The electroencephalographic substratum of the awakening

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Abstract

The aim of the present study was to characterize the regional electroencephalographic substratum of the awakening process by means of a Hz-by-Hz EEG spectral power analysis. For this purpose, we recorded a group of 25 female subjects who slept for at least two consecutive nights in the laboratory. The postsleep waking EEG was compared to the one recorded during the presleep wakefulness from four midline derivations (Fz–A1, Cz–A1, Pz–A1, Oz–A1). Results indicated that the first 10 min after awakening are characterized by an increase of EEG power in the low-frequency range (1–9 Hz) compared to the corresponding presleep waking period, and by a significant decrease of EEG power in the beta range (18–24 Hz). As regards topographic differences, the increase of EEG power upon awakening in the delta–theta range showed a parieto-occipital prevalence. Moreover, the occipital derivation showed a larger decrease of power in the beta range as compared to the other derivations.

In conclusion, the EEG substratum of the sleep offset period is characterized by a pattern of increased EEG power in the delta–theta and low-alpha bands, and of decreased power in the beta range. This pattern could be considered as the spectral EEG signature of the sleep inertia phenomenon. The state of postsleep EEG hypoarousal does not subside in the first 10 min period after awakening considered in the present analysis. Finally, according to our results, the more posterior scalp locations show stronger EEG signs of sleep inertia, and could be the last ones to properly wake up.

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1. Introduction

Sleep inertia denotes a period of transitory hypovigilance, confusion and impaired cognitive and behavioral performance that immediately follows awakening. While the variables influencing sleep inertia have largely been explored (reviewed in [12,37]), its physiological underpinnings have received scarce attention. Nevertheless, the physiological data available clearly show that the sleep-to-wake transition is not a rapid shift from one state of consciousness to another, but a complex process that takes some time to be completed. As an example, it has incidentally been reported that cerebral blood flow (CBF) [28] and CBF velocity [22,26] immediately following awakening are reduced in comparison to daytime wakefulness. In addition, Hajak et al. [22] showed that, upon morning awakening, subjects required up to half an hour to reach CBF velocity values corresponding to the waking state of the previous evening. The delayed increases in CBF velocity after awakening strongly suggest an uncoupling of cerebral electrical activity and cerebral perfusion. More recently, H215 O PET was used to assess changes in regional CBF upon awakening from stage 2 sleep [3]. It was found that global CBF rates did not change during the first 20 min of wakefulness, indicating that the dissipation of sleep inertia is not a function of generally increasing levels of brain activation. Rather, the reactivation of local brain areas was critical. In fact, CBF was most rapidly reestablished in centrencephalic regions (e.g., brainstem and thalamus), suggesting that the reactivation of these regions underlies the reestablishment of conscious
awareness. Then, across the ensuing 15 min of wakefulness, further increases in CBF were evident primarily in anterior cortical regions (prefrontal association cortices).

Electrophysiological studies also support the notion that brain activation levels upon awakening largely differ from those characterizing wakefulness. Visual evoked potentials (VEP) recorded upon awakening have long been recognized as having decreased amplitude and increased latency of 100–300 ms components relative to waking [5]. Moreover, following one-fourth of the arousals from slow-wave sleep (SWS), VEP contained an apparent carry-over of typical SWS components. No similar changes in VEP were observed after awakenings from REM sleep [5]. A decrease of P300 amplitude immediately upon awakening as compared to the presleep measure has also been observed [39]; this effect persisted for up to 15 min following awakening. A similar amplitude drop of the P300 wave has recently been found after sudden awakening following as short as 3 min of sleep [4]. In addition, an amplitude reduction of the N1–P2 complex of the auditory evoked potential (AEP) during the sleep–wake transition as compared to presleep wakefulness has been reported [13]. Moreover, during recovery sleep the N1–P2 amplitude decreased at parieto-occipital sites, while it increased at the frontal midline derivation as compared to baseline, as a possible effect of a compensatory effort of the frontal areas for the increased homeostatic drive for sleep during the recovery night [16].

The only study that examined EEG power spectra during behaviorally identified (button pressing to stop a tone) spontaneous arousals from sleep, showed a non-predicted, gradual and continued drop of power in the delta, theta and sigma bands, extending well into the first few minutes of wakefulness [29].

Altogether, these studies confirm that a state of cortical hypopausal characterizes the waking period in the first few minutes after awakening from sleep. Moreover, it seems that the transition between sleep and wakefulness may show large regional differences, since the reactivation of some subcortical and cortical areas is faster than with other brain areas.

The first aim of the present study is to assess, for the first time, the EEG correlates of the awakening period after a whole night of sleep by means of a Hz-by-Hz EEG spectral power analysis. Moreover, the presence of regionally graded differences along the antero–posterior axis in the EEG substratum of the sleep offset period have also been evaluated.

