



Episodic memory causes a slow oscillation of EEG, awakening and performance recovery from sleep episodes during monotonous psychomotor test

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Received 14 November 2023 / Accepted 7 December 2023

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Abstract The aim of this study is to detect changes in a functional state during the performance of monotonous psychomotor test. We propose that these changes represent the repeated episodes of short sleep and spontaneous awakenings from them. We also argue that spontaneous awakenings from sleep stage 2 are accompanied by high-amplitude slow oscillations (SO). 20 subjects participated in 27 experiments during daytime. They performed continuous psychomotor test in a supine position with eyes closed for 1 h. Expert scoring of SOs in the EEG, including K-complexes and other single slow waves, was performed in sleep episodes reaching sleep stage 2, defined as pauses in task performance longer than 3 s. 248 episodes with SOs occurred in time intervals without activity lasting from 3 s to 10 min. In 195 cases, at least one SO was recorded before the test performance was resumed. 248 sleep episodes with 1255 SOs were taken into analysis. It was shown that SOs occur much more frequently just before awakenings (SO¹, 12 or less seconds before awakening) than within sleep episodes (12 or more seconds before awakening, SO²). Some SOs were recorded within a few seconds after behavioral awakening (SO³), which has not been previously reported. We propose the hypothesis that SOs which occurred just before resuming performance (SO¹) are associated with the unconscious episodic memory that triggers awakening followed by recovery of conscious activity performed prior to falling asleep. We also describe the novel type of SO (SO³) which occurs just after awakening.

1 Introduction

In the field of research on neural criticality, the word *critical* is used in the sense of statistical physics, which is distinct from other meanings, including the colloquial use. In statistical physics, criticality is defined as a specific type of behavior observed when a system undergoes a *phase transition*. This state on the edge between two qualitatively different types of behavior is called the *critical state*, and in this state, the system is *at criticality*. Because phase transitions usually break certain symmetries of the system, they often separate an ordered state from a less ordered state. Critical states are therefore said to be on the *edge of chaos*. These findings inspired the *criticality hypothesis*, which proposes that the brain operates in a critical state because the associated optimal computational capabilities should be evolutionarily selected for [1].

Since sleep and wakefulness are fundamentally different brain states, we can extend this hypothesis to the process of falling asleep and especially to brief wake–sleep transitions. It was previously shown [2] that durations of such sleep episodes tend to be exponentially distributed, while durations of wake episodes tend to be power-law distributed; this behavior resembles the dynamics of certain physical systems exhibiting self-organized criticality. Such systems are characterized by recurring avalanches (excitations from a “quiescent” state) triggered by the accumulation of incoming energy.

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A notable example of this process can be observed during the performance of monotonous operator activity, which can lead to accidents in transportation and production, including fatalities. Long episodes of sleep (up to tens of minutes) are probable in monotonous operator activities that do not require a quick response.

We assume that the rapid resumption of operator activity after episode of falling asleep is due to the preservation of instructions about operator's work in the episodic memory, which remains active even during sleep. We hypothesize that high-amplitude slow oscillations (SO) that include K-complexes facilitate awakening and work resumption. In our previous work we used the term "K-complex" [3, 4], which is replaced by modern term SO (including K-complexes and other single slow waves) in the research presented here [5, 6].

Although episodic memory is one of the most studied topics in cognitive neuroscience, the answers to these questions are still unclear, in large part because episodic memory research has traditionally focused on experiments using simple, well-controlled stimuli, where participants receive clear instructions about when to encode and retrieve [7]. These experiments have the potential to shed light on when encoding and retrieval take place during event perception in a naturalistic context, where no one is explicitly instructing participants about how to use episodic memory [8].

Also, many researchers study episodic memory and its possible interaction with sleep [9–15]. We have not found any direct experimental evidence for the possibility of retrieving information from episodic memory during sleep that causes awakening in modern scientific literature. Nevertheless, there is well-known phenomenon of morning awakening at a predetermined time, which some authors [16, 17] interpret as a cognitive activity related to the retrieval of instructions to wake up at the right time from memory.

We have developed a continuous bimanual psychomotor test [3] that allows us to observe several episodes of short sleep episodes during a 1 h-long experiment performed at the daytime. We hypothesize that during the performance of the test, information about the nature of its performance is stored in episodic memory. During short sleep episodes, this memory wakes up the subject, triggering SO, and returns him to conscious activity. This test activity is similar to the activity performed to driving various vehicles that cause monotony, such as driving a car or train. Previously, we showed a reliable increase in the probability of K-complex appearance before the moments of behavioral awakenings (the first button press after a pause in test performance), as well as their difference from "baseline" K-complexes which appear long before awakening [4]. It has been shown in a number of papers that driving a vehicle takes no more than half of time behind the wheel, and the rest of the time driver is distracted [18, 19]. In a collection of publications devoted to establishing a link between mental state and human behavior, it has been shown that reactions to external stimuli (stimulus presentation, driving initiation of inhibition, etc.) are linked to reproducing or reactivating the contents of episodic memory. However, no technologies have been developed yet that can accurately measure and log information during the formation or reactivation of episodic memory [19].

In the scientific literature about sleep, the K-complex is considered as a pattern that has a dual function [20]. Andrillon and Kouider [21] discuss the dual function of the K-complex: on the one hand, the K-complex can inhibit responses elicited by a single stimulus, but on the other hand, it can create "wakefulness windows" in sleep, where sleeper can monitor relevant internal and environmental inputs and wake up if necessary [22].

The aim of this study is to detect changes in a functional state during the performance of monotonous psychomotor test. We propose that these changes represent the repeated episodes of short sleep and spontaneous awakenings from them. We also argue that spontaneous awakenings from sleep stage 2 are accompanied by high-amplitude slow oscillations (SO).

