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Reduced Alpha power associated with the recall of mentation from Stage 2 and Stage REM sleep

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Abstract

Relationships between Alpha (8–12 Hz) activity and cognitive processes during wakefulness raise the possibility of similar relationships between Alpha and cognitive activity during sleep. We hypothesized that Alpha power decreases during both Stage 2 and REM sleep would index the presence of sleep mentation in these stages. Absolute power for six classical EEG bands and three Alpha subbands was calculated for Stage 2 and REM sleep awakenings both with and without mentation recall. In both stages, recall was associated with lower Alpha power, especially with middle Alpha power (9.5–11.5 Hz). Unexpectedly, a similar effect for Delta power (0.5–4.0 Hz) was also observed. The Alpha effect may reflect cognitive elaboration active in the minutes preceding awakening; however, attention and memory processes cannot be excluded. The Delta effect is consistent with prior observations of regular linkages between Alpha and Delta power during sleep.

Descriptors: Alpha power, Delta power, Quantitative EEG, Sleep mentation, Dreaming, REM sleep, Stage 2 sleep

After almost 50 years of research since the discovery of vivid dreaming during REM sleep (Aserinsky & Kleitman, 1953; Dement & Kleitman, 1957), the psychophysiological correlates of dreaming remain unclear. One of the most debated issues concerns whether the recall of sleep mentation from both REM and NREM sleep is better explained by the existence of a single dream generator (one-generator model) or two different generators (two-generator model) active during the two types of sleep. With the exception of models proposed by Nielsen (2000) and Solms (2000),[1] one-generator models stem from a cognitive perspective in which physiological activity is seen to be related to dreaming in only a general sense, for example, cortical "activation" may be related to the length or complexity of a dream report (Cicogna & Bosinelli, 2001; Foulkes, 1985). In contrast, the most representative two-generator model (Hobson, Pace-Schott, & Stickgold, 2000) is a neurocognitive model in which sleep mentation is thought to be a direct function of the different physiological profiles characterizing REM and NREM sleep. For example, the high cholinergic and low aminergic neuromodulation characterizing REM sleep is thought to determine formal features of dreaming recalled from that state (e.g., hallucinosis, delusion, intense emotion, bizarreness), whereas the intermediate levels of both types of neuromodulation characterizing NREM sleep is thought to determine the less dreamlike mentation from that state (Hobson et al., 2000).

Considering that both one- and two-generator models are supported by some kinds of empirical evidence (see Nielsen, 2000, for review), questions about the psychophysiological correlates of sleep mentation remain pertinent and in need of further study.

Previous research attempting to discern relationships between EEG spectral power and dream recall have produced mixed results. A relationship between Beta mean frequency and dream recall in Stage 2 sleep was reported (Williamson, Csima, Galin, & Mamelak, 1986) as was a relation between reduction in postawakening Sigma (12–16 Hz) activity and dream recall after awakenings from Stage 2 sleep (Morel, Hoffmann, & Moffitt, 1991). Other authors reported a relation between suppression of Alpha power (8–12 Hz) over EEG sites corresponding to Broca's and Wernicke's areas and the degree of language content in dream reports in an intensive single case study (Hong et al., 1996). More recently, Alpha power was found to be inversely correlated with visual content in both blind and sighted subjects (Bertolo et al., 2003). Incongruities among these and other studies (Rochlen, Hoffmann, & Armitage, 1998) are likely due, in part, to the use of different methods of EEG quantification (Armitage, Hoffmann, Fitch, Morel, & Bonato, 1995).

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[1]Solms does not refer specifically to a single-generator model. Rather, he identifies activation of dopaminergic circuits in ventromesial forebrain as a necessary step in dreaming occurrences, an activation that is common in all sleep stages and that could thus be construed to be a one-generator explanation.

Since Berger (1929) first described Alpha activity in the human EEG, measures of Alpha activation have been associated with eyes closed, relaxed wakefulness, whereas measures of Alpha suppression have been related to the perceptual and/or cognitive treatment of external stimuli. For example, much evidence supports the possibility that fluctuations in Alpha and Theta activity reflect cognitive and memory performance during wakefulness (see Klimesch, 1999, for review). Klimesch suggests that during REM sleep, when Alpha activity in most (Benca et al., 1999; Hadjiyannakis, Ogilvie, Alloway, & Shapiro, 1997), but not all (Dumermuth, Langer, Lehmann, Meier, & Dinkelmann, 1983), studies has been shown to be generally lower than in other sleep stages, Alpha reduction could also be interpreted to be an event-related suppression. In others words, an Alpha decrease could be viewed as analogous to the Alpha desynchronization observed when an awake, alert subject performs a task. To the extent that dreaming involves mechanisms similar to waking task performance, Alpha reduction during sleep may have a similar significance. On the other hand, some authors suggest that fundamentally different mechanisms are responsible for Alpha activity in different sleep–wake states (Cantero, Atienza, & Salas, 2002).

Relationships between Alpha power and cognitive elaboration have been described in several domains. Magnetoencephalography reveals Alpha suppression in occipital sites during mental imagery and visual memory scanning (Kaufman, Schwartz, Salustri, & Williamson, 1990). An inverse relationship between the magnitude of Alpha activity and an increase in working memory load has also been described (Gevins et al., 1998). Lower Alpha power has been observed (1) over the left hemisphere during a verbal task, in particular, during delivery of a speech about a specific topic chosen by the subject; and (2) over the right hemisphere during a spatial task, that is, during a construction of block designs (Galin, Herron, Johnstone, Fein, & Yingling, 1988). More recently, task-related desynchronization in 8–10 Hz activity was observed exclusively for accurate performers during a cognitive flexibility task (Verstraeten & Cluydts, 2002).