2. Materials and methods

2.1. Subjects

Twenty-five normal right-handed female university students (age range: 20–26 years; mean age = 23.4 ± 1.08 years) were selected as paid volunteers for the study. They signed an informed consent, according to the Declaration of Helsinki. Their habitual sleep duration was between 7 and 8 h per night, their body mass index ranged from 17.5 to 23.0 kg/m², and their smoking habits were non-smokers or light smokers (less than 5 cigarettes per day). The subjects did not take any psychoactive medications, and were not involved in any clinical or experimental study involving drugs known to affect sleep or wakefulness. The data from 23 subjects were finally included in the analyses.

2.2. Procedure

The subjects participated in different research protocols, all approved by the local Institutional Review Board, sleeping for three to four consecutive nights in a sound-proof, temperature controlled room. All the subjects were recorded during the follicular phase of their menstrual cycle. The data reported here were collected during the second night that was an undisturbed baseline, always preceded by an adaptation night. Every night, the subjects arrived in the laboratory at about 21:30 h for electrode hook-up. Polygraphic sleep recordings started at 23:30 h (±30 min), according to the participants’ habitual sleep patterns and ended after 7.5 h of accumulated sleep, when an experimenter called the subjects via intercom (total sleep time was computed online by disregarding time spent in wakefulness). During the baseline night, just before lights-off, the subjects were requested to lay down in bed in the dark, and the presleep waking EEG was recorded for 10 min. The first 5 min were recorded with eyes-closed, while the following 5 min were recorded with eyes-open. Within 30 s of the final morning awakening (awakening time range: 7:50 to 8:40 h), the presleep waking EEG was again recorded for 10 min, as before. EEG recordings were checked online to guarantee that subjects did not fall asleep. Five subjects (two stage REM and three stage 2 awakenings) showed signs of incipient sleep (theta rhythm, stage 1) during the eyes-closed condition. In those cases, the stage 1 epochs were discarded from the analyses. Subjects were never called by name during the EEG recording, to avoid any arousing stimulus. Consequently, all the presleep and postsleep artifact-free EEG epochs considered in the analyses corresponded to the waking state, according to Rechtschaffen and Kales [31]. Twelve subjects were awakened from stage REM, while 13 subjects were awakened from stage 2.

An Essette Biomedica VEGA 24 polygraph was used for polygraphic recordings. EEG signals were high-pass filtered with a time constant of 0.3 s and low-pass filtered at 30 Hz. EEG was recorded from Fz, Cz, Pz and Oz sites, referred to the left mastoid (A1). Submental EMG was recorded with a time constant of 0.03 s. Bipolar horizontal and vertical eye movements were recorded with a time constant of 1 s. Bipolar horizontal EEG was recorded from electrodes placed about 1 cm from the medial and lateral canthus of the dominant eye, and bipolar vertical EEG from electrodes located about 3 cm above and below the right eye pupil. Electrode impedance was kept below 5 kΩ.

2.3. Quantitative analysis of signals

EEG, EMG and EOG signals were digitized online at a 128 Hz sampling rate, and stored on the hard disk of a computer. Artifacts were excluded offline on a 2 s basis by visual inspection. Power spectra of the four derivations along the antero–posterior axis (Fz–A1, Cz–A1, Pz–A1, Oz–A1) were computed by a Fast Fourier Transform routine for consecutive 2 s epochs, resulting in a frequency resolution of 0.5 Hz. Values above 25 Hz were not used in the analysis. By collapsing two adjacent 0.5-Hz bins (between 1 and 24 Hz), the data were reduced to a 1-Hz bin-width. Power spectra were calculated separately for eyes-closed and eyes-open wakefulness. Bins are referred to and plotted in this study by the lowest frequency included (e.g., the 1–5 Hz bin refers to the averaged values of the bins centered at 1.0 and 1.5 Hz).

2.4. Data analysis

Absolute power values were log-transformed before the statistical tests in order to approximate a normal distribution. Since preliminary mixed design ANOVAs showed that stage 2 and REM awakenings did not significantly differ for any single-bin Hz, the EEG power values of the two awakenings were averaged. Consequently, the differences between presleep and postsleep wakefulness in Hz-by-Hz EEG power were assessed by means of two-way ANOVAs. Recording Time (presleep versus postsleep by Derivation (Fz, Cz, Pz, Oz)) was the within-subjects factor, while Sleep Stage (stage 2, stage REM) was the between-subjects factor. When significant, the interaction between the two factors was strictly relevant to the aims of this study. In the absence of a significant interaction, the main effect for Recording Time was considered. The two 5-min waking periods were analyzed separately, given the different physiological state in which the EEG recordings were carried out (with eyes-closed, 1–5 min after awakening, and eyes-open, 6–10 min after awakening, respectively). When significant, the interaction was analyzed by post hoc tests.

