Electroencephalography and clinical Neurophysiology, 91 (1994) 140–148

EEG 92657

Is the appearance of mismatch negativity during stage 2 sleep related to the elicitation of K-complex?

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(Accepted for publication: 2 March 1994)

Summary There is no convincing evidence for the occurrence of mismatch negativity (MMN) elicited by infrequent deviant tones in a homogenous tone stream during sleep in adult humans. Also the data presented here failed to show an MMN during any stage of sleep when event-related potentials (ERPs) were averaged across all trials of the same sleep stage. The aim of the study was to determine whether the MMN appearance during sleep is related to the variations in microstates of sleep that differ in terms of stimulus elicited phasic EEG events. The focus was on stage 2 sleep. The single responses to a deviant tone were classified into 3 types during stage 2 prior to averaging ERPs. These 3 response types included K-complex, other phasic EEG events and no visually discernible phasic EEG events. The results showed that an MMN-like deflection indeed appeared during stage 2 but only when the deviant tone also elicited a K-complex. This type of deflection was not seen when the deviant tone was presented without the intervening standard tones. This supports the hypothesis that a true MMN to the deviant tone was seen during stage 2 sleep preceding a K-complex.

Key words: Event-related potentials; Mismatch negativity; Phasic EEG events; K-complex; Sleep

There are two main types of event-related potential (ERP) studies that attempt to illuminate the ways external stimuli are "processed" during sleep. The first identifies sleep-specific ERP deflections during different stages of sleep and their relation to information processing (Williams et al. 1962; Weitzman and Kremen 1965; Kevanishvili and Von Specht 1979; Campbell et al. 1985; Ujszászi and Halász 1986, 1988; Halász and Ujszászi 1988) whilst the second compares ERP deflections which are well known in the awake state to those found during sleep (Paavilainen et al. 1987; Campbell et al. 1988, 1992; Wesensten and Badia 1988; Nielsen-Bohlman et al. 1991). One of these latter deflections, that can be considered to occur also during sleep, is the mismatch negativity (MMN). The MMN has been thought to represent a difference found in a comparison process between a neuronal trace created by the homogenous repetitive standard stimuli and an input of the rare deviant stimulus (see Näätänen 1990). Many waking studies have supported the contention that the MMN is an automatic response, i.e., that it occurs even in the absence of attention (e.g., Sams et al. 1985b; Mäntysalo and Näätänen 1987; Alho et al. 1989; Kaukoranta et al. 1989; Lounasmaa et al. 1989; Lyytinen et al. 1992). However, results of some recent waking studies suggest that the amplitude of the MMN could be influenced by attention (Woldorff et al. 1991; Alho et al. 1992; Woods et al. 1992). One very critical test for the MMN's automaticity is to examine whether it occurs during sleep. In sleep, a subject can be considered to be in an extreme state of inattention.

The findings on the occurrence of MMN during sleep have been thus far quite contradictory [1]. Paavilainen et al. (1987) and Nielsen-Bohlman et al. (1991) failed to show an MMN during sleep. Paavilainen et al. assumed that their stimulus deviation (standard 1000 Hz and deviant 1050 Hz) was probably too small to elicit an MMN during sleep. Nielsen-Bohlman et al. suggested that the MMN may be masked by "an increased noise level" during sleep. In contrast to these studies, there are a few studies in which signs of sleep MMN have been obtained. Campbell et al. (1992) found an MMN-like response during the late portions of stage 2 and REM sleep under 1 out of 4 stimulus conditions. The stimulus conditions differed in terms

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[1] Very recent results of Campbell et al., presented at EPIC X in Hungary 1992, suggest that the detection of MMN is difficult in NREM sleep but that in REM sleep the MMN can be seen although reduced in amplitude. The results of Winter et al. presented at the same congress failed to show an MMN during stage 2 sleep. This supports the proposition that it is difficult to demonstrate the MMN in NREM sleep.

SSDI 0013-4694(94)00066-T

of probability and magnitude of stimulus deviation. These authors assumed that they probably used a too long interstimulus interval (ISI; 0.5 sec) which made it difficult to demonstrate the MMN during sleep. According to Alho et al. (1990), it is possible to record an MMN-type deflection from newborns who are in "quiet sleep." Csépe et al. (1987) reported that in cats the MMN is elicited also during sleep but its cortical distribution is more restricted and its latency is longer than in wakefulness.

Consistent with the studies of Paavilainen et al. (1987) and Nielsen-Bohlman et al. (1991), we failed to show an MMN during any stage of sleep when ERPs were averaged across all trials of the same sleep stage (Fig. 1) (Sallinen et al., unpublished paper). However, one possibility is that a single sleep stage may not be a homogenous state with regard to the occurrence of MMN. This means that the MMN may only occur during certain moments of sleep but not consistently resulting in the failure to observe the MMN in the ERPs averaged across all trials of a single sleep stage. Because of this possibility the data used in our above-mentioned unpublished study were reanalyzed.

