

Auditory Evoked Potentials and Impairments to Psychomotor Activity Evoked by Falling Asleep

V. B. Dorokhov, Yu. S. Verbitskaya, and T. P. Lavrova

UDC 612.821.7

Translated from Zhurnal Vysshei Nervnoi Deyatel'nosti imeni I. P. Pavlova, Vol. 59, No. 2, pp. 133–143, March–April, 2009. Original article submitted January 28, 2008. Accepted June 9, 2008.

Sounds provide the most suitable stimuli for studies of information processes occurring in the brain during falling asleep and at different stages of sleep. The widely used analysis of evoked potentials averaged for groups of subjects has a number of disadvantages associated with their individual variability. Thus, in the present study, measures of the individual components of auditory evoked potentials were determined and selectively summed for individual subjects, with subsequent analysis by group. The aim of the present work was to identify measures of auditory evoked potentials providing quantitative assessment of the dynamics of the brain's functional state during the appearance of errors in activity associated with decreases in the level of waking and falling asleep. A monotonous psychomotor test was performed in the lying position with the eyes closed; this consisted of two alternating parts: the first was counting auditory stimuli from 1 to 10 with simultaneous pressing of a button, and the second was counting stimuli from 1 to 5 silently without pressing the button, and so on. Computer-generated sound stimuli (duration 50 msec, envelope filling frequency 1000 Hz, intensity 60 dB) were presented binaurally with interstimulus intervals of 2.4–2.7 sec. A total of 41 subjects took part (both genders, mean age 25 years), of which only 23 fell asleep; data for 14 subjects with sufficient episodes of falling asleep were analyzed. Comparison of measures of auditory evoked potentials (the latencies and amplitudes of the N1, P2, N2, and P3 components) during correct and erroneous psychomotor test trials showed that decreases in the level of consciousness elicited significant increases in the amplitudes of the components of the vertex N1-P2-N2 complex in series without button pressing. The greatest changes in auditory evoked potentials in both series were seen in the N2 component, with latency 330–360 msec, which has a common origin with the EEG theta rhythm and is characteristic of the first stage of sleep.

KEY WORDS: sleep, falling asleep, auditory evoked potentials, performance errors.

The formation of critical levels of sleepiness in humans performing monotonous work activities is the cause of numerous transportation and factory accidents. The appearance of conditions for this is currently regarded as associated with the development of drowsiness, a process supporting the transition from waking to sleep. Some authors believe [15, 16, 22] that drowsiness alters the state of consciousness. The concept of “consciousness” [15] helps to provide a theoretical basis for the relationship and between selective attention and sleep and the transition

between them, as well as between subjective and objective factors inducing impairments to activity during falling asleep. The presence of selective attention provides the subject with conscious perception only of significant stimuli and, at the same time, unconsciously inhibits responses to insignificant stimuli.

The most suitable external stimuli are sound stimuli delivered via headphones, such that constant intensity was maintained independently of the subject's head position, which is a necessary condition for analysis of changes in the characteristics of auditory evoked potentials (EP) during falling asleep and increases in the depth of sleep.

Intermediate- and long-latency auditory EP in waking consist of the following components: P1, with a latency of

Institute of Higher Nervous Activity and Neurophysiology,
Russian Academy of Sciences, Moscow;
e-mail: vbdorokhov@mail.ru.

about 50 msec from the onset of the sound stimulus, N1, with a latency of about 100 msec, P2, with a latency of 180–200 msec, and P3, with a latency of 300–400 msec [9, 17]. The negative N350 and N550 components (latent periods of about 350 and 550 msec) arising as the depth of sleep increases, along with the positive P2 (latency about 200 msec) and P900 (latency about 900 msec) components, have been suggested by several authors [5, 7, 25] to be linked with increases in information processing time during sleep and to serve as markers for the onset of the deeper stages of sleep.

However, existing methods of analyzing EP [1, 4] have a number of limitations associated with the averaging of numerous single EP for identification of low-amplitude components from EEG recordings containing spontaneous activity. Correction of changes in brain information processes during falling asleep with corresponding impairments to psychomotor activity in subjects requires monitoring of sleep onset states. A number of reports [13, 20] have demonstrated that many subjects can respond to external stimuli during the first stage of sleep and even partially at the onset of the second. However, these data were criticized on the basis that existing methods of identifying sleep stages require visual analysis of 30-sec polygraph traces. The transition from waking to sleep is accompanied by alternating transient (shorter than 30 sec) periods of sleep and waking.

The transition from waking to sleep, when subjects are still seen to respond to external stimuli, is quite brief, so an experimental protocol was developed with falling asleep and repeated waking [20]. This approach was quite laborious. Thus, studies reported in [10] recorded a total of 24 repeated wakings during three different nights.

These experimental difficulties were overcome using the psychomotor test developed by ourselves [2], which allows the subject to reach the state of drowsiness in 10–15 min; in this state, impairments to activity induced by periodic falling asleep are seen. When this test was used, subjects could, over 40 min, show quite prolonged periods with decreased levels of consciousness and periods with erroneous test performance during which the number of recorded EP was sufficient for quantitative analysis of averaged EP.

