EEG Characteristics Prior to and Following the Evoked K-Complex

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Abstract This study was designed to determine if the K-Complex reflects an arousal from sleep or a sleep protection mechanism. Ten participants were presented auditory stimuli every 20 s while asleep. Trials were sorted according to the presence or absence of a K-Complex. A fast Fourier Transformation of the data was computed on EEG segments prior to and following stimulus onset. The log power of activity in delta, theta, alpha, sigma, and beta bandwidths was computed. When a K-Complex was elicited, there were no differences in EEG activity prior to and following the stimulus. However, during slow wave sleep, when a K-Complex was not elicited, there was a significant overall increase in theta, alpha, sigma, and beta activity following stimulus. These results tend to support the notion that the K-Complex appears to prevent arousal.

During sleep, information processing from the external environment is largely inhibited by a thalamo-cortical loop gating system (Steriade, McCormick, & Sejnowski, 1993). All but the most relevant of information is gated from entry to consciousness, presumably to prevent sleep from being disturbed. It is thus somewhat of a paradox that the largest response to an external stimulus that can be recorded from the human scalp occurs during sleep. Because of its very large amplitude, it was first noticed in some of the earliest electrical recordings of the human sleep patterns using the newly discovered electroencephalogram (EEG) (Davis, Davis, Loomis, Harvey, & Hobart, 1939; Loomis, Harvey, & Hobart, 1939). This large amplitude response was labelled the K-Complex, for reasons that are still largely unknown. There is some belief (with little support) that it derived from the fact that a simple knock on an adjacent wall might elicit the K-Complex in the sleeper. Strangely, the K-Complex might be elicited by a stimulus one time and when the same, identical stimulus is later again presented, the K-Complex no longer can be elicited. At times, the K-Complex occurs spontaneously in the ongoing EEG in the absence of any apparent external stimulus. Of course, this does not necessarily mean that there was no stimulus. It is possible that the K-Complex might be elicited by internal consciousness or perhaps by something like an unobserved muscle twitch. Colrain and his colleagues have indicated that the K-Complex can be elicited by interruptions of breathing (Colrain, Bell, & Gora, 1998).

Most human evoked potentials recorded within conscious, waking, and alert states measure from 1 to 15 μV. The K-Complex often exceeds 100 μV. The K-Complex consists of a very large amplitude negative deflection peaking between 500 and 600 ms (thus the label, N550) followed by a positive wave peaking between 800 and 1,200 ms (P900) (Halász, Pàl, & Rajna, 1985; Ujszászi & Halász, 1988). The K-Complex appears as a phasic event: It is a transient EEG activity that occurs perhaps once every 2-3 minutes, although its frequency of occurrence can vary widely within and between sleepers. Sleep is conveniently divided into two markedly different states, REM (rapid eye movement) and non-REM. There are many differences between REM and non-REM sleep. Among the markers of non-REM sleep is the appearance of the K-Complex. In REM sleep, the K-Complex cannot be elicited by an external stimulus nor does it occur spontaneously. Non-REM sleep is further subdivided into Stage 2, 3, and 4 of sleep, Stage 4 being associated with deeper sleep than Stage 2.

We now know a good deal about the types of stimuli and paradigms – the antecedent conditions – that will elicit the K-Complex. The K-Complex is most often elicited by a psychologically relevant stimulus, such as the participant's own name; or a biologically relevant stimulus, such as a relatively loud and abrupt tone that is presented infrequently. Altering the participant's ability to breathe is obviously biologically relevant. This "stimulus" will elicit a K-Complex, even if the change in breathing occurs as often as every 5 s. While the antecedent conditions that elicit a K-Complex are fairly well understood, we know very little about the consequences of the K-Complex. What is its functional significance? What changes are we likely to see in behaviour or physiology once the K-

Complex is elicited? Will the participant awaken or at least will sleep be altered? Will the participant become conscious of the stimulus? Are these changes different from when the K-Complex is not elicited? The present study attempts to answer this question by examining the characteristics of the ongoing EEG prior to and following the presentation of a stimulus. Some of these stimuli will elicit a K-Complex, some will not. Will the poststimulus EEG pattern be different when the K-Complex is elicited compared to when it is not?

Two prominent and opposing theories have attempted to explain the functional role of the K-Complex. Almost all theorists agree that the K-Complex serves in the detection of either biologically or psychologically relevant stimuli. Where the theorists disagree is on what happens subsequentially. What are the consequences of the detection? One theory maintains that the K-Complex is an arousal mechanism that disturbs/interrupts sleep and perhaps allows the sleeper to become conscious of the external environment (e.g., Roth, Shaw, & Green, 1956). The opposing theory maintains that the K-Complex is a mechanism that, by inhibiting conscious awareness, protects the sleeper from awakening (Hess, 1965; Walter, 1963). Thus, the K-Complex serves to detect external stimuli but determines that the stimulus is not so relevant that sleep should be disturbed.