The variation of single EEG responses during sleep is a well-known phenomenon. Especially Ujzsászi and Halász (1986) have stressed this issue. These authors showed that single brain responses of stage 2 sleep to an invariable stimulus vary considerably from K-complex-like responses of high amplitude (> 121 μV) to responses of low amplitude (< 80 μV) which are some-

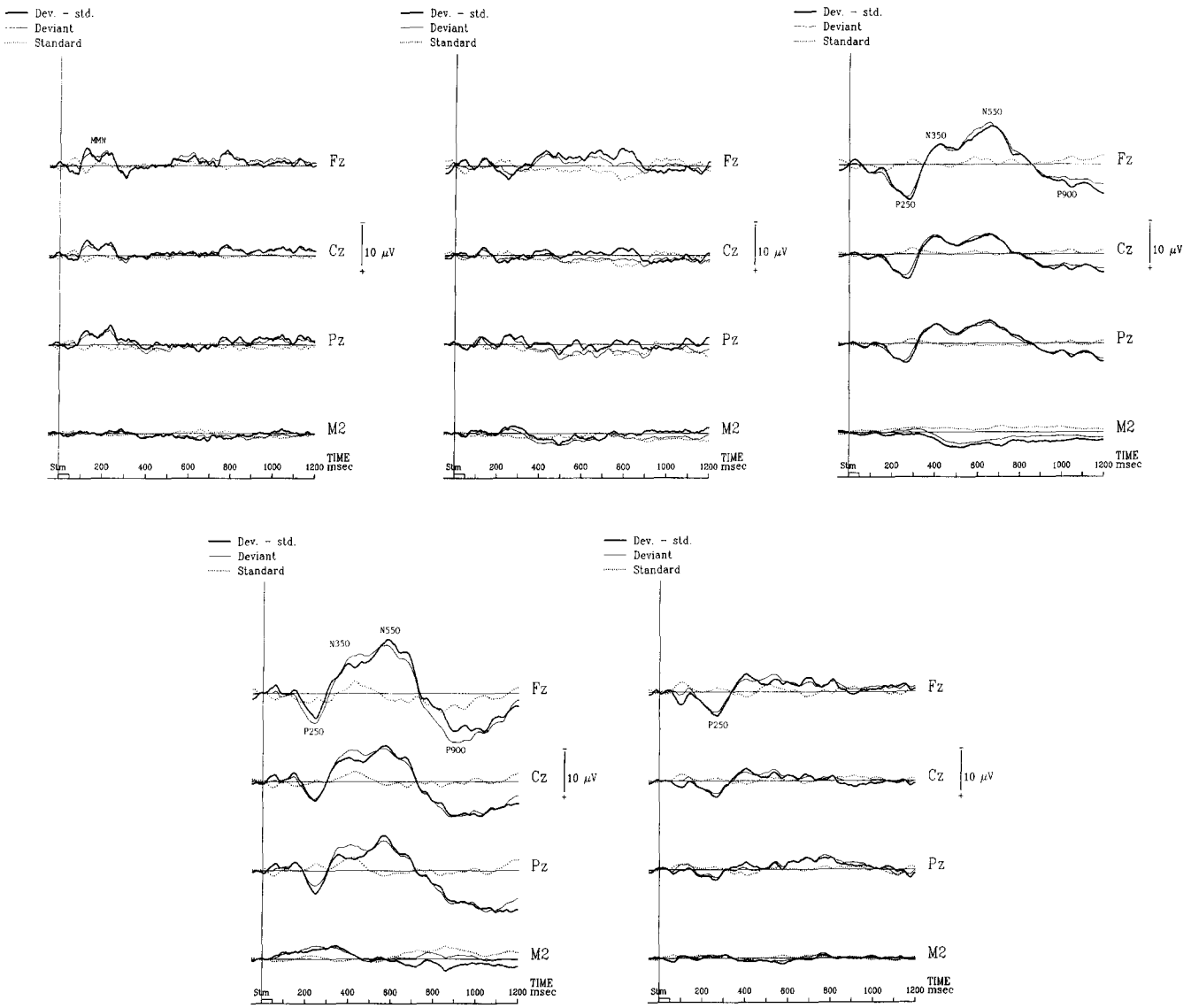

Fig. 1. Grand average ERPs across subjects to the standard (dotted line) and deviant stimuli (thin line) during the reading session (up left), stage 1 (up middle), stage 2 (up right), stage 3/4 (down left), and REM sleep (down right). The difference curves (deviant-standard) are marked with a thick line. During the reading session, stage 1 and stage 2 sleep N = 10. During stage 3/4 and REM sleep N = 9.

times invisible. The authors suggested that this variation reflects the existence of different microstates within stage 2. Ujzsászi and Halász (1986) speculated that the variation may indicate differences in the depth of sleep or in momentary capacity for cortical information processing. If MMN does occur during sleep it can be considered to be most probable when the unaveraged EEG shows a large response (such as the K-complex) to a deviant stimulus. In order to determine whether the occurrence of MMN during sleep varies as a function of such microstates, single evoked responses to a deviant stimulus were classified before averaging ERPs in the present study.

