Sleep, 15(3):236–245

Fundamental Research

The Evoked K-Complex: All-or-None Phenomenon?

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Summary: The functional significance and topographical variation of the different components of the evoked K-complex were examined. In the first experiment, the intensity of the stimulus (80 and 60 dB SPL) and its rise-and-fall time (2 and 20 milliseconds) were manipulated during nonrapid eye movement sleep. In the second experiment the tonal frequency (500, 1,000 and 2,000 Hz) of the stimulus was manipulated. In the first experiment, nine stimuli were presented every 10 seconds, whereas in the second, 20 consecutive stimuli were presented. The evoked K-complex consisted of two different negative components peaking at approximately 350 and 550 milliseconds, respectively, and followed by a positive component peaking at approximately 900 milliseconds. K-complexes were easier to elicit for high-intensity fast rise-and-fall time stimuli than for low-intensity slow rise-and-fall time stimuli. The probability of occurrence was not affected by the tonal frequency of the stimulus. When a K-complex was evoked, the amplitude and latency of N350, N550 and P900 remained invariant regardless of its intensity, rise-and-fall or its tonal frequency. The N550–P900 portion of the K-complex therefore appears to be an all-or-none phenomenon. On trials in which a K-complex could not be elicited, N350 was still visible although much attenuated. In these trials, its amplitude was further reduced when stimulus intensity was lowered. N350 might need to reach a certain critical threshold before the much larger N550–P900 complex is elicited. **Key Words:** NREM sleep—K-complex—Stimulus parameters—All-or-none phenomenon.

Résumé: Le rôle fonctionnel et les différences topographiques des composantes du complexe-K évoqué étaient examinés. Deux intensités (80 et 60 dB SPL), deux temps de montée (2 et 20 millisecondes) et trois fréquences (500, 1,000 et 2,000) étaient manipulés dans les stades 2, 3 et 4 du sommeil. L'intervalle interstimulus était de 10 secondes. Les résultats démontraient que le complexe-K évoqué consistait en deux pics négatifs ayant respectivement une latence approximative de 350 et 550 millisecondes et suivis d'un pic positif apparaissant vers 900 millisecondes. Les complexes-K étaient plus facilement évoqués sous les stimuli d'intensité élevée et de temps de montée rapide que sous les stimuli d'intensité faible et de temps de montée lent. La fréquence des tons n'affectait pas la probabilité d'évoquer un complexe-K. Lorsqu'un complexe-K était évoqué, l'amplitude et la latence des pics N350, N550 et P900 demeuraient invariables sous les différentes conditions. Le complexe-K semblait donc obéir à la loi du "tout-ou-rien". Lors des essais où des complexes-K ne purent être identifiés, N350 était visible malgré une atténuation d'environ 50%. Lors de ces essais, son amplitude était encore plus atténuée lorsque l'intensité était diminuée. N350 devrait donc avoir à atteindre un certain seuil d'amplitude critique afin que le complexe N550–P900 soit subséquemment évoqué.

More than 50 years ago, Loomis et al. (1) noted that the presentation of an external auditory stimulus to the sleeping subject could elicit a very large amplitude waveform, the K-complex. In the same year, Davis et al. (2) observed that this waveform could also occur spontaneously without any apparent external stimulation. It is now known that K-complexes appear in stages 2, 3 and 4 of sleep (3,4). They do not occur spontaneously and cannot be evoked in rapid eye movement (REM) sleep or while the subject is awake.

The morphology of the K-complex consists of a well-delineated negative wave peaking at approximately 550 milliseconds (N550) followed by a positive wave peaking at approximately 900 milliseconds (P900). An earlier negative component peaking at about 350 milliseconds (N350) has also been considered to be part of the K-complex by some authors (5). The N350, N550 and P900 peaks have different scalp topographies. N550 and P900 are frontally and frontocentrally distributed, respectively, whereas N350 is more evenly distributed over midline areas of the scalp (5).

A number of studies have now examined the types of stimuli that can elicit a K-complex. The intensity of the evoking stimulus plays a critical role, the probability of eliciting a K-complex varying directly with the intensity of the stimulus (4,6,7). Campbell et al.

Accepted for publication January 1992.
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(7) have also noted that tone pips having fast rise-and-fall times are more likely to evoke a K-complex than those having slow rise-and-fall times. Tonal frequency and stimulus duration have no significant effect (7,8).

There has been considerable debate about possible variation of the amplitude of the K-complex as a function of stimulus parameters. In a classic article, Roth et al. (3) claimed that the K-complex was an all-or-none response. The usage of this term was probably adopted from the all-or-none law describing the action potential of a neuron. Roth et al. (3) claimed that the single trial K-complex was an all-or-none response, as manipulation of a wide range of stimulus parameters apparently did not alter its amplitude. On any trial, any stimulus might elicit a K-complex, and when it did, its amplitude did not vary.

Later research was more equivocal. For example, Johnson and Karpan (9) also observed that a stimulus will not elicit a K-complex on every trial. However, when it is elicited, its amplitude does not vary in spite of manipulation of the physical qualities of the stimulus. On the other hand, Oswald et al. (10) presented much more complex stimuli—spoken names. Contrary to what would be expected of an all-or-none phenomenon, they found that the amplitude of the K-complex increased in amplitude the more the spoken name resembled the subject's own name.