To further explore the antero–posterior EEG power differences during the sleep offset period, an ANOVA was carried out on relative power values obtained by calculating, for each 1-Hz bin and for each derivation, the postsleep/presleep EEG power ratio. This analysis is particularly well suited to highlight the changes
in EEG power upon awakening as a function of the presleep EEG power levels (i.e., regardless of the absolute EEG power, which may largely differ between derivations). Even in this case, a preliminary mixed design ANOVA with Stage (REM, NREM) as between subjects factor and Derivation (Fz, Cz, Pz, Oz) as repeated measure factor did not show any significant main effect or interaction involving the Stage factor for any single-Hz bin. For this reason, the sleep Stage factor was collapsed and the relative values were compared by means of one-way repeated measure ANOVAs with Derivation. These analyses were again carried out separately for the eyes-closed and the eyes-open 5-min periods. Also in this case, post hoc t-tests were used to analyze significant main effects.

### 3. Results

Table 1 reports means (and standard errors) of the main sleep parameters during the experimental night, visually scored on the central derivation according to the standard criteria [31].

#### 3.1. Eyes-closed

Fig. 1 shows the EEG power in the 1–24 Hz range for each of the four midline derivations in the eyes-closed condition, recorded in the presleep period and immediately after awakening.

The 1 Hz bin showed a significant **Recording Time × Derivation** interaction ($F_{3,69} = 6.30; p < 0.001$), with increased EEG power upon awakening on all derivations (all $p < 0.01$). The interaction was present also for the 2 Hz bin ($F_{3,69} = 5.49; p < 0.002$), with t-test showing a significant increase of power upon awakening only on the parieto-occipital sites (Pz, $p < 0.05$; Oz, $p < 0.01$). As regards the 3 Hz bin ($F_{3,69} = 11.31; p < 0.0001$), post hoc tests indicated a significant postsleep increase of power only on the occipital lead ($p < 0.01$); on the contrary, the more anterior derivations showed a higher EEG power during the presleep wakefulness as compared to the awakening (Fz and

### Table 1

Visually scored sleep EEG parameters

<table>
<thead>
<tr>
<th>Stage</th>
<th>Means (S.E.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1 (min)</td>
<td>27.0 (4.8)</td>
</tr>
<tr>
<td>Stage 2 (min)</td>
<td>252.6 (9.8)</td>
</tr>
<tr>
<td>Stage 3 (min)</td>
<td>42.9 (2.9)</td>
</tr>
<tr>
<td>Stage 4 (min)</td>
<td>39.2 (6.1)</td>
</tr>
<tr>
<td>REM (min)</td>
<td>104.2 (3.4)</td>
</tr>
<tr>
<td>Intra-sleep wake (min)</td>
<td>36.0 (7.1)</td>
</tr>
<tr>
<td>Total sleep time (min)</td>
<td>466.0 (5.6)</td>
</tr>
<tr>
<td>Sleep efficiency index</td>
<td>0.91 (0.01)</td>
</tr>
<tr>
<td>Stage 1 latency</td>
<td>12.4 (2.7)</td>
</tr>
<tr>
<td>Stage 2 latency</td>
<td>16.4 (2.9)</td>
</tr>
<tr>
<td>REM latency</td>
<td>116.4 (10.9)</td>
</tr>
</tbody>
</table>

Means and S.E. (within brackets).

Fig. 1. Absolute EEG power ($\pm$ S.E.M.) in the 1–24 Hz range (plotted on a logarithmic scale) in the eyes-closed condition of presleep wakefulness (open circles) and of postsleep wakefulness (filled circles, recorded in the first 5 min after awakening).
Cz, \( p < 0.01 \). Similarly, the 4 Hz bin (\( F_{3,69} = 7.44; p < 0.0002 \)) was characterized by a decrease of EEG power on the fronto-central derivations in the postsleep as compared to the presleep period (Fz and Cz, \( p < 0.01 \)).

ANOVA also showed a significant interaction at 6 and 7 Hz (\( F_{3,69} = 2.64; p < 0.05 \) and \( F_{3,69} = 3.24; p < 0.05 \), respectively), with higher power upon awakening on all the derivations (6 Hz: Fz, Cz: \( p < 0.05 \), Pz, Oz: \( p < 0.01 \); 7 Hz: all \( p < 0.01 \)), and a main effect for Recording Time at 8 and 9 Hz (\( F_{1,23} = 28.29; p < 0.0001 \) and \( F_{1,23} = 19.08; p < 0.0002 \), respectively), again indicating a generalized higher EEG power upon awakening.

The interaction was significant also at 11 and 12 Hz (\( F_{3,69} = 2.66; p < 0.05 \) and \( F_{3,69} = 5.59; p < 0.01 \), respectively), with a decrease of EEG power upon awakening on all the derivations except the central at 12 Hz (all \( p < 0.01 \)).

Finally, the same effect was present for several bins in the beta (18–23 Hz) range (18 Hz: \( F_{3,69} = 3.43, p < 0.05 \); 19 Hz: \( F_{3,69} = 5.53, p < 0.