Our study was limited to stage 2 sleep because different microstates are most easily detected during this sleep stage. During slow wave sleep (SWS), stimulus-elicited phasic EEG changes become easily confused with spontaneous delta activity. In addition, the MMN is very small in amplitude and its separation from the high amplitude spontaneous EEG during SWS may be difficult.

Two kinds of stimulus paradigms were used in the present study: the oddball paradigm (infrequent deviant tones presented with frequent standard tones) and OR paradigm (infrequent tones presented without intervening standard tones). The OR paradigm was included because the MMN is shown to disappear when standard tones are removed around deviant tones (no memory trace exists for a comparison process) (Sams et al. 1985a; Näätänen et al. 1989). Thus the OR paradigm makes it possible to evaluate whether a possible MMN-like deflection elicited by deviant tones in the oddball paradigm during sleep is a true MMN.

Methods

Subjects

Ten healthy subjects (5 females) without any history of hearing problems, between the ages of 16 and 33 years, slept 2 nights in the laboratory. Five of the subjects had participated earlier in a psychophysiological study. Subjects were told to refrain from use of alcohol for 24 h prior to the experiment. None of them used drugs.

Procedure and stimuli

During half of the nights, a tone was presented as a deviant tone with homogenous standard tones (oddball paradigm) and during the other half the same tone was presented alone, i.e., without the intervening standard tones (OR paradigm). The order of the nights was balanced across the subjects.

The subjects slept about 8 h (typically from 11 p.m. to 7 a.m.) in the laboratory. The stimuli were presented through a loudspeaker set behind the lying subject's head. ERPs were also recorded during a reading session before falling asleep or in the morning, after a whole night sleep. During the reading period, the subjects were instructed to concentrate on text material they had chosen in advance. In the oddball paradigm, the standard stimuli of 1000 Hz and deviant stimuli of 1200 Hz were presented with 97.5% and 2% probabilities, respectively.

The subject's own and other names were also presented at 0.25% probability for each. The responses to the name stimuli are not reported here. Both standard and deviant stimuli had an intensity of 45 dB and 10 msec rise and fall time with a 30 msec plateau (total duration 50 msec). The fixed interstimulus interval was 625 msec. The interval between two consecutive deviating stimuli (i.e., deviants and names) varied pseudorandomly between 20 and 45 sec so that the probabilities were as mentioned above. In the OR paradigm the deviant stimuli (called the OR stimuli in this paradigm) were presented using an identical schedule. The only difference was that the standard stimuli were omitted.

Recording

ERPs were recorded from Fz, Cz, Pz and M2 (right mastoid) with silver-silver chloride electrodes, referenced to the nose. M2 was included because the MMN has been reported to reverse polarity below the sylvian fissure (Alho et al. 1986; Paavilainen et al. 1991). The time constant and low-pass filter were 1 sec and 100 Hz, respectively. EEG epochs of 3 sec (1.5 sec before and after the deviant/OR stimuli, depending on the paradigm) were digitized at the 200 Hz sampling rate (i.e., in the oddball paradigm, within one trial the standard stimulus occurred 4 times and the deviant stimulus once). Sleep stages were scored visually according to the criteria of Rechtschaffen and Kales (1968). EEG, EOG and EMG (placement of electrodes according to Rechtschaffen and Kales 1968) epochs of 20 sec (10 sec before and after the deviant/OR stimuli) were digitized at the 100, 50 and 200 Hz sampling rates, respectively.

ERP analysis

The trials were divided into 3 classes according to phasic EEG events following the deviant/OR stimuli prior to averaging ERPs. These 3 classes, which are illustrated in Fig. 2, were as follows: K-complex (KC), other phasic EEG events (OPE), and no visually discernible phasic EEG events (no response, NR). To become selected into the KC class, an event following the deviant/OR stimulus had to meet the criteria of morphology and duration of the K-complex adopted from Rechtschaffen and Kales (1968). According to these authors the K-complex is a "negative sharp wave which is immediately followed by a positive component." A total duration has to exceed 0.5 sec. More-