Evidence in support of both positions has come from an analysis of the frequency and amplitude characteristics of the EEG prior to and following the K-Complex. The EEG has traditionally been divided into a series of higher- and lower-frequency bands. These bands include beta (15 Hz and above), sigma (12-15 Hz), alpha (8-12 Hz), theta (4-7 Hz) and delta (0.5-2 Hz). During the waking and sleeping states, all frequency bands will be recorded. However, the relative balance of higher and lower frequencies will vary dramatically during the waking and sleeping states. Higher frequencies (associated in general with lower amplitude EEG) are a sign of arousal, wakefulness, and consciousness. Thus, a participant who is awake, alert, and highly attentive might display an EEG that is largely composed of beta frequencies. A participant who is also awake but very relaxed might display a slower alpha frequency. Lower frequencies (associated with higher amplitude EEG) are a sign of a lack of arousal, sleepiness, and unconsciousness. During the transition from a waking state to a drowsy-sleep state, the participant might lose the dominant beta/alpha frequencies and display theta activity (see Colrain, di Parsia, & Gora, this issue). As the participant becomes less conscious of the external environment and enters deeper sleep (or coma), large amplitude delta waves may be observed. Stage 4 contains more than 50% delta activity while Stage 2 contains less than 25%. There are exceptions to the association of higher frequencies with higher arousal. Most notably for an understanding of sleep, a 12-14 Hz rhythmic waveform, known as a spindle, occurs during non-REM sleep. Even though the sleep spindle does have a higher frequency content, it is not a sign of arousal. Rather, the spindle is associated with an inhibition of sensory processing (Cote, Epps, & Campbell, 2000; Steriade et al., 1993).

It might thus be expected that if the K-Complex is associated with a disturbance of and/or arousal from sleep, the EEG would manifest an increase in the amount of higher-frequency activity and/or a decrease in the amount of lower-frequency activity. On the other hand, if the K-Complex is associated with the prevention of arousals/disturbances, it might be expected that the EEG would manifest an increase in the amount of lower-frequency activity and/or a decrease in the amount of higher-frequency activity. An analysis of the frequency content of the EEG prior to and following a K-Complex can be employed to test these hypotheses. Prior to the advent of powerful and low-cost computers, this was a difficult task. Older studies typically employed visual analysis of the EEG. Roth et al. (1956) claimed that there was a tendency for the EEG to move toward higher-frequency activity following the occurrence of a K-Complex. The function of the K-Complex was thus hypothesized to be a correlate of a "crude perceptual process which tended to initiate arousal." They did not however carry out a statistical analysis of these observations. Moreover, they did not indicate the duration of the changes that they observed. Ehrhart, Ehrhart, Muzet, Shieber, and Naitoh (1981) suggested that spontaneous K-Complexes appear to precede phases of transient activation. These phases are transient signs of arousal in which (a) the EEG increases in frequency and decreases in amplitude, (b) alpha and other waking EEG patterns appear, and (c) body movements appear along with an increase in heart rate (Schieber, Muzet, & Ferrière, 1971). Other studies have shown that when the phases of transient activation are occurring, K-Complexes hold an antagonistic relationship with inhibitory spindles (Antony-Baas, 1975; Naitoh, Antony-Baas, Muzet, & Ehrhart, 1982). An increase in the number of K-Complexes leads to a decrease in spindles which, in turn, leads to a phase of transient activation (Antony-Baas, 1975; Naitoh et al., 1982). From these studies, it would appear that whereas spindles serve as inhibitory mechanisms, K-Complexes allow arousal.

A problem with early studies is that the human eye can detect only the largest and most obvious changes in the EEG. In more recent studies, researchers have relied on computerized analyses involving Fast Fourier Transformations and spectral analyses of the EEG. Spectral analysis provides a measure of power in the different frequency bands in the EEG. Power can be conceived as a measurement of area under the curve. An increase in power within a particular frequency band is generally interpreted to

reflect an increase in the amount of that frequency. Thus, an increase in power in higher-frequency bands following the occurrence of a K-Complex would be reflective of lighter sleep and increased arousal. An increase in the power in lower frequencies would be reflective of deeper sleep and decreased arousal.

Pál, Simon, and Halász (1985) noted that the occurrence of a K-Complex could be predicted from the power density function of a 1.8-s EEG segment preceding the stimulus. They suggested that evoked K-Complexes are linked to "finely graded microstates" of arousal (short and fast power elevation in higher frequencies) that can be measured in Stage 2 and possibly in Stages 3-4. Halász (1991) observed slight arousal shifts (i.e., microarousals) for 10-15 s *following* presentation of a stimulus. A consequence of the K-Complex was thus a poststimulus pattern characterized by a short initial power elevation (e.g., increases in the EEG activity within the alpha, and beta frequency bands) followed by a reduction of the delta, theta, alpha, and beta frequency bands along with a gradual reduction of the 13-14 Hz spindle band. Halász and Ujszászi (1991) used a more precise second-by-second Fast Fourier Transformations analyses of the poststimulus period. Their results showed both a power elevation for the delta, theta, alpha, and beta frequency bands and a power reduction for the spindle activity. The return to baseline values extended beyond the 10-s analysis period. Power in the delta, theta, alpha, and beta bands showed large increases in the first 2 s, slowly decreasing back toward baseline values, while spindle power gradually decreased following the stimulus. A problem with the Halász and Ujszászi (1991) studies is that they did not sort trials on the basis of whether the stimulus actually elicited a K-Complex or not. The authors failed to examine trials on which a K-Complex could not be elicited. Thus, the changes in the EEG could be due to the stimulus per se and not the K-Complex.

In the present study, the functional role of the K-Complex will be evaluated by analyses of the frequency content of the EEG prior to and following presentation of a stimulus that elicits a K-Complex. These will be compared to trials in which the stimulus does not elicit a K-Complex. The examination of trials in which K-Complexes are not elicited will be made in order to determine the effects of the stimulus itself on the EEG power spectra. The EEG will be divided into 4-s time intervals prior to and following the onset of the stimulus.