In light of this literature, and especially in light of results reported by Hong et al. (1996) and Bertolo et al. (2003), we speculated that decreased Alpha power during a given sleep stage might be a marker of mentation that is recalled following awakening from that stage. If a decrease in Alpha power were found to be related to recall of mentation from both REM and NREM sleep awakenings, this would be consistent with the interpretation of a similar process associated with dreaming in the two stages and would support a one-generator model of sleep mentation. It would also support the more general notion that Alpha activity has the same or similar functional significance in both waking and sleep states. Further, in view of evidence that highly specific Alpha frequency subbands are related to cognitive functions (e.g., Klimesch, Sauseng, & Gerloff, 2003; Verstraten & Cluydts, 2002), including recall of sleep mentation (Takeuchi, Ogilvie, Murphy, & Ferrelli, 2003), we assessed three commonly used Alpha subbands in relation to recall of sleep mentation.

The objective of this study was thus to determine if a decrease in Alpha power is associated with successful recall of mentation following awakenings from REM and NREM (Stage 2) sleep. We predicted that decreases in Alpha activity would be associated with successful mentation recall from both sleep stages. Although some previous research suggests that activity in Delta activity may also predict recall of mentation (e.g., Waterman, 1992), we made no specific prediction for this frequency band.

Methods

Participants

All EEG records were selected from the EEG data bank of the Dream and Nightmare Laboratory in the Sleep Research Center of Sacré-Coeur Hospital in Montréal. Participants were volunteers who were recruited by advertisement in newspapers and word of mouth, and who reported themselves to be healthy, that is, free from any sleep, psychiatric, or physical illnesses, to have normal sleep schedules, and to be free from medications prior to the experiment. No measures were taken to assure the verity of this information. Participants were reminded several days prior to participating to abstain from alcohol for at least 24 h, and from caffeine for at least 6 h, prior to arriving at the laboratory.

The volunteers were among 56 who had participated in a previous unpublished study of mental activity reported from REM and NREM (Stage 2) sleep (Faucher, Nielsen, Bessette, Raymond, & Germain, 1999; Raymond, Nielsen, Bessette, Faucher, & Germain, 1999). They were awakened by an auditory signal when a trained judge using the standard Rechtschaffen and Kales (1968) sleep staging criteria and unaware of the hypotheses of the study determined that either a REM or Stage 2 episode had fulfilled the requirements of a counterbalanced schedule (see Table 1). This schedule was designed so that (a) participants would each undergo an early and a late REM awakening and an early and a late Stage 2 awakening over two consecutive nights, (b) each pair of REM and Stage 2 awakenings would occur as close to the same time of night as possible over the two nights, (c) the duration of elapsed sleep stage (e.g., 5 min, 10 min, 15 min) would be similar for each pair of awakenings, (d) the order of REM and Stage 2 awakenings would be counterbalanced over the two recording nights, and (e) sleep times would conform as closely as possible to participants' habitual sleep times. PSG tracings for these 56 participants were

Table 1. *Awakening Schedule for Elicitation of REM and Stage 2 Mentation Reports*

	Target sleep stage	
Time elapsed in sleep stage before awakening	First experimental night	Second experimental night
5 min	1st REM episode	Descending Stage 2 prior to 1st REM episode
10 min	Descending Stage 2 after 2nd REM episode	2nd REM episode
15 min	3rd REM episode	Descending Stage 2 after 3rd REM episode
15 min	Ascending Stage 2 prior to 4th REM episode	4th REM episode
Variable	Morning awakening	Morning awakening

evaluated by the first author to screen out participants for whom sleep stage classification prior to the experimental awakening was doubtful or ambiguous or for whom anomalies in the EEG were apparent. This included PSG tracings that contained microarousal events (American Sleep Disorders Association, 1992), alpha bursts in REM sleep (Cantero et al., 2002), and wakefulness epochs occurring within the target sleep stage.

Finally, because not all of the selected participants were awakened in all four conditions on each night, and because not all participants recalled mentation on at least one of their awakenings, the sample was further reduced to a total of 11 participants in the Stage 2 group and 8 participants in the REM group. For Stage 2, there were 8 female and 3 male participants (mean age: 23.6 ± 3.33 yrs; 2 left-handed) who had at least two awakenings in Stage 2 sleep, one with and one without mentation recall. Three of these participants had both awakenings occur on the same night. For stage REM, there were 6 female and 2 male participants (mean age: 27.4 ± 4.61 yrs; 0 left-handed) who had at least two awakenings in REM sleep, one with and one without mentation recall. Seven of these participants had both awakenings occur on the same night.

Recording Apparatus

All-night EEGs were recorded using a Grass Model 12 Neurodata Acquisition System (− 6 dB filters with cutoffs at 0.30 [time constant 0.4 s] and 100 Hz) and archived under the control of Rhythm version 9.1 software. EEG signals were sampled at 128 Hz using a linked ear reference with a 10 KΩ resistance.

EEG Quantitative Analysis

We assessed the 3 min of EEG immediately preceding each awakening tone from REM (either phasic or tonic) and Stage 2 sleep. From these 3 min, we selected epochs that were free from body movement, eye movement, and ECG and EMG artifacts (Rechtschaffen & Kales, 1968) for analysis by fast Fourier transform (FFT) using Harmonie version 5.0b software. Absolute Power for 19 standard EEG sites (Fp1, Fp2, F3, F4, F7, F8, C3, C4, P3, P4, O1, O2, T3, T4, T5, T6, Fz, Cz, Pz; American Electroencephalographic Society, 1994) was computed on successive 4.0-s epochs using a cosine window with 50% overlap and a spectral resolution of 0.25 Hz. Periodograms for these computations were averaged to obtain absolute amplitude power spectra. Frequency bandwidths were defined as: Delta (0.50–4.00 Hz), Theta (4.00–8.00 Hz), Alpha (8.00–12.00 Hz), Sigma (12.00–14.00 Hz), Beta 1(14.00–20.00 Hz), Beta 2 (20.00–32.00 Hz). The Alpha band was further subdivided into slow Alpha (7.50–9.50 Hz), middle Alpha (9.50–11.50 Hz), and fast Alpha (11.50–13.50 Hz) according to published criteria (Cantero, Atienza, Gómez, & Salas, 1999; Takeuchi et al., 2003; Tanaka, Hayashi, & Hori, 1997).

