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VEP, physiological and psychological circadian variations in humans*

G. Stolz[1], J. C. Aschoff[1], J. Born[2], and J. Aschoff[3]

[1] Abteilung Neurologie, [2] Angewandte Physiologie, Universität Ulm, D-7900 Ulm, Federal Republic of Germany
[3] Max-Planck-Institut für Verhaltensphysiologie, D-8131 Andechs, Federal Republic of Germany

Summary. Amplitudes and latencies of components of visual evoked potentials (VEPs) were analysed during the 24-h cycle in humans. Circadian variations of other physiological parameters (oral temperature, urine volume and urinary potassium excretion) and of psychomotor performance (grip strength, tapping rate, visual reaction time and performance on a letter cancellation test) were also assessed. Eight male volunteers (aged 20–34 years) were tested twice over a period of 30 h. Test sessions took place every 3 h (1100 hours, 1400 hours etc.). VEPs were elicited by checkerboard pattern reversals. Significant circadian variations in the VEPs were found for the latencies of the P100 and N140 components, which were longest between 2 a.m. and 5 a.m. (P100: 103 ms; N140: 138 ms) and shortest at about 5 p.m. (P100: 97 ms; N140: 130 ms). Pronounced circadian variations were also observed in physiological parameters and in psychomotor performance measures. Circadian variation of oral temperature was correlated with the 24-h profiles of most of the other variables. Time courses of VEP latencies and oral temperature were moderately negatively correlated. However, from the data presented it seems premature to conclude that there is a common pacemaker for the circadian variations of the different parameters investigated.

Key words: Circadian rhythm – VEP – Body temperature – Psychomotor performance

Introduction

It is well known that spontaneous EEG activity changes during the 24-h cycle, evidently associated with the sleep-wake rhythm. However, little is known of circadian variations of stimulus-evoked electrical activity of the brain.

Visual evoked potentials (VEPs) are widely used in revealing neurological disorders of the visual pathways, but few studies have been concerned with circadian variations in its components. Heninger et al. [14] investigated the influence of time of day on visual, auditory and somatosensory evoked potentials in two sessions, one in the morning and one in the evening. Additionally, 17-hydroxycorticoid levels in serum and urine were assessed. Significant changes were found for components of the VEPs and somatosensory evoked potentials, showing longer latencies and higher amplitudes in the morning sessions. Kerkhof et al. [17] demonstrated that morning-type subjects, as determined by a questionnaire, displayed higher amplitudes of auditory evoked potentials as well as VEPs in the morning than in the evening, whereas evening-type subjects showed higher amplitudes in the evening. Owing to the low sampling rate used in this experiment, no conclusions were drawn from changes in the latencies of VEP components.

Although these previous investigations suggest the VEP to be sensitive to circadian rhythms, none of the studies strictly tested the dependence of the VEP response on time of day during the 24-h cycle. In a recent study, however, Marshall and Donchin [18] demonstrated that latencies of auditory brainstem potentials distinctly vary with the time of day. Furthermore, the changes in latencies were found to be negatively correlated with body temperature.

The aim of the present study was to examine whether evoked potentials in the visual modality are subject to similar circadian variations. VEPs obtained by repeated reversals of a checkerboard pattern display a first prominent negative component at about 80 ms, a subsequent positive deflection about 100 ms, and a second negative one about 140 ms after stimulus onset. The generators of the N80 potential may be located in the primary visual cortex; the subsequent components P100 and N140 appear to reflect activity from associated cortical areas (areas 18 and 19) [12, 13, 22].

Moreover, our study was designed to compare 24-h time courses of VEP components with variations of other parameters for which stable circadian rhythms have been repeatedly demonstrated in previous studies [2, 3, 11]. Therefore, grip strength, tapping rate, simple reaction time and performance on a cancellation test were evaluated in addition to VEPs. Besides psychomotor performance, peripheral physiological parameters such as body temperature, urine volume and urine potassium were assessed.