A number of methodological differences and limitations could account for the discrepancies among the various studies. There has been little consistency across studies with respect to the types of stimuli (clicks, tones, spoken words) used to elicit the K-complex or the method of transducing the auditory signal (loudspeakers, earphone inserts). Furthermore, sample sizes tend to be small, thus limiting the statistical power to find differences. A major problem is that the amplitude of the ongoing electroencephalogram (EEG) is very large in the sleeping subject, particularly during slow-wave sleep (SWS). This ongoing, random EEG will overlap and sum to the K-complex. A good portion of the variation in the amplitude of the K-complex will thus be due to the random, overlapping background EEG. It is thus highly unlikely that single-trial K-complexes can be reliably measured in the background EEG.

To overcome this problem, we have relied on signal averaging techniques to reduce the background noise. The evoking stimulus is presented repeatedly. The average of the random background activity should tend to cancel out. The constant response, the K-complex, will remain. Campbell et al. (7) used such an averaging technique to determine the effects of different stimulus parameters on the K-complex. As mentioned, they observed that the probability of evoking a K-complex increased as stimulus intensity increased, or as the rise-time of the tone pip became shorter. The actual amplitude of the averaged K-complex was also dependent on the physical qualities of the evoking stimulus, contrary to the all-or-none principle. Its amplitude decreased as the intensity of the evoking stimulus decreased. Further, the amplitude of the K-complex was dependent on the rise-and-fall time of the stimulus. Its amplitude was higher with fast rise-times and smaller with slow rise-times. There is, however, a confound in this study. Single trials were averaged regardless of whether they elicited a K-complex or not. The averaged K-complex will be higher in amplitude when a train of stimuli elicit many K-complexes than when few are elicited. It is thus quite possible that the amplitude of a single K-complex is consistent with the all-or-none principle even though the average is not. Clearly, the indiscriminate use of averaging techniques is inappropriate in these circumstances.

Finally, many studies measure the K-complex from the peak of the large N550 wave to the peak of the large P900 wave. Such peak-to-peak measurement assumes a common functional significance of both peaks. This need not be the case. Furthermore, N550 and P900 have different scalp distributions. Peak deflections having different scalp distributions must have different intracranial generators (11). A baseline-to-peak measurement technique permits separate measurement of each peak deflection and thus does not make the assumption that the two peaks are generated by identical cerebral processes.

The present study was designed to overcome many of these methodological limitations. The physical qualities of the evoking stimulus will be manipulated in different stages of sleep. Trials will be sorted according to those in which a K-complex can and cannot be identified in the background noise following stimulus presentation. Signal averaging techniques will be employed to reduce background EEG activity that overlaps the K-complex. To determine if the K-complex is morphologically invariant in the different stages of sleep, single trials in which a K-complex was evoked will be averaged and compared in the different conditions. Similarly, trials in which a K-complex could not be elicited will also be averaged. The effects of the manipulation of the physical qualities of the stimulus on the amplitude and latency of the N350, N550 and P900 deflections will then be determined.

METHODS

Subjects

Nineteen young adults (5 males, 14 females) between the ages of 20 and 35 ($\bar{x}$ = 24) participated in these studies. They were tested in a single all-night session. They were instructed to refrain from alcohol and drug

use for 24 hours prior to the experiment. All subjects were asked to read and sign a consent form that provided details of the experimental paradigm and procedures. Each subject received an honorarium for their participation in this study.

EEG recording

The EEG was recorded with Grass gold cup electrodes placed at midline frontal, central and parietal sites (Fz, Cz and Pz). The reference was the left mastoid. The electrooculogram was recorded with electrodes fixed at the supraorbital ridge of one eye and the infraorbital ridge of the other. This permitted the recording of horizontal and vertical eye movements on a single polygraphic channel. Interelectrode impedance was maintained below 5 kOhms.

A pilot study was conducted to determine appropriate filter settings for the recording of the K-complex. Four subjects were tested during stage 2 sleep. In these subjects, EEG was limited to a single Fz channel where the K-complex tends to be at its maximum amplitude. This recording was fed into eight different polygraphic channels. In five of these, the high filter was held constant at 15 Hz but the time constant was varied, being either 5.0, 2.0, 1.0, 0.3 or 0.1 seconds. In the other three channels, the time constant was held constant at 1 second, and the high filter settings were varied, being either 35, 70 or 500 Hz. Single K-complexes were evoked by an 80-dB SPL tone pip. Trials were sorted according to those in which a K-complex was either identified or not identified in the background noise. Those single trials in which a K-complex was elicited were then averaged for each subject to reduce background EEG noise. A minimum of 10 K-complexes was used for the calculation of the average.

As illustrated in Fig. 1, the filter settings had a marked effect on the K-complex. The amplitudes of N550, P900 and the subsequent slow wave (between 1,000 and 1,500 milliseconds) were attenuated by shorter time constants. Increasing the time constant beyond 1 second had minimal effect on the morphology of the K-complex. A small amount of high-frequency background noise was visible with filter settings above 35 Hz. A time constant of 1 second and a high-frequency filter of 35 Hz was therefore selected as a reasonable compromise for the recording of the K-complex.

Procedure

Each subject was individually fitted with a hearing-aid device through which the auditory stimuli were presented. The hearing-aid system assured constancy of stimulus input in spite of changes in the subject's head position during the night (12).