Method

PARTICIPANTS

Ten adults (3 men, 7 women) between the ages of 18 and 34 years ($\overline{X}$ = 24) were tested in a single all-night session. They were advised they should refrain from alcohol or drug use within 24 hours of the experimentation. Prior to being enrolled in the study, all participants were asked to read a consent form that explained the experimental procedures. Each participant received an honorarium for his or her participation in the study.

EEG RECORDING

The EEG was recorded with Grass gold cup electrodes placed at midline frontal, central, and parietal sites (Fz, Cz, and Pz, respectively). The reference was the mastoid contralateral to the ear of stimulus presentation. Eye movement and blink artifact were monitored with electrodes fixed at the supra-orbital ridge of one eye and the infra-orbital ridge of the other. Interelectrode impedance was maintained below 5 kOhms.

PROCEDURE

Each participant was individually fitted with a hearing-aid/ear-mould device through which the auditory stimuli were presented. The hearing-aid device assured a constancy of stimulus input in spite of the changes in the participant's head position during the night (Campbell & Bartoli, 1986).

K-Complexes were elicited by auditory stimuli. The stimuli were 80 dB SPL 2000 Hz tone pips having a total duration of 52 ms with a rise-and-fall time of 2 ms. Stimuli with these characteristics are most likely to elicit a K-Complex (Bastien & Campbell, 1992). Stimuli were presented in blocks. Within a block, 15 stimuli were presented at an interstimulus interval (ISI) of 20 s. Time between blocks varied from 2 to 5 minutes. Stimuli were delivered during definite (uninterrupted and artifact-free) stage 2, 3, and 4 of sleep.

One experienced rater later classified the stages of sleep according to the standard Rechtschaffen and Kales (1968) method. A 30-s period of EEG activity (or "epoch") was used for staging purposes. Stage 2 was defined by a relatively small percentage of delta activity (less than 25%) and the presence of spindles and K-Complexes. During Stage 3, the delta percentage increased from 25 to 50% of the 30-s epoch. During Stage 4, the delta percentage increased to more than 50%. In the rare cases of stage classification ambiguity (less than 5% of conditions), records were later scored by a second, experienced rater. If the raters disagreed, the condition was rejected from further analysis. Stage 2 was subdivided into early (2E) and late (2L) halves of the night to examine possible time-of-night effects. Stages 3 and 4 were combined to form slow wave sleep.

Time spent in the different stages of sleep varied between about 15 to 180 minutes for Stage 2E and 2L and 25 to 100 minutes in Stages 3 and 4. Because the effect of stimulus presentation is assessed in definite stages of sleep only, stimulus presentation was halted and the data removed from further analysis when the EEG pattern showed signs of artifacts (e.g., body movement) or the onset of

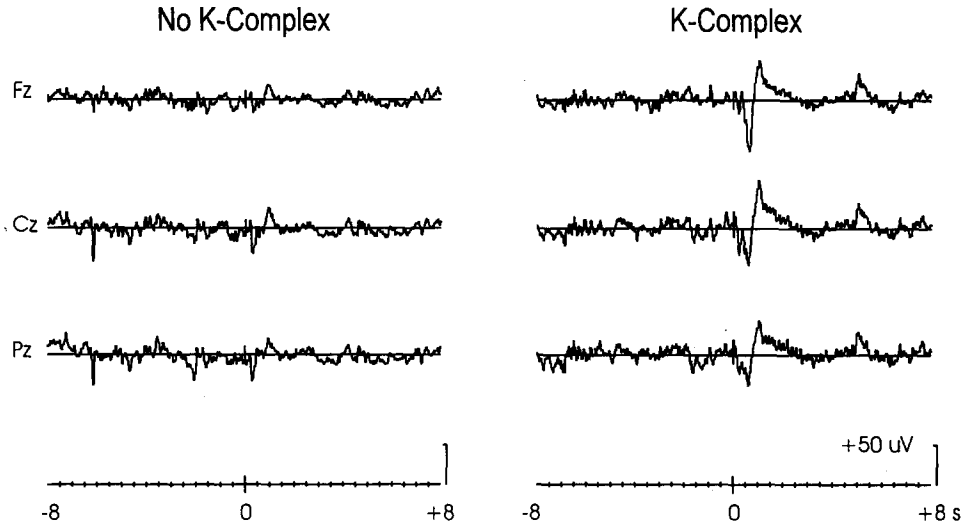

Figure 1. Single trial in which a K-Complex was not elicited (left portion) and when it was elicited (right portion). Negativity at the scalp relative to the reference is shown by a downward deflection. A 16-s sweep occurring during Stage 2 of sleep is illustrated. At time point 0, an auditory stimulus is presented. In the right portion of the figure, approximately 500-600 ms following the stimulus, a large amplitude negative peak (N550) followed by a positive wave is elicited. This is the K-Complex. It is maximal over the frontal electrode site. Prior to the N550, another negative wave peaking between 300 and 350 ms is apparent. This N350 is maximal over the central site. In the left portion of the figure, the large N550 is not visible. A smaller negative wave peaking at about 350 ms remains visible following stimulus onset. Spectral analyses were carried out in the 8-s period prior to the onset of the stimulus and from 1.25 to 9.25 s following its presentation..

waking. Each block of 15 stimuli was repeated at least one more time in each stage of sleep to ensure replicability of the results. For most participants, the available time allowed at least three repetitions of each block. A total of between 90 and 255 stimulus presentations were available for analysis for each participant.