The monotonous nature of the test and the limited sensory afferentation (eyes closed and the subject being in the lying position) accelerates the appearance of errors in activity; on the other hand, the discrete nature of the test allows analysis of data, along with self-monitoring and subjective perception of errors during performance at different stages of development of the state of drowsiness. The selection of counting to ten in series with joystick button pressing and counting to five without button pressing was based on the decimal counting system natural to humans, using the number of fingers, which simplifies the instructions given to the subject.

A number of authors have suggested [14, 16] that drowsiness is a transitional state from waking to sleep and,

on the other hand, that drowsiness is a separate state distinct from waking and sleep and consists of up to seven sequential stages [25]. In terms of electrophysiological measures, this transitional state corresponds almost completely to the first stage of sleep as defined by the usual criteria [24]. Some authors [6, 13, 20, 21] have used EEG criteria to discriminate the first stage of sleep into substages alpha-1 and theta-1, differing in terms of the level of the EEG alpha and theta rhythms.

In recent studies of changes in information processes during falling asleep, many authors [6, 9, 13, 23] have used the oddball paradigm, when two different stimuli are presented with different probabilities and the subject has to press a button in response to the deviant (significant) stimuli. In these experiments, particular attention is paid to changes in the P3 (P300) component at substages alpha-1 and theta-1 of the first stage of sleep [8].

Prolongation of the latent period (LP) when increases in reaction times (RT) occur is explained in terms of slowing of signal processing.

The psychomotor test used in our experiments corresponds better to real monotonous activity than the oddball paradigm described above. On performance of this test, the significance of a given stimulus in two alternating series with and without button pressing is determined by the instructions given.

The aims of the present work were to identify measures of auditory evoked potentials allowing quantitative analysis of the dynamics of the brain's functional state on appearance of errors in activity associated with the decreased level of consciousness and falling asleep and to assess the contribution of the mechanisms regulating sleep and waking to the appearance of the state of monotony.

METHODS

Subjects. A total of 41 healthy subjects (both genders, mean age 25 years) took part in the present study; errors induced by decreases in the level of consciousness were observed in 23 subjects. Data from 14 subjects with sufficient numbers of episodes of falling asleep and with a clear alpha rhythm were analyzed. Voluntary consent to take part in the experiments was obtained from all subjects.

Recording of electrophysiological measures. EEG, auditory evoked potentials (AEP), and other polygraph measures were recorded using a Leonardo (MKE Medizintechnik GmbH, Germany) multichannel electroencephalograph with polygraph channels. The EEG was recorded from eight EEG electrodes positioned in accordance with the international 10–20% system (F3, F4, C3, C4, P3, P4, O1, O2). The electrooculogram (EOG) was recorded from two electrodes: one located 2 cm above the lateral angle of the orbital bone of the right eye, and the other 2 cm below the lateral angle of the left eye. The reference electrode con-

sisted of combined mastoid electrodes A1 and A2. Recordings were made using gold-plated cup electrodes and adhesive electrode gel from Grass (USA). Apart from the EEG and EOG, polygraphic measures were also recorded: the electromyogram, the electrocardiogram, and respiration and pulseoximetry traces. The present study included analysis of EP only from two EEG recording points, i.e., F3 and C3, while polygraph data were used for visual analysis of sleep stages. The signal digitization frequency was 200 Hz, and a 12-bit analog-to-digital converter was used.

Sound stimulation was with short computer-generated sound tone bursts presented binaurally via headphones. Sound stimulation parameters were: tone duration 50 msec, filling frequency 1000 Hz, intensity 60 dB, and interstimulus interval varying over the range 2.4–2.7 sec.

Experimental protocol. Experiments were performed in the evening, i.e., from 17:00 to 20:00. Subjects were placed in a dark, soundproofed room and were in the lying position with the eyes closed.

Subjects performed a psychomotor test which consisted of two continuous sequences of actions: initially counting stimuli silently from 1 to 10 with simultaneous button pressing and then counting stimuli from 1 to 5 without button pressing, and so on. Sound stimuli were presented with intervals of 2.4–2.7 sec. Test duration was 40 min. The correctness of test performance was assessed using behavioral and physiological measures: the correct numbers of stimuli in series were 10 with button pressing and five without button pressing. A necessary condition for identification of activity impairments as test performance errors consisted of deviations from instructions by one stimulus or more. An additional condition for error identification was the appearance at this time of EEG and EOG drowsiness patterns: substitution of EEG alpha activity by a rhythm in the theta range and the appearance of slow, high-amplitude oscillations, corresponding to slow horizontal eye movements, on the EOG.

Statistical analysis. EP configurations were analyzed using two leads, i.e., F3 and C3, which were the most informative for studies of the late components of EP in impairments to activity induced by falling asleep. EEG traces were filtered using digital filters to select the frequency range 0.5–30 Hz prior to analysis. Statistical analysis was restricted to data from those subjects showing errors induced by falling asleep and corresponding to the criteria given above and lacking recording artifacts. EEG trace segments with individual EP of total duration 4 sec were then selected, including 1 sec before the stimulus and 3 sec after the stimulus, and these segments were then used for selective averaging of EP in different groups using the criteria described below.

Four groups were defined for selective averaging of EP:

BC (button, correct) – with button pressing, correct (sequences of 10 stimuli with pressing);

NBC (no button, correct) – without button pressing, correct (sequences of five stimuli without pressing);

BE (button, erroneous) – with button pressing, erroneous (sequences of N stimuli with button pressing, where the number of presses differed from 10 by one press or more);

NBE (no button, erroneous) – without joystick button pressing, erroneous (sequences of N stimuli without button pressing, where the number of stimuli without pressing differed from five by one or more stimuli).