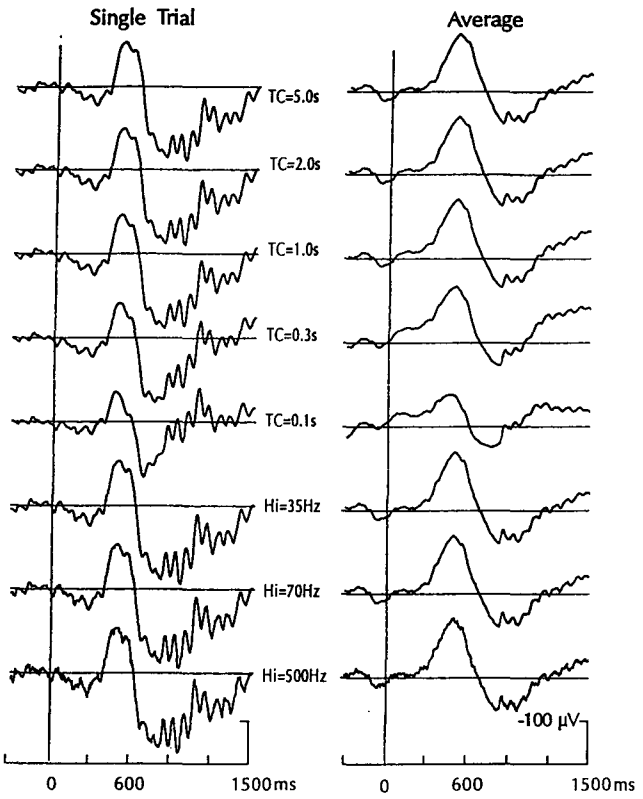

FIG. 1. The effect of high- and low-pass filters on single trial (left) and averaged (right) K-complexes from one subject (AB). The plotted waveforms are from frontal recordings. For this subject, the average on the right-side of the figure is the average of 14 single trials containing a K-complex. In five channels, the high filter was held constant at 15 Hz, but the time constant was varied, being either 5.0, 2.0, 1.0, 0.3 or 0.1 seconds. In the other three channels, the time constant was held constant at 1 second and the high filter settings were varied being either 35, 70 or 500 Hz. In both the single trial and averaged waveforms, a time constant below 1 second resulted in the attenuation of the large negative wave at approximately 500 milliseconds and of a late slow wave in the 1,000–1,500 millisecond interval. High frequency background noise can be observed in filter settings above 35 Hz.

Experiment 1

In Experiment 1, the intensity of the eliciting stimulus and its rise-and-fall time were manipulated. Seven subjects participated in this experiment. In the standard high-intensity fast rise-time condition, nine consecutive 80 dB SPL 2,000 Hz tone pips having a rise-and-fall time of 2 milliseconds were presented monaurally to the right ear of the subject. The interstimulus interval was 10 seconds. In the low-intensity condition, the intensity of the tone pips was lowered to 60 dB SPL. All other stimulus parameters were, however, held constant. In the slow rise-time condition, the rise-and-fall time was increased to 20 milliseconds, stimulus intensity being held at 80 dB. The duration was increased to 70 milliseconds to control for the amount of energy elicited by the short and long rise-and-fall time stimuli (13).

Experiment 2

In Experiment 2, the tonal frequency of the eliciting stimulus was manipulated. Twelve subjects participated in this study, none of whom had participated in the first experiment. Stimulus transduction and electrode placement were identical to Experiment 1. Twenty consecutive 80 dB SPL 52 millisecond tone pips having a rise-and-fall time of 2 milliseconds were presented monaurally to the right ear of the subject. The interstimulus interval was 10 seconds. The frequency of the stimulus was either 500, 1,000 or 2,000 Hz in different conditions.

In both experiments, stimulus presentation began 15 minutes after the beginning of sleep onset (defined as the appearance of stage 2 or SWS). Each condition was presented in the different stages of sleep and repeated at least one more time to ensure replicability of the results. For most subjects, time permitted three repetitions of each of the different conditions.

EEG analysis

The different stages of sleep were classified on-line by an experienced rater according to standard scoring criteria (14). Testing occurred during definite stage 2, 3 and 4 of sleep. Stage 2 was subdivided into early (2E) and late (2L) halves to examine possible time-of-night effects. Stages 3 and 4 were combined to form SWS. 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. Stimulus presentation was discontinued and rejected when the EEG sleep pattern showed signs of a stage change or upon subject movement.

EEG analysis began 300 milliseconds prior to stimulus onset and continued for 1,800 milliseconds (i.e. 1,500 milliseconds poststimulus). A total of 300 data points were digitized for each channel (i.e. the sampling rate was every 6 milliseconds). The average of the prestimulus activity served as baseline from which peak deflections were measured.

Single trials were stored on-line and subsequently plotted. They were sorted into those trials identified as containing and not containing a K-complex (in this case, a large amplitude N550–P900 wave). A set of algorithms was employed for the definition of a K-complex. The trial had to have a negative peak between 400 and 700 milliseconds (N550) followed by a positive peak between 700 and 1,200 milliseconds (P900). Their peak-to-peak amplitude had to exceed 75 μV. Furthermore, the negative peak had to have a frontocentral maximum distribution. This algorithm was implemented to reject possible inclusion of random background noise as a K-complex. This was especially necessary in SWS sleep during which isolated delta waves might be mistakenly identified as K-complexes. An automatic computer scoring routine was used for the purposes of pattern recognition following these criteria (15).

Single trials were sorted by condition, stage of sleep and whether they contained a K-complex or not. They were then averaged to reduce random background noise.