2 Methods

All procedures performed in this nap study of human participants were in accordance with the ethical standards of the 1964 Helsinki Declaration and its later amendments, and in accordance with the ethical standards of the institutional research committee. The Ethics Committee of the Institute approved the experimental protocol in June 2019 (Approval#12402-02-7112). The study participants were informed in detail about all procedures, and informed written consent was obtained from each participant.

2.1 Participants

Unpaid volunteers of the 60-min nap studies were 20 (9 male and 11 female) students with mean age of 19.3 and 20.0 years and standard deviation of 1.3 and 1.1 years, respectively. After the structured interview with a sleep researcher, they were invited to participate in the nap study.

The exclusion criteria were: age either younger than 18 or older than 23 years, pregnancy or breastfeeding (for female participants), history of mental or sleep disorders, any complains about poor physical condition and functioning, current mild cold and missing classes due to any sickness in 2 previous weeks, involvement in shift or night work, crossing several time zones in the previous month.

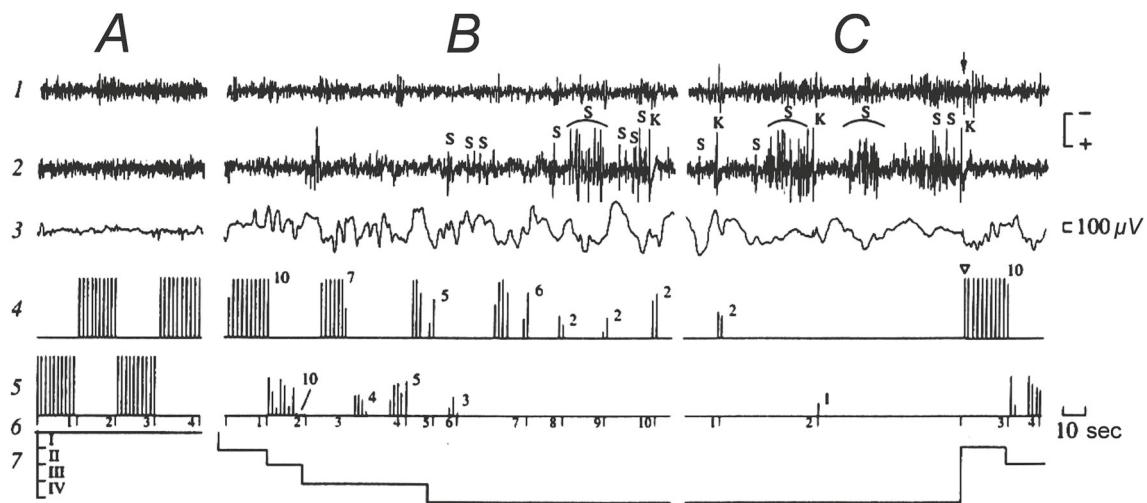


Fig. 1 EEG correlates of psychomotor test errors for various short episodes of falling sleep observed in single experiment. **A** wakeful state, **B** drowsiness and light sleep (1st stage), **C** deeper sleep, (sleep stage 2). 1,2—O1 and C3 EEG electrodes, 3—EOG (left eye), 4,5—button presses for right and left hands, 6— number of presses in the batch, 7—performance level. EEG electrodes: S—vertex sharp waves, K—K-complexes. Arrow marks awakening sound. Performance levels are the following: I—correct task performance, II—weaker pressing force, III—weaker pressing force with errors, IV—sleep stage 2

2.2 Study protocol

Participants completed questionnaires to assess their habitual sleep patterns, sleep quality, sleepiness, and general condition before the start of the experiment. All experiments were conducted during the daytime, starting from 1 to 3 pm. The duration of the experiment was about 1.5 h. The EEG was recorded for 60 min. During the recording, the participant was in a supine position on a couch with closed eyes in a darkened, soundproofed and ventilated room.

For each participant, a button was fixed on the index finger of each hand, which the participant had to press with the thumbs of the same hand. The participants were instructed to press the buttons 10 times with a frequency of one press per second, alternating presses with the right and left hand.

The alternating phases with right and left hand presses lasted until the subjects fell asleep. After sleep followed by spontaneous awakening, participants were required to resume the psychomotor test and perform it until they fell asleep again. The alternation of episodes of falling asleep and waking up accompanied by psychomotor activity makes it possible to study neural correlates of changes in the functional state and level of human consciousness. The first pressing of the button with any hand was considered to be the awakening and the beginning of activity.

Several behavioral measures of the subject's consciousness state were available during the experiment: counting accuracy, time between presses, pressing force. Electromyogram (EMG) from both thumb fingers (*musculus abductor pollicis brevis*) was recorded in addition to button mechanograms. Raw data extracted during the test procedure are schematically shown in Fig. 1.

In each successful experimental session several short episodes without any activity were observed. Duration of these lapses varied from 3 s to several minutes, with median value about 52 s. Five subjects that didn't fall asleep at all were excluded from further analysis.

2.3 Polysomnographic recordings

The polysomnographic recordings were performed via a 16-channel wireless system ("Neuropolygraph 24," Neuretech, Taganrog, Russia). A standard monitoring montage was used for polysomnographic recordings, namely, the EEG channels, one chin electromyogram channel, and two electro-oculogram channels. All electrodes were placed in accord with the international 10–20 system of electrode placement. To provide a possibility of conventional sleep stage scoring, the placement included two central channels referenced to ear mastoid sites (C4–M1 and C3–M2) and two occipital channels referenced to an ear mastoid sites (O1–M2 and O2–M1). The recorded signals were preprocessed using the high-pass, low-pass, and notch filters (frequencies of 0.5, 35, and 50 Hz, respectively). The signals were sampled and stored on a hard drive with a sampling rate of 500 Hz.

2.4 Sleep episodes

The data of 27 experiments from right-handed subjects were selected for the analysis. The selection criterion was the onset of the sleep stage 2 and subsequent awakening while pressing the button with the hand during the psychomotor test. 248 sleep episodes (pauses between button presses) with slow wave oscillations (SOs, previously known as K-complexes and other SOs), were identified, their duration varied from 3 s to 10 min. We describe several types of SOs depending on their time of occurrence—more on that in the Results section.

