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# Topographic Cortical Mapping of EEG Sleep Stages During Daytime Naps in Normal Subjects

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**Summary:** Computer-generated cortical maps of power spectral estimates derived from 16 leads were drawn based on daytime sleep recordings in four normal volunteers. These data were compiled from nine 10-s artifact-free, EEG epochs from awake, stages 1–4 and REM sleep in each volunteer. EEG leads were placed on the left hemisphere and midline according to the 10–20 system with four additional interpolated posterior locations. Magnitude spectral estimates with 1 Hz resolution and adjacent frequencies (delta 2–4, alpha 8–12, beta 13–18) were analyzed with two-way ANOVA (lead by sleep stage). Delta activity was relatively uniform and of low amplitude in awake, eyes-closed subjects, and REM. Delta power increased at the vertex in stage 1. With progressing, non-REM sleep stages, it increased in power and enlarged radially to the intraparietal sulcus posteriorly, and the superior frontal gyrus anteriorly. Comparison of maps with ear and a computed average reference yielded similar topographic patterns. Alpha activity was expectedly maximal occipitally in awake subjects, but surprisingly a frontal area appeared in slow wave sleep. Beta activity in awake subjects was low and maximal parietally; stages 1 and REM showed even lower and more uniform distribution. Stage 2 showed the greatest power, concentrated at the vertex, with stages 3 and 4 diminishing. These data suggest that sleep stages are not completely uniform electrophysiologically across the cortex. This opens the possibility for a new method for the diagnosis of sleep disorders and alternatives in sleep staging.
**Key Words:** Sleep—Sleep stages—EEG spectral analysis—Cortical localization.

Traditionally, sleep stages have been defined on the basis of EEG data gathered from a single unipolar electrode, usually located at $C_3$ over the central cortex (1),

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but vertex–occipital derivations have also been employed (2). This practice derives from the practical difficulties of recording many leads, and from the suggestion that regional differences may be relatively unimportant for the scoring of sleep stages (e.g. (1)).

Although patients with sleep disorders and psychiatric illnesses have been reported to have abnormal proportions of sleep stages, the possibility has never been explored that they also differ in the topographic distribution of sleep EEG patterns. Since advances in computer technology have made the development of topographic maps feasible, we have now begun to evaluate cortical distributions of EEG activity during the various stages of sleep. Previous studies have documented the utility of mapping the waking EEG and evoked potential (3–5), but detailed sleep studies have not appeared. Recently, Findji et al. (6) have reported eight channel comparisons of delta rhythm powers indicating a regional increase with stages 3 and 4. In order to characterize the human cortex with greater resolution and to clarify issues of appropriate EEG reference raised by Findji et al. (6), we have studied sleep using 16 leads on the left hemisphere with two reference systems.

## METHODS

Because of the more extensive and novel recording technique, we elected to do our initial study during daytime. Four adult volunteers, ages 21 to 25, were studied during a 3 to 4-h recording session, beginning at noon. Three subjects arose at 6 AM to facilitate daytime sleep.

### Recordings

Traditional sleep recording procedures, consisting of unipolar EEG, EOG, and EMG, were used for determining sleep stage. A paper record was made on a Grass Model 78 polygraph with sensitivity of 50 $\mu$V/7.5 mm and a paper speed of 10 mm/s. Sleep stages were determined by a single independent reader for 30-s epochs according to standard criteria (1).

A set of 16 electrodes was placed on the left hemisphere, 12 according to the International 10–20 system (left hemisphere and midline: $FP_1$, $F_Z$, $C_Z$, $P_Z$, $O_Z$, $F_3$, $C_3$, $P_3$, $O_1$, $F_1$, $T_3$, $T_5$) and four additional locations posteriorly (at centers of squares formed by the regular positions; see reference 5). Recordings were referenced to left ear. EEG was recorded on FM tape. In each subject, nine representative 10-s epochs were selected for each stage. All 16 leads for each epoch were inspected for artifacts. Although traditional sleep criteria allow staging of a 30-s epoch on the basis of the majority of activity (e.g., EEG stage 1 may contain alpha, but must be less than 50% of total epoch), we chose to use 10-s epochs that were uniform. Thus, stage 1 epochs were without any alpha and stage 2 epochs always contained at least one spindle, with or without K complexes. As described in the scoring manual, stage 3 epochs contained 20–50% delta activity, while stage 4 had greater than 50%. Presence of a sleep spindle in an epoch which would otherwise be scored as stage 4 was not an exclusionary factor. All REM epochs were selected to exclude large eye movement contamination in the EEG during that particular 10 s.