Data analysis

N350 was defined as the maximum negative peak in the 300–450 millisecond range, N550 as the maximum negative peak in the 450–700 millisecond range and P900 as the maximum positive peak in the 700–1,200 millisecond range. To determine the effects of stimulus intensity a four-way ANOVA was run having repeated measures on intensity (high, low), scalp site (Fz, Cz, Pz), stage of sleep (2 early, SWS, 2 late) and appearance of the K-complex (present, absent). Similar four-way repeated measures ANOVAs were run for the comparison of the effects of stimulus rise-and-fall time (fast, slow) and the effects of tonal frequency (high, medium and low). The analyses were carried out using the SAS PROC GLM procedure. Wilk's Lambda was employed to test for sphericity. Greenhouse–Geisser corrections were employed when appropriate. Significant differences in probability of occurrence of K-complexes across conditions were determined using multiple one-tailed t tests. One-tailed directional t tests were used, as a priori expectations could be established on the basis of previous literature. For all comparisons, the significance level was set at $p < 0.05$.

RESULTS

Experiment 1

Across all conditions and stages of sleep, a K-complex was elicited on approximately one-third of trials. The probability of occurrence in the standard condition was 0.50 compared to 0.20 for the low intensity. The probability of occurrence of a K-complex for the slow rise-and-fall time condition was 0.30. No significant differences were, however, observed among the different conditions. K-complexes were more likely to occur in stages 2 early and SWS (0.42 and 0.35 probability, respectively) than in stage 2 late (0.23), but the differences were again not significant.

As mentioned, trials were sorted into those in which a K-complex was identified and those in which it could not be identified. They were then averaged by electrode site, condition and stage of sleep. Figure 2 presents single trial data (randomly selected) for one subject

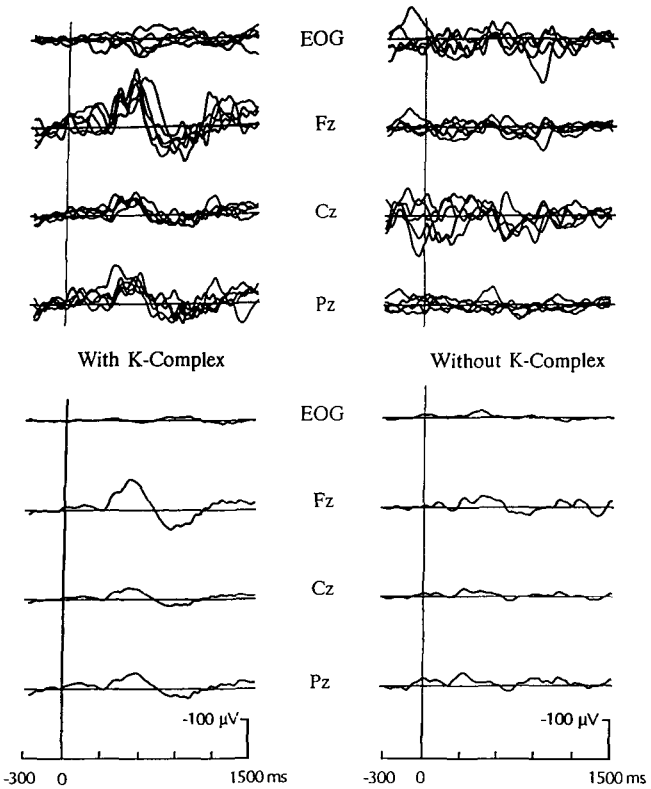

FIG. 2. Single trials obtained from one subject (DC) for the standard condition on trials in which a K-complex could be elicited (left) and could not (right). The recordings were obtained during slow-wave sleep where background noise is especially apparent. The average of these single trials is presented at the bottom of the figure. As can be seen, the average is a good reflection of the single trials.

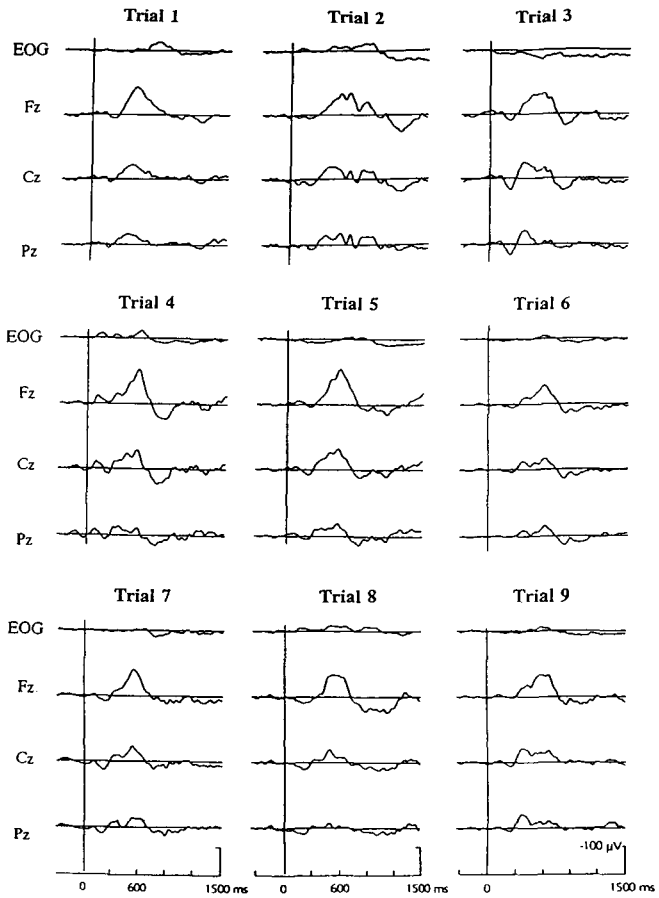

FIG. 3. Grand averages (average of all subjects' averages) on each of the nine trials for the standard condition during early stage 2 sleep. No sequential effects were observed for the probability of occurrence of a K-complex within the nine consecutive trials. Furthermore, neither the amplitude nor the latency of the various components of the K-complex varied significantly.

