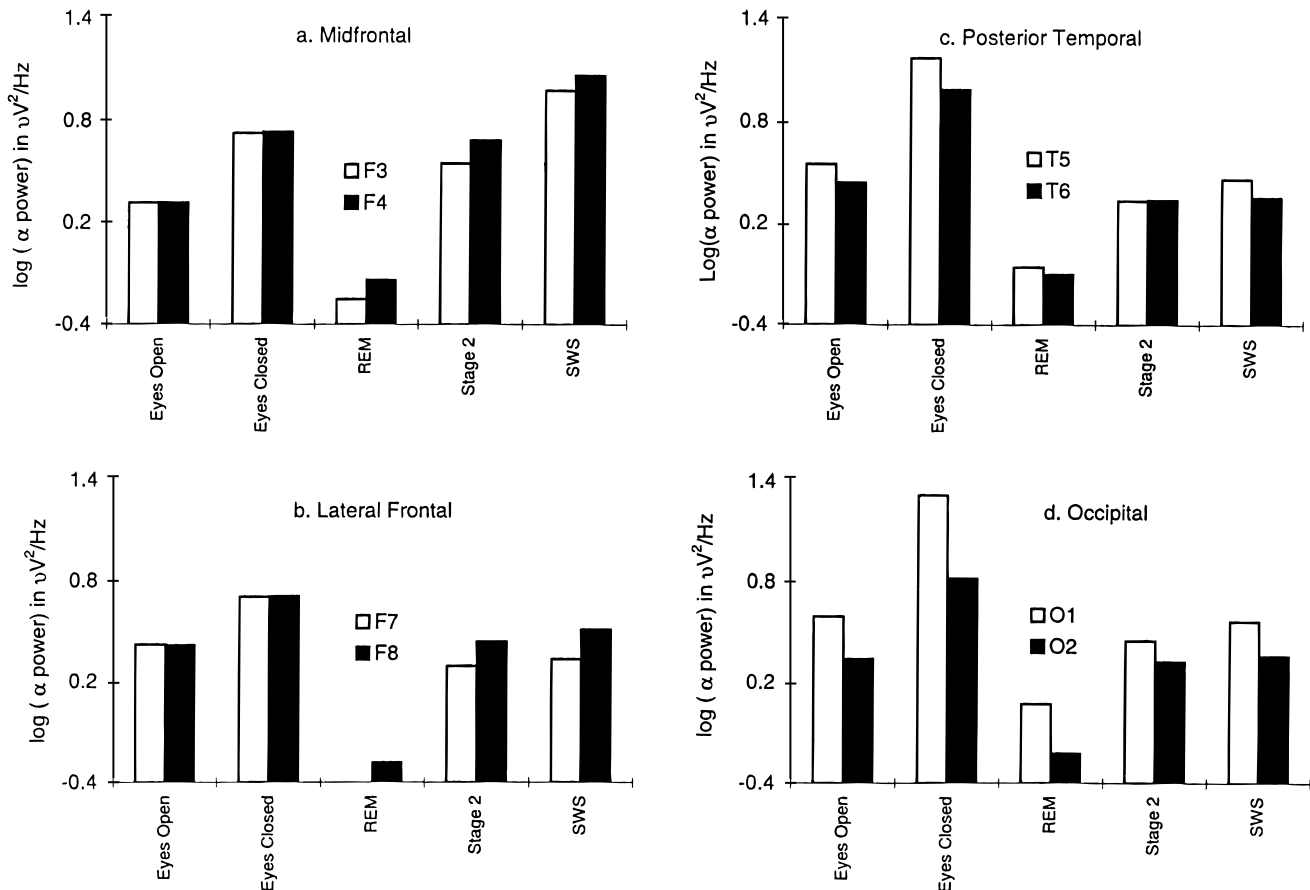


Figure 2. Log alpha band power for specific brain regions during eyes open and eyes closed waking, rapid eye movement sleep, stage 2 sleep, and slow-wave sleep. Open bars denote left hemisphere; shaded bars denote right hemisphere. Significant Condition \times Hemisphere interactions were found for the midfrontal and lateral regions, reflecting greater right than left alpha power during sleep compared with waking conditions.

emotional content of dream reports from sleep (Donzella et al., 1994). The magnitude of the known association between the PANAS and alpha power precludes definitive conclusions regarding the relationship between PANAS scores and sleeping or waking alpha asymmetry. More negative affect in dream reports, such as waking negative affect, is associated with decreased relative waking right frontal alpha band power, which is thought to reflect increased activation in this scalp region. The association between increased frontal asymmetry in waking and affective content of dream reports could be related to a reporting bias related to emotional reactivity at the time of the dream report and/or activation patterns of mood-regulatory systems during sleep. Results of the present study suggest that the affective content of dreaming may be related in part to frontal alpha asymmetry during sleep. It is therefore not surprising that frontal alpha asymmetry is more highly correlated between waking and REM sleep, because dreaming most commonly occurs during this state. Although NREM sleep-waking regional asymmetry correlations were lower than those between REM sleep and waking, the stability of asymmetry patterns between eyes-closed waking and NREM sleep was nonetheless particularly marked in frontal and temporal regions.

Despite the stability of alpha power asymmetry between waking and sleep there was a distinct tendency for a rightward shift in alpha power during sleep, particularly in the frontal regions. This

relative increase in relative right-sided alpha power was observed in both REM and NREM sleep. If increased alpha power within sleep is a reflection of decreased brain activation, our results are consistent with functional imaging studies, which have suggested increases in left-sided thalamic activation during REM sleep (Maquet et al., 1996) and decreased activation of right-sided structures (precuneus and mediotemporal cortex) during SWS (Maquet et al., 1997).