2.5 EEG analysis

SOs were scored in two steps [5, 6]. Firstly, signal was filtered in a frequency range of 0.5–2 Hz using the 4th order Butterworth filter. After that, we detected waves for which the discrepancy between the adjacent minimum and maximum was more than 70 μV , excluding epochs previously marked by the expert as the sleep stage 3. Then the SOs on different electrodes, the time difference between which was less than 0.5 s, were combined into one, the minimum of which was calculated from the electrode for which the amplitude difference of the detected wave was maximum. Secondly, two experts supervised the scored EEG and removed marks that were not close enough to SOs. We considered all episodes where there were no presses for 3 s or more as short sleep episodes. First button press after such pause was considered a behavioral awakening. We have used a similar design in [4].

Weighted histograms obtained from these markers were taken for further analysis. Descriptive statistics of sleep episodes and SO occurrence times were calculated. The paired samples *t* test was used to show statistically significant difference of SO frequency within sleep and before awakening. Detailed analysis of several time intervals before and just after awakening was also performed.

3 Results

All sleep episodes for each experiment were categorized as either sleep stage 1 and sleep stage 2. Their number and maximal episode duration for each participant are summarized in the Table 1.

Time proportions of correct task performance, incorrect task performance and lack of presses (microsleep episodes) are shown for each subject in Fig. 2.

From the sleep episodes that were categorized as the sleep stage 2, SO were scored at the intervals of -12 and -3 until the moment of the 1st pressing of the button (0) and 4 s after pressing the button (0–4). Only single SO values were selected, since this stage was necessary to select the SO that trigger the first press on the button; one SO was selected for each interval before pressing the button and after pressing the button. There were 195 such SOs in total. Figure 3 shows a histogram of these data.

Since sleep episodes varied highly in duration, the following intervals were chosen to analyze the relative frequency of SO occurrence before behavioral awakenings within them:

- 1: – 12 to – 8 s before activity resumption;
- 2: – 8 to – 4 s before activity resumption;
- 3: – 4 to 0 s before activity resumption;
- 4: 0 to + 4 s after activity resumption.

For all of these time intervals, the frequency of SO occurrences was compared in a pairwise fashion. Because the proportion of sleep for each subject differed significantly, the SO histogram for each experiment was normalized by the total number of sleep episodes with SO in that experiment. Histograms for subjects with repeated experiment series were preliminarily averaged.

The means for normalized SOs in each interval were: 0.22 for the interval 1, 0.27 for the interval 2, 0.68 for the interval 3, and 0.18 for the interval 4. Using Student's *t* test for paired samples with Bonferonni correction for multiple comparisons, we obtained these *p* values: 1 and 2—0.59; 1 and 3—0.0002 $^{**}(p < 0.01/6)$; 1 and 4—0.57; 2 and 3—0.00007 $^{***}(p < 0.001/6)$; 2 and 4—0.14; 3 and 4—0.000016 $^{***}(p < 0.001/6)$. Interval 3 showed the highest probability of SO occurrence compared to other intervals. In general, the normalized number of SOs increases with the proximity to wakefulness at the selected time interval.

To compare frequency of all SO occurrences before and after awakening, we analyzed a histogram of the occurrence times of all SOs at the interval – 60... + 4 s before and after behavioral awakening. The total number of SOs was 1255. This distribution is presented in Fig. 4.

Based on the appearance of the histogram, we chose the time interval – 32... – 20 s before behavioral awakening as a “baseline” sleep, where the average number of SOs is more or less constant. We compared it with the interval – 12... 0 s before behavioral awakening by performing normalizations similar to the previous point. Using Student's *t* test for paired samples, we obtained the *p* value of 0.00001 $^{***}(p < 0.0001)$. Thus, it can be argued that the probability of SO occurrence before awakening has significantly increased compared to the “baseline” SOs.

Table 1 Total number of microsleep episodes during sleep stages 1 and 2 and maximum episode duration for each participant

Experiment	Stage 1	Stage 2	Max ep duration
Al	25	6	532.05
Ar	40	13	616.84
Br	81	15	119.69
Bul	26	6	332.37
Fal	21	3	534.99
Gan	22	17	161.43
Gor	9	5	628.86
Gr	22	10	621.47
Kal	89	10	354.35
Kar	23	8	347.84
Kar	36	6	633.05
Koh	22	8	520.42
Kom	68	6	238.51
Kom	17	7	538.33
Kuz	23	14	226.24
Kuz	20	3	46.10
Pil	32	7	621.80
Pil	38	8	478.99
Pil	22	11	575.96
Pil	49	0	382.86
Pr	33	7	327.83
Ryab	107	8	52.88
Saz	27	16	172.12
Saz	45	22	436.90
Saz	48	21	566.15
Tar	54	9	389.64
Zat	8	2	1063.83

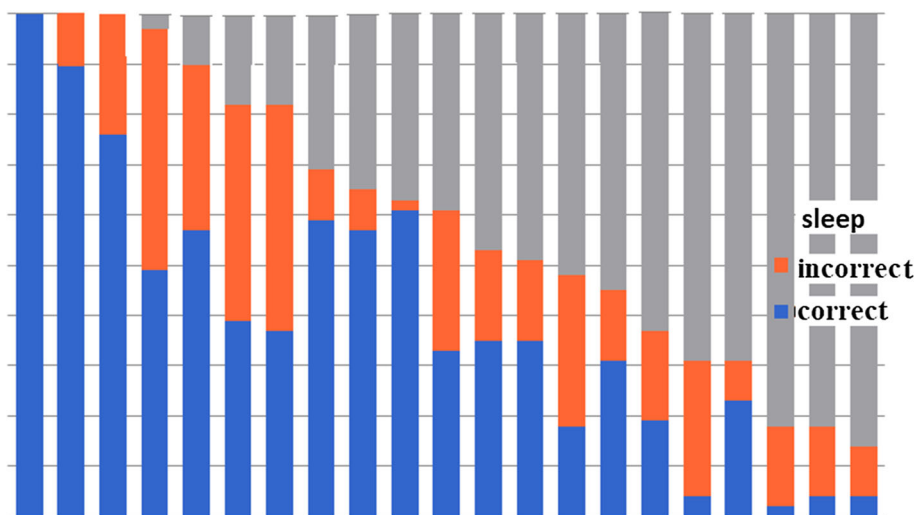


Fig. 2 Proportion of correct press batches (blue color), incorrect press batches (orange color) and sleep (grey color) for each subject