EEG activity was digitized at 102.4 Hz (1024 samples/epoch), and low frequency subharmonics were removed by an autoregressive filter (7). A window function consisting of a 10% cosine taper was obtained by weighing the 50 points at either end of the epoch by a cosine bell. A standard fast Fourier transform was applied and the power spectrum estimates were computed at 0.1 Hz steps. For smoothing 10 adjacent estimates were summed to yield 1 Hz resolution with the final estimates expressed as magnitude values in microvolts (square root of power). Thus, a peak-to-trough 75 $\mu$V delta wave series throughout the 10-s EEG epoch would yield a value of 37.5. If only 25% of the epoch contained delta activity, the value would be approximately 10. Additionally, the activity estimates for the following bands were computed by summing adjacent values: delta, 2.1–5.0 cps; theta, 5.1–8.0; alpha, 8.1–13.0; beta 1, 13.1 to 18.0; beta 2, 18.1 to 30.0.

Topographic maps were computed by linear interpolation from the four nearest electrodes (5). For each of the 4,000 picture elements in the map, four corresponding weights were multiplied by the appropriate four spectral power values. Interpolated values were displayed on an approximately equal area map of the lateral surface of the human cortex, using dot-density representation of power values. Figure 1 illustrates the map of awake eyes closed alpha activity in the four subjects, computed from the 16 mean values for each EEG electrode position.

Data were analyzed using a two-way ANOVA with repeated measures (stages by lead position), and averages for each subject across the nine epochs were entered. Individual subjects were similarly analyzed with the nine epochs entered separately. A second analysis was done on spectral estimates from EEG data expressed as a difference from the mean value across all 16 leads.

## RESULTS

### Delta activity

Waking, stage 1 and REM showed relatively low activity (mean across all leads 1.02 to 1.0 $\mu$V) which was greatest at $C_z$ and least in the temporal lobe (Fig. 2). Delta increased progressively in stage 2 (1.82), stage 3 (2.30), and stage 4 (2.52) ($F = 21.32$, $df = 1,3$, $p < 0.05$ for stage effect). Power tended to be highest on the midline ($F_z$-$P_z$-$C_z$-$O_z$) falling off in concentric circles; anterior and central midline values tripled from awake to stage 4, whereas occipital and temporal power merely doubled (lead by stage interaction, $F = 8.39$, $p < 0.10$ with most conservative $df = 1,3$ and $p < 0.05$ with 5 and 15 df). This was most evident at 3 cps ($F = 10.65$, $df = 1,3$, $p < 0.05$). When each subject was analyzed separately (nine epochs, per stage) three of the four had significant lead by stage interactions ($F = 12.7$, $p < 0.05$). The fourth subject had high levels of waking delta (2.02) and showed little change. (This individual had not been adequately sleep deprived, which might explain several differences observed.)

### Theta activity

Group analysis showed neither significant regional changes nor sleep stage changes. Individual analysis showed some subjects with lower theta in stage 1, but this was not a uniform pattern.