(DC) and the average of these trials for the standard condition. As may be observed, although there is some variability due to the overlapping large amount of background noise, the average is a good reflection of the single trials. Although the large negative–positive complex is visible on most single trials, an earlier negative wave peaking between 300 and 400 milliseconds could be discerned following averaging. As can also be observed, this earlier negative peak remains visible on the average of trials in which no K-complexes could be identified.

For the standard condition, no significant differences were found for the probability of occurrence of K-complexes among the nine consecutive trials within a block. From the first to the ninth trials, K-complexes were identified on 61, 48, 48, 33, 61, 48, 61, 38 and 52% of the trials, respectively. When only trials containing a K-complex were averaged, no significant differences were observed at Fz for either the amplitude or the latency of N550 and P900 among the nine trial positions. K-complexes for each of the nine trials for the standard condition are illustrated in Fig. 3.

Across both conditions and all stages of sleep, when a K-complex was elicited, it consisted of a biphasic negative complex having peaks at 365 and 565 milliseconds (N350 and N550, respectively) and followed by a later positive wave, peaking at 935 milliseconds (P900). N350 could only be discerned following averaging. It was embedded in the background EEG on single trials. For trials not containing the large N550–P900 complex, the N350 deflection still remained apparent. Different scalp topographies were observed for the different deflections. N350 tended to be uniformly distributed over midline areas of the scalp. It was maximum at Cz, declining in amplitude by 16% at Fz and 33% at Pz. These differences did not reach significance. N550 was markedly frontally distributed, declining in amplitude by 41% and 61% at Cz and Pz, respectively ($F = 79.06$, $p < 0.01$). P900 had a more frontocentral distribution. It tended to be maximum at Fz, declining in amplitude by 21% and 33% at Cz and Pz, respectively. These differences were, however, not significant. The amplitudes of the different components (N350,

TABLE 1. *Effects of stage of sleep and scalp site on the mean amplitude (in μV) of the different components of the K-complex (SDs are in parentheses) for the standard condition (80 dB SPL intensity, 2 milliseconds rise-and-fall time)*

	2 early	2 late	Slow-wave sleep
N350			
Fz	−26.7 (21.9)	−35.5 (28.1)	−38.9 (22.1)
Cz	−28.3 (19.7)	−42.5 (24.6)	−49.3 (23.2)
Pz	−21.5 (17.1)	−26.9 (22.2)	−32.5 (15.0)
N550			
Fz	−67.7 (17.5)	−70.8 (17.4)	−93.9 (37.8)
Cz	−37.4 (15.5)	−46.2 (11.5)	−53.4 (21.2)
Pz	−22.4 (14.8)	−33.5 (8.4)	−33.2 (10.4)
P900			
Fz	36.6 (25.5)	38.9 (13.6)	69.1 (18.9)
Cz	28.6 (21.7)	28.6 (12.1)	54.3 (20.9)
Pz	19.3 (13.3)	15.5 (8.8)	39.0 (10.1)

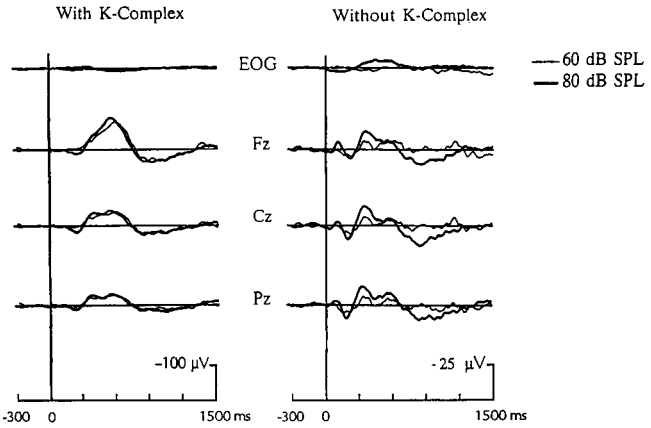

FIG. 4. Grand average of all subjects for all trials containing (left) and not containing (right) a defined K-complex. Note that trials in which a K-complex could not be discerned are plotted at a gain of ×4. Stimulus intensity was either 80 (thick line) or 60 dB (thin line). The K-complex consists of an early negative peak N350, a second negative peak N550 and followed by a positive peak P900. N350 remains visible (although attenuated), whereas N550 is difficult to discern, and P900 is markedly attenuated in single trials in which a K-complex could not be identified.

N550 and P900) at each electrode placement are shown in Table 1 for the standard condition.

The effects of stimulus intensity are illustrated in Fig. 4. Amplitudes of the different components for the two intensities (60 and 80 dB) are presented in Table 2.

When a K-complex was observed (again defined on the basis of N550–P900) (left-hand portion of the figure), N350 varied in amplitude from 14 to 45 μV in individual subjects or across conditions ($\bar{x}$ = 35.1, SD = 16.3 μV). In these trials, its amplitude was not significantly affected by the intensity (60 or 80 dB) or the rise-and-fall time (2 or 20 milliseconds) of the stimulus ($F < 1$ in both cases). Stage of sleep had no main or interacting effects. Its amplitude was reduced by approximately 50% in trials in which no K-complex could be identified in the background noise. Moreover, in these trials, a decrease in stimulus intensity resulted in a significant attenuation (by, on average, 26%) of N350 amplitude ($F = 3.20$, $p < 0.02$). Manipulation of the rise-and-fall time had no significant effect on N350 amplitude. Stage of sleep had no main or interacting effects. No latency shifts were observed for any of the conditions or in any stage of sleep.