Analysis of mean EP parameters and averaging for individual subjects. Selection of subjects for analysis was based on the minimum number of stimuli in series with errors with and without button pressing; the number needed to be above 50 stimuli for each of the erroneous series. Initially, EP in these groups were averaged separately for each subject. The amplitudes and latent periods of the components of the averaged EP were then determined for each subject. The latent period (LP) of the P1 (70–80 msec), N1 (140–150 msec), P2 (220–250 msec), N2 (330–360 msec), and P3 (460–550 msec) peaks were measured from the start of the stimulus, while peak amplitudes were measured “from peak to peak” as the absolute values of the differences in amplitudes from the peak of the preceding peak: $N1 = N1 - P1$, $P2 = P2 - N1$, $N2 = N2 - P2$, and $P3 = P3 - N2$. The amplitude of component P1 was measured relative to the preceding negative peak (N0). The mean latencies and amplitudes of EP components of each group were compared using the statistics program Statistica 6.0 (Microsoft). To confirm the applicability of standard parametric statistical tests, normal distributions were verified using the Lilliefors (L) and Shapiro–Wilk (S–W) tests. These tests are in principle valid for sets of greater than 30. Nonetheless, using them in parallel to each of the study sets showed that in most cases (more than 90%), set distributions could be successfully approximated to the normal distribution.

The contributions of each of the factors (lead \times task \times performance quality) on peak amplitudes were assessed by three-factor analysis of variance. Mean displacements were assessed using Student’s test for paired comparisons and the nonparametric Wilcoxon rank test for paired comparisons. As a rule, the results obtained using these two tests agreed qualitatively.

RESULTS

The following designations will be used: lead F3 is identified as F and lead C3 as C. Figure 1 shows histograms of the numbers of erroneous series for series with different numbers of stimuli with and without pressing. Series with pressing contained more erroneous series (BE) with decreased numbers of stimuli in series, i.e. fewer than 10 (to the left of 0), while without pressing there were more erroneous series (NBE) with increased numbers of stimuli, i.e., more than five (to the right of 0). Column height in the null bin corresponds to the number of correctly performed series (BC and NBC).

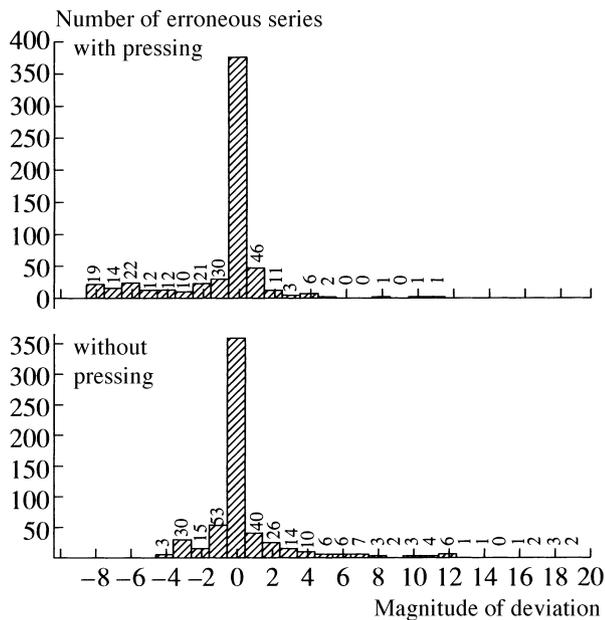


Fig. 1. Histogram showing numbers of correct and erroneous series for all subjects ($N = 14$) of the four groups of the psychomotor test. The horizontal axis shows the magnitude of deviations from the correct number of stimuli in series. The vertical axis shows numbers of series. Above: histogram of series with pressing. The null bin shows the number of correctly performed series (BC) with $n = 10$ stimuli; numbers to the left and right of the null bin are the numbers of series with errors (BE), with $n < 10$ to the left and $n > 10$ to the right. Below: histogram of series without pressing. The null bin shows the number of correctly performed series (NBC) with $n = 5$ stimuli; numbers to the left and right of the null bin are the numbers of series with errors (NBE), with $n < 5$ to the left and $n > 5$ to the right.

Results were analyzed from a total of 14 subjects who satisfied the criterion of a minimum number of erroneous series (more than 50) used to select data for analysis. Figure 2 shows superimposed AEP for leads F3 and C3 averaged for each subject and selectively chosen for each of the four groups of psychomotor test results.

Statistical analysis of the amplitudes and latent periods of AEP components averaged for individual subjects yielded data on changes in the peaks of individual AEP components in the groups studied. The results of this analysis are presented in the Tables with mean amplitudes (Table 1) and LP (Table 2) and on plots (Figs. 3 and 4) constructed on the basis of these Tables. In Fig. 3, the data from Table 1 are presented graphically as changes in the mean amplitudes of AEP components.

Analysis of variance of mean P1 amplitude for individual subjects demonstrated a significant increase in this component in lead C3 when errors appeared in series with button pressing. In series without button pressing, the appearance of errors, conversely, was accompanied by a decrease in P1 amplitude, though this reduction did not reach significance.