To facilitate the use of parametric statistics, we used amplitude power, which is calculated as the square root of power. To facilitate comparisons of mean Alpha power between recall and no recall awakenings from both Stage 2 and Stage REM sleep, as well as of t and p values from multiple t tests, StatMap+3D version 1.1 was used to generate EEG cartographic maps. Our mean maps (Duffy, Bartels, & Burchfiel, 1981) consist of color representations of average amplitude power (in microvolts) for 19 original data points with quadratic interpolation of all other points. Mean maps were compared statistically using paired t tests, the results of which were graphically represented with probability maps. The latter are similar to mean maps except that the raw data before interpolation consists of p values from the 19 paired t test comparisons, rather than average power values.

Sleep Mentation Collection

Participants were awakened using a standard acoustic stimulus. By intercom, the experimenter asked the participant what was going on in his/her mind just before the awakening. Verbal reports were tape-recorded and later transcribed. These reports were assessed by a blind judge using the following criteria: (a) *Recall*, a report in which the participant described the content of some previous mentation; (b) *White dream*, a report in which the participant indicated that some mentation had occurred prior to awakening but was not able to describe it; (c) *No recall*, in which the participant indicated that he/she could not recall any mentation. Awakenings followed by white dreams were excluded from further consideration.

Statistical Analyses

Statistica 5.1 and SPSS 10.0 were used to perform statistical tests for EEG measures. To assess main effects for sleep stage and mentation recall we used 2 × 2 × 19 ANOVAs with sleep stage (REM, Stage 2) as a between-subjects variable, mentation (recall, no recall) and EEG (electrodes 1–19) as repeated measures variables, and six classical frequency bands (Delta, Theta, Alpha, Sigma, Beta 1, Beta 2) as separate dependent measures. Topographic differences between recall and no-recall conditions were then subject to topographic mapping procedures for select frequency bands as described above using correction for Type I error based on the threshold of $p < .01$ for each of the eight sets of comparisons. Effect sizes for F and t tests were calculated and classified according to Cohen's (1998) system, that is, F/t: small = 0.10/0.20; medium = 0.25/0.50; large = 0.40/0.80.

EEG samples for Stage 2 awakenings followed by either recall (St2R) or no recall (St2N) were compared. Similarly, samples for REM awakenings followed by either recall (REMR) or no recall (REMN) were compared. Effect sizes were calculated for significant recall/no recall comparisons ($p < 0.01$) observed in the Alpha band (8–12 Hz), Alpha subbands, and Delta band for both Stage 2 and REM sleep conditions.

Although amplitude power involves a root square transformation that tends to normalize skewed distributions, we nevertheless tested the normality of our sample distributions using Shapiro–Wilks tests; p values equal to or less than .05 were taken to indicate nonnormal distributions.

Results

The absence of a confound between the mentation condition and the time-of-night of awakenings was confirmed by a lack of difference between the clock time of awakenings for the recall and no recall groups in Stage 2 sleep, $t_{10} = 1.324$, $p = .215$, and REM sleep, $t_7 = 1.165$, $p = .282$. There were also no between-groups differences when clock time was adjusted to each participant's sleep onset time, that is, time from lights out to awakening for each participant (Stage 2: recall: 5:23 ± 1:48; no recall: 4:22 ± 2:09; $t_{10} = 1.323$, $p = .215$ and REM: recall: 6:07 ± 1:52; no recall: 4:54 ± 3:01; $t_7 = 1.119$, $p = .300$).

For the Alpha band, a main effect for mentation, $F_{1,17} = 14.683$, $p = .001$, $d = .879$, indicated that Alpha power was lower for awakenings with recall ($M = 1.750 \pm 0.069$) than

for awakenings with no recall ($M = 2.032 \pm 0.101$). The direction of the mentation effect (no recall > recall) was *the same* in both stages (see Figure 1) as indicated by significant mentation effects for 2 × 19 ANOVAs conducted separately for REM, $F_{1,7} = 6.676$, $p = .036$, $d = 0.913$, and Stage 2, $F_{1,10} = 9.570$, $p = .011$, $d = 0.933$, sleep. Figure 1 also displays Alpha power averaged over the 19 electrodes for individual participants by mentation and stage conditions; the mentation effect (no recall > recall) can be observed for 10 of 11 participants in Stage 2 sleep and for 6 of 8 participants in REM sleep.

When assessed by subbands, the mentation difference was robust for slow Alpha, $F_{1,17} = 14.669$, $p = .001$, $d = 0.879$, and middle Alpha, $F_{1,17} = 13.850$, $p = .002$, $d = 0.854$, but marginal for fast Alpha, $F_{1,17} = 4.175$, $p = .057$, $d = 0.469$.

For other frequency bands evaluated, parallel mentation main effects were also observed for Delta, $F_{1,17} = 11.354$, $p = .004$, $d = 0.773$, and Theta, $F_{1,17} = 7.093$, $p = .016$, $d = 0.611$. For Delta, the mentation effect was present for both REM, $F_{1,7} = 12.780$, $p = .009$, $d = 1.264$, and Stage 2, $F_{1,10} = 5.410$, $p = .042$, $d = 0.701$, sleep; for Theta, the effect was not significant in either of the separate analyses, $p = .081$ and .130, respectively. No effects or interactions involving mentation were observed for the other bands.