Subjects and methods

Eight healthy male adults selected according to age (20–34 years) and handedness (right-handed) voluntarily participated in the study. The subjects' vision was normal ($n = 6$) or had been corrected to normal ($n = 2$). Funduscopy did not indicate any pathology. During the test period, the subjects lived in a clinical unit. Two days before and at the time of the experiment, sleeping time was between 11.30 p.m. and 7.30 a.m. Each subject was tested twice during a period of 30 h. Test sessions took place every 3 h (1100, 1400, 1700 hours etc.). The results from the first three test sessions were not subjected to data analysis, but served to habituate the subject to the experimental situation. To avoid affecting the potassium levels, subjects were not allowed to consume foodstuffs that contain a high potassium concentration (nuts, bananas, cocoa etc.).

Each test session lasted about 15–20 min so that nocturnal sleep interruption was minimal. Within test sessions the pro-

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Offprint requests to: G. Stolz, Abteilung Neurologie, Universität Ulm, Steinhövelstrasse 9, D-7900 Ulm, Federal Republic of Germany

cedure was as follows: (1) collection of urine; (2) measurement of oral temperature (Temp 1); (3) recording of an averaged VEP response (VEP 1); (4) determination of psychomotor performance (first grip strength, then tapping rate, simple visual reaction time and performance in a letter cancellation test); (5) recording of a second VEP response (VEP 2); and (6) measurement of oral temperature (Temp 2).

VEPs were recorded in an electrically shielded room with constant background illumination (100 lux, at the subject's eye). The subjects sat facing a video monitor located at a distance of 130 cm from their eyes. They fixated their eyes on a dot which was always visible in the centre of the monitor. Black and white checkerboard patterns (square size 2 cm^2) were projected on the screen. The square sizes subtended an angle of 55′, the total screen an angle of 22° at the subject's eye. Pattern reversals were produced at a rate of 2 Hz. VEPs were recorded by non-polarizable needle electrodes (Beckmann) attached at Oz and Cz (right earlobe: ground). Recordings were filtered at 1.6 Hz (high pass) and at 300 Hz (low pass). The sampling rate was 2.5 kHz. The sampling epoch started with stimulus-onset and lasted for 200 ms. Trials in which the voltage exceeded 80 μV in the sampling epoch were automatically rejected. VEPs were averaged from EEG responses to a series of 256 pattern reversals.

Maximal grip strength for the right and left hand was determined by a Bettendorf dynamometer. The mean strength for six short trials on each hand was determined. To assess the tapping rate, the subject was required to press a key repeatedly as quickly as possible with the middle finger of the right hand. Trials started with a pretest period of 5 s, followed by a test period of 10 s. The visual reaction time task required the subject to press a button as soon as a light next to the button went on. Light stimuli were presented with irregular interstimulus intervals between 1.0 and 5.5 s. The median reaction time was computed from ten subsequent trials. In the letter cancellation test (Test d2, [5]) subjects had to cross specially marked letters within 14 lines, as fast and as accurately as possible within 280 s. The number of correctly identified letters minus the number of incorrectly crossed letters was used as the performance score.

Data reduction and analysis. VEP components were determined visually. Latencies for the N80, P100, and N140 component and peak-to-peak amplitudes for the N80-P100 and the P100-N140 difference were measured.

Prior to statistical analysis, data for all parameters were transformed to eliminate interindividual differences, because interindividual variance typically exceeds the variance determined by circadian variations. Thus, statistical analysis was based on scores indicating the deviation (in per cent) from the individual 24-h mean of each subject and test session.

For each parameter circadian variations were statistically assessed by Friedmann's test. Changes between VEPs and temperature measures within one test session (VEP 1 vs VEP 2; Temp 1 vs Temp 2) were evaluated by analysis of variance. The stability of circadian variations during the two 24-h experimental periods for each subject was assessed by product-moment correlation. Principal component analysis was used to reveal clusters of correlated 24-h time courses among the parameters investigated.