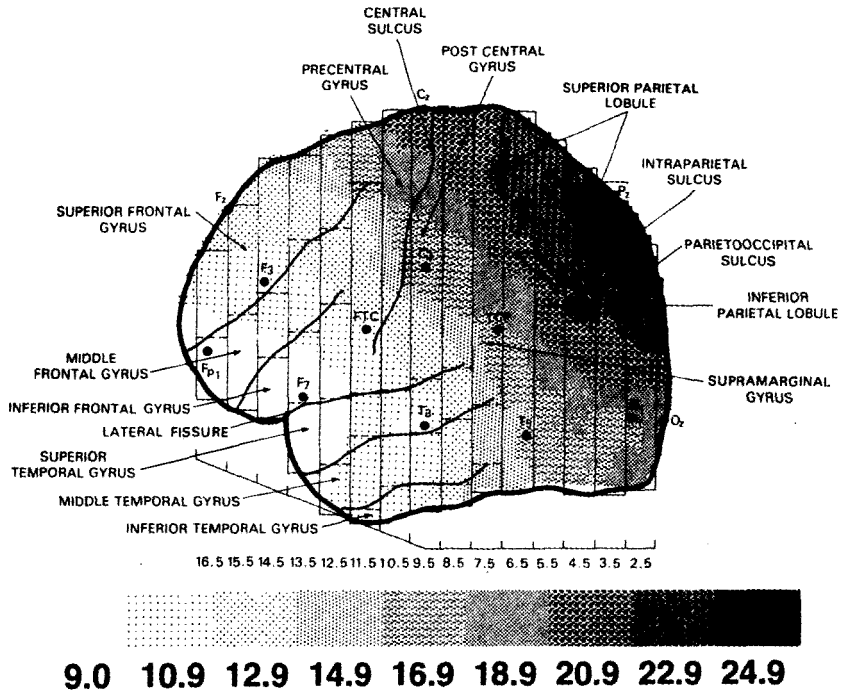


FIG. 1. Alpha power map for mean data for four awake, eyes-closed subjects. Labels are located on anatomical reconstruction from whole head sections (see 5). Numerical scale at bottom gives distance in cm from occiput. Gray scale gives alpha magnitude in microvolts.

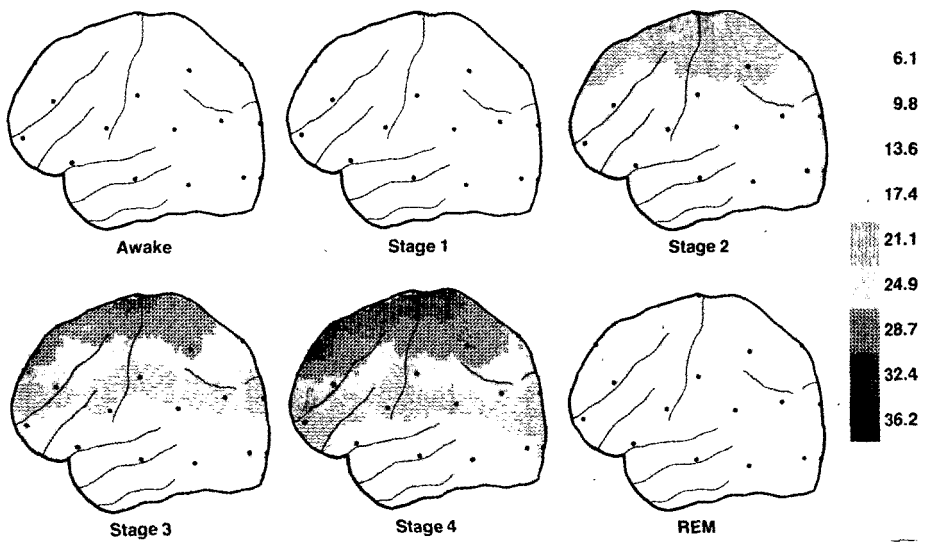


FIG. 2. Delta activity maps for mean data for four subjects. Delta is highest and covers the widest area in stage 4. Midline areas have higher delta in all stages. The downward spread of delta from stages 1 to 4 resembles a nightcap.

### Alpha activity

As expected, in restful waking with eyes closed, alpha energy was greatest in parietal-occipital areas (e.g., $P_z$, 2.65; $P_3$, 2.30; $F = 10.7$, lead effect, $p < 0.05$). In the group analysis, alpha diminished in all sleep stages ($F = 5.84$, $df = 5,15$, $p < 0.05$; conservative $df = 1,3$, $p < 0.10$). In individual analyses on the four subjects, this was more marked ($F = 18, 27, 1.1, 10.32$, respectively, $p < 0.05$ for three of four subjects). Alpha tended to move to a more anterior position in stages 3 and 4 ($F = 20, 43$, and 19 in three of four subjects).