EEG ANALYSIS

To assess the impact of stimulus presentation on the ongoing EEG activity, the single trials were sorted into those in which a K-Complex was and was not elicited. Figure 1 illustrates trials in which a K-Complex was and was not elicited. A set of algorithms was employed for the definition of a K-Complex. Following stimulus presentation, the EEG sweep had to have a negative peak occurring between 450 and 650 ms followed by a positive peak occurring between 700 and 1,200 ms. Peak-to-peak amplitude of the negative-positive deflection of the K-Complex had to exceed 75 μV. Finally, the negative peak had to have a frontal-central maximum distribution. This negative peak has been shown to be maximum over fronto-central areas of the scalp (Colrain, Webster, & Hirst, 1999; Cote, Langley, de Lugt, & Campbell, 1999). The pattern of the K-Complex was recognized by an automatic computer scoring routine following these criteria (Bell, Campbell, Deacon-Elliott, & Noldy-Cullum, 1988). This method reduced the likelihood of inclusion of random background noise being misclassified as a K-Complex. These criteria were particularly required in slow wave sleep, when large amplitude delta waves (often measuring more than 100 μV) could be mistakenly identified as a K-Complex.

The continuous EEG signal was divided into 4-s segments relative to stimulus onset. Fast Fourier Transformations were computed for the following intervals: from 8.00 to 4.00 s, from 3.99 to 0.00 s prior to stimulus onset, and from 1.25 to 5.25 s and from 5.26 to 9.25 s following stimulus onset. A 1.25-s delay following stimulus onset was employed to avoid including portions of the K-Complex (having a duration of approximately 1 s) in the Fast Fourier Transformations analyses. This procedure is illustrated in Figures 1 and 2. The Fast Fourier Transformations were run at each electrode site. The resolution of the Fast Fourier Transformations was 0.25 Hz, the frequency range being from 0 to 20 Hz. Trials were sorted and analyzed separately for those in which a K-Complex was identified and those in which it was not identified.

The EEG was then divided into five frequency bands. Although a particular frequency band may dominate a particular stage of sleep, all frequencies are, in fact, present in all stages of sleep. The following frequency bands were defined: delta (0.75-3.75 Hz), theta (4.00-7.75 Hz), alpha (8.00-11.75 Hz), sigma (12.00-14.00 Hz), and beta (14.25-20.00 Hz). The sigma band corresponded (but was not necessarily identical) to spindle activity. An increase in activity in higher-frequency bands (alpha, beta) would be interpreted as reflecting a movement toward arousal and wakefulness. An increase in activity in the lower-frequency bands (theta, delta) would be interpreted as a movement

toward sleep.

Slow frequencies such as delta activity tend to have large amplitude EEG. Thus, slower frequencies contain considerably more power than higher frequencies. A logarithmic transform of the power was considered necessary to compensate for the disproportionate skewing effects of delta activity. Ratios of the low- to- high-frequency bands were then computed. The first ratio ("SW1") was the quotient of the sum of delta and theta divided by the sum of alpha and beta (SW1 = (delta+theta)/(alpha+beta)). The second ratio ("SW2") was the quotient of theta divided by alpha (SW2 = theta/alpha). The two ratios provide a convenient and often-used summary of the proportion of lower- to- higher-frequency activity. A higher ratio is reflective of less arousal since there would be more power in the lower than the higher frequencies.

DATA ANALYSIS

Fast Fourier Transformations and spectral analyses for each of the five frequency bands were computed prior to and following each of the 15 stimulus presentations. Some of the stimuli will elicit a K-Complex while some will not. The data were then averaged across each of the trials in which a K-Complex could be identified and the trials in which a K-Complex could not be identified. It is possible that the stimulus per se causes an arousal independent of the K-Complex. Thus, in those trials in which the K-Complex is not elicited, it would be expected that there would be an increase in power in the higher-frequency bands or, alternatively, a decrease of power in the lower frequency bands following stimulus presentation. If the K-Complex serves an arousal role, the increase in power in higher frequencies and/or decrease in power in lower frequencies would have to be greater on trials in which a K-Complex occurred compared to when it did not occur. On the other hand, if the K-Complex serves to prevent arousals caused by the stimulus, then it would be expected that there would be a decrease in power for higher frequencies and/or an increase in power for lower frequencies.

A massive amount of data accumulates during long-duration sleep studies. The data were made more manageable by considering each stage of sleep separately. To determine the effects of stimulation on the log power of the different EEG bands, two-way repeated measures ANOVAs were performed. An initial two-way ANOVA with repeated measures on K-Complex (present or absent) and scalp site (Fz, Cz, Pz) was run for each of the time intervals (8.00 to 4.00, 3.99 to 0.00 s prior to stimulus onset and 1.25 to 5.25, 5.26 to 9.25 s following stimulus onset) for each frequency band for each stage of sleep. A second two-way ANOVA was run to compare time intervals (8.00 to 4.00 vs 3.99 to 0.00 s; 3.99 to 0.00 vs 1.25 to 5.25 s; 1.25 to 5.25 vs 5.26 to 9.25 s) and scalp site. Separate two-way ANOVAs were run when a K-Complex was elicited and another when it was not. For each ANOVA, the dependent variables were: the activity of the delta, theta, alpha, sigma, beta bands, the total absolute activity, SW1, and SW2.

For all comparisons, the significance level was set at $\alpha = .05$. An assumption for use of repeated measures ANOVA is that of sphericity. This assumption was tested using the Wilk's Lambda procedure. When this assumption was violated, Greenhouse-Geisser correction procedures were applied when appropriate.