Results

VEPs from all subjects showed distinct variations during the 24-h cycle. The VEP responses from a single subject during two 24-h cycles are illustrated in Fig. 1.

Mean circadian variations (averaged across all subjects and both 24-h periods) for latencies of the N80, P100, and N140 components are shown separately in Fig. 2 for VEP 1 and VEP 2 of each test session. Circadian variations of latencies seemed the more distinct the later the component occurred. The most prominent and significant circadian variations were seen for the P100 and N140 latencies. (Refer to Table 1 for the statistical significance of circadian variations for all parameters.) Variations in N80 latency were less clear. For all components, the longest mean latencies were found in the early morning hours between 2 a.m. and 5 a.m. (averaged across VEP 1 and VEP 2; N80: 72 ms; P100: 103 ms; N140: 138 ms); the shortest latencies were measured at 5 p.m. (P100: 97 ms; N140: 130 ms). For the N80 no definite minimum latency

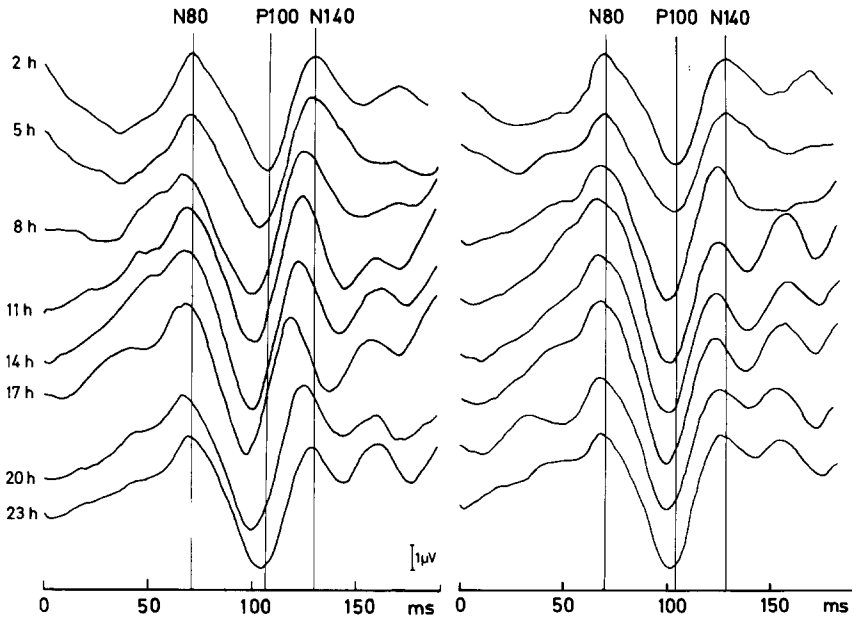

Fig. 1. Variations in visual evoked potential (VEP) responses over two 24-h cycles recorded from a single subject

Table 1. Means, standard errors (SE), and the mean range of fluctuations within subjects across the 24-h time course (range) for all parameters. Statistical significance (Friedmann tests: χ^2, and *P*-level of significance) of 24-h variations is also indicated. Right column indicates correlations (*r*) between two 24-h time courses measured in each subject. NS = not significant