Main effects for stage were found for Delta, $F_{1,17} = 14.249$, $p = .002$, $d = 0.866$, and for bands encompassing frequencies from 11.5 to 20.0 Hz, that is, fast Alpha, $F_{1,17} = 16.091$, $p = .001$, d = 0.920, Sigma, $F_{1,17} = 18.925$, $p = .0004$, $d = 0.998$, and Beta 1, $F_{1,17} = 7.052$, $p = .017$, $d = 0.609$. In all cases, the direction of differences was Stage 2 > REM.

To further clarify the Alpha mentation effect for the two sleep stages, we examined subbands and brain topography separately for Stage 2 and REM sleep EEG samples.

Stage 2 Mentation Recall

A total of 157 4-s EEG epochs was analyzed for the Stage 2 recall condition (St2R) and 150 epochs for the Stage 2 no-recall condition (St2N). The mean number of epochs analyzed per participant did not differ by condition (St2R: 14.00 ± 1.48; St2N: 13.58 ± 2.15; $F_{1,11} = 0.270$; $p = .614$). Shapiro–Wilks tests indicated that 73.5% of the EEG distributions examined were normal; thus, parametric statistics were applied to the original distributions.

Results for the topographic analyses of Alpha and its three subbands are summarized in Table 2 and the left panels of Figure 2. The relative decrease in overall Alpha power was apparent in seven electrodes at the $p = .01$ threshold, in six trends at $p = .05$ and in effect sizes that were medium (Fp1: $d = 0.694$) to large (T4: $d = 2.497$). The effect was topographically most widespread for slow Alpha, with six electrodes attaining $p = .01$, seven trends, and effect sizes ranging from medium (T6; $d = 0.626$) to large (F4; $d = 2.213$) and middle Alpha, with five electrodes attaining .01, seven showing trends, and effect sizes ranging from medium (Fp1; $d = 0.597$) to large (T4; $d = 2.605$). The effect was least pervasive for fast Alpha, with only five trends and effect sizes from small (Fp1; $d = 0.170$) to large (T4; $d = 1.433$).

To determine whether the mentation effect was due to Alpha activity in the range of sleep spindles (12–14 Hz), all 4-s epochs bearing spindles (Rechtschaffen & Kales, 1968) were removed from the Stage 2 sample and FFTs were recalculated. This reselection led to the loss of 44 and 58 (or 28% and 39%) of all 4-s epochs for the recall and no-recall conditions, respectively. The mentation effect was still apparent in the slow Alpha subband, $F_{1,10} = 6.944$, $p = .025$, $d = 0.795$, but not in middle ($p = .180$) and fast ($p = .463$) Alpha subbands. Further, assessment of the contribution of epochs containing spindles—calculated by subtracting, for each electrode and each participant, power values for "spindle-free" epochs from those for the original epochs—revealed indications of the mentation effect for middle Alpha, $F_{1,10} = 5.295$, $p = .044$, $d = 0.694$, and fast Alpha, $F_{1,10} = 5.236$, $p = .045$, $d = 0.690$, but not for slow Alpha, $F_{1,10} = 1.397$, $p = .265$, d = 0.357. In short, the mentation effect was apparent in *both* background EEG (slow Alpha) and sleep spindles (middle and fast Alpha).

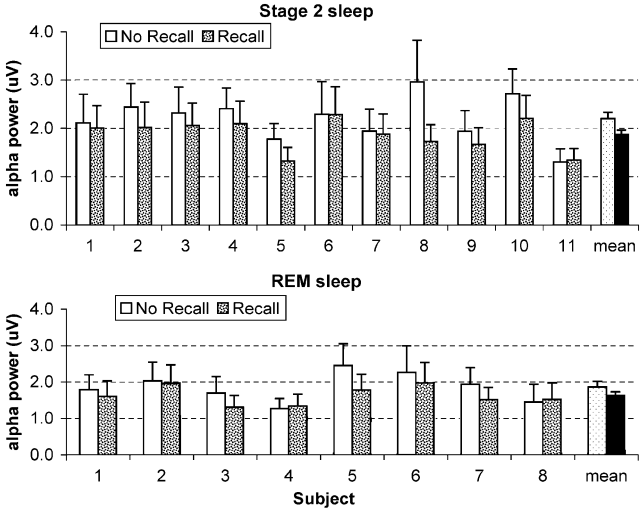

Figure 1. Per subject Alpha power for 19-electrode means (white and pale blue bars) and group means (gray and dark blue bars) for no-recall and recall conditions by sleep stage. Per subject Alpha power for recall was lower than that for no recall for 10 of 11 participants in Stage 2 sleep and for 6 of 8 participants in REM sleep. Mean power for recall is lower than for no recall in both sleep stages, although the effect is somewhat larger for Stage 2 sleep.

REM Sleep Mentation Recall

A total of 80 epochs were analyzed for the REM recall condition (REMR) and 84 epochs for the REM no-recall condition (REMN). The number of epochs analyzed per participant did not differ by condition (REMR: 10.00 ± 3.93; REMN: 10.50 ± 2.39; $F_{1,7} = 0.071$; $p = .798$). The overall number of scoring epochs bearing rapid eye movements was low, 6 out of 164, with 3 each in the REMR and REMN groups. These epochs were not removed from the EEG samples. Shapiro–Wilks tests indicated that 75.2% of the EEG distributions examined were normal and parametric statistics were applied to the original distributions.