When a K-complex was elicited, N550 amplitude varied from 45 to 164 μV ($\bar{x}$ = 77.5, SD = 26.6 μV) at Fz. No significant differences were found for either its amplitude ($F < 1$) or its latency ($F < 1$) for either the manipulation of stimulus intensity or its rise-and-fall time. It tended to peak earlier and was larger in SWS compared to 2 early and 2 late, although the differences were not significant ($p > 0.05$). When no K-complex could be identified, N550 was not visible.

When a K-complex was elicited, P900 amplitude varied from 15.5 to 74 μV ($\bar{x}$ = 47.5 μV, SD = 29.5). The P900 component followed the same pattern as the N550 on trials in which a K-complex could be identified. Thus, neither the manipulation of the intensity of the stimulus nor its rise-and-fall time significantly affected the K-complex's amplitude or latency ($F < 1$ in both cases). On the other hand, in single trials in which a K-complex could not be identified in the background noise, only a very small amplitude P900 was visible in the averaged waveform.

Experiment 2

Across all conditions and stages of sleep, a K-complex was elicited on approximately half of all trials (0.50). The probability of occurrence in the 2,000 Hz condition was 0.53 compared to 0.50 and 0.49 for the 1,000 and 500 Hz conditions, respectively. No significant differences were observed among the different conditions. K-complexes were more likely to occur in stages 2 early and 2 late (0.51 and 0.56 probability, respectively) than in stage SWS (0.45) but the differences were again not significant.

The averaging procedure of Experiment 2 was identical to that used in Experiment 1. Thus, trials were again sorted into those in which a K-complex was identified and those in which it could not be identified. They were then averaged by condition and stage of sleep.

Across all conditions and all stages of sleep, when a K-complex was elicited, it consisted of a biphasic negative complex having peaks at 387 and 615 milliseconds (N350 and N550, respectively) and followed by a later positive wave, peaking at 954 milliseconds (P900). Again, N350 could only be discerned following averaging. For trials not containing the large N550–

TABLE 2. *The effects of stimulus intensity on the mean amplitude (in μV) of the different components of the K-complex (SDs are in parentheses)*[a]

	80 dB			60 dB		
	2 early	Slow-wave sleep	2 late	2 early	Slow-wave sleep	2 late
N350	−26.8 (21.9)	−38.9 (22.1)	−35.5 (28.1)	−14.4 (4.7)	−28.4 (17.5)	−42.0 (32.7)
N550	−67.7 (17.5)	−93.9 (37.8)	−70.8 (17.4)	−68.6 (33.1)	−82.1 (23.9)	−78.4 (33.8)
P900	36.6 (25.5)	69.1 (18.9)	38.9 (13.6)	38.4 (30.2)	33.2 (21.8)	69.2 (20.2)

[a] Data are from frontal recordings.

P900 complex, the N350 deflection still remained apparent. Different scalp topographies were again observed for the different components. N350 tended to be uniformly distributed over midline areas of the scalp. It was maximum at Cz declining in amplitude by 30% at Fz and 27% at Pz. These differences did not reach significance. N550 was markedly frontally distributed, declining in amplitude by 32% and 45% at Cz and Pz, respectively ($F < 1$). P900 had a more frontocentral distribution. It tended to be maximum at Fz, declining in amplitude by 7% and 46% at Cz and Pz, respectively. These differences were, however, not significant.

The effects of tonal frequency on the averaged K-complex are illustrated in Fig. 5 and the mean amplitudes of the different components are shown in Table 3. When a K-complex was observed (left-hand portion of the figure), N350 varied in amplitude from 13 to 78 μV in individual subjects or across conditions ($\bar{x}$ = 32.7, SD = 24.6 μV). In these trials, its amplitude was not significantly affected by the frequency (2,000, 1,000 or 500 Hz) of the stimulus ($F < 1$). Stage of sleep had no main or interacting effects. Its amplitude was reduced by approximately 60% in trials in which no K-complex could be identified in the background noise. Stage of sleep had no main or interacting effects.

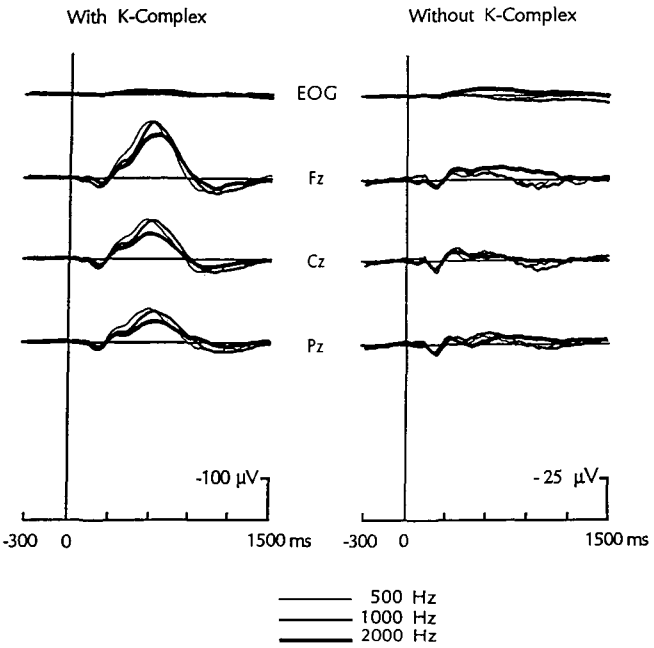

FIG. 5. Grand average of all subjects for all trials containing (left) and not containing (right) a defined K-complex. Note that trials in which a K-complex could not be discerned are plotted at a gain of ×4. Tonal frequency was either 500 (thin line), 1,000 (medium line) or 2,000 Hz (thick line). Again, the K-complex consists of an early negative peak N350, a second negative peak N550 and followed by a positive peak P900. N350 remains visible (although attenuated), whereas N550 is difficult to discern, and P900 is markedly attenuated in single trials in which a K-complex could not be identified.