Results

Across all conditions and stages of sleep, a K-Complex was elicited on approximately 50% of trials. The number of elicited K-Complexes did not differ across the stages of sleep ($F < 1$).

PRESENCE OR ABSENCE OF A K-COMPLEX

Some authors have reported that one of the consequences of the K-Complex was a poststimulus pattern characterized by a short initial power elevation followed by a reduction in power in other frequency bands. This would suggest that the K-Complex reflects an arousal mechanism. On the other hand, if the K-Complex reflects a sleep protection mechanism, it is expected that the EEG would show a reduction in power density in the different frequency bands instead of an elevation. The first analysis thus compared the amount of power in each of the five frequency bands on trials in which a K-Complex was elicited by the stimulus and those trials in which it was not elicited.

STAGE 2

Tables 1 and 2 present the results of the analysis of the influence of stage of sleep and the consequences of a K-Complex. In stage 2 sleep (early and late), the presence or absence of a K-Complex had no effect on the EEG. Thus, the EEG spectrum prior to the presentation of the stimulus did not differ ($F < 1$ in most cases) on trials in which a K-Complex was elicited compared to when it was not. Similarly, the EEG spectrum following the presentation of the stimulus did not differ ($F < 1$ in most cases) on trials in which a K-Complex was elicited compared to when it was not. Thus, there were no significant differences in any of the different bandwidths (activity of the delta, theta, alpha, sigma, beta bands) and the two ratios (SW1, SW2) on trials in which a K-Complex was elicited compared to when it was not.

SLOW WAVE SLEEP (STAGES 3 AND 4)

During slow wave sleep, the EEG spectrum prior to the presentation of the stimulus did not significantly differ ($F < 1$) on trials in which a K-Complex was elicited compared to when it was not. Thus, there were no signifi-

TABLE 1
Mean Log Power (μV^2 Units, *SD* is in Parentheses) for the 1.25- to 5.25-s Interval as a Function of K-Complex Presence

	Stage 2E		Slow Wave Sleep		Stage 2L	
EEG ACTIVITY	+	-	+	-	+	-
Delta	101.3	101.8	107.4	108.2	92.7	95.4
	(5.7)	(7.7)	(4.5)	(3.7)	(8.5)	(5.1)
Theta	76.6	78.0	78.0	78.6*	73.1	73.9
	(5.2)	(4.0)	(4.1)	(4.1)	(4.0)	(4.0)
Alpha	65.6	66.7	64.0	65.6*	64.4	63.9
	(1.2)	(3.2)	(2.4)	(2.9)	(2.1)	(4.1)
Sigma	55.4	56.0	50.2	53.4*	55.9	56.5
	(4.1)	(4.2)	(6.3)	(6.4)	(6.1)	(5.7)
Beta	49.1	51.0	47.0	48.9*	50.3	50.1
	(5.9)	(4.9)	(6.1)	(7.9)	(7.4)	(6.6)
Total	102.5	103.4	108.0	109.2	95.5	97.5
	(5.6)	(6.6)	(4.2)	(3.7)	(6.8)	(4.7)
SW1	1.5	1.5	1.7	1.6	1.5	1.5
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
SW2	1.2	1.2	1.2	1.2	1.1	1.2
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)

Note. Data are from frontal recordings; + : presence of a K-Complex; - : absence of a K-Complex.
* $p < .05$.

cant differences in any of the different bandwidths (activity of the delta, theta, alpha, sigma, beta bands) and the two ratios (SW1, SW2) prior to stimulus onset for K-Complex and no K-Complex trials. However, differences did emerge following stimulus presentation. For the poststimulus intervals, the log power of theta, alpha, sigma, and beta activity was significantly larger on trials in which a K-Complex was not elicited compared to when it was (theta: $F(1,8) = 6.50$, $MSE = 4.11$, $p < .05$; alpha: $F(1,8) = 3.76$, $MSE = 6.37$, $p < .05$; sigma: $F(1,8) = 4.60$, $MSE = 14.99$, $p < .05$; beta: $F(1,8) = 4.06$, $MSE = 4.11$, $p < .05$). There was no significant difference in delta activity following stimulus presentation when the stimulus elicited the K-Complex compared to when it did not.

PRESTIMULUS AND POSTSTIMULUS DIFFERENCES

Some authors have also noted that the occurrence of a K-Complex could be predicted from the power density function of an EEG segment preceding the stimulus. The time interval immediately prior to the stimulus (i.e., 3.99 to 0.00 s) and following it (i.e., 1.25 to 5.25 s) were thus compared. Tables 3 and 4 summarize these findings.

No significant pre- and postdifferences were found for any stage of sleep and any of the different bandwidths (activity of the delta, theta, alpha, sigma, beta bands) and the two ratios (SW1, SW2) on trials in which a K-Complex was elicited ($F < 1$ in all cases) during either Stage 2 or slow wave sleep. On the other hand, during slow wave sleep when no K-Complex was elicited, significantly greater log power was found for theta, alpha, sigma, and beta frequency bands in the post- compared to the prestimulus intervals (theta: $F(1,8) = 4.17$, $MSE = 1.00$, $p < .05$; alpha: $F(1,8) = 9.56$, $MSE = 0.38$, $p < .05$; sigma: $F(1,8) = 10.89$, $MSE = 0.75$, $p < .05$; beta: $F(1,8) = 5.21$, $MSE = 1.87$, $p < .05$). No significant delta frequency band differences were found. During Stage 2 (early and late), no pre- and poststimulus differences were found for any of the frequency bands ($F < 1$ in all cases) when the K-Complex could not be elicited for any frequency band.