Analysis of variance of mean N1 amplitude for individual subjects did not identify any significant differences in any lead in either series.

Analysis of variance of mean P2 amplitude demonstrated significant increases in P2 amplitude in lead F3 when errors appeared in series without pressing (NB series), with an almost identical increase, though not significant, in lead C3. In series with errors without button pressing, as compared with series with pressing, there was an insignificant increase in P2 amplitude.

Analysis of variance of the N2 peak amplitude showed that all three factors (lead, type of task, quality of performance) were significantly reflected in the mean amplitude of the N2 peak. The amplitude of the N2 peak in lead C3 was, on average, greater than that in lead F3. In erroneous performance of tasks, the amplitude of the N2 peak increased, significantly in series without pressing and almost significantly in lead F3 in series with pressing. Finally, in series without pressing, the mean amplitude of the N2 peak was greater than that in series with pressing, significantly in lead F3 and significantly in lead C3 only when errors appeared.

Analysis of variance of the amplitude of the P3 peak, unlike the very informative N2 peak amplitude, demonstrated low sensitivity (though the mean peak amplitude in lead F3 in correct performances of both tasks was nonetheless almost significantly lower than that in lead C3). The main influence on the mean amplitude of the P3 peak was the quality of performance, especially in series without pressing – when errors were made, the amplitude of this peak in lead C3 increased significantly. As this was not observed for series without pressing, the interaction “task \times quality of work” was significant. It should be noted that although the amplitude of this peak differed on performance of different tasks without reaching the level of significance, the amplitude of the peak in lead C3 on performance of tasks with errors in series without pressing, was, on average, greater than that in series with pressing.

Changes in the latent periods of EP components when errors appeared in both series are shown in Table 2 and the plot in Fig. 4, based on this Table. “Smearing” of measures was seen as LP increased – the EP components further from the onset of stimulation became more different from each other in different leads, experimental conditions, and quality of task performance not only in terms of mean values, but also in terms of the error of the mean (SE or m).

It follows from Table 2 that significant changes in LP were only seen for the P3 peak. The LP of this component in lead F3 in series with button pressing increased significantly when errors appeared. The opposite changes were seen in series without pressing – the appearance of errors decreased the LP of P3.

Thus, these results lead to the conclusion that impairments to the performance of the psychomotor test induced by decreases in the level of consciousness are reflected in the structure of AEP components (N1, P2, N2, and P3).

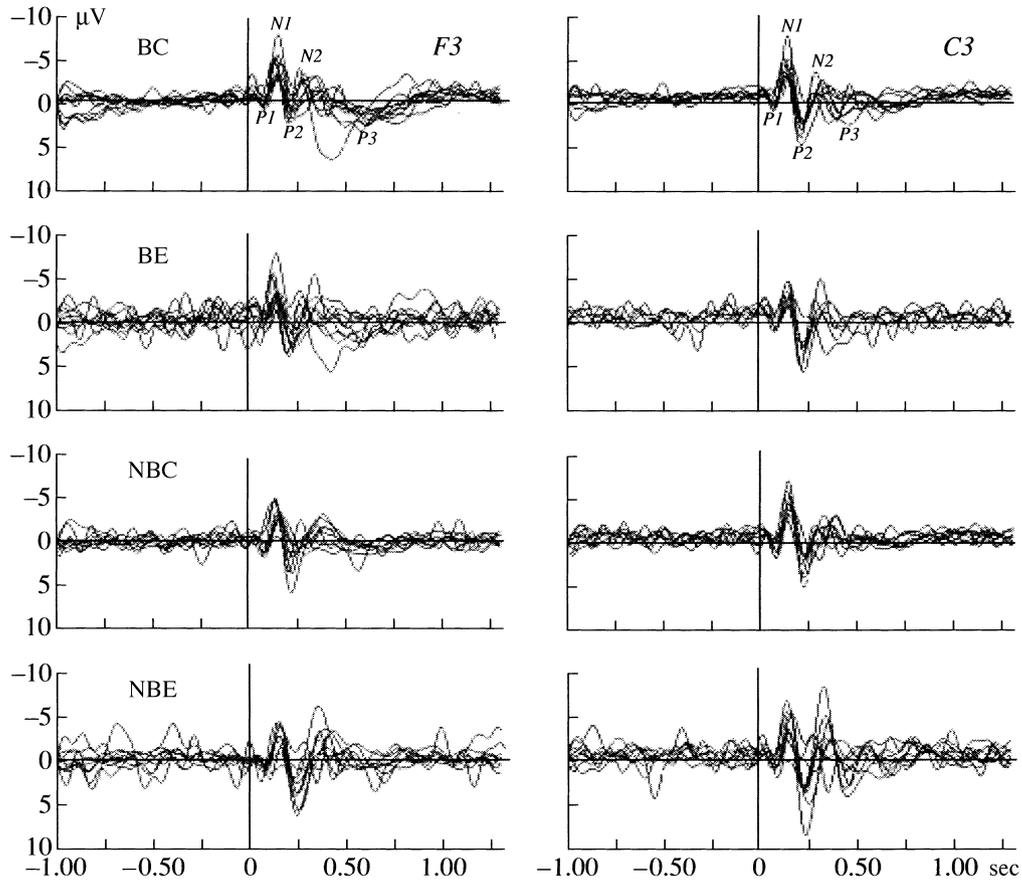


Fig. 2. Superimposed AEP for leads F3 and C3 averaged for each subject and selectively chosen for each of the four groups of the psychomotor test. For further details see caption to Fig. 1.