Parameter	Mean	SE	Range	χ^2	*P*	*r*
VEP 1						
N80 latency (ms)	70.1	1.7	2.2	15.9	0.05	0.25
P100 latency (ms)	100.4	2.5	6.5	50.4	0.01	0.52
N140 latency (ms)	134.2	7.4	8.9	30.6	0.01	0.33
N80–P100 amplitude (μV)	3.7	0.53	1.3	29.4	0.01	0.45
P100–N140 amplitude (μV)	3.5	0.42	0.6	8.0	NS	0.45
VEP 2						
N80 latency (ms)	70.3	1.9	3.7	24.6	0.01	0.38
P100 latency (ms)	98.8	1.8	5.1	48.4	0.01	0.42
N140 latency (ms)	133.5	6.9	8.5	30.6	0.01	0.41
N80–P100 amplitude (μV)	3.5	0.53	0.6	19.1	0.01	0.02
P100–N140 amplitude (μV)	3.7	0.45	0.7	9.2	0.10	0.30
Psychomotor performance						
Grip strength (N)	468	33	98	36.7	0.01	0.60
Tapping rate (taps/10 s)	51.5	2.3	7.0	24.0	0.01	0.14
Reaction time (ms)	202.5	7.0	18.0	25.0	0.01	0.51
Cancellation test (score)	593	20.1	90	44.2	0.01	0.68
Peripheral physiological parameters						
Temperature 1 (°C)	36.1	0.14	0.7	37.5	0.01	0.64
Temperature 2 (°C)	36.3	0.14	0.9	48.7	0.01	0.82
Urine volume (ml)	213	46.6	169	19.2	0.01	0.44
Potassium concentration (mg)	369	75.6	393	38.2	0.01	0.60

could be determined. Thus, the ranges of variation in mean latencies during the day were about 3 ms for the N80, 6 ms for the P100, and 8 ms for the N140 (Table 1).

A comparison of VEPs from the beginning (VEP 1) and the end of each session (VEP 2) indicated that latencies (in particular of the P100 and N140) were longer at the beginning than the end of a test session, with this effect being more pronounced in nocturnal test sessions [Time of day × VEP interaction, P100: $F(1,7) = 7.94$; N140: $F(1,7) = 23.66$, $P < 0.05$].

Circadian variation in amplitudes of VEP components was generally less prominent than for latencies (Fig. 3). Only circadian variations in N80-P100 amplitude were statistically significant (Table 1). The lowest N80-P100 amplitudes consistently occurred at 5 a.m. (3.0 μV) and these amplitudes reached a maximum at 11 p.m. (3.9 μV). Variations during the 24-h cycle of P100-N140 amplitudes did not reach statistical significance.

N80-P100 amplitudes were in general larger at the beginning of a test session than at the end [$F(1,7) = 5.61$, $P < 0.05$]. P100-N140 amplitudes, by contrast, were higher at the end than at the beginning of a session [$F(1,7) = 10.9$, $P < 0.05$].

For psychomotor performance measures, i.e. grip strength, tapping, simple reaction time and letter cancellation, consistent variations during the 24-h cycle could be confirmed by Friedmann's tests (Table 1, Fig. 4). Psychomotor performance improved during the day until the early evening hours (5–8 p.m.) and then declined to reach its minimum in the early morning hours (2–5 a.m.). Time courses of grip strength were identical for the right and left hand with maximum strength at 8 p.m. (512 N) and minimum strength at 5 a.m. (414 N). Tapping speed was fastest at 2 p.m. (54 taps/10 s) and slowest at 2 a.m. (47 taps/10 s). Simple visual reaction time was shortest