Results are shown in Table 2 and in the right panels of Figure 2. The relative decrease in overall Alpha power was less widespread than for Stage 2, both topographically and in the frequency domain, but nevertheless present. No electrodes attained the $p = .01$ threshold, but 10 showed trends at $p = .05$ and effect sizes were medium (Cz: $d = 1.471$) to large (P3: $d = 4.806$). When assessed by subbands, the effect was most apparent for middle Alpha, with two electrodes attaining $p = .01$, four showing trends, and effect sizes all large in magnitude (Cz: $d = 0.928$ to T5: $d = 2.128$). No effects or trends were observed for slow or fast Alpha.

Table 2. *p Values Resulting from Paired t Tests on Recall versus No Recall Conditions for Classical Alpha Power and Slow, Middle, and Fast Alpha Power over Different Brain Regions*

		Stage 2 sleep				REM sleep				
Brain region	EEG	Alpha (8.0–12.0 Hz)	Slow (7.5–9.5 Hz)	Middle (9.5–11.5 Hz)	Fast (11.5–13.5 Hz)	EEG	Alpha (8.0–12.0 Hz)	Slow (7.5–9.5 Hz)	Middle (9.5–11.5 Hz)	Fast (11.5–13.5 Hz)
Left frontal	Fp1	0.145	0.044[a]	0.198	0.704	Fp1	0.072	0.156	0.152	0.499
	F3	0.066	0.011[a]	0.100	0.154	F3	0.025[a]	0.097	0.045[a]	0.143
	F7	0.107	0.059	0.158	0.204	F7	0.033[a]	0.065	0.066	0.106
Right frontal	Fp2	0.023[a]	0.043[a]	0.028[a]	0.600	Fp2	0.039[a]	0.141	0.127	0.487
	F4	0.003*	0.001*	0.006*	0.051	F4	0.048[a]	0.161	0.174	0.337
	F8	0.003*	0.011[a]	0.003*	0.305	F8	0.030[a]	0.109	0.148	0.525
Central	C3	0.035[a]	0.018[a]	0.046[a]	0.071	C3	0.039[a]	0.064	0.031[a]	0.155
	C4	0.004*	0.003*	0.009*	0.030[a]	C4	0.064	0.077	0.072	0.426
Left temporo-parietal	P3	0.007*	0.011[a]	0.010*	0.069	P3	0.019[a]	0.067	0.009*	0.135
	T3	0.044[a]	0.053	0.048[a]	0.072	T3	0.079	0.106	0.137	0.203
	T5	0.015[a]	0.018[a]	0.018[a]	0.048[a]	T5	0.024[a]	0.100	0.006*	0.179
Right temporo-parietal	P4	0.007*	0.006*	0.023[a]	0.059[a]	P4	0.077	0.177	0.057	0.174
	T4	0.000*	0.001*	0.001*	0.031[a]	T4	0.253	0.395	0.218	0.120
	T6	0.077	0.175	0.124	0.046[a]	T6	0.086	0.094	0.049[a]	0.094
Midline	Fz	0.046[a]	0.008*	0.061	0.164	Fz	0.067	0.185	0.092	0.084
	Cz	0.050[a]	0.059	0.040[a]	0.079	Cz	0.358	0.199	0.283	0.663
	Pz	0.006*	0.008*	0.013[a]	0.083	Pz	0.203	0.085	0.140	0.341
Occipital	O1	0.127	0.137	0.156	0.137	O1	0.031[a]	0.089	0.026[a]	0.160
	O2	0.083	0.121	0.059	0.087	O2	0.044[a]	0.110	0.056	0.165

[a]Trend at $p < .05$ threshold.
*Significant difference at $p < .01$ threshold; mean absolute power with mentation recall is lower than for power without mentation recall.

Visual examination of the Stage 2 and REM maps reveals that the mentation effect was shared by six electrode derivations grouped in two distinct regions: right frontal (Fp2, F4, and F8) and left centro-temporo-posterior (C3, T5, and P3). When Alpha subbands are also considered in the topographic comparisons, this interstage consistency is apparent only for middle Alpha in the left centro-temporo-posterior cluster (C3, T5, and P3).

Delta Power Assessments

Because of these observed consistencies, we conducted post hoc assessments of the topographic distributions of Delta power differences for REM and Stage 2 sleep to determine if the consistencies held also for the Delta band (see Table 3 and Figure 3). For the 19 Stage 2 comparisons, only one electrode surpassed the $p < .01$ threshold (F4) whereas five others showed trends (F3, F8, Fz, Fp1, T4). For the 19 REM sleep comparisons, seven electrodes surpassed $p < .01$ (C3, C4, Cz, F3, Fz, T4, T3) and four showed trends (P3, P4, Pz, T5). No electrodes revealed a significant Delta power difference for both Stage 2 and REM sleep, although Fz, F3, and T4 revealed both trends for Stage 2 ($p = .017$, .027, and .050) and significant differences for REM ($p = .007$, .001, and .007), T5 revealed a trend for both states ($p = .051$ and .037) and several others revealed weak, albeit consistent, trends for one or both states, for example, P3 (Stage 2: $p = .063$; REM: $p = .019$) and Pz (Stage 2: $p = .061$; REM: $p = .033$). In sum, the right-frontal (Fp2, F4, and F8) and centro-temporo-parietal (C3, T5, and P3) consistencies observed for Alpha power comparisons were not clearly evident for Delta power.

Discussion

In general, Alpha power behaves similarly in Stage 2 and REM sleep relative to the success or failure of recalling prior sleep mentation. In both states, lower overall Alpha power, and lower middle Alpha power in particular, is related to successful mentation recall. However, in Stage 2, slow and fast Alpha also differentiate between conditions to some extent, whereas in REM sleep these differences are more marginal. Topographic distributions of the effect are not identical in the two states, but there are two consistent areas of overlap.