TABLE 1. Mean Amplitudes of AEP Components (P1, N1, P2, N2, P3) in Leads F3 (F) and C3 (C) Selectively Averaged for Individual Subjects ($N = 14$) Depending on the Task and Quality of Task Performance

	P1		N1		P2		N2		P3	
	$X \pm m$	Me								
FBC	1.41 ± 0.28	1.42	3.80 ± 0.67	3.24	4.91 ± 0.78	4.51	2.71 ± 0.26	2.67	4.34 ± 0.55	4.72
FBE	2.07 ± 0.24	2.17	4.19 ± 0.93	3.02	5.29 ± 0.73	5.42	3.96 ± 0.67	4.34	4.13 ± 0.72	3.76
FNBC	1.36 ± 0.20	1.32	4.02 ± 0.43	3.92	5.46 ± 0.86	5.09	3.99 ± 0.74	3.34	3.39 ± 0.29	3.34
FNBE	1.60 ± 0.31	1.59	3.73 ± 0.69	3.51	6.31 ± 1.14	5.52	7.82 ± 1.74	6.93	4.98 ± 0.95	3.92
CBC	1.42 ± 0.24	1.50	4.45 ± 0.45	4.09	6.78 ± 0.51	6.68	4.21 ± 0.56	3.84	3.29 ± 0.42	3.00
CBE	2.52 ± 0.32	2.84	4.67 ± 0.84	3.76	6.65 ± 0.71	6.10	5.72 ± 0.70	5.17	3.54 ± 0.56	3.09
CNBC	1.82 ± 0.40	1.92	5.19 ± 3.85	0.81	7.08 ± 0.76	6.94	4.92 ± 0.82	4.68	2.85 ± 0.32	2.28
CNBE	2.10 ± 0.30	1.84	3.82 ± 0.61	4.08	7.86 ± 0.88	8.35	9.22 ± 1.34	8.10	6.37 ± 1.29	4.85

Note. Amplitudes were measured “from peak to peak,” μV . $X \pm m$ is the arithmetic mean \pm error of the mean. Me is the median; P1, N1, P2, N2, and P3 are the components of the auditory evoked potential; F and C are leads F3 and C3; NBC, NBE, BC, and BE are identified in the caption to Fig. 1.

In series with button pressing, decreases in the level of consciousness increased the mean LP of the positive P3 component in lead F3 (by 90 ± 30 msec), decreased the amplitude of the negative N1 component, and increased the

amplitude of the positive P2 component and the mean amplitude of the negative N2 component (by $1.25 \pm 0.56 \mu\text{V}$).

Statistically significant features accompanying the appearance of errors in series without button pressing were

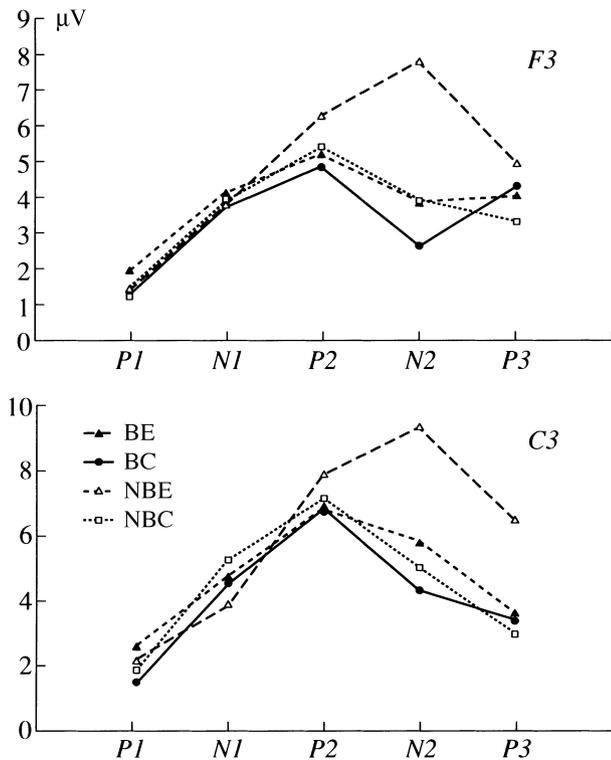


Fig. 3. Plots of mean amplitudes of AEP components (P1, N1, P2, N2, and P3) at different stages depending on the instructions and quality of following them (for details see Fig. 1) in leads F3 and C3. The horizontal axes show AEP components (P1, N1, P2, N2, and P3); the vertical axes show mean amplitudes of AEP components, μV . At right: key for type of task and quality of performance.

a reduction in the mean LP of the positive P2 component (by 110 ± 30 msec) in lead F3, increases in the amplitudes of the positive P2 component (by 0.85 ± 0.35 μV) and the negative N2 component (by 3.8 ± 1.3 μV), and an increase in the amplitude of the P3 peak (more significant in lead C3 – by 3.5 ± 1.3 μV).