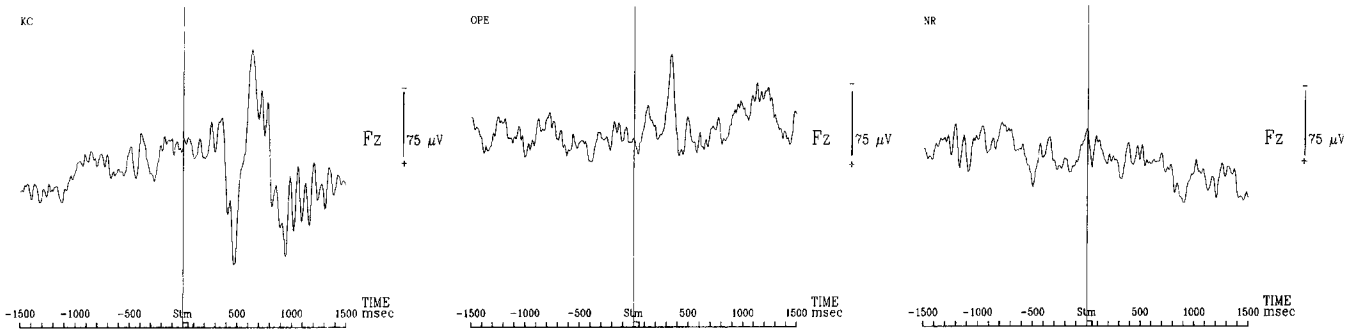

Fig. 2. Examples of the 3 different types of trials recorded from Fz. KC, K-complex (left); OPE, other phasic EEG event (middle); and NR, no response (right). Subject M.K. Vertical lines indicate deviant stimulus onset.

over, in the present study a minimum peak-to-peak amplitude for a K-complex event was 75 μV. This amplitude criterion had to be met either at Fz, Cz or both. In the OPE class were those EEG events following the deviant/OR stimuli, which did not meet all these criteria of the K-complex but clearly deviated from the ongoing EEG. In the NR class were those trials, during which the EEG following the deviant/OR stimulus did not deviate from the ongoing EEG. The phasic EEG events had to emerge within the latency range of 200–1000 msec to be classified as stimulus-elicited.

Secondly, prior to averaging ERPs, two criteria were applied to the interval from the onset of the deviant/OR stimulus to 200 msec after the stimulus. Trials including either a maximum peak-to-peak amplitude over 75 μV or a sleep spindle (or both) within this interval were excluded. This was done because otherwise a possible MMN-like deflection might have been overlapped by the large EEG events occurring often spontaneously during stage 2.

MMN was identified by subtracting ERPs to the standard stimulus from ERPs to the deviant stimulus. ANOVA was used to test whether the negative peak amplitude of the ERPs to the deviant stimulus differed significantly from the corresponding amplitude of the ERPs to the standard stimulus within the 100–250 msec latency range (the waking MMN latency range). ANOVA was performed when the difference curve deviant-standard showed a clear negative deflection within this latency range. The peak amplitudes were computed by subtracting the mean value of the −50–0 msec prestimulus time from the negative peak of the 100–250 msec poststimulus time.

Results

Oddball paradigm

Six out of 10 subjects had a sufficient number of all trial types (> 20 each) during stage 2 sleep for averaging. Statistical analyses were conducted on the data from these subjects.

ERPs during reading. ERPs of the reading session showed a negative displacement within the waking MMN latency range reaching significance at Fz (F (1, 5) = 13.3, P = 0.015), Cz (F (1, 5) = 18.5, P = 0.008), and Pz (F (1, 5) = 14.5, P = 0.013) (Fig. 3). The polarity of this frontally distributed displacement did not clearly reverse at M2. Beyond the waking MMN latency range, the ERPs revealed a small frontally distributed positivity following the negative displacement.

Phasic EEG events following standard and deviant stimuli during stage 2. The average proportions of KC, OPE and NR events following the stimuli are shown in Table I. It shows that K-complexes followed the deviant stimuli with a considerably higher probability than the standard stimuli. Interestingly, the type of

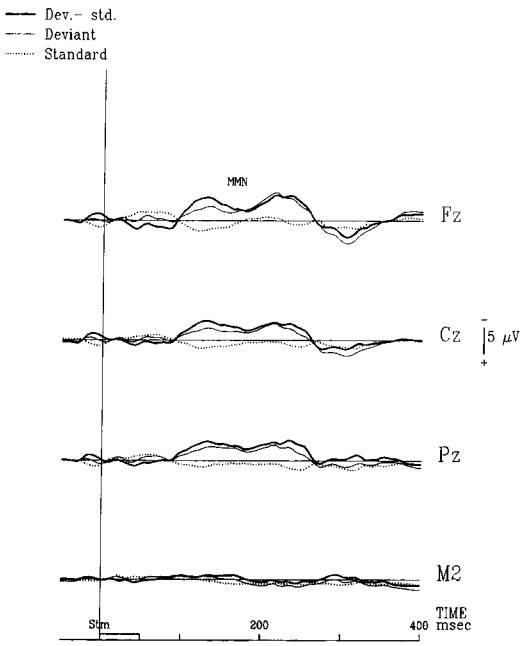

Fig. 3. Grand average ERPs (505 trials) to the standard (dotted line) and deviant stimuli (thin line) in the reading situation. The deviant-standard difference curve is marked with a thick line.

TABLE I

The average of probabilities of K-complexes (KC), other phasic EEG events (OPE) and no responses (NR) following standard and deviant stimuli during stage 2 sleep (oddball paradigm).