It is known that predominant Alpha frequency varies as a function of age, brain volume, and other factors (Köpruner, Pfurtscheller, & Auer, 1984; Niedermeyer, 1993; Nunez, 1995). Because our Stage 2 and REM groups consisted of different participants it is possible that differences in the patterns of results for these two states reflect intersubject differences. For example, some individuals may have had more characteristically prominent activity in the Alpha frequency range than others (Klimesch, 1996). However, the relative consistency in Alpha reduction that we observed for mentation recall in REM and Stage 2 sleep suggests that a common mechanism is mediating both EEG modulation and mentation recall.

Our results do not, of course, indicate what this mediating mechanism might be. However, they do support our hypothesis and notions proposed by other authors about the relationship between Alpha activity and sleep mentation. Cantero, Atienza, and Salas (2000), in distinguishing among different kinds of Alpha activity characteristic of REM sleep, suggested that a

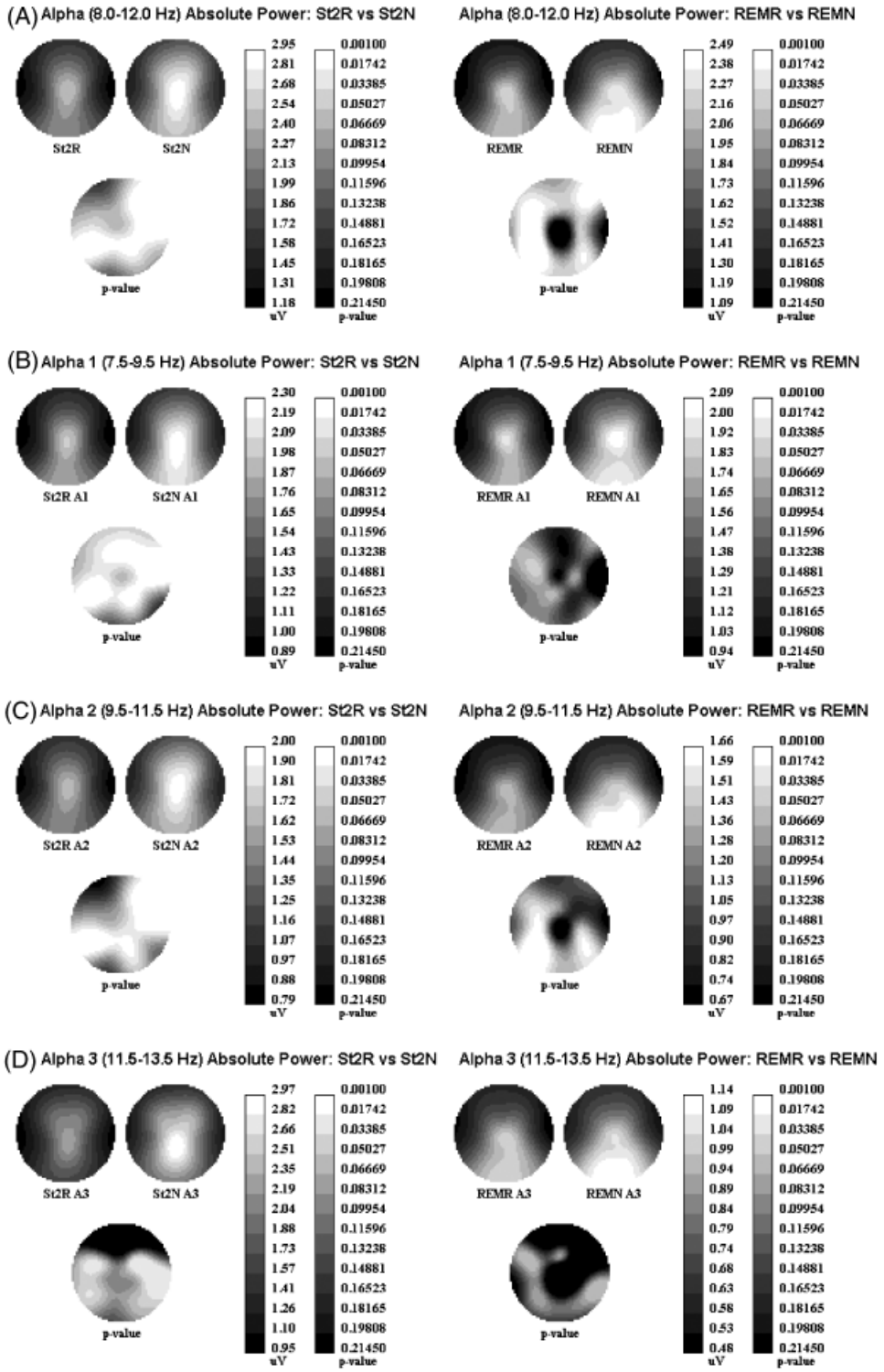

Figure 2. Topographic EEG maps for recall versus no recall comparisons of Alpha power. In each of eight sets of comparisons, two types of maps are shown, with two corresponding legends to their right: *mean maps* (in pairs), illustrating mean absolute power scores for recall and no-recall conditions for the 19 electrode sites (with interpolation), use red and orange hues to represent higher power; and *p maps*, illustrating *p* values for each of the paired *t* test comparisons of the 19 electrode pairs (with interpolation), use red hues to indicate significant differences ($p < .01$ to $p < .001$) and trends ($p < .05$ to $p < .01$). The four rows of maps depict (A) paired comparisons (recall vs. no recall) for total Alpha power (8.00–12.00 Hz) for Stage 2 sleep (left panel) and REM sleep (right panel). Predominantly red hues for *p* maps in both instances indicate relatively widespread reduction of Alpha power as a function of mentation recall in both Stage 2 and REM sleep; (B, C, and D) paired comparisons for slow (7.50–9.50 Hz), middle (9.50–11.50 Hz), and high (11.50–13.50 Hz) Alpha power for Stage 2 sleep (left panel) and REM sleep (right panel). The differences shown in panel A are reflected most robustly for both sleep stages in the middle Alpha power band (panel C). St2R: Stage 2 awakening followed by mentation recall; St2N: Stage 2 awakening followed by no recall; REMR: REM sleep awakening followed by mentation recall; REMN: REM sleep awakening followed by no recall.