DISCUSSION

Analysis of auditory evoked potentials (AEP) on appearance of errors in performing the psychomotor test showed that the most marked changes in AEP form were seen at time intervals corresponding to the P1 (70–80 msec), N1 (140–150 msec), P2 (220–250 msec), N2 (330–360 msec), and P3 (460–550 msec) components. Similar changes in AEP form were also seen in control series of experiments using spontaneous falling asleep during the transition from waking to sleep, as described in our previous report [3]. In both series, the greatest changes in AEP were in the areas of the N2 component, which has a latency of 330–360 msec.

Comparison of AEP configuration on appearance of errors in series with performance of the psychomotor test

and the control series with spontaneous falling asleep showed that the greatest changes in AEP occurred in relation to the N2 component, with latency 330–360 msec, which has an origin similar to that of the EEG theta rhythm and is characteristic of the first stage of sleep [10, 11, 13].

This result points to the involvement of sleep/waking mechanisms in the formation of the state of monotony induced by uniform activity leading to impaired performance of this activity. In terms of electrophysiological measures, this state transition almost completely corresponds to the first stage of sleep according to the usual criteria of sleep [23]. Some authors [13, 19, 22] have used EEG criteria to divide the first stage of sleep into the alpha-1 and theta-1 substages, which differ in terms of the intensity of the alpha and theta EEG rhythms. Many authors [6, 13, 21, 24] use the experimental oddball paradigm in studies of changes in information processes on falling asleep, where two different stimuli are presented with different probabilities and the subject has to press a button in response to the deviant (significant) stimulus. In these experiments, special attention is paid to changes in the P3 (P300) component at the alpha-1 and theta-1 substages of the first stage of sleep. Increases in the LP when reaction times increase are explained in terms of slowing of signal processing; two sub-components of P300 were identified, with different functions [6, 13, 21, 24].

The psychomotor test used here allows better modeling of real monotonous activity than the oddball paradigm described above. When the psychomotor test using two alternating series with and without button pressing is performed, the significance of a given stimulus was related to the instruction received and its performance. Switching from performing one series to the other requires a certain, quite high level of consciousness. The multitude of brain systems involved in regulating the level of consciousness produces different possible routes to the transition from waking to sleep [15]. The cause of the appearance of errors in performing different series may therefore have features linked with the fact that changes in the states of brain regulatory systems occur at different times as the level of consciousness decreases and sleep develops. Erroneous increases in the number of presses ($n > 10$) in series with pressing may be associated with the slower development of the processes underlying the transition to sleep. Some authors [18] have suggested that the brain systems responsible for motor behavior are different from the systems monitoring behavior. In this situation, decreases in the level of consciousness impaired only the monitoring function in erroneous sequences with increased numbers of presses, without altering the performance of counting and the formation of motor commands. Performance of erroneous series with reduced numbers of presses ($n < 10$) evidently involved faster deepening of sleep processes and the impairment to mental processes was more significant. These errors were seen more frequently than increases in

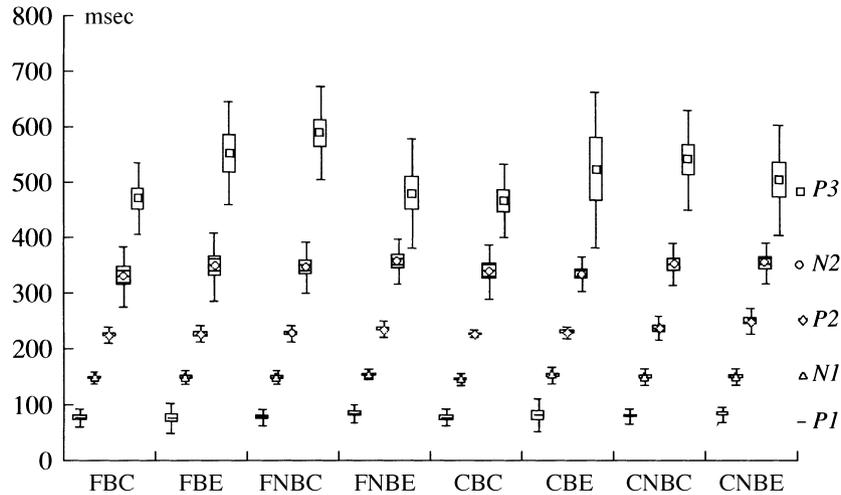


Fig. 4. Summary plot of latent periods of all EP components for the four groups of the psychomotor test. For details see Fig. 1. The vertical axis shows latent periods (see Table 2). The horizontal axis shows identifiers for the four groups of the psychomotor test (for details see Fig. 1) in leads F3 and C3, shown on the horizontal axis as F and C. EP components are identified at right.

TABLE 2. Mean Latent Periods of AEP Components (P1, N1, P2, N2, P3) in Leads F3 (F) and C3 (C) Selectively Averaged for Individual Subjects (*N* = 14) Depending on the Task and Quality of Task Performance