Most previous studies that have examined EEG power lateralization during sleep have examined REM versus NREM sleep rather than comparisons between sleep and wakefulness. One early study assessing EEG power across all frequencies found greater integrated amplitude over the left hemisphere in REM sleep and over the right hemisphere during NREM sleep (Goldstein et al., 1972). Studies that have assessed asymmetry within discrete EEG frequency bands have not yielded consistent findings regarding the effects of sleep states on EEG power asymmetry. Armitage et al. (1989) found greater absolute hemispheric asymmetry (irrespective of direction) in NREM sleep, particularly SWS, in comparison with REM sleep. In contrast to the present results, these differences were noted only in the slower theta and delta rhythms. In a subsequent study (Armitage et al., 1993), groups of depressed patients were found to have consistent increases in beta, theta, and delta power in the right hemisphere during REM sleep versus NREM

Table 2. *F Ratios for Tests of EEG Power Differences Across Condition (C), Region (R), and Hemisphere (H)*

	<i>F</i>	<i>df</i>	<i>p</i>
Condition × Region × Hemisphere ANOVA			
C	7.77	4, 48	.005
R	3.56	3, 36	.02
H	1.00	1, 12	<i>ns</i>
C × R	14.47	12, 144	.001
C × H	4.19	4, 48	.02
R × H	9.73	3, 36	.001
C × R × H	0.96	12, 144	<i>ns</i>
Condition × Hemisphere ANOVA for Each Region			
F3/4			
C	10.43	4, 48	.001
H	1.94	1, 12	<i>ns</i>
C × H	3.07	4, 48	.045
F7/8			
C	9.80	4, 48	.001
H	4.61	1, 12	.05
C × H	3.55	4, 48	.04
T5/6			
C	9.52	3, 36	.002
H	0.85	1, 12	<i>ns</i>
C × H	1.62	3, 36	<i>ns</i>
O1/2			
C	9.73	3, 36	.002
H	15.81	1, 12	.002
C × H	3.59	3, 36	.06

Note: EEG = electroencephalogram; ANOVA = analysis of variance; *ns* = not significant.

sleep, resulting in greater asymmetry at these frequencies in REM sleep. No previous studies have assessed sleep-wake effects on alpha power asymmetry over specific scalp regions.

Although increased alpha power during waking is thought to indicate decreased cortical activation, the significance of alpha EEG activity during sleep is unknown. Consistent with previous studies (Buchsbaum et al., 1982; Tanaka, Hayashi, & Hori, 1997), we found that alpha power was during waking with eyes closed was greater in posterior than in anterior regions (Figure 2). It has been shown that at sleep onset, posterior alpha decreases and alpha activity in frontal areas increases (Buchsbaum et al., 1982; Tanaka et al., 1997).

Thus, the decrease in cortical activation in the transition from waking to sleep is not reflected by an increase in alpha power at a given site. It is possible, however, that the relationship between alpha and cortical activation within sleep may be similar to the relationship within waking. We found that alpha power was lowest during REM sleep over all sites recorded in this study. NREM values were increased significantly in comparison to REM sleep,

with SWS usually showing the greatest values for alpha power. A previous study that failed to find significant differences in alpha power across sleep states recorded EEG only over central regions (Armitage, 1995).

Studies assessing brain activation in sleep have found REM sleep to be the state of greatest cortical activation, based on global measurements of cerebral blood flow and metabolic rate (Buchsbaum et al., 1989; Madsen et al., 1991; Madsen & Vorstrup, 1991; Sakai et al., 1980). Functional imaging studies have identified more localized patterns of cerebral activity. REM sleep is specifically associated with increased activation of limbic structures and extrastriate cortex, but decreased activation of lateral orbital and dorsolateral prefrontal cortex (Braun et al., 1998; Maquet et al., 1996). Our finding of decreased frontal alpha power during REM sleep may reflect increased activation in central areas, such as limbic and paralimbic regions. In contrast, SWS is characterized by decreased cerebral blood flow in comparison to waking, particularly in orbitofrontal and thalamic regions (Hofle et al., 1997; Maquet et al., 1997).

The current data are in accord with these findings in that alpha power was greatest in SWS and least overall in REM sleep. As EEG does not have the spatial resolution of imaging techniques, it is not possible to determine which structures contribute most to the overall level of frontal alpha during REM sleep. Thus, decreased alpha may reflect increased activation of underlying limbic and paralimbic regions.

Waking alpha power values were not significantly different from values during NREM sleep stages, which suggests that the relationship between alpha power and cortical activation may not be identical across states of sleep and waking at all recording sites. It is possible and even likely that variations in frontal alpha power may reliably indicate relative variations in cortical activation across sleep stages. Our results suggest the possibility that increased alpha power may represent cortical deactivation during sleep, as it does during waking. When alpha intrusion occurs during sleep, it is usually most prominent during SWS, for example. A study of insomniacs found that those with higher amounts of alpha during sleep had significantly more SWS and overestimated their sleep time in comparison to those with low amounts of alpha during sleep, suggesting that alpha sleep may not be less deep or restorative sleep (Schneider-Helmert & Kumar, 1995). It remains to be determined whether increased alpha power during sleep in patients with alpha-delta sleep is the cause or effect of their nonrestorative sleep and reported daytime fatigue.

Previous work has demonstrated that waking frontal EEG alpha power asymmetry is a consistent, traitlike feature that correlates with various aspects of emotional reactivity during waking and sleep. The current results extend the previous findings by demonstrating the stability of frontal alpha asymmetry across states of sleep and wakefulness. Further studies with larger numbers of subjects will be needed to determine the relationship between sleeping asymmetry and affect in waking and in sleep.

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