Table 3. *p Values Resulting from Paired t Tests on Recall versus No-Recall Conditions for Delta Power over Different Brain Regions*

	Stage 2 sleep		REM sleep	
Brain region	EEG	Delta (0.5–4.0 Hz)	EEG	Delta (0.5–4.0 Hz)
Left frontal	Fp1	0.040[a]	Fp1	0.124
	F3	0.027[a]	F3	0.001*
	F7	0.147	F7	0.503
Right frontal	Fp2	0.062	Fp2	0.735
	F4	0.006*	F4	0.086
	F8	0.045[a]	F8	0.573
Central	C3	0.101	C3	0.002*
	C4	0.091	C4	0.008*
Left temporo-parietal	P3	0.063	P3	0.019[a]
	T3	0.293	T3	0.001*
	T5	0.052	T5	0.038[a]
Right temporo-parietal	P4	0.103	P4	0.027[a]
	T4	0.050[a]	T4	0.007*
	T6	0.313	T6	0.059
Midline	Fz	0.017[a]	Fz	0.007*
	Cz	0.126	Cz	0.007*
	Pz	0.062	Pz	0.033[a]
Occipital	O1	0.268	O1	0.125
	O2	0.123	O2	0.070

[a]Trend at $p < .05$ threshold.
*Significant difference at $p < .01$ threshold; mean absolute power with mentation recall is lower than for power without mentation recall.

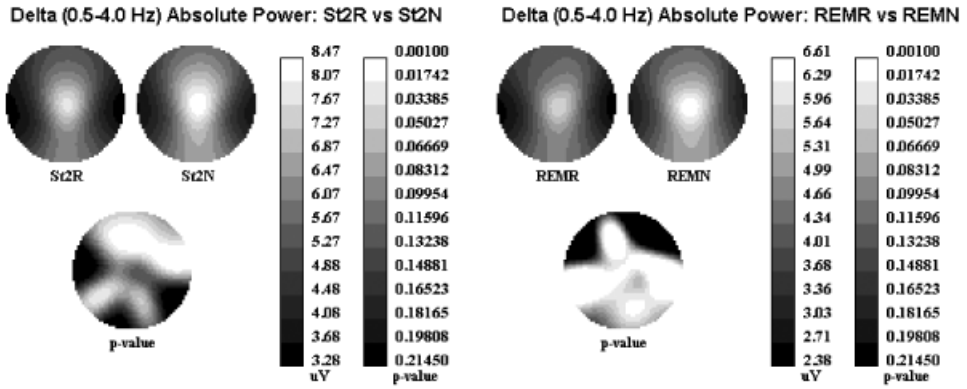

Figure 3. Topographic EEG maps for recall versus no recall comparisons of Delta power (0.50–4.00 Hz). Map definitions and legends are as for Figure 2. Red hues for *p* maps in both instances again indicate reduction of Delta power as a function of mentation recall in both Stage 2 and REM sleep.

decrease in background Alpha activity could be associated with visual imagery in dreams. Jouny, Chapotot, and Merica (2000) explained a decrease in Alpha power during phasic REM sleep as a possible correlate of visual cortex activation during information processing and, in particular, during visual imagery. These authors have focused principally on REM sleep; our findings provide grounds for extending this reasoning to include Alpha-linked information processing during NREM sleep as well.

It is possible that the mechanism signaled by Alpha reduction is that of mentation production. However, because mentation production and mentation retrieval are completely confounded variables in this study (as they are in most such studies), it is also possible that Alpha reduction signals the operation of some cognitive process *other* than production, that is, mentation retrieval or processes that might influence the salience or memorability of mentation (orienting, attending, affect, sensory quality, etc.). The fact that a similar association between abrupt Alpha power decreases and the recall of hypnagogic images from sleep onset (NREM) was recently observed (Germain & Nielsen, 2001), regardless of whether images were visual, auditory, or kinesthetic in nature, supports our inclination to view Alpha power as an index of mentation production as opposed to sensory quality of the mentation. However, further assessments of mentation are needed to clarify this issue. Further study is also needed to determine whether the results generalize to Stages 3 and 4 sleep, for which there is also much evidence of mentation recall (e.g., Cavallero, Cigogna, Natale, Occhionero, & Zito, 1992; Cicogna, Natale, Occhionero, & Bosinelli, 2000; Natale, 2000; Pivik & Foulkes, 1968; Purcel, Mullington, Moffitt, Hoffmann, & Pigeau, 1986; Tracy & Tracy, 1974).

In a more general sense, our findings might be taken to support the view that Alpha activity, or at least some subset of the classically defined Alpha band (e.g., middle Alpha), may have the same functional significance during both wakefulness and sleep. For example, an increase in Alpha power has been suggested to reflect cortical deactivation in both sleep and waking (Benca et al., 1999). In this respect, it may prove useful to further differentiate subtypes of Alpha activity in sleep, either by virtue of their presumed origins in thalamic versus occipital generators (Lopes da Silva, Vos, Mooibroek, & Van Rotterdam, 1980; Pivik

& Harman, 1995) or by the association between specific Alpha subfrequencies and different classes of cognitive processes. For instance, in line with Klimesch's (1997) hypothesis that different waking state memory processes are indexed by upper (encoding) and lower (attention) Alpha power, our current findings might be attributed more to attentional processes because mentation recall/no-recall differences for both sleep stages were distinguished most clearly by middle and lower Alpha frequencies. However, until more fine-grained analyses of Alpha activities during wakefulness and sleep and their associations with varieties of mentation are undertaken, this possibility remains speculative.