	Standard (%)	Deviant (%)	*t* value	*df*	*P*
KC	1.8	22.2	8.22	5	< 0.001
OPE	23.3	23.5	0.08	5	ns
NR	74.9	54.3	−3.16	5	= 0.015
Total	100	100			

stimuli had no effect on the probability of the occurrence of OPE events. When the OPE events following the deviant and standard stimuli were averaged separately the ERPs were, however, very different (Fig. 4). The ERPs of the OPE events following the standard stimuli showed a frontally distributed long lasting negativity. The corresponding ERPs to the deviant stimuli revealed P250, N350, N550 and P900 deflections.

ERPs during stage 2 across the KC, OPE and NR trials. When ERPs were averaged across all trials (i.e., averaged without classification of trials) during stage 2 no deflection resembling the MMN could be detected (Fig. 5). There was only a small negative deflection at Pz but it failed to reach significance. Otherwise, only the K-complex deflections (N350, N550, and P900) and a P250 were seen.

ERPs during the KC trials. The difference curve of the KC trials showed a negative deflection within the waking MMN latency range (called MMN-like deflection here) that preceded K-complex deflections N350, N550 (polarity reversal at M2) and P900 and a P250 (Fig. 6). The ERPs to the deviant stimulus differed significantly from the ERPs to the standard stimulus at

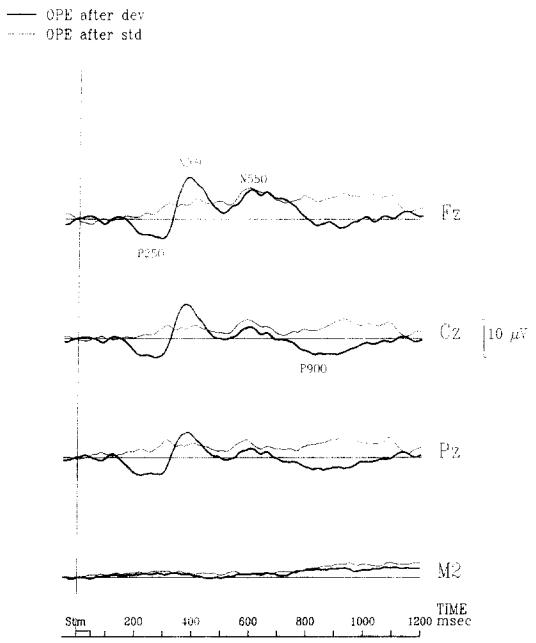

Fig. 4. Grand average ERPs of the other phasic EEG events (OPE) following the standard (thin line, 229 trials) and deviant (thick line, 219 trials) stimuli during stage 2 sleep.

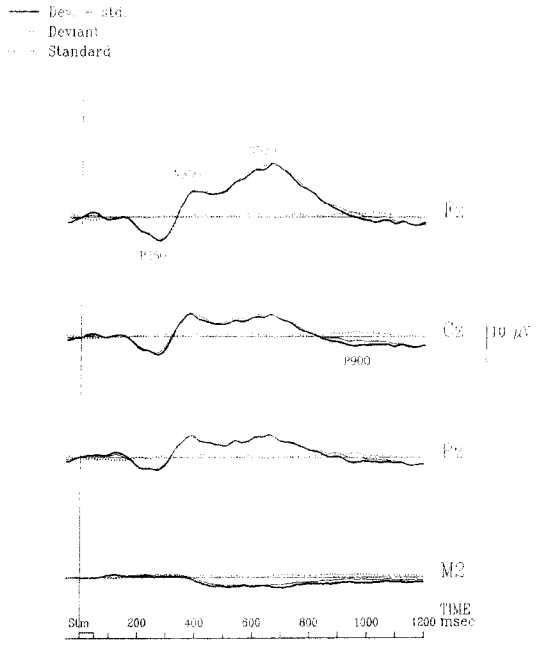

Fig. 5. Grand average ERPs to the standard (dotted line) and deviant stimuli (thin line) averaged across KC (207 trials), OPE (219 trials), and NR trials (507 trials) during stage 2 sleep. The deviant-standard difference curve is marked with a thick line. The critical latency range is from 100 to 250 msec.

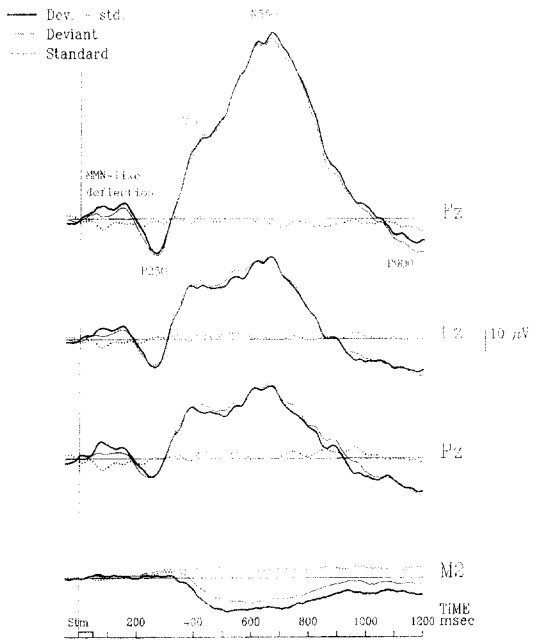

Fig. 6. Grand average ERPs (207 trials) to the standard (dotted line) and deviant stimuli (thin line) during the KC trials of stage 2 sleep. The deviant-standard difference curve is marked with a thick line. The critical latency range is from 100 to 250 msec.