INITIAL STIMULUS

During slow wave sleep, when a K-Complex could not be elicited, the log power of theta, alpha, sigma, and beta activity was significantly higher in the poststimulus intervals compared to the prestimulus intervals. On trials on which a K-Complex was elicited, the EEG power was not significantly different in the pre and poststimulus intervals during either Stage 2 or slow wave sleep.

Averaging procedures (data were collapsed across all trials) may have masked differences that occurred within single trials. It is possible, for example, that arousal/inhibition changes might occur only on the first trial but not on subsequent trials. A separate analysis of the initial trial was therefore carried out. This analysis has another advantage. There is a possible contamination in the pre, poststimulus analysis. The postinterval for one stimulus could be considered to be the preinterval for the next stimulus. Only on the first trial is the prestimulus period not also a poststimulus interval. Fast Fourier Transformations of the pre and poststimulus intervals were therefore compared for the initial trial. Again trials in which a K-Complex was and was not elicited were considered separately.

The initial stimulus presentation elicited a K-Complex

TABLE 2
Mean Log Power (μV^2 Units, *SD* is in Parentheses) for the 5.26- to 9.25-s Interval as a Function of K-Complex Presence

	Stage 2E		Slow Wave Sleep		Stage 2L	
EEG ACTIVITY	+	-	+	-	+	-
Delta	99.3	101.7	107.8	109.0	94.2	96.2
	(6.1)	(7.5)	(4.4)	(5.5)	(6.9)	(7.3)
Theta	76.2	77.7	77.9	79.2*	73.0	75.1
	(4.4)	(4.3)	(3.9)	(4.3)	(3.6)	(3.8)
Alpha	65.5	66.2	64.1	65.9*	63.7	65.0
	(3.4)	(3.7)	(2.7)	(2.8)	(4.4)	(3.5)
Sigma	53.8	54.6	51.9	54.3*	55.9	56.7
	(5.5)	(3.9)	(7.0)	(6.4)	(7.0)	(4.9)
Beta	49.3	49.7	48.6	50.3*	50.4	51.3
	(6.3)	(4.6)	(7.6)	(7.4)	(8.0)	(6.3)
Total	100.6	103.3	108.6	109.3	96.4	98.6
	(4.9)	(6.7)	(3.9)	(5.2)	(6.2)	(6.5)
SW1	1.5	1.5	1.7	1.6	1.5	1.5
	(0.1)	(0.1)	(0.2)	(0.1)	(0.1)	(0.1)
SW2	1.2	1.2	1.2	1.2*	1.1	1.2
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)

Note. Data are from frontal recordings; + : presence of a K-Complex; - : absence of a K-Complex
* $p < .05$.

on 40% of trials. K-Complexes were elicited more often in Stage 2E and 2L than in slow wave sleep, although this difference was not significant ($F < 1$ in all cases).

When a K-Complex was elicited, there were no significant differences ($F < 1$) in any bandwidth for any stimulus interval across all stages of sleep. No power changes in any of the frequency bands, or in any of the ratios, were observed on the pre and poststimulus interval ($F < 1$).

When K-Complexes were not elicited, as with the global analyses of all trials, during slow wave sleep, the log power of alpha and beta activity as well as SW2 significantly increased following stimulus onset compared to prior to its onset (alpha: $F(2,10) = 3.79$, $MSE = 26.97$, $p < .05$; beta: $F(2,10) = 4.81$, $MSE = 0.57$, $p < .05$; SW2 = $F(2,10) = 4.58$, $MSE = 0.004$, $p < .05$). No significant changes were observed in Stage 2 sleep ($F < 1$).

Discussion

The functional significance of the K-Complex has long been a subject of debate. One theory postulates that the function of the K-Complex is to arouse the participant from sleep and perhaps become aware of relevant external stimuli. If this is the case, it would be expected that following the occurrence of the K-Complex, the EEG would show movement toward higher frequencies. A second, alternative theory postulates that the function of the K-Complex is to protect the sleeper from the disturbing effects of irrelevant stimuli and prevent consciousness of these stimuli. If this is the case, it would be expected that following the occurrence of a K-Complex, the EEG would show movement toward lower frequencies.

The significant changes that were observed should be interpreted with some caution. During slow wave sleep when a K-Complex was not elicited, a significant change in the power of higher frequencies was observed. Nevertheless, the magnitude of the changes was very small. Differences were observed in other stages, conditions, and in the delta frequency band that were, in fact, larger but not significant.

Previous studies have shown changes in EEG power following stimulus presentation, whether a K-Complex was elicited or not. The effects of stimulus presentation itself must therefore also be considered. For this reason, in the present study, trials in which the K-Complex was elicited were analyzed separately from those in which a stimulus was presented, but no K-Complex was elicited. During slow wave sleep, on trials in which a K-Complex was not elicited, there was greater power in the higher-frequency bands. (While slow wave sleep is dominated by low-frequency delta activity, it should be noted that all frequency bands are in fact present. Of course, the total power in higher frequencies will be much reduced compared to that observed in the waking state.) The log power of theta, alpha, sigma, and beta activity was slightly but significantly higher in the poststimulus intervals. No significant changes were observed for delta activity. It is thus the higher-frequency, nondelta bands that show changes following stimulus onset. Most EEG researchers agree that a movement toward higher frequencies reflects cortical arousal. This does not imply that the participant was awakened by the stimulus. Rather, the stimulus itself appeared to elicit a small, brief arousal in the sleeping participant. The arousal was so brief that sleep was not interrupted. On the other hand, during slow wave sleep, in