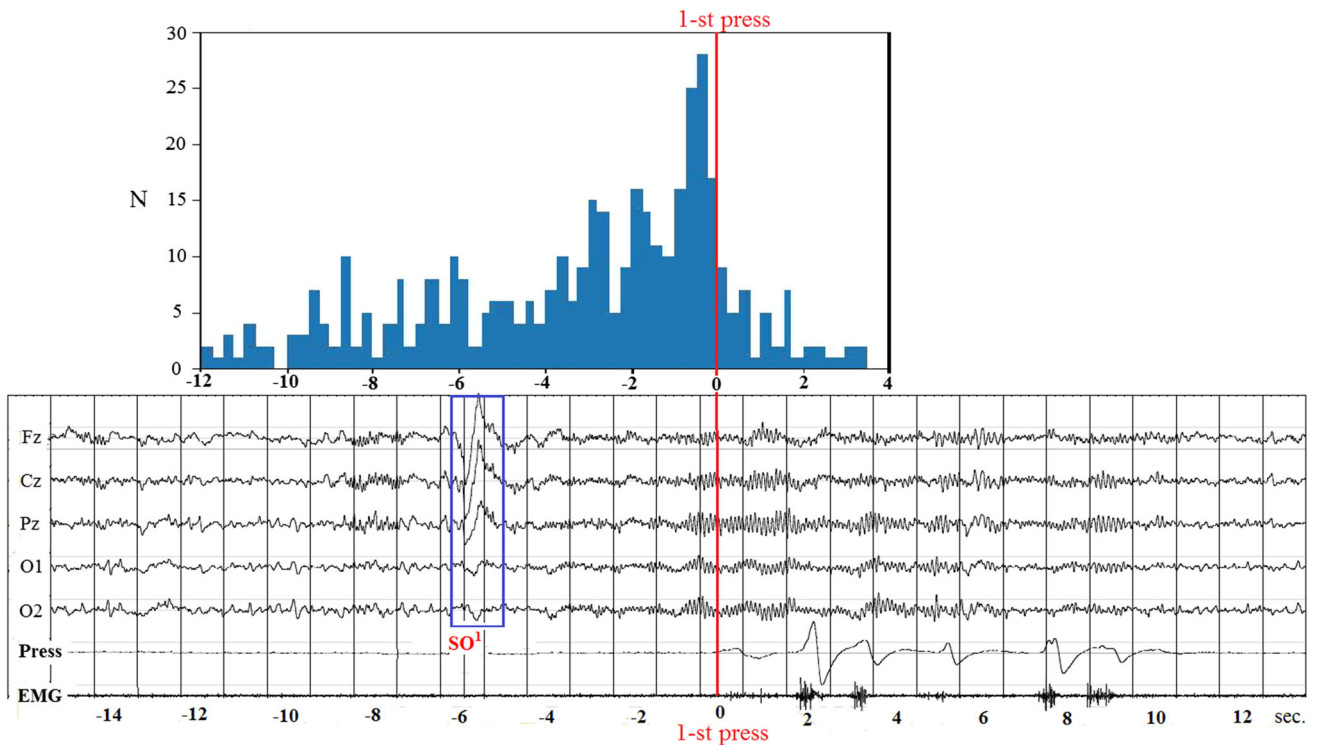


Fig. 3 Top: histogram of the times between the last SO at the interval $-12 \dots +4$ s around the moment of behavioral awakening and the awakening moment. The vertical line shows the moment of the first button press after a pause in presses. X-axis: time in seconds, 0 corresponds to the first button press. The histogram resolution window is 250 ms. Y-axis: number of SOs in the 250 ms hypnogram window. Data for 20 subjects and 27 experiments are shown, total number of awakenings included in the analysis is 195. Bottom: example of a polysomnogram with the appearance of SO 6 s before the first press. Fz, Cz, O1, O2—EEG channels; Press—mechanogram signal, showing button presses and their strength; EMG—electromyogram of the short muscle withdrawing the thumb of the right hand (musculus abductor pollicis brevis). It can be seen that alpha activity appears after SO (highlighted in a box), followed by the first press

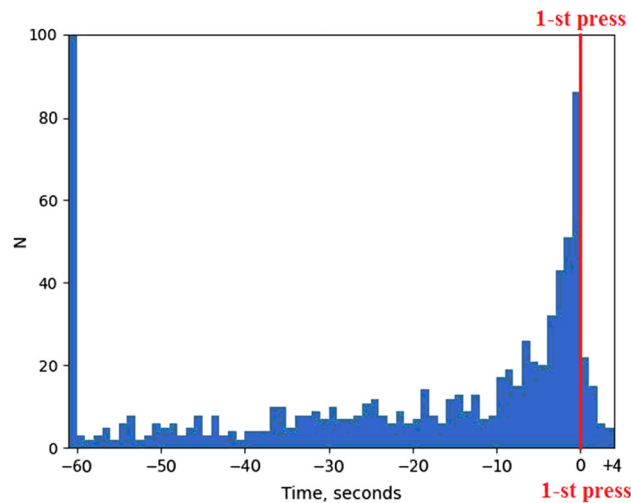


Fig. 4 Histogram of appearance times for all SOs on the interval $-60 \dots +4$ s before and after behavioral awakening. X-axis: time in seconds, where 0 is the moment of the first pressing of the button; hypnogram window is 1 s. Y-axis: number of SOs in the time interval of 1 s