### Beta activity

Beta 1 was least in stage 1 (0.35) and REM (0.34), highest in stage 2 (0.71), and then progressively decreased in stages 3 (0.57) and 4 (0.52) ($F = 12.2$, $p < 0.05$). Waking beta was maximal in the parietal region (Fig. 3) but tended to move anteriorly in slow wave sleep (Fig. 3) ($F = 8.25$, lead by stage interaction, $p < 0.10$, $df = 1,3$). Only one subject significantly confirmed this trend on individual analysis ($F = 15.3, 8.9, 1.1$, and 9.6) suggesting that topographic beta effects are minor and certainly are less strong than the delta or alpha distributions.

### Fast activity

Activity over 30 Hz was minimal (mean 0.15 over all leads and stages). It was slightly higher in awake subjects (0.26) than in all sleep stages (0.16 to 0.12) and showed no significant topographic distribution ($F = 1.06$, lead by stage interaction).

### Average reference

Topographic analysis of data computed from average reference EEG revealed similar patterns of distribution; ear and average reference delta patterns were

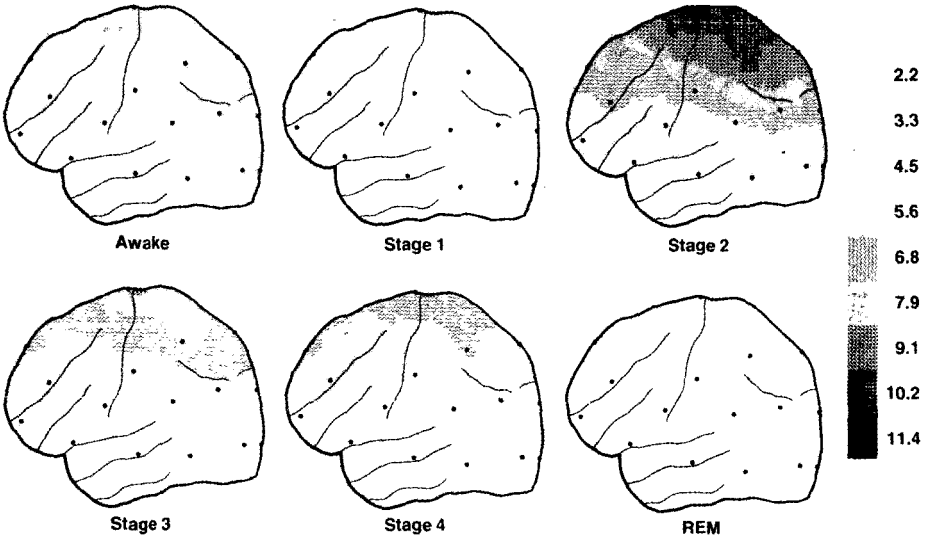


FIG. 3. Beta 1 activity maps for mean of four subjects. Waking beta is maximal in parietal areas and shifts to a more anterior position in slow wave sleep. Note resemblance of REM and stage 1, as was also noted with delta (Fig. 2).

quite similar (Fig. 4). ANOVA showed a stage effect ($F = 14.03$, $df = 5,15$ $p < 0.05$) and a stage by area interaction ($F = 3.91$, $df = 5,15$, $p < 0.05$; $df = 1,3$, $p = ns$).

## DISCUSSION

These data confirm that sleep stages, as defined by traditional criteria, differ in the quantitative and topographic distribution of EEG frequency bands. That quantitative EEG techniques can separate sleep stages is well known, and forms the basis for attempts at automatic sleep staging in both animals (8) and man (2,9,10). However, this work has been limited to one or two leads. In contrast, our data provide an opportunity to see localized effects. These include the "nightcap" movement of delta activity and the anterior movement of alpha in the sequential non-REM sleep stages. In general, our recordings support the choice of $C_3$ by Rechtschaffen and Kales (1) for sleep staging. However, for automated analysis $C_z$ might prove superior and occipital recordings less desirable as a single choice (see Figs.1 and 2).