TABLE 3
Mean Log Power (μV^2 Units, *SD* is in Parentheses) in Prestimulus and Poststimulus Intervals in Which a K-Complex was Identified

	Stage 2E		Slow Wave Sleep		Stage 2L	
EEG ACTIVITY	Pre	Post	Pre	Post	Pre	Post
Delta	101.3	101.3	106.9	107.4	93.6	92.7
	(7.4)	(5.7)	(3.7)	(4.5)	(9.4)	(8.5)
Theta	76.4	75.0	77.3	78.0	73.0	73.1
	(5.0)	(5.2)	(4.0)	(4.1)	(4.7)	(4.0)
Alpha	65.0	65.6	65.0	64.0	65.0	64.4
	(2.9)	(1.8)	(3.1)	(2.4)	(2.3)	(2.1)
Sigma	54.1	55.4	52.2	50.2	55.9	55.9
	(4.8)	(4.1)	(6.5)	(6.3)	(4.9)	(6.1)
Beta	49.6	49.1	47.2	47.0	50.2	50.3
	(6.7)	(5.9)	(6.0)	(6.1)	(7.5)	(7.4)
Total	102.7	102.5	107.7	108.0	96.2	95.5
	(6.4)	(5.6)	(3.6)	(4.2)	(7.6)	(6.8)
SW1	1.5	1.5	1.6	1.7	1.4	1.4
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
SW2	1.2	1.2	1.2	1.2	1.1	1.1
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)

Note. Data are from frontal recordings; Pre: 4.1 to 0.0 s; Post : 1.25 to 5.25 s.

trials in which the stimulus also elicited a K-Complex, no changes in the EEG were noted following stimulus presentation. Thus the brief arousal that was apparent when the stimulus did not elicit a K-Complex, was not apparent when the stimulus did elicit the K-Complex.

These findings support theories that claim that the K-Complex serves as a mechanism that protects against sleep disturbances. An auditory stimulus increases arousal from slow wave sleep (although it does not necessarily cause a change of stage). However, if it also elicits a K-Complex, this subsequent arousal does not occur. The small changes that were observed could not have been detected using conventional visual analysis of the EEG. This probably explains why many earlier studies relying on human scoring techniques may not have found similar significant effects.

The present results are not consistent with those of Halász and Ujszászi (1991). They also noted small but significant changes in the spectrum of the EEG. An increase in the power of higher frequencies was observed following the occurrence of the K-Complex. Several differences in the physical parameters of the stimuli and in the analyses of the data could account for these apparent contradictions. The rise-and-fall time of the stimuli was much longer in their study; stimuli were presented much more slowly; and their spectral analysis began at stimulus onset. The K-Complex consists of a series of slowly developing negative and positive peaks. A spectral analysis beginning at stimulus onset will also include the K-Complex and this will appear as lower-frequency, mainly delta activity. Finally, their FFTs were based on a 1-s analysis over a 10-15 s time interval, compared to the present 2-s analysis over 4-s intervals. What is consistent between the two studies is that whatever changes occur following stimulus presentation will be very small.

Sallinen, Kaartinen, and Lyytinen (1994) have reported the appearance of the mismatch negativity (difference wave computed by subtracting a standard from a target waveforms in the same condition) preceding the evoked K-Complex. The mismatch negativity is usually elicited by auditory stimuli deviating with respect to tonal frequency, intensity, duration, spatial location, interstimulus interval, and complex phonetic changes (Naatanen & Lyytinen, 1994; Sams, Aulanko, Aaltonen, & Naatanen, 1990). The mismatch negativity is thought to reflect an automatic detection of the deviant. This occurs in the preconscious although it might later trigger consciousness (see Sabri, de Lugt, & Campbell, this issue, for a discussion of the effects of sleep onset on the mismatch negativity). Although the mismatch negativity might trigger an arousal response, it would appear that the K-Complex inhibits this arousal. On the other hand, if the same identical stimuli do not elicit a K-Complex, a general arousal pattern of the EEG (perhaps reflecting higher cortical processing) will occur. The K-Complex may therefore serve to gate or attenuate processing of information that may be relevant but not so relevant as to cause the participant to move toward wakening, thus reaching full awareness and consciousness.

The increase in power following stimulation on trials on which a K-Complex was not elicited was observed only in Stages 3 and 4. This might be attributed to a first-night effect because participants spent only one single night in the laboratory. It is possible that adaptation to the stimulus could occur on subsequent nights. There is some evidence to suggest this is not the case. Pál et al. (1985) have

TABLE 4
Mean Log Power (μV^2 Units, *SD* is in Parentheses) in Prestimulus and Poststimulus Intervals in Which a K-Complex was Not Identified