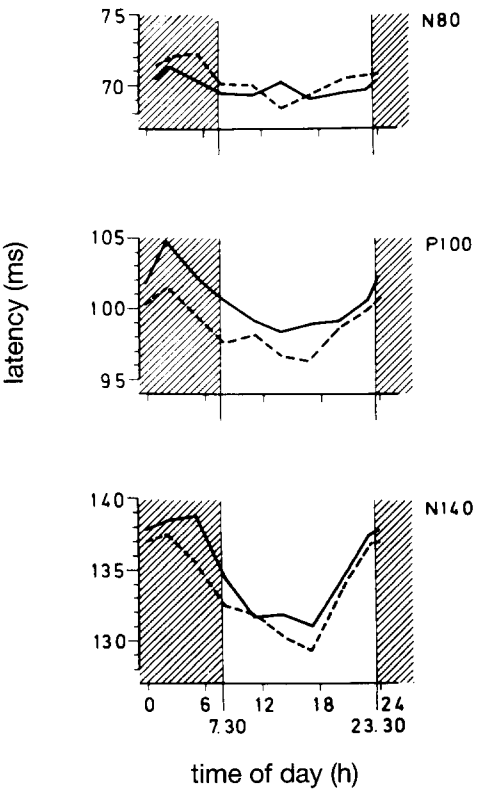

Fig. 2. Mean latencies of the N80, P100, and N140 components of VEPs during the 24-h cycle. *Solid line:* VEP 1 recorded in the beginning of each test session; *dashed line:* VEP 2 recorded at the end of each test session. *Hatched areas* indicate sleeping time. The data represent means for all subjects and both 24-h periods of each subject

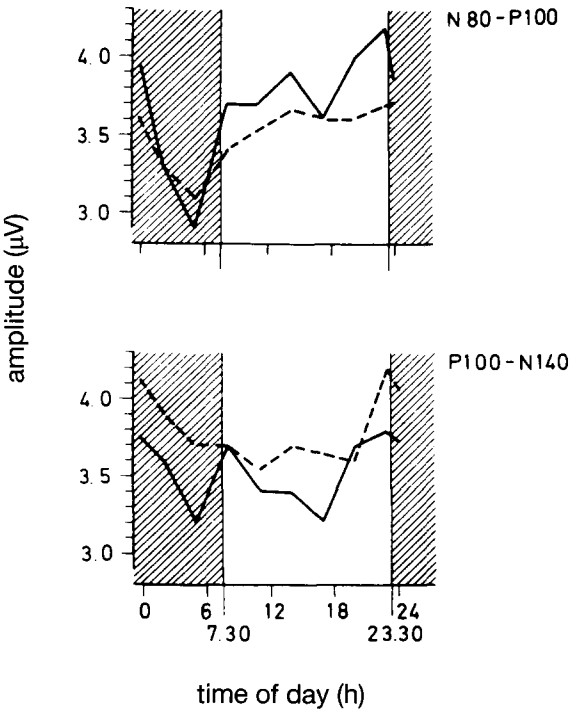

Fig. 3. Circadian variations in mean amplitudes (averaged across subjects and both 24-h cycles) of the N80-P100 and P100-N140 components of VEPs. *Solid line:* VEP 1; *dashed line:* VEP 2

at 5 p.m. (194 ms) and longest at 5 a.m. (212 ms). Highest performance scores in the letter cancellation test were obtained at 5 p.m. (632 letters) while at 2 a.m. performance was worst (542 letters).

As expected, fluctuations of oral temperature displayed a highly regular 24-h rhythm (Fig. 4, Table 1). Highest mean temperatures were recorded at 2 p.m. for both Temp 1 and Temp 2, with means of 36.4°C and 36.7°C respectively. The mean temperature was lowest for Temp 1 at 5 a.m. (35.7°C) and for Temp 2 at 2 a.m. (35.8°C). Furthermore, Temp 2 was significantly higher than Temp 1 [$F(1,7) = 68.74$, $P < 0.01$], and this difference was greater during the day than at night [Time of day × Temp interaction, $F(1,7) = 9.4$, $P < 0.05$].

For potassium, mean urine concentrations were highest at 5 p.m. (594 mg) and lowest at 8 a.m. (201 mg). The largest mean urine volumes were collected at 2 p.m. (297 ml) and the smallest at 5 a.m. (128 ml) (Table 1, Fig. 4).