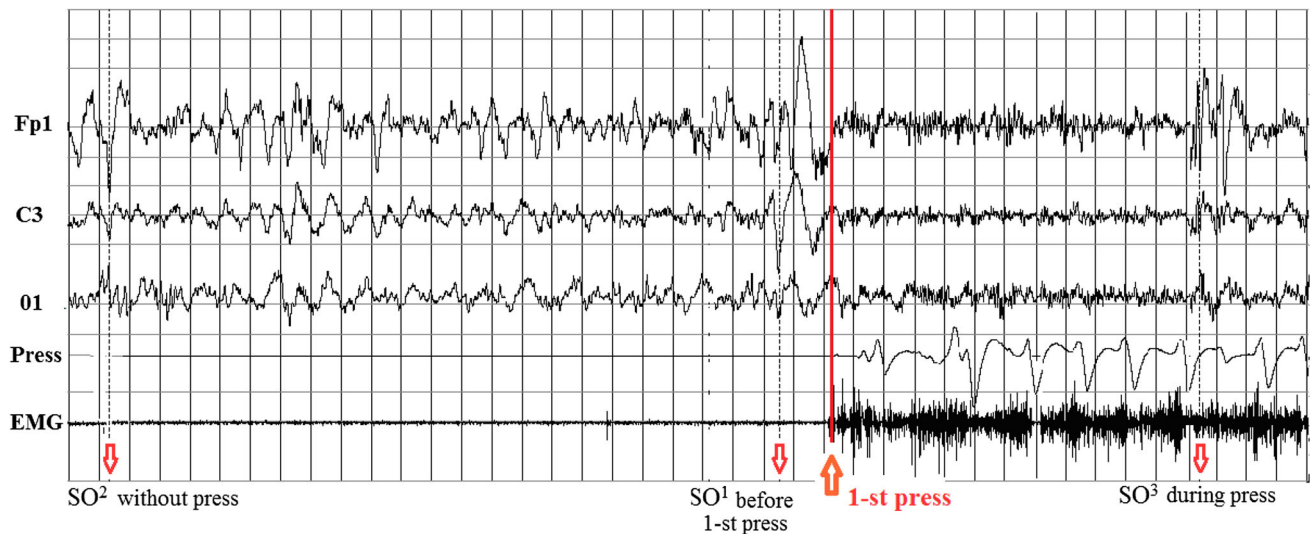


Fig. 5 Polysomnogram showing all three types of SOs: **a** “baseline” SO² with further EEG of sleep stage 2; **b** activating SO¹, co-occurring with EEG alpha rhythm and the first button press; **c** SO³ appears around 13–14 s during wakefulness. Fp1, C3, O1 are corresponding EEG electrodes, Press is the mechanogram signal from the button, EMG is the electromyogram from the thumb of the hand. The signal from the pneumatic button shows that the first button press was very weak, although the EMG was well pronounced

Consequently, we categorized SOs into three types: SO¹—preceding behavioral awakening and co-occurring with alpha activity; SO²—“baseline” SOs corresponding to the second stage of sleep; SO³—appearing at 0...4 s interval, after the first button press. SO³ often appeared immediately after SO², as it is shown in Figs. 2 and 3. Isolated SO³, occurring more than one second from the first press, were rare. All these types of SOs are shown in Fig. 5.

4 Discussion

We obtained the following results: (1) at the $-4...0$ s time interval before behavioral awakening, the probability of SO¹ appearance was maximal compared to the $-12...-8$ s and $-8...-4$ s intervals; (2) the frequency of SO¹ appearance at the $-12...0$ s time interval before awakening was significantly increased compared to the SO² at the $-32...-20$ s interval. We also detected SO³, slow oscillations appearing right after behavioral awakening (see Figs. 2, 3, 4). We hypothesize that they could signify additional brain activation after awakening.

Earlier in our studies, evidence was obtained for the possible involvement of K-complexes in the awakening of people who fell asleep while performing a psychomotor test with closed eyes [3]. In this work we obtained the quantitative confirmation of this hypothesis. We replaced the old term “K-complex” with SO (slow-wave oscillations). It includes K-complexes and other slow waves of similar amplitudes and frequencies and is actively used by many authors [5, 6].

A total of 248 sleep episodes with SOs were analyzed. 195 episodes had at least one SO¹ occurrence, while 53 episodes had only background SO²s. We proposed the hypothesis that SO¹ occurred during 2 sleep stage the instruction to perform a psychomotor test remains active in the subject’s unconscious episodic memory, prompting the subject to awaken and continue the task. In a series of research articles Pudiel [14, 15] showed in a similar experimental design that during brief episodes of falling asleep the coherence in thalamic structures is disrupted, but that excitation remains in cortical structures. This increased high-frequency activity supports that this activity reflects unconscious ‘cognitive’ processes aimed at returning the brain to consciousness and responsiveness.

The appearance of an alpha rhythm before resuming test performance was described by us earlier in [3]. The alpha rhythm observed in the EEG is a correlate of brain activation while awakening. It is triggered by endogenous mechanisms and provides spatial and temporal binding of various brain structures into a unified network, which is necessary for the recovery of conscious activity [23–25]. In particular, the alpha rhythm creates conditions for accessing instructions from episodic memory to executive motor structures.