TABLE 3. *The effects of tonal frequency (in Hz) on the mean amplitude (in μV) of the different components of the K-complex (SDs are in parentheses)*[a]

	500			1,000			2,000		
	2 early	Slow-wave sleep	2 late	2 early	Slow-wave sleep	2 late	2 early	Slow-wave sleep	2 late
N350	−50.1 (38.6)	−22.1 (49.9)	−29.9 (45.8)	−33.6 (28.6)	−32.7 (37.8)	−25.3 (34.4)	−12.8 (14.8)	−10.1 (12.9)	−19.2 (25.1)
N550	−116.6 (19.3)	−82.9 (31.2)	−118.7 (33.5)	−104.3 (27.2)	−86.1 (30.1)	−122.2 (23.2)	−110.3 (24.2)	−71.2 (17.9)	−95.1 (18.6)
P900	39.4 (10.5)	27.8 (11.5)	24.1 (11.5)	22.5 (13.9)	32.9 (5.2)	24.1 (22.7)	19.1 (17.3)	40.5 (15.5)	13.5 (19.2)

[a] Data are from frontal recordings.

No latency shifts were observed for any of the conditions or in any stage of sleep.

When a K-complex was elicited, N550 amplitude varied from 47 to 163 μV ($\bar{x}$ = 94.6, SD = 31.2 μV) at Fz. No significant differences were found for either its amplitude ($F < 1$) or its latency ($F < 1$) for the manipulation of the stimulus frequency. When no K-complex could be identified, N550 was not visible.

The P900 component varied from 13 to 75 μV ($\bar{x}$ = 28.3, SD = 16.5 μV) and followed the same pattern as the N550 on trials in which a K-complex could be identified. Thus, the manipulation of the frequency of the stimulus did not significantly affect the K-complex's amplitude or latency ($F < 1$ in both cases). On the other hand, in single trials in which a K-complex could not be identified in the background noise, only a very small amplitude P900 was visible in the averaged waveform.

DISCUSSION

Across the two experiments, in the standard 80 dB 2 millisecond rise-time conditions, the overall probability of eliciting a K-complex was approximately 0.50. The probability of eliciting it decreased with a decrease in stimulus intensity and an increase in rise-time. Although these probabilities were not significantly different, they are consistent with other studies (4,6,7). The second experiment indicated that tonal frequency had no significant effect on the probability of eliciting the K-complex, replicating similar findings by Campbell et al. (7).

In the initial experiment, the K-complex was elicited more frequently in the first half of the night than in the second. This was not the case in the second experiment. The K-complex was elicited more often in stage 2 (both early and late) than in SWS. Halász et al. (4) found that the number of evoked K-complexes decreased from the early to the later portion of sleep and, furthermore, from cycle to cycle. The probability of its elicitation was higher in ascending slopes of sleep stages than in descending ones. They interpreted this as being related to the deepness of stages of sleep, as K-complexes were more frequently observed when stage 2 was followed by stages 3–4 than by the REM stage. However, some authors (16,17) have reported that a significantly higher density of K-complexes can be counted in the 10 minute time period prior to REM sleep. On the other hand, Paiva and Rosa (18) have indicated that the frequency of occurrence of a K-complex does not vary among the stages or cycles of sleep. Our present results thus are in agreement with their findings. Paiva and Rosa (18) have nevertheless observed that the number of K-complexes markedly increases in the period prior to any stage transition (i.e. not just REM or when the transition is into a deeper stage of sleep).

When the K-complex was elicited, it consisted of a biphasic negative deflection (N350, N550) followed by a late positive deflection (P900). N350, N550 and P900 were found to have different topographies. These results are in agreement with those obtained by Ujszaszi and Halász (5). They also noted that the N350 component was evenly distributed over midline areas, whereas the N550 component was markedly frontally distributed. Paiva and Rosa (18) have also reported that the spontaneous K-complex (N550–P900) is usually distributed frontally or frontocentrally. In fewer cases, it was distributed maximally at the vertex and, in rare cases, over posterior sites. It is possible that differences in scalp topography are due to a comparison of spontaneous versus evoked K-complexes, peak-to-peak versus baseline-to-peak measurements or measurement of single trials (and the possibility of overlapping background noise) versus averaged K-complexes. The P900 was distributed frontocentrally. When the large N550–P900 could not be discerned in the background noise (i.e. when their peak-to-peak amplitude did not exceed 75 μV), only small amplitude N350 and P900 waveforms were visible following averaging. N550 was not visible in averages of individual subjects or in the grand averages of these individual averages in any stage of sleep. On this basis, it can be concluded that N350, N550 and P900 reflect functionally distinct processes.