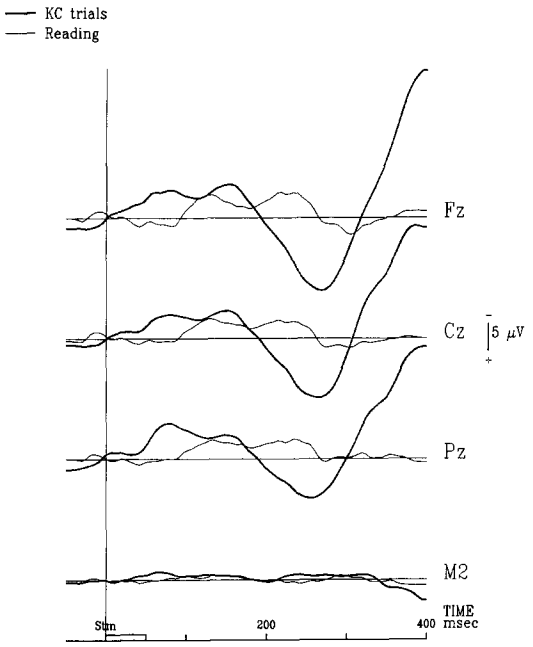

Fig. 7. The deviant-standard difference curves of the reading situation (thin line, 505 trials) and the KC trials of stage 2 sleep (thick line, 207 trials). Notice the change in the time scale compared to the previous figures.

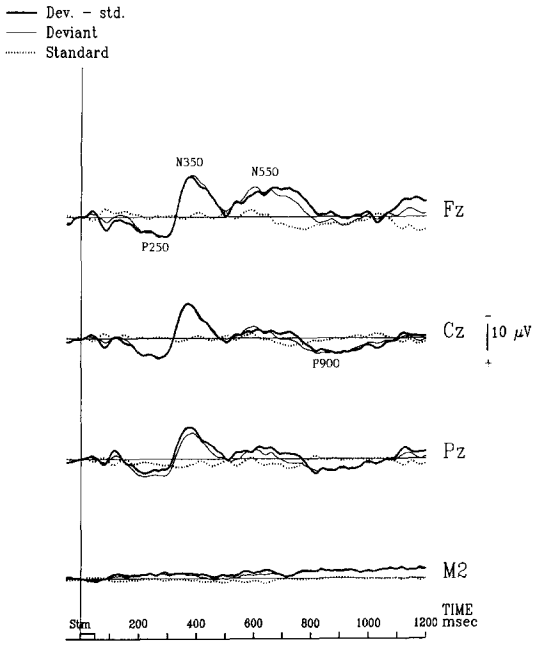

Fig. 8. Grand average ERPs (219 trials) to the standard (dotted line) and deviant stimuli (thin line) during the OPE trials of stage 2 sleep. The deviant-standard difference curve is marked with a thick line. The critical latency range is from 100 to 250 msec.

Fz (F (1, 5) = 7.0, $P = 0.045$) and Cz (F (1, 5) = 26.6, $P = 0.004$). The peak amplitude of this frontally distributed deflection did not differ from the awake state's MMN. The latency of the MMN-like deflection seemed to be shorter than that of the awake state's MMN but the difference failed to reach significance (at Fz F (1, 5) = 6.56, $P = 0.051$) (Fig. 7). The MMN-like deflection consisted of two peaks as did the MMN in wakefulness. The peak amplitudes and latencies of the MMN-like deflection and the awake state's MMN at Fz, Cz, and Pz are shown in Table II.

ERPs during the OPE trials. The difference curve of the OPE trials revealed no negative deflection within the waking MMN latency range at Fz or Cz (Fig. 8). There was a small, negative deflection at Pz within this latency range but it failed to reach significance. Outside the waking MMN latency range, the grand average difference curve showed the same long-latency deflections as during the KC trials. In this curve, their amplitudes were smaller than during the KC trials.

ERPs during the NR trials. There was no sign of any early negativity seen at Fz or Cz in the difference curve of the NR trials (Fig. 9). A small, non-significant

TABLE II

The peak amplitudes and latencies of the negativities within latency range of 100–250 msec during reading and stage 2 sleep in the K-complex (KC) trials (oddball paradigm).