It should be noted that when subjects perform the psychomotor test in the sitting position, unlike the supine position, SOs are not registered [26]. Performing the test in the supine position causes a rapid onset of drowsiness and the occurrence of episodes of falling asleep, but the recovery of activity is also faster than in a number of

studies that investigated the recovery of activity against the background of sleep inertia [27, 28]. In order not to miss weak presses, we used a pneumatic button sensitive to the force of pressing, as well as an EMG signal [29]

As Andrillon and Kouider [21] assume, the sleeping subjects maintain a certain amount of minimal vigilance, a stand-by mode allowing the quick reversal to wakefulness if necessary. It is well-known that K-complex can be caused not only by environmental, but also by internal stimuli [30]. Hypothetical instruction “count and press buttons” remaining in an episodic memory could act as such internal stimuli, given it is retained in the memory and gains access to brain executive structures when the “windows of wakefulness” open [22]. Hence, its emergence just before spontaneous awakenings in our experiment could signify the test instruction retrieval followed by further cortex activation and neuronal activity integration. We therefore suggest that “spontaneous” awakenings occur when hypothetical instruction “count and press button” successfully gains access to brain executive structures. Thus, it could play its role in urgent activation and further synchronization of these executive neural networks. We argue that subject’s button presses after sleep stage 2 could be used as an objective, “no-report” measure of consciousness level [31, 32]. We also believe that SO^1 during spontaneous awakenings represent a binding process [23–25] between neurons of various brain regions, that in turn builds a foundation to gradual consciousness activation.

The term “episodic memory” refers to the recollection of personally experienced life episodes [33]. It remains unclear whether and under what circumstances the most sophisticated form of human learning, namely episodic memory formation, can proceed during deep sleep. Episodic memory formation depends on hippocampal–neocortical interactions [34, 35]. Because episodic memory belongs to declarative memory, episodic memory was long associated with wakefulness and conscious awareness of events [33, 36, 37]. In the meantime, counterevidence suggests that hippocampal-assisted episodic memory formation may also proceed without conscious awareness of the learning material [38, 39]. These findings confirm newer theoretical claims that the sole premise for episodic encoding through hippocampal–neocortical interactions is a task that calls upon the core computational features of episodic memory and the hippocampus: rapid formation of new and flexible associations. It was also confirmed in [40, 41]. Many studies have been published on the creation of episodic memory during sleep and waking, which have been linked to the occurrence of SOs and sleep spindles [7, 12, 25, 42, 43]

The mechanisms of spontaneous awakening are described in detail in [44, 45]. Vocc [46] distinguishes two components of awakening: (1) cognitive component, shifting attention from internal to external focus, which is manifested in changes in EEG patterns, in particular, in the appearance of alpha rhythm (arousal response), and (2) behavioral component, which is expressed in the resumption of conscious activity. It should be noted that it is difficult to determine the moment of return of consciousness upon awakening without recording the behavioral task. In our case, we use the resumption of psychomotor test performance as such marker [3, 31, 32].

Our approach is consistent with many current studies of local sleep and transitions between sleep and wakefulness states. In particular Windt [47] discusses a continuum of states of consciousness within the sleep–wake cycle. The presence of an instruction to perform a test, prompting the subject to wake up, in unconscious episodic memory fits well into this paradigm.

We suggest that the brain state prior to the onset of wakefulness can be considered as a critical in the context of works in self-organizing criticality [1]. The sensitivity of such critical state to external stimuli may explain the fact that in some cases no slow waves (SO^1) were recorded before awakening (in 53 out of 248), although background slow waves (SO^2) were also recorded, indicating sufficient sleep depth in these cases.

To our knowledge, this is the first time that the occurrence of SO after recovery of conscious activity (SO^3) has been reported, which is a fundamentally new result that requires further investigation.

5 Conclusion

When performing a monotonous bimanual psychomotor test in the supine position with eyes closed, subjects experienced periodic alternation of sleep and wakefulness. Before awakening, the occurrence frequency of slow-wave oscillations (SO) increased statistically significantly. We hypothesized that the occurrence of SO before awakening (SO^1) is associated with the activity of an unconscious episodic memory containing instructions to perform the test. This unconscious episodic memory prompts the subject to continue performing button presses with simultaneous counting, which is manifested in the EEG by the SO^1 followed by the appearance of an alpha rhythm, which is a marker of awakening and is necessary for the recovery of conscious activity. Further investigation of unconscious episodic memory during episodes of falling asleep requires the development of new methods and approaches.

For the first time, the occurrence of SO^3 after recovery of conscious activity has been documented.

Author contributions

Conceptualization VBD; funding acquisition VBD and ONT; data curation VBD and ONT; resources VBD, AOT, GNT, EOG and NVL; supervision VBD; software ONT and VBD; investigation VBD, and ONT; methodology VBD, and ONT; sleep scoring AOT, GNT and EOG; spectra calculation AOT, GNT and EOG; validation VBD; visualization VBD and ONT; writing—review and editing VBD, ONT, AOT, GNT, EOG and NVL.

Funding This study was funded by Russian Science Foundation (Grant #22–28–01769).

Data availability The data sets generated during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors declare no competing interests. The funders had no role in the design of the study, the collection, analyses, or interpretation of data, the writing of the manuscript, and the decision to publish the results.