Each subject was run on two 24-h experimental periods to assess the stability of circadian variations. The correlations between the two 24-h time courses are summarized for all parameters in Table 1 (right column). The retest reliabilities of circadian variations in peripheral physiological parameters (in particular temperature) and in psychomotor performance were of considerable magnitude. The latencies and amplitudes of VEP components appeared to be somewhat less stable. Nevertheless, high retest correlations were observed for the time courses of P100 and N140 latencies, which also displayed the most distinct circadian variations of all VEP parameters. However, these coefficients were still substantially lower than those obtained from correlating 24-h time courses of P100 and N140 latencies recorded at the beginning and at the end of one test session (VEP 1 vs VEP 2), which were $r = 0.84$ for the P100 and $r = 0.83$ for the N140 latency.

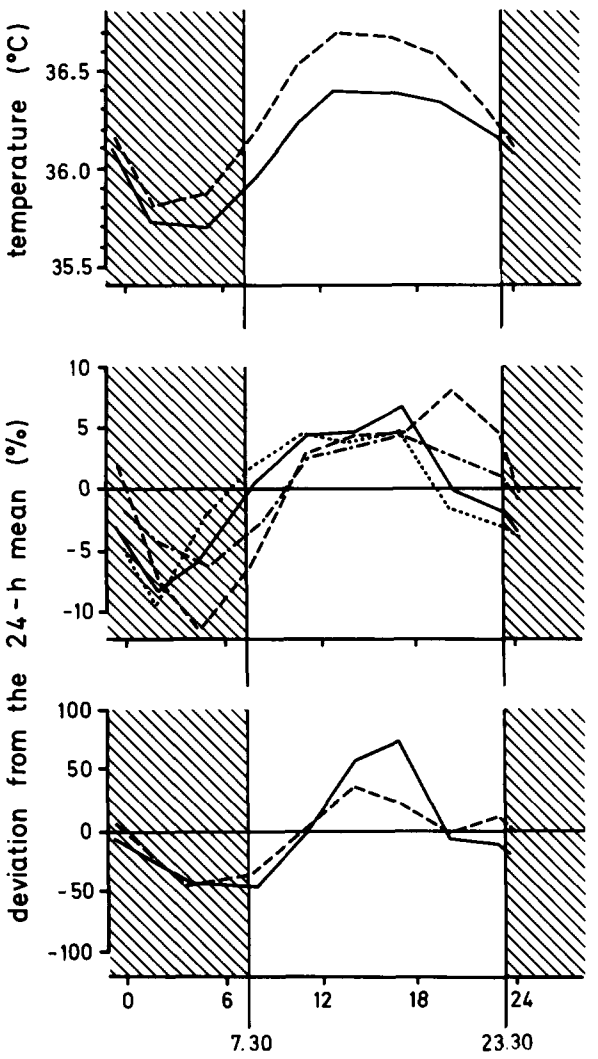

Fig. 4. *Top panel:* Mean circadian variation (all subjects and both 24-h test periods) of oral temperature recorded at the beginning (Temp 1, *solid line*) and at the end (Temp 2, *dashed line*) of each test session. *Middle panel:* Mean circadian variations in grip strength (right hand), tapping rate, the reciprocal of visual reaction times, and performance on a letter cancellation test during the 24-h cycle (—— letter cancellation; ----- grip strength; ..... tapping rate; —·—·— reaction time). *Bottom panel:* Mean circadian variations of urine volume *(solid line)* and of urinary potassium concentration *(dashed line)*. For middle and bottom panel data represent the percentage deviation from the individual 24-h mean. *Hatched areas* indicate sleeping time

Principal component analysis should reveal clusters of parameters with fluctuations correlated over the 24-h cycle and was applied to the correlation matrix derived from z-transformed data (to eliminate interindividual variance) for all parameters. The component structure was subsequently varimax rotated. Nine principal components were extracted, explaining 70% of the total variance. Table 2 summarizes loadings for components marked by at least one variable with a loading greater than 0.5. Figure 5 shows the time courses (estimated from component scores) for components 1, 2, and 4, representing different but distinct circadian rhythms. Component 1 appeared to be determined by synchronized variations during the 24-h cycle of oral temperature and of performance on most of the psychomotor tasks. In addition, N80 and P100 latency showed considerable negative loadings on that compo-