Many areas remain to be explored before the sleep stages are completely characterized topographically. The finding of high beta during stage 2 in these data

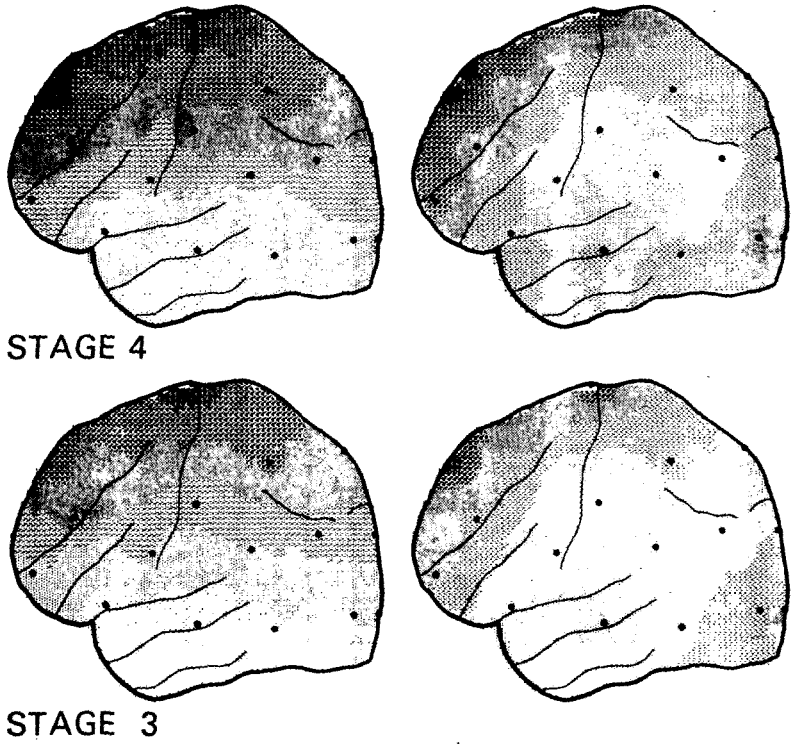


**FIG. 4.** Mean topographic maps for delta activity in the four subjects from the ear (left) reference and calculated average (right) reference. Maps are expressed on same scale. Average reference shows somewhat less power, but very similar topographic pattern.

may be a product of our selection of epochs containing spindle activity. Epochs with only K complexes (or neither transient event) could alternatively have been selected to determine their relative topographic contribution to stage 2. The absence of any effects in the fast band (over 30 Hz) suggests that we have been successful in removing muscle and movement artifacts as contributors to stage differences.

It should also be noted that these recordings were made during daytime naps and that there is some possibility that nocturnal sleep may differ. Similarly, sleep at different times of the night, or REM cycle positions, may differ in future studies.

As is well known, individual subjects differ in the amount of waking alpha activity. The possibility that low alpha/high delta subjects show less pronounced topographic distributions during sleep is suggested by EEG from one of our subjects, but needs further study. Individual differences are also emphasized in the observation that F ratios for lead by stage interactions, calculated with individuals across nine epochs, tended to be higher than F ratios calculated across individuals.

The only previous multilead data available to us are the pioneering 8 lead data of Findji et al. (6) in which delta amplitude from each of four electrodes was examined. For the left hemisphere, biauricular reference, a ranking of delta activity $F_3$, $P_3$, $C_3$, $O_1$ was found, similar to our Fig. 2. However, for the average reference, the ranking $O_1$, $F_3$, $P_3$, $C_3$ was observed. While we agree with the low $C_3$ ranking, our average reference map strongly resembles our ear reference map. In addition, it seems to suggest a more complex underlying distribution with $O_1$ values about 75% of frontal values. This ranking, $F_3$, $O_1$, $P_3$, $C_3$, is not all that dissimilar. With the 8 lead data of Findji et al. (6) a number of possible field distributions are possible and even our use of 16 leads may be insufficient to resolve the exact distribution. A distribution with a frontal and vertex peak could also be consistent with the Findji rankings. Other differences between our studies include the Findji et al. (6) report of rank order rather than microvolt data, younger subjects including infants, and a slightly different delta frequency band (0.5 to 4.5 cps).

It has already been shown that a number of disorders including narcolepsy, affective illness, and schizophrenia have abnormal proportions of sleep stages as determined by single lead studies. It is possible that these abnormalities may yet be further clarified by topographic distinctions.

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