References

1. J. Hesse, T. Gross, Self-organized criticality as a fundamental property of neural systems. *Front. Syst. Neurosci.* **23**(8), 166 (2014). <https://doi.org/10.3389/fnsys.2014.00166>
2. C.C. Lo, T. Chou, T. Penzel, T.E. Scammell, R.E. Strecker, H.E. Stanley, PCh. Ivanov, Common scale-invariant patterns of sleep-wake transitions across mammalian species. *Proc. Natl. Acad. Sci. U.S.A.* **101**(50), 17545–17548 (2004). <https://doi.org/10.1073/pnas.0408242101>
3. V.B. Dorokhov, Alpha-bursts and K-complex: phasic activation pattern during spontaneous recovery of correct psychomotor performance at difference stages of drowsiness. *Zh. Vyssh. Nerv. Deiat. Im. I P Pavlova* **53**(4), 503–512 (2003)
4. V.B. Dorokhov, A. Runnova, O.N. Tkachenko, A.O. Taranov, G.N. Arseniev, A. Kiselev, A. Selskii, A. Orlova, M. Zhuravlev, Analysis two types of K complexes on the human EEG based on classical continuous wavelet transform. *Chaos* **33**(3), 031102 (2023). <https://doi.org/10.1063/5.0143284>
5. F. Siclari, G. Bernardi, B.A. Riedner, J.J. LaRocque, R.M. Benca, G. Tononi, Two distinct synchronization processes in the transition to sleep: a high-density electroencephalographic study. *Sleep* **37**(10), 1621–1637 (2014). <https://doi.org/10.5665/sleep.4070>
6. G. Bernardi, F. Siclari, G. Handjaras, B.A. Riedner, G. Tononi, Local and widespread slow waves in stable nrem sleep: evidence for distinct regulation mechanisms. *Front. Hum. Neurosci.* **19**(12), 248 (2018). <https://doi.org/10.3389/fnhum.2018.00248>
7. Q. Lu, U. Hasson, K.A. Norman, A neural network model of when to retrieve and encode episodic memories. *Elife* **10**(11), e74445 (2022). <https://doi.org/10.7554/eLife.74445>
8. S. Sonkusare, M. Breakspear, C. Guo, Naturalistic stimuli in neuroscience: critically acclaimed. *Trends Cogn. Sci.* **23**(8), 699–714 (2019). <https://doi.org/10.1016/j.tics.2019.05.004>
9. T. Schreiner, T. Staudigl, Electrophysiological signatures of memory reactivation in humans. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **375**(1799), 20190293 (2020). <https://doi.org/10.1098/rstb.2019.0293>
10. T. Schreiner, M. Petzka, T. Staudigl, B.P. Staresina, Endogenous memory reactivation during sleep in humans is clocked by slow oscillation-spindle complexes. *Nat. Commun.* **12**(1), 3112 (2021). <https://doi.org/10.1038/s41467-021-23520-2>
11. Y. Norman, E.M. Yeagle, S. Khuvis, M. Harel, A.D. Mehta, R. Malach, Hippocampal sharp-wave ripples linked to visual episodic recollection in humans. *Science* **365**(6454), eaax1030 (2019). <https://doi.org/10.1126/science.aax1030>
12. S. Berres, E. Erdfelder, The sleep benefit in episodic memory: an integrative review and a meta-analysis. *Psychol. Bull.* **147**(12), 1309–1353 (2021). <https://doi.org/10.1037/bul0000350>
13. T. Andriillon, A. Burns, T. Mackay, J. Windt, N. Tsuchiya, Predicting lapses of attention with sleep-like slow waves. *Nat. Commun.* **12**(1), 3657 (2021). <https://doi.org/10.1038/s41467-021-23890-7>
14. M.H. Zaky, R. Shoorangiz, G.R. Poudel, L. Yang, C.R.H. Innes, R.D. Jones, Increased cerebral activity during microsleeps reflects an unconscious drive to re-establish conscious awareness. *Int. J. Psychophysiol.* **S0167–8760**(23), 00429–00434 (2023). <https://doi.org/10.1016/j.ijpsycho.2023.05.349>
15. G.R. Poudel, C.R. Innes, P.J. Bones, R. Watts, R.D. Jones, Losing the struggle to stay awake: divergent thalamic and cortical activity during microsleeps. *Hum. Brain Mapp.* **35**(1), 257–269 (2014). <https://doi.org/10.1002/hbm.22178>
16. W. Moorcroft, J.L. Breitenstein, Awareness of time during sleep. *Ann. Med.* **32**(4), 236–238 (2000)
17. S. Malloggi, F. Conte, B. Albinni, G. Gronchi, G. Ficca, F. Giganti, Sleep and psychological characteristics in habitual self-awakeners and forced awakeners. *Chronobiol. Int.* **39**(4), 547–556 (2022). <https://doi.org/10.1080/07420528.2021.2003375>
18. K. Christoff, Z.C. Irving, K.C. Fox, R.N. Spreng, J.R. Andrews-Hanna, Mind-wandering as spontaneous thought: a dynamic framework. *Nat. Rev. Neurosci.* **17**(11), 718–731 (2016). <https://doi.org/10.1038/nrn.2016.113>

