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Normal Sleep

Topographical Distribution of Spindles and K-complexes in Normal Subjects

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Summary: To assess the topographical distribution of sleep spindles and K-complexes, four 15-minute samples of stage 2 sleep in a group of eight healthy young adults were analyzed. Results show that a majority of spindles generated are detected over central regions, and that K-complexes are markedly predominant over prefrontal and frontal regions. These findings are consistent with the single-spindle generator hypothesis and raise questions concerning the Rechtschaffen and Kales rules for scoring K-complexes. **Key Words:** Sleep spindles—K-complex—Topographical distribution—Humans—Normal subjects.

Spindles and K-complexes are well known to sleep investigators as the hallmark of stage 2 non-rapid eye movement sleep. For many years, investigation of these events was confined to central electroencephalogram (EEG) channels, in agreement with the standard method for scoring sleep stages (1), and was based on the assumption that the central placement of electrodes is the best location for detection of spindles and K-complexes. Recently, the topographical distribution of spindles and K-complexes on the scalp has become the subject of increasing research interest. To our knowledge, however, there exists no detailed analysis of the topographical distribution of spindle density. Further, the single study found in our literature review of the distribution of K-complexes reports their amplitude distribution rather than the topographical distribution of K-complex density per se (2). The goal of the present study, therefore, was to determine the percentage of spindles and K-complexes detected by each individual EEG channel in a topographic 10-20 montage to establish which one of these channels is most likely to detect a maximum of spindles and a maximum of K-complexes.

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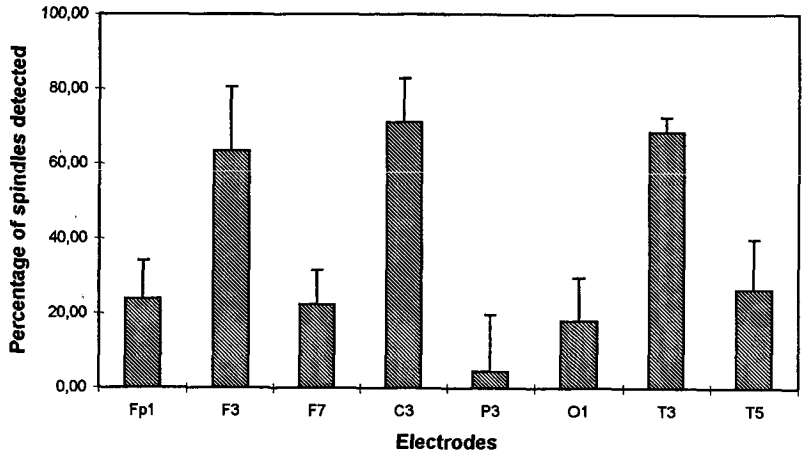
METHODS

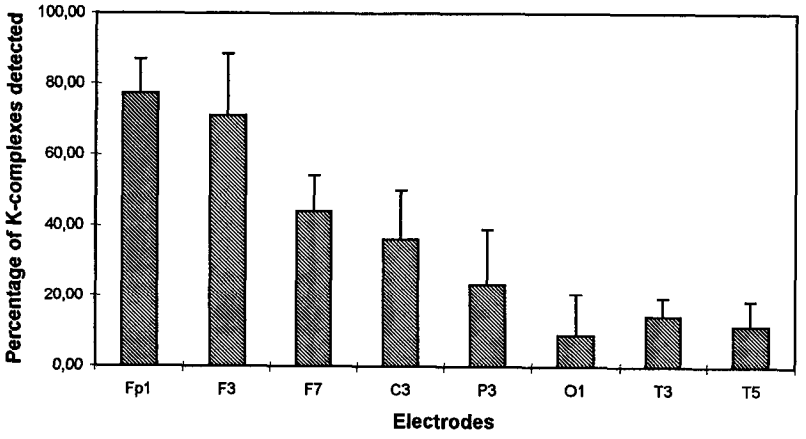
Eight healthy right-handed subjects (two males and six females) aged 14 to 28 years (mean = 22.2 years, SD = 6.3) participated in the investigation. Criteria for "healthy" subjects included the following: a negative history of sleep, neurological, or psychiatric problems; no use of medications; and no recent drug or alcohol dependence.

Subjects slept for three consecutive nights in the sleep laboratory. Sixteen EEG channels (Fp1, Fp2, F3, F4, F7, F8, C3, C4, P3, P4, O1, O2, T3, T4, T5, T6) were applied using the international 10-20 system (3). In addition, sleep was monitored with two electrooculogram (EOG) channels and one submental electromyogram (EMG). Channels were recorded continuously on an on-line computer system with a linked ear reference (A1 + A2). The first two nights allowed for adaptation to the laboratory. The third night was for the study of sleep architecture and phasic events.