Our findings also provide limited support for an inverse relationship between slow wave activity and sleep mentation. We found high Delta activity for our no-recall condition in both sleep states. Early studies found similar inverse relationships for more broadly defined frequency bands, for example, 0.4–14.0 Hz (Lehmann, Dumermuth, Lange, & Meier, 1981) and 2.0–25.0 Hz (Meier, 1989). More recently, Waterman (1992) reported an inverse correlation between Delta power and the length of mentation reports (word information count) for REM sleep, but not for Stage 2. In the present study, the effect was, in fact, much more robust for REM sleep. Thus, there is some consistency among studies supporting Delta power as an index of sleep mentation that deserves further study. On the other hand, it should be noted that Waterman (1992) did not find relationships between mentation length and other EEG frequencies, including Alpha power, which is not consistent with our findings for Alpha power in the present study.

Our results for REM sleep Alpha power are presumably not due to Alpha power decreases that occur during phasic REM sleep (Jouny et al., 2000; Waterman, Elton, Hofman, Woestenberg, & Kok, 1992) or to the Alpha power decline between early, middle, and late REM periods (Waterman et al., 1992) because our samples of recall and no-recall EEG sections included few, albeit equal numbers of phasic REM sleep epochs and were sampled homogenously across the night. For similar reasons, our results for REM sleep Delta power are not likely due to known decreases in Delta power across the night (Dijk, Beersma, & van de Hofdakker, 1989; Waterman et al., 1992). Because Delta power decreases occurred predominantly over frontal and central derivations, it is possible that the differences are due to eye movement artifact—which is usually predominant over frontal derivations. However, such eye movement artifact would not be expected to occur in Stage 2 sleep.

Because our observed changes in Alpha and Delta activity were similar across sleep states, these findings support one-generator models of sleep mentation to some degree. One-generator models that assume psychophysiological isomorphism (e.g., Nielsen, 2000) imply that common physiological processes are involved in mentation production across sleep states. For example, Nielsen (2000) has suggested that mentation recall in Stage 2 sleep might be due to activation of processes normally linked to REM sleep—so-called covert REM sleep processes. In the present study, we did not specifically score the presence or absence of the most obvious covert REM sleep signs in Stage 2 sleep (eye movements and EMG phasic activity) and our results do not address the model in this respect. Nevertheless, Alpha reduction is known to be a typical characteristic of REM sleep. For example, Alpha power is reduced during recovery sleep following selective REM sleep deprivation (Brunner, Dijk, & Borbely, 1993; Endo et al., 1998), when REM sleep pressure is high and sleep mentation is more vivid (Weinstein, Schwartz, & Ellman, 1991). Endo et al. (1998) even propose that Alpha power may be a biomarker of REM sleep homeostasis. Alpha power decreases have also been found to index both verbal (Hong et al., 1996) and visual (Bertolo et al., 2003) imagery in dreams. In fact, our finding of cross-state consistencies in middle Alpha decreases for derivations over Wernicke's area (T5, P3) parallels Hong et al's finding that dream imagery is linked preferentially to Alpha activity in this area. Decreases in Alpha power over left posterior cortex have also been found during intense visual imagery in the waking state (Engelkamp, 1995). Together, these findings suggest that Alpha decreases, and particularly decreases in left temporo-parietal regions, could be further explored as markers of covert REM sleep processes.

Findings also implicate Delta power changes. Although it is known that Delta and Alpha coherence covary in NREM sleep (Achermann & Borbely, 1998) and that the dominance of Delta and Alpha power varies in parallel over sleep states, both as a function of the underlying ultradian rhythm (Shannahoff-Khalsa, Gillin, Yates, Schlosser, & Zawadzki, 2001) and in response to sleep deprivation (Borbely, Baumann, Brandeis, Strauch, & Lehmann, 1981; Dijk, Brunner, & Borbely, 1990), there is still relatively little literature in which the Delta frequency band is validated as a measure of cognitive processes.

Our findings could also support a one-generator model proposed by Cicogna and colleagues (Cicogna & Bosinelli, 2001; Cicogna et al., 2000) in which quantitative differences detected among dream measures collected from different sleep stages (e.g., differences in report length) are taken to reflect differential engagement of cognitive systems, especially memory systems. In fact, our findings indicated lower mean Alpha power values in REM than in Stage 2 sleep—as might be predicted by their model. However, to conclusively validate Alpha activity as an indicator of cognitive activity in this quantitative sense, we would need to make direct within-subjects comparisons between Stage REM and Stage 2 groups to determine linkages between levels of cognitive elaboration (e.g., total word count) and levels of Alpha power. Such comparisons were not possible with the present design.

Finally, it should be noted that many of the recall/no-recall differences in Alpha and Delta power observed in the present study were not consistent topographically or in the frequency domain over the two sleep stages. Although such inconsistencies might be taken to support two-generator models of mentation production, it nevertheless remains possible that much variability in Alpha frequency is due to either (1) uncontrolled differences in dominant Alpha frequency, the latter varying with participant age and memory ability, memory load, brain topography, and other factors (Klimesch, 1997), or (2) stage-specific modulation of an otherwise common mentation generator. Until such possibilities are more carefully considered, the interpretation of between-stage inconsistencies may be premature.

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