19. E.M. Robertson, L. Genzel, Memories replayed: reactivating past successes and new dilemmas. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **375**(1799), 20190226 (2020). <https://doi.org/10.1098/rstb.2019.0226>
20. P. Halász, R. Bódizs, L. Parrino, M. Terzano, Two features of sleep slow waves: homeostatic and reactive aspects—from long term to instant sleep homeostasis. *Sleep Med.* **15**(10), 1184–1195 (2014). <https://doi.org/10.1016/j.sleep.2014.06.006>
21. T. Andrillon, S. Kouider, The vigilant sleeper: neural mechanisms of sensory (de) coupling during sleep. *Curr. Opin. Phys.* **15**, 47–59 (2020). <https://doi.org/10.1016/j.cophys.2019.12.002>
22. A. Destexhe, S.W. Hughes, M. Rudolph, V. Crunelli, Are corticothalamic “up” states fragments of wakefulness? *Trends Neurosci.* **30**(7), 334–342 (2007). <https://doi.org/10.1016/j.tins.2007.04.006>
23. J. Feldman, The neural binding problem(s). *Cogn. Neurodyn.* **7**(1), 1–11 (2013). <https://doi.org/10.1007/s11571-012-9219-8>
24. R. Jerath, C. Beveridge, Multimodal integration and phenomenal spatiotemporal binding: a perspective from the default space theory. *Front. Integr. Neurosci.* **5**(13), 2 (2019). <https://doi.org/10.3389/fnint.2019.00002>
25. A.P. Yonelinas, C. Ranganath, A.D. Ekstrom, B.J. Wiltgen, A contextual binding theory of episodic memory: systems consolidation reconsidered. *Nat. Rev. Neurosci.* **20**(6), 364–375 (2019)
26. V.B. Dorokhov, V.V. Dementienko, L.G. Koreneva, A.G. Markov, V.M. Shakhnorovich, The electrodermal indices of the subjective perception of performance errors during drowsy changes in consciousness. *Zh. Vyssh. Nerv. Deiat. Im. I P Pavlova* **50**(2), 206–218 (2000)
27. P. Tassi, A. Bonnefond, O. Engasser et al., EEG spectral power and cognitive performance during sleep inertia: the effect of normal sleep duration and partial sleep deprivation. *Physiol. Behav.* **87**(1), 177–184 (2006). <https://doi.org/10.1016/j.physbeh.2005.09.017>
28. R. Vallat, D. Meunier, A. Nicolas, P. Ruby, Hard to wake up? The cerebral correlates of sleep inertia assessed using combined behavioral, EEG and fMRI measures. *Neuroimage* **184**, 266–278 (2019). <https://doi.org/10.1016/j.neuroimage.2018.09.033>
29. V. Dorokhov, S. Gruzdeva, O. Tkachenko, E. Cheremushkin, N. Petrenko, Experimental model of consciousness in the sleep-wake paradigm: determining consciousness activation using behavioral and Electromyographic indicators. *Proc. Comput. Sci.* **169**, 840–844 (2020). https://doi.org/10.1007/978-3-030-71637-0_49
30. I.M. Colrain, The K-complex: a 7-decade history. *Sleep* **28**(2), 255–273 (2005). <https://doi.org/10.1093/sleep/28.2.255>
31. I. Duman, I.S. Ehmman, A.R. Gonsalves, Z. Gültekin, J. Van den Berckt, C. van Leeuwen, The no-report paradigm: a revolution in consciousness research? *Front. Hum. Neurosci.* **11**(16), 861517 (2022). <https://doi.org/10.3389/fnhum.2022.861517>
32. N. Tsuchiya, M. Wilke, S. Frässle, V.A.F. Lamme, No-report paradigms: extracting the true neural correlates of consciousness. *Trends Cogn. Sci.* **19**(12), 757–770 (2015). <https://doi.org/10.1016/j.tics.2015.10.002>
33. E. Tulving, Episodic memory: from mind to brain. *Annu. Rev. Psychol.* **53**, 1–25 (2002). <https://doi.org/10.1146/annurev.psych.53.100901.135114>
34. N.J. Cohen, H. Eichenbaum, *Memory, amnesia, and the hippocampal system* (MIT press, Cambridge, 1993)
35. K. Henke, A model for memory systems based on processing modes rather than consciousness. *Nat. Rev. Neurosci.* **11**(7), 523–532 (2010). <https://doi.org/10.1038/nrn2850>
36. J.D. Gabrieli, Cognitive neuroscience of human memory. *Annu. Rev. Psychol.* **49**, 87–115 (1998). <https://doi.org/10.1146/annurev.psych.49.1.87>
37. M. Moscovitch, The hippocampus as a “stupid,” domain-specific module: Implications for theories of recent and remote memory, and of imagination. *Can. J. Exp. Psychol.* **62**(1), 62–79 (2008). <https://doi.org/10.1037/1196-1961.62.1.62>
38. S.B. Duss, T.P. Reber, J. Hänggi, S. Schwab, R. Wiest, R.M. Müri, P. Brugger, K. Gutbrod, K. Henke, Unconscious relational encoding depends on hippocampus. *Brain* **137**(Pt 12), 3355–3370 (2014). <https://doi.org/10.1093/brain/awu270>
39. T.P. Reber, K. Henke, Integrating unseen events over time. *Conscious. Cogn.* **21**(2), 953–960 (2012). <https://doi.org/10.1016/j.concog.2012.02.013>
40. L. Pacozzi, L. Knüsel, S. Ruch, K. Henke, Inverse forgetting in unconscious episodic memory. *Sci. Rep.* **12**(1), 20595 (2022). <https://doi.org/10.1038/s41598-022-25100-w>
41. I. Lerner, P.K. Pilly, A.A. Moustafa, Editorial: mechanisms contributing to sleep-dependent memory generalization. *Front. Neurosci.* **20**(16), 1106577 (2022). <https://doi.org/10.3389/fnins.2022.1106577>
42. S.A. Hall, D.C. Rubin, A. Miles, S.W. Davis, E.A. Wing, R. Cabeza, D. Berntsen, The neural basis of involuntary episodic memories. *J. Cogn. Neurosci.* **26**(10), 2385–2399 (2014). https://doi.org/10.1162/jocn_a_00633
43. E. van der Helm, N. Gujar, M. Nishida, M.P. Walker, Sleep-dependent facilitation of episodic memory details. *PLoS ONE* **6**(11), e27421 (2011). <https://doi.org/10.1371/journal.pone.002742>
44. L. Peter-Derex, M. Magnin, H. Bastuji, Heterogeneity of arousals in human sleep: a stereo-electroencephalographic study. *Neuroimage* **123**, 229–244 (2015). <https://doi.org/10.1016/j.neuroimage.2015.07.057>
45. P. Ruby, M. Eskinazi, R. Bouet, S. Rheims, L. Peter-Derex, Dynamics of hippocampus and orbitofrontal cortex activity during arousing reactions from sleep: an intracranial electroencephalographic study. *Hum. Brain Mapp.* **42**(16), 5188–5203 (2021). <https://doi.org/10.1002/hbm.25609>
46. U. Voss, Change in EEG pre and post awakening. *Int. Rev. Neurobiol.* **93**, 23–55 (2010). [https://doi.org/10.1016/S0074-7742\(10\)93002-X](https://doi.org/10.1016/S0074-7742(10)93002-X)

47. J.M. Windt, How deep is the rift between conscious states in sleep and wakefulness? Spontaneous experience over the sleep-wake cycle. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **376**(1817), 20190696 (2021). <https://doi.org/10.1098/rstb.2019.0696>

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