	Peak amplitude (μV)		Peak latency (msec)	
	Reading	KC (stage 2)	Reading	KC (stage 2)
Fz	−5.04	−7.67	200	140
Cz	−3.56	−6.04	200	135
Pz	−3.70	−4.95	200	135

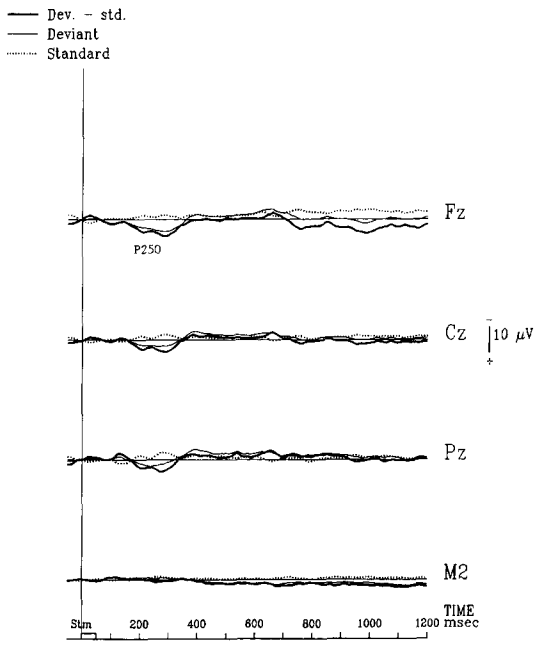

Fig. 9. Grand average ERPs (507 trials) to the standard (dotted line) and deviant stimuli (thin line) during the NR trials of stage 2 sleep. The deviant-standard difference curve is marked with a thick line. The critical latency range is from 100 to 250 msec.

negativity was seen at Pz within this latency range. The negative peak amplitude of this difference curve significantly differed from that of the KC trials at Fz (F (1, 5) = 16.9, P = 0.009) and Cz (F (1, 5) = 10.3, P = 0.024) within the waking MMN latency range. Beyond this interval, a P250 was also seen as in the averages of other trial types (KC and OPE).

OR paradigm vs. oddball paradigm

Phasic EEG events following tones presented alone during stage 2. Eight out of 10 subjects had a sufficient number of all trial types (> 20 each) for averaging. Only data from these subjects were examined. The average proportions of the different trial types were the following: KC trials 35.2%, OPE trials 34.2%, and NR trials 30.6%.

ERPs during reading. Fig. 10 clearly shows that the MMN disappeared when the standard stimuli were removed from the stimulus sequence. ERPs to the tone presented alone showed only a N1 deflection peaking at 130 msec during the reading session. The mean amplitude of the 100–150 msec latency range differed significantly from zero at Fz (F (1, 7) = 28.0, P = 0.001), Cz (F (1, 7) = 34.1, P = 0.001), and Pz (F (1, 7) = 24.7, P = 0.002). The maximum amplitude (7.72 μV) was reached at Fz. There was a small positive deflection at this latency range at M2 but it failed to reach significance. The N1 was followed by two positive deflections of which the later one reached significance at Fz (F (1, 7) = 7.6, P = 0.028), Cz (F (1, 7) = 7.6, P = 0.028), Pz (F (1, 7) = 8.5, P = 0.022), and M2 (F (1, 7) = 13.9, P = 0.007).

ERPs of the KC, OPE and NR trials during stage 2. Fig. 10 shows that no MMN-like deflection was elicited by the tone present alone during the KC trials. The same held true for the OPE and NR trials. Otherwise ERPs of these different trial types consisted of the same deflections as during the oddball paradigm.

Discussion

The only condition where an MMN appeared without any doubt was the reading session. In addition to an MMN, the first part of the negative deflection within the 100–250 msec interval was possibly partly caused by an N1 enhancement due to the clear and very rare stimulus deviation. The results of stage 2 sleep showed that the appearance of the MMN-like ERP deflection is related to the variability of the stimulus-elicited phasic EEG events. The MMN-like deflection only appeared in trials containing a K-complex as a response to the deviant stimulus. K-complexes occurred clearly more frequently after the de-