	P1		N1		P2		N2		P3	
	<i>X</i> ± <i>m</i>	Me	<i>X</i> ± <i>m</i>	Me	<i>X</i> ± <i>m</i>	Me	<i>X</i> ± <i>m</i>	Me	<i>X</i> ± <i>m</i>	Me
FBC	74.5 ± 5.19	72.5	148 ± 3.27	150	224 ± 4.76	225	328 ± 17.11	330	466.5 ± 20.4	450
FBE	76.0 ± 8.59	85	149 ± 4.07	150	226 ± 4.76	220	345.5 ± 19.0	340	548.6 ± 35.2	575
FNBC	77.0 ± 4.73	80	149 ± 3.79	150	227 ± 4.48	230	344 ± 14.3	360	584.5 ± 26.2	600
FNBE	83.0 ± 5.39	85	154 ± 3.06	155	235 ± 5.00	240	355 ± 12.7	360	476.5 ± 31.3	450
CBC	75.5 ± 4.74	80	145 ± 3.73	145	226 ± 2.21	225	336 ± 15.07	335	462.5 ± 21.0	440
CBE	80.0 ± 9.55	85	152 ± 4.90	150	229 ± 4.38	225	331.5 ± 10.1	330	519.2 ± 57.0	480
CNBC	78.0 ± 3.89	80	149 ± 4.82	150	236 ± 7.02	230	349.0 ± 12.2	355	536.0 ± 28.8	555
CNBE	81.0 ± 4.07	80	149 ± 4.82	150	248 ± 7.42	240	351.0 ± 11.5	340	499.5 ± 31.3	450

Note. Latent periods were measured from the stimulus to the peak of the corresponding component, msec. For further details see caption to Table 1.

the number of presses. When series without pressing were impaired, the transition from waking to sleep had a number of features; large numbers of stimuli in series (*n* > 5) were seen more frequently than shortening of series (*n* < 5).

Comparison of AEP configurations (Fig. 3) in series with and without pressing for series performed correctly and erroneously with and without pressing demonstrated a significant increase in the amplitudes of the N1-P2-N2 components of the vertex complex in series without pressing. The increases in the amplitudes of the P2 and N2 components on comparison of erroneous series with and without pressing were particularly large. Similar increase in the amplitudes of the P2 and N2 components were also seen on erroneous performance of the test in both series (Fig. 4). In both cases, the

increase in the amplitude of the negative N2 component was accompanied by increases in its latent period.

On the one hand, this result is consistent with the view of Näätänen [19], that the slow negative wave, termed “processing negativity,” of the vertex complex is summed with the N1 and P2 components and reflects information processing processes when attention is attracted to a significant stimulus. It has been suggested [7] that decreases in the amplitude of the N1 component and increases in the amplitude of the P2 component in the first stage of sleep are evoked by the decrease in stimulus significance on transition from waking to sleep and subtraction of processing negativity from the vertex complex when attention-related information processes are inhibited.

On the other hand, the significant increase in the amplitude of the negative N2 component and the increases in its latent period in erroneous series without pressing showed that the corresponding functional state of the brain was close to that in the first stage of sleep. The negative N2 component is known to be replaced by the new N350 component during the first stage of sleep, and that the latency and amplitude of this are increased, such that it is regarded as the new N350 component typical of sleep [26]. The N350 component has recently been regarded as an analog of sharp waves appearing on falling asleep, and the common origin of this component and the EEG theta rhythm has been identified [10, 11, 13].

Most authors link the positive P3 component with the processes of attention and identification of stimulus significance. Most studies [7, 8, 12, 24] indicate that increases in the LP of the P3 component at stage alpha-1 is accompanied by increases in the reaction time, which the authors suggested was linked with an increase in the time required for analysis of signal significance. We suggest that this explanation also applies to the results obtained in our studies: impairments of test performance in series with pressing involve increases in the latency of the P3 component.

The present study shows that the psychomotor test used here provides an effective model for studies of the neurophysiological mechanisms of impairments to activity evoked by the development of monotony, with reductions in the level of consciousness and the transition to sleep.

CONCLUSIONS

1. The nature of errors in performing a psychomotor test induced by a reduction in the level of consciousness differed in series with and without button pressing. In series with button pressing, impairments to oral counting were apparent as a decrease in the number of stimuli counted (less than the 10 stimuli instructed), while in series without pressing there was an increase in the number of stimuli in the series (more than the five stimuli instructed).

2. Comparison of auditory evoked potentials in correctly and erroneously performed tests showed that impairments to the performance of the psychomotor test both in series with button pressing and in series without pressing were accompanied by increases in the latent period and increases in the amplitude of the P1 (70–80 msec), N1 (140–150 msec), P2 (220–250 msec), and N2 (330–360 msec) components. The greatest changes in AEP parameters were seen when errors appeared in series without pressing. The most significant changes were increases in the amplitude of the N2 component, which were accompanied by increases in the latent period. The change in the amplitude of the P3 component (460–550 msec) was less informative. P3 amplitude showed some decrease in erroneous counting in series with and without pressing and increased in series without pressing.

3. The most significant changes in the parameters of auditory evoked potentials when errors appeared in performance of the psychomotor test were seen in the negative N2 component, with latency 330–360 msec, which is characteristic of the first stage of sleep and which, according to current concepts, has a common origin with the EEG theta rhythm. We suggest that the appearance of errors is associated with the development of the state of monotony in the subjects, induced by the uniform nature of the performance of the psychomotor test. Thus, changes in the negative N2 component can be regarded as a measure of the involvement of sleep mechanisms in the development of the state of monotony.

This study was supported by the Russian Humanities Scientific Foundation (Project Nos. 08-06-00598a and 08-06-0412a).