Neither the peak amplitude nor latency of N550 or P900 was significantly affected by either the manipulation of stimulus intensity, its rise-and-fall time or its tonal frequency. Moreover, their morphology remained unaltered throughout the different stages of sleep. This provides powerful support for the classic notion that the K-complex (at least the large amplitude N550–P900 complex) is an all-or-none phenomenon (4). An external stimulus either elicits a K-complex or it does not. When it does, the N550–P900 complex does not vary in amplitude regardless of manipulation of the parameters of the evoking stimulus. Such findings contradict results obtained by Campbell et al. (7) and Church et al. (6) who showed variation in the amplitude of the K-complex with either manipulation of the intensity or the rise-and-fall time of the stimulus. This controversy may be explained by the fact that these previous studies averaged all trials regardless of whether a K-complex was evoked or not. The indiscriminate use of averaging probably accounted for their effects.

It is possible that the arbitrary decision to select only frontocentral responses exceeding 75 μV might have confounded these results. This does not appear to be the case. When averaged, trials in which N550–P900

did not exceed 75 μV contained no visible evidence of an N550 deflection. On the other hand, it is also possible that in some trials noise was considered to be a true K-complex. In such cases, the background noise had to exceed 75 μV in the time interval used to define a K-complex and, moreover, the noise had to have a frontocentral distribution. This was highly unlikely.

Residual background noise can, however, be used to explain the variation in the K-complex within and between conditions. Although averaging techniques will reduce background noise, it will not eliminate it altogether. Individual subject averages for a single condition were at times based on a relatively small number of trials (as few as four or five in some cases), depending on the number of K-complexes that were elicited. The amplitude of the background noise is reduced in a nonlinear asymptotic manner. Although the background noise will therefore be markedly reduced after a small number of trials (even with the large amplitude background EEG seen in sleep), a significant amount of noise will remain in the waveform following averaging procedures.

In trials in which the large N550–P900 complex was not identified, N350 was still visible. Its amplitude was, however, markedly reduced compared to trials in which N550–P900 was elicited. In these trials, N350 varied directly as a function of the intensity of the stimulus. On the other hand, in trials in which the N550–P900 complex was elicited, N350 was not affected by stimulus intensity. It would thus appear that N350 continues to increase in amplitude with increases in stimulus intensity until it reaches a certain critical threshold amplitude at which point the invariant all-or-none N550–P900 is triggered. As mentioned, neither N550 nor P900 was affected by manipulation of stimulus intensity.

A number of negative waves in the 350–450-millisecond latency have been reported in the sleeping subject. For example, a late negative wave, peaking at about 350 milliseconds, increases in amplitude at sleep onset (19,20). Picton and Hillyard (21) suggested that a sleep N2 might play a role in arousing the subject from sleep or alternatively as a means to prevent arousal from it. Others have observed a vertex sharp wave at sleep onset, occurring again in the 350–450-millisecond range, following stimulus presentation. Broughton (22) has suggested that N2 at sleep onset may be related to the presence of vertex sharp waves. It may well be that the sleep-onset N2, the vertex sharp wave and the N350 wave reflect the same process. However, N350 and N2 have different scalp distributions. N2 is markedly frontally distributed, whereas N350 is evenly distributed over midline areas (and tends to be largest at the vertex). Moreover, N2 decreases in amplitude later in the night (20,23), whereas N350 remains relatively unaltered. In any case, the present findings suggest that N350, although perhaps being part of the K-complex, is not unique to it. It is possible that N350's role could be limited to the initiation of the N550–P900 complex, and it may be this complex that either assists or prevents the arousal from sleep.

Hess (24) has suggested that the K-complex (the N550–P900 complex) may play an important role in the prevention of awakenings. Recently, Ujszaszi and Halász (5) claimed that information processed by the K-complex would provide an indication of nonspecific arousal mechanisms and could play an important role in orientation in sleep. In this context, McDonald and Carpenter (25) reported that the K-complex can be elicited to meaningful stimuli (such as the subject's name). Oswald et al. (10) claimed that the more significant the stimulus (the more it resembled the subject's name), the larger the amplitude of the K-complex. Oswald et al. (10) and McDonald and Carpenter (25) therefore suggested that the K-complex was an orienting response (OR). The probability of evoking a K-complex increases with the intensity of the stimulus (4,6,7). This is also the case with an OR. However, the probability of eliciting a K-complex also increases with a decrease in the rise-and-fall time of the stimulus. Stimuli that are loud (> 100 dB) and abrupt (i.e. having fast rise-times) may evoke a defensive response (DR) (26). In the present study, stimulus intensity did not exceed 80 dB SPL. This intensity rarely elicits a DR in the waking subject. The effects of loudness might, however, vary from the waking to the sleeping states. Indeed, Church et al. (6) have indicated that lower intensity 44-dB SPL tones cause an acceleration in the heart-rate in the sleeping subject. Heart-rate acceleration is considered to be a component of the DR (rather than an OR). It would therefore seem to be possible to elicit DRs with even relatively low-intensity stimuli in the sleeping subject. The Johnson and Karpan study (9) also noted that heart-rate acceleration occurred when K-complexes did not. From this perspective, although the K-complex may occur in association with other indices of a DR, DRs also occur in the absence of the K-complex.

In conclusion, our results suggest that the K-complex consists of three components, N350, N550 and P900. These components seem to be morphologically and functionally independent. The three components have different scalp topographies. Moreover, N350 was still apparent on trials in which N550–P900 could not be clearly identified in the background noise. In these trials, the amplitude of N350 varied directly with the intensity of the evoking stimulus. The amplitude of N550–P900 did not vary on trials in which a K-complex was elicited. This complex therefore appears to be an all-or-none phenomenon.

Acknowledgements: This research was partially supported by funds provided by the National Science and Engineering Research Council (NSERC) of Canada. The authors acknowledge the contributions of Louise Rouillard, Ian Bell, Herman van den Bergen, Madan Makasare and Robert Spratt.

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