Table 2. Matrix of component loadings derived from principal component analysis. Only loadings greater than 0.23 are presented. Since loadings for components 3, 8 and 9 never exceeded 0.5, these components were omitted

Component 1	
Temperature 1	0.79
Temperature 2	0.70
Grip strength	0.74
Letter cancellation	0.65
Reaction time	−0.59
VEP 2: N80 latency	−0.48
VEP 1: P100 latency	−0.39
VEP 2: P100 latency	−0.39
VEP 1: N80–P100 amplitude	0.35
VEP 2: N80–P100 amplitude	0.23
Component 2	
VEP 1: N140 latency	0.58
VEP 2: N140 latency	0.59
VEP 1: P100 latency	0.43
VEP 2: P100 latency	0.50
Temperature 2	−0.35
Component 4	
Potassium concentration	0.78
Urine volume	0.58
Reaction time	−0.44
Component 5	
VEP 1: P100–N140 amplitude	0.59
VEP 2: P100–N140 amplitude	0.31
VEP 1: N80–P100 amplitude	0.53
Component 7	
VEP 1: N80 latency	0.57
VEP 2: N80 latency	0.42

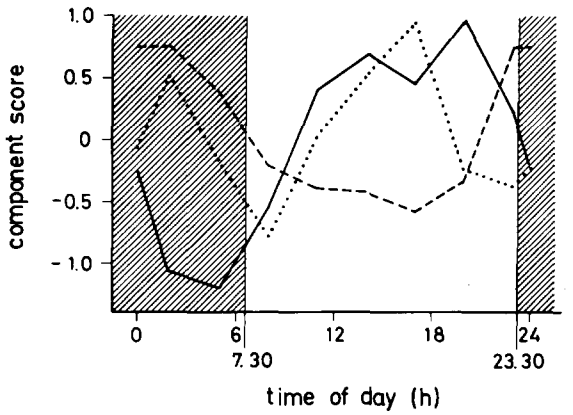

Fig. 5. Mean time courses estimated from component scores for components 1 (——), 2 (– – –), and 4 (·····) derived from principal component analysis. These components appeared to reflect specific circadian variations in oral temperature and psychomotor performance (component 1), VEP latencies (component 2), and potassium excretion and urine volume (component 4)

nent. Component 2 seems to reflect common variations during the 24-h cycle of P100 and N140 latency. Component 4 represents a time course over the 24-h cycle unique to potassium excretion and urine volume. The remaining components did not appear to reflect circadian variations of the different parameters. Cross-correlation functions determined from component scores did not indicate a timed relationship between the 24-h time courses represented by the different components.

Discussion

The results demonstrate that VEPs recorded from the human scalp are subject to systematic circadian variations. P100 and N140 latencies were substantially longer at night than during the day. The amplitudes of these VEP components increased in the evening between 6 p.m. and 11 p.m. and subsequently diminished in the course of the night. However, circadian variations of amplitudes were less prominent than those in latencies. Furthermore, our results suggest that circadian fluctuations of VEP components cannot be sufficiently described by investigations based on only two sessions per day, one in the morning and one in the evening [6, 7, 9, 14, 17]. The present data show most pronounced changes particularly of VEP latencies when recordings are compared from test sessions in the middle of the day and in the middle of the night.

The finding that circadian variations in VEP latencies were more distinct the later the component occurred suggests a neurogenic origin within the central visual pathways. Concomitant changes at the receptor level may have contributed to the 24-h fluctuations in latencies; the electroretinogram, for example, has been shown to depend on plasma glucocorticoid levels [24], which are known to display distinct circadian rhythmicity. Variations in intraocular pressure, on the other hand, may not account for the observed changes in VEPs. Given a normal autoregulation, these variations are small and have not been found to affect VEP latencies consistently (see, for example, [21, 23]). Slight but unsystematic eye movements during the subject's fixation, too, may not explain the regular circadian changes in VEP responses. Thus, supported by similar results on VEPs in rabbits [4], we assume that the present findings of shorter latencies (and higher amplitudes) of VEP components during the day than at night reflect an increased sensitivity of the visual system in humans during the period of high visual activity.