REFERENCES

1. V. V. Gnezditskii, *Evoked Brain Potentials in Clinical Practice* [in Russian], Taganrog Radiotechnical University Press, Taganrog (1997).
2. V. B. Dorokhov, "Analysis of the psychophysiological mechanisms of impairment to activity in drowsiness-related changes in consciousness," *Vestn. Ros. Guman. Nauchn. Fonda*, **4**, 137–144 (2003).
3. V. B. Dorokhov and Yu. S. Verbitskaya, "Changes in the components of auditory long-latency evoked potentials at different stages of slow-wave sleep," *Zh. Vyssh. Nervn. Deyat.*, **55**, 29–38 (2005).
4. A. M. Ivanitskii, V. B. Strelets, and I. A. Korsakov, *Brain Information Processes and Mental Activity* [in Russian], Nauka, Moscow (1984).
5. C. H. Bastien, K. E. Crowley, and I. M. Colrain, "Evoked potential components unique to non-REM sleep: relationship to evoked K-complexes and vertex sharp waves," *Int. J. Psychophysiol.*, **46**, 257–274 (2002).
6. H. Bastuji, L. Garcia-Larrea, C. Franc, and F. Mauguiere, "Brain processing of stimulus deviance during slow-wave and paradoxical sleep: a study of human auditory evoked responses using the oddball paradigm," *J. Clin. Neurophysiol.*, **12**, 155–167 (1995).
7. K. B. Campbell and I. M. Colrain, "Event-related potential measures of the inhibition of information processing: II. The sleep onset period," *Int. J. Psychophysiol.*, **46**, 197–214 (2002).
8. I. M. Colrain, "P300 and the daytime consequences of disturbed nocturnal sleep: easy to measure but difficult to interpret," *Sleep*, **28**, No. 7, 790–792 (2005).
9. I. M. Colrain and K. B. Campbell, "The use of evoked potentials in sleep research," *Sleep Med. Rev.*, **11**, No. 4, 277–293 (2007).
10. I. M. Colrain, P. Di Parsia, and J. Gora, "The impact of prestimulus EEG frequency on auditory evoked potentials during sleep onset," *Can. J. Exp. Psychol.*, **54**, 243–254 (2000).
11. K. E. Crowley, J. Trinder, and I. M. Colrain, "An examination of evoked K-complex amplitude and frequency of occurrence in the elderly," *J. Sleep Res.*, **11**, 129–140 (2002).
12. C. C. Conchin and M. G. H. Coles, "Precommentary: Is the P300 component a manifestation of context updating?" *Behav. Brain Sci.*, **11**, 355–425 (1988).
13. J. Harsh, U. Voss, J. Hull, S. Schrepfer, and P. Badia, "ERP and behavioral changes during the wake-sleep transition," *Psychophysiology*, **31**, 244–252 (1994).
14. Y. Hiroshige and V. B. Dorokhov, "Hemispheric asymmetry and regional differences in electroencephalographic alpha activity at the wake-sleep transition," *Japan Psychol. Res.*, **39**, 75–86 (1997).

15. J. A. Hobson, E. F. Pace-Schott, and R. Stickgold, "Dreaming and the brain: toward a cognitive neuroscience of conscious states," *Behav. Brain Sci.*, **23**, 793–842 (2000).
16. W. T. Liberson and C. W. Liberson, "EEG records, reaction times, eye movements, respiration and mental content during drowsiness," *Rec. Adv. Biol. Psychiatry*, **8**, 295–302 (1965).
17. A. Muller-Gass and K. Campbell, "Event-related potential measures of information processing: I. Selective attention in the walking state," *Int. J. Psychophysiol.*, **46**, 177–195 (2002).
18. R. Näätänen, "The role of attention in auditory information as revealed by event-related potentials and other brain measures of cognitive brain function," *Behav. Brain Sci.*, **13**, 201–288 (1990).
19. Y. Niiyama, R. Fujiwara, N. Satoh, and Y. Hishikawa, "Endogenous components of event-related potential appearing during NREM stage 1 and REM sleep in man," *Int. J. Psychophysiol.*, **17**, 165–174 (1994).
20. R. D. Ogilvie, I. A. Simons, R. H. Kuderian, T. MacDonald, and J. Rustenberg, "Behavioral event related potential and EEG/FFT changes at sleep onset," *Psychophysiology*, **28**, 54–64 (1991).
21. T. W. Picton, "The P300 wave of the human event-related potential," *J. Clin. Neurophysiol.*, **9**, 456–479 (1992).
22. R. T. Pivik, "Psychophysiology of dreams," in: *Principles and Practice of Sleep Medicine*, M. T. Kryger, T. Roth, and W. C. Dement (eds.), W. B. Saunders Company, Philadelphia, Third Edition (2000), pp. 491–501.
23. A. Rechtschaffen and A. Kales, *A Manual of Standardized Terminology: Techniques and Scoring System for Sleep Stages of Human Subjects*, US Government Printing Office (1968).
24. P. Ruby, A. Caclin, S. Boulet, C. Delpuech, and D. Morlet, "Odd sound processing in the sleeping brain," *J. Cogn. Neurosci.*, **20**, No. 2, 296–311 (2008).
25. H. Tanaka, M. Hayashi, and T. Hori, "Statistical features of hypnagogic EEG measured by a new scoring system," *Sleep*, **19**, 731–738 (1996).
26. C. M. Yang and C. S. Wu, "The effects of sleep stages and time of night on NREM sleep ERPs," *Int. J. Psychophysiol.*, **63**, No. 1, 87–97 (2007).