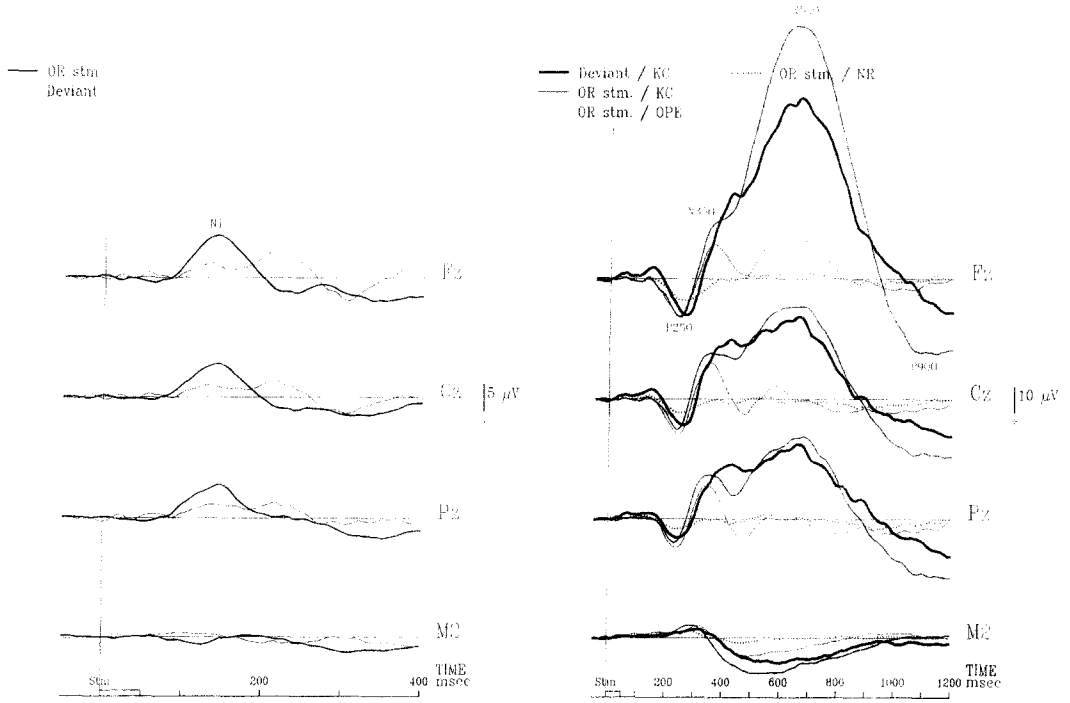

Fig. 10. Left: grand average ERPs to tones presented *without* standard tones (thick line, 595 trials) and to deviant tones presented *with* standard tones (thin line, 505 trials) during the reading situation. Right: grand average ERPs to tones presented *without* standard tones during the KC (the second thickest line, 543 trials), OPE (thin line, 528 trials), and NR trials (dotted line, 472 trials) of stage 2. The thickest line is grand average ERPs to deviant tones presented *with* standard tones during the KC trials of stage 2 (207 trials). The focus is on the waking MMN latency range (100–250 msec).

viant stimuli than after the standard stimuli, which proved that K-complexes following the deviant stimuli were really elicited by them.

The critical question is whether the observed negative deflection preceding the K-complex in the oddball paradigm experiment is a true MMN. The main finding supporting an affirmative answer was that the ERPs to the tone presented alone (OR paradigm) showed no MMN-like deflection during the KC trials. The N1 was also absent which is not a surprising finding in the light of previous studies (Anch 1977; Kevanishvili and Von Specht 1979; Paavilainen et al. 1987). This well-known attenuation of N1 during sleep has not, however, prevented researchers from studying MMN during sleep. The disappearance of the MMN-like deflection after the removal of the standard tones suggests that it is dependent on a memory trace created by the homogenous standard tones (in the same manner as the MMN). At least one can say that the MMN-like deflection acts uniformly with the waking MMN with regard to the removal of the standard tones (Sams et al. 1985a; Näätänen et al. 1989). In addition, the OR paradigm experiment also demonstrates that the MMN-like deflection cannot be considered as an early component of the K-complex which would otherwise has been a possible interpretation for the MMN-like deflection. The fact that both the MMN-like deflection and waking MMN had a frontal distribution also supports the idea that the MMN-like deflection is a genuine MMN.

A negative answer to the above-mentioned critical question is supported by the short onset and peak latencies of the MMN-like deflection during the KC trials. Especially, the onset latency of the MMN-like deflection is difficult to explain because at Fz it seems to start immediately after the deviant stimulus onset. The exact point of the onset is, however, difficult to determine. When one looks at Cz and Pz the actual response seems to start about 45 msec after the stimulus onset (Fig. 7) which is not an exceptional onset latency for the MMN. The short onset latency of MMN during sleep is also consistent with the study of Campbell et al. (1992). The second finding supporting the negative answer was that the MMN-like deflection did not show polarity reversal below the sylvian fissure as the MMN has been demonstrated to show (Alho et al. 1986; Paavilainen et al. 1991). However, this also held true for the reading situation. One explanation for this result may be a poor signal-to-noise ratio of our data at M2 which possibly prevented us from seeing the polarity reversal of these small-amplitude ERP shifts.

In conclusion, the present oddball and OR paradigm experiments mostly support the hypothesis that a true MMN can be found during stage 2 sleep. In order to observe this response it seems necessary to take into account the incessant fluctuation of microstates of sleep. However, replication is needed to confirm this new result because of the small number of subjects in the present study.

We wish to thank Professors Risto Näätänen, Peter Halász, Heikki Lang, Dennise Molfese and Juri Allik for their valuable comments on this study.

This study was supported by grants from the Academy of Finland.

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