EEG recordings were scored for sleep stages by an experienced polysomnographer. Four 15-minute episodes were sampled from stage 2 sleep for counting spindles and K-complexes. These artifact-free episodes were free of any fluctuations in sleep stage and were distributed homogeneously over the first 7 hours of recording in all subjects. The criteria for spindle activity was that such activity be visually homogeneous in



(A)



(B)

FIG. 1. Percentage of phasic events detected nonexclusively by each electrode in normal subjects. (A) Spindles, (B) K-complexes.

the sigma band (12–15 Hz) and that it last at least 0.5 seconds (1). K-complexes were defined as large amplitude biphasic or triphasic slow waves of at least 75 μV and with a duration of 0.5–3.0 seconds (4). A certified technician and an experienced polysomnographer, both of whom had extensive experience in scoring stage 2 phasic events, first identified all spindles and K-complexes that occurred within the four periods by visually scoring each of the left (dominant) hemisphere channels separately to prevent any influence on scoring from the simultaneous occurrence of phasic events over other channels. Interjudge reliability was at least 90% for spindles and within the 80–85% range for K-complexes. All channels were then displayed together to count in turn the total number of spindles and the total number of K-complexes that occurred simultaneously and in any combination of channels. This approach made possible determination of the total number of generated events, that is, the number of times the generator(s) produced potentials with the morphological characteristics of spindles or K-complexes regardless of the topographical location of their occurrence.

RESULTS

The total number of spindles was variable among subjects, with a mean of 316.1 and a standard deviation of 119.9 (mean density per minute = 5.2 ± 2.0). As illustrated in Fig. 1A, the topographical distribution of the mean number of spindles detected nonexclusively by each channel was found to be heterogeneous. C3 detected most spindles (71.0%), followed by T3 (68.4%) and F3 (63.3%). The total number of K-complexes was also found to be variable among subjects, with a mean of 138.0 and a standard deviation of 67.8

(mean density per minute = 2.3 ± 1.1). However, the topographical distribution of the mean number of K-complexes detected by each channel revealed a striking majority of K-complexes recorded by prefrontal (Fp1 = 77.3%) and frontal (F3 = 71.0%) electrodes. Further, the number of K-complexes detected was found to decrease in a homogeneous fashion from anterior to posterior channels (see Fig. 1B).

DISCUSSION

Spindles

Mean spindle density (5.2 per minute) was found to be consistent with previous reports (5.2 per minute) (5). Further, a majority of spindles was detected over central regions, a finding consistent with both anatomical work suggesting that there exists only one spindle generator and pacemaker located in the reticular nucleus of the thalamus (6), and phase-delay investigation of spindle frequency bands suggesting a pattern of spindle activity propagation from central areas to all other cortical regions (7). In addition, the finding that most spindles are recorded over central areas strongly indicates that scoring of overall spindle density on the basis of central electrode scanning should be the method of choice.

K-complexes

Results of the present study provide baseline data for the mean density and topographical distribution of K-complexes in healthy young adults. Difficulties in interjudge agreement owing to the lack of precise definitions of K-complex morphology are well described (8). This difficulty, combined with the fact that the counting of K-complexes on all channels is a time-consuming task, has likely dissuaded researchers from studying K-complexes topographically in a comparative fashion. To our knowledge, there exists no other account in the literature of the striking predominance of K-complexes recorded over prefontal and frontal regions in normal subjects. However, findings are consistent with previous descriptions of larger amplitudes of K-complexes recorded over frontal derivations (2,9). In addition, a recent description in the literature on bipoles points toward Fz when examining K-complexes with magnetoencephalography technology (10). Findings of the present study, therefore, provide additional evidence for involvement of frontal regions in the expression of K-complexes.

Further, the predominance of K-complexes recorded over prefrontal and frontal regions throws into question application of the Rechtschaffen and Kales rules for scoring K-complexes with central channels (1). Traditionally, the scoring of K-complexes has relied on information from only the central EEG leads. However, if more than twice the total number of K-complexes are detected by Fp1 (77.3%) than by C3 (36.1%), one should carefully scrutinize clinical and research contentions that are based on only C3/C4 scoring. Because the onset of stage 2 sleep is based on the occurrence of spindles or K-complexes, stage 2 onset latency may be delayed and stage 2 percentage may be underestimated if only central channels are scored. This may have consequences for the quantification of sleep stages over the life span (11) and in conditions such as dementia of the Alzheimer's type, in which the number of K-complexes is diminished (12).

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