That circadian variations were by far more distinct and replicable for latencies than for amplitudes may have been due to the fact that amplitudes are determined by overlapping potential fields from several generators at a given moment. Thus, their multifactorial determination may have blurred a circadian periodicity. The latency of a component is less affected by overlapping potential fields. That P100 and N140 components showed more distinct circadian variations than N80 may hint at differences in the sensitivity of component generators to circadian pacemakers. Changes in N80 latency may be primarily related to the specific processing of sensory stimuli in the primary visual cortex. Further processing of visual stimuli in adjacent areas generating the P100 and N140 components may be more sensitive to more generalized influences subjecting activity of the visual system to a distinct circadian rhythm.

The VEPs measured at the beginning (VEP 1) and at the end of each test session (VEP 2) showed similar time courses. However, latencies were generally shorter for VEP 2 than for VEP 1. For P100 and N140 this within-session effect was even more prominent at night, when VEP 1 latencies were longest. Given that during sessions at night light adaptation is completed within a few seconds, these effects cannot be due to

adaptation because subjects were adjusted to the luminance conditions for about 5 min prior to the recording of VEP 1 (see also [10]). Moreover, to diminish adaptation processes in both directions, background luminance was held constant at an intermediate level. Thus, the decrease in latency of P100 and N140 components in VEP 2 compared with VEP 1 more likely reflects an increase in cortical activation across the session. The more pronounced effect at night compared with the day may have damped down circadian variations in VEP 2. In the same way, the arousing effect of waking the subject at night prior to sessions may have reduced circadian oscillations.

Pronounced circadian variations were also observed for performance on all psychomotor tasks. Performance generally peaked in the afternoon and was poorest at night, as has been described by others [2, 3, 8, 15, 16]. The oscillations proved to be rather stable with retest correlations greater than 0.5. As the circadian variations were similar for all measures of psychomotor performance (i.e. on principal component analysis they were described by a single component) these tests may reflect changes in the general level of cortical activation, in addition to specific functions.

Physiological parameters such as oral temperature, urine volume and potassium excretion also indicated prominent and stable circadian fluctuations, which is in accord with results from previous studies [11, 15, 16, 19, 20]. The mean time courses of peripheral physiological parameters appeared to be similar; however, they were not correlated within subjects, because principal component analysis resulted in different components describing the changes of the three parameters during the 24-h cycle. In addition to its circadian variation, body temperature (like VEP latencies) indicated an increase in activation during each test session with higher temperatures at the end than at the beginning of a session (Fig. 4) (see also [1]).

The time course of oral temperature was correlated with the time courses of most of the other variables, i.e. positively with psychomotor performance and negatively with the time courses of VEP latencies. This may suggest that the time-dependent changes in oral temperature best represent the influence of a superior pacemaker that synchronizes circadian physiological rhythms and which may be localized in the nucleus suprachiasmaticus [20]. Nevertheless, the correlation coefficients and results from principal component analysis (Fig. 5) also indicate that circadian variations of the various parameters – particularly of temperature and VEP latencies – were to a great extent independent of rather than dependent on each other.

In summary, our study provides evidence for prominent circadian rhythms in latencies of the P100 and N140 components of the human VEP. Circadian variations were also found for peripheral physiological measures such as body temperature, as well as for psychomotor performance. The observed circadian changes in VEPs complement previous results by Marshall and Donchin [18], who demonstrated similar variations for the auditory evoked potential. The results suggest that sensory neuronal processing is controlled by specific circadian influences.

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