	Stage 2E		Slow Wave Sleep		Stage 2L	
EEG ACTIVITY	Pre	Post	Pre	Post	Pre	Post
Delta	101.5	101.8	108.2	108.2	95.2	95.4
	(7.1)	(7.7)	(3.6)	(3.8)	(5.2)	(5.1)
Theta	77.9	78.0	78.3	78.6*	73.9	73.9
	(4.5)	(4.0)	(4.5)	(4.1)	(3.7)	(4.0)
Alpha	66.6	66.7	64.7	65.6*	64.0	63.9
	(2.7)	(3.2)	(3.5)	(2.9)	(4.1)	(4.1)
Sigma	55.8	56.0	52.4\	53.4*	56.9	56.5
	(4.4)	(4.2)	(6.8)	(6.4)	(6.8)	(5.7)
Beta	51.0	51.0	47.8	48.8*	51.1	50.1
	(5.7)	(4.9)	(8.0)	(7.9)	(7.4)	(6.6)
Total	103.2	103.4	108.9	109.2	97.3	97.5
	(6.6)	(6.6)	(3.8)	(3.7)	(4.7)	(4.7)
SW1	1.5	1.5	1.7	1.6*	1.5	1.5
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
SW2	1.2	1.2	1.2	1.2	1.1	1.2
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)

Note. Data are from frontal recordings; Pre: 4.1 to 0.0 s; Post : 1.25 to 5.25 s.
* $p < .05$.

demonstrated that presenting stimuli during a single night session had no effect on the mean duration or on the percentages of sleep stages compared to undisturbed baseline nights. The differences observed in slow wave sleep are not thus easily attributed to a first-night effect.

Analysis of the initial stimulus revealed that no change in the EEG could be observed in any frequency bands on trials in which a K-Complex was elicited. It followed the same pattern as the overall analysis on trials in which a K-Complex was not elicited – thus an elevation in higher frequency bands following stimulus presentation. The impact of the first stimulus on the EEG is less marked than what might have been expected. Prepost differences that were observed when the data were collapsed across all trials could not be due to an apparent confound – the interval following a stimulus could be considered to be also the interval prior to the subsequent stimulus. The post-stimulus EEG, thus, had enough time (8 s) to return to baseline values. Therefore, in the present study, the post-stimulus did not contaminate the subsequent prestimulus interval.

In conclusion, this study tends to support the notion that the K-Complex serves as a protector of sleep and does prevent or gate awareness of the external environment. Nevertheless, a change in the EEG toward lower frequencies was not apparent following the elicitation of a K-Complex. The EEG remained unchanged in poststimulus trials in which a K-Complex was elicited. Rather, the conclusion is based on the fact that the presence of a K-Complex appears to block the general movement toward increased power for higher-frequency bands which otherwise occurs in response to a stimulus. The changes in the EEG were however very small. Due caution is required in their interpretation.

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Sommaire

Le Complexe-K est une composante de l'électroencéphalogramme (EEG) qui apparaît pendant le sommeil lent (les stades 2, 3 et 4) en réponse à un stimulus externe ou avec une spontanéité apparente. Il a préalablement été démontré que le Complexe-K évoqué répondait à la loi du tout-ou-rien, ses différentes composantes ne variant pas en amplitude ni en latence lors de variations dans les paramètres physiques de stimuli auditifs (Bastien & Campbell, 1992). Malgré ces résultats nous informant sur les antécédents du Complexe-K, le débat sur le rôle fonctionnel de celui-ci demeure vif. Certains auteurs considèrent le Complexe-K comme un signe d'éveil pendant le sommeil. D'autres lui assignent la fonction de mécanisme protecteur pendant le sommeil. L'objectif de la présente étude est de vérifier ces théories opposées. L'analyse des spectres de l'EEG qui précèdent et suivent la présentation d'un stimulus auditif est effectuée sur des essais où un Complexe-K est évoqué et des essais où un complexe-K n'est pas évoqué par la stimulation auditive. Si le Complexe-K est un signe d'éveil pendant le sommeil, les spectres de l'EEG démontreront une augmentation dans l'activité des fréquences élevées de l'EEG (par exemple, alpha et bêta). Cependant, si le Complexe-K reflète un mécanisme de protection du sommeil, une activité moins intense des spectres de fréquences élevées de l'EEG et/ou une augmentation dans les spectres de fréquences lentes de l'EEG (par exemple, delta) seront présentes. L'EEG de dix participants est enregistré pendant une nuit. Les stimuli auditifs consistaient en des sons (52 ms, 80 dB SPL, 2000 Hz) présentés à toutes les 20 s pendant le sommeil à ondes lentes (stades

2, 3 et 4). L'EEG fut fragmenté en intervalles de 4 s relatifs à la présentation du stimulus. Des transformations de Fourrier (FFTs) sont analysées pour les intervalles suivants: de 8.00 à 4.00 et 3.99 à 0.00 s avant la présentation du stimulus et, de 1.25 à 5.25 et 5.26 à 9.25 s à la suite de la présentation du stimulus. La valeur logarithmique des spectres de l'activité delta, thêta, alpha, sigma et bêta est obtenue. Lors des essais où un Complexe-K est évoqué, aucun changement n'est détecté dans l'activité spectrale des bandes de fréquences avant ou après la présentation des stimuli. Cependant, lorsqu'un Complexe-K n'est pas évoqué, une augmentation générale significative apparaît dans l'activité spectrale des bandes de fréquences thêta, alpha, sigma et bêta à la suite de la présentation du stimulus dans les stades 3 et 4 du sommeil. Cette augmentation n'apparaît pas en stade 2. Lorsque l'analyse spectrale de l'EEG est effectuée sur le premier stimulus de chaque bloc de présentation de stimuli, les résultats sont similaires à tous les essais additionnés. Ces résultats semblent supporter la théorie selon laquelle, pendant le sommeil à ondes lentes (3 et 4), le Complexe-K agirait en tant que filtre des stimuli perturbateurs en prévenant l'éveil qui apparaîtrait sinon à la suite de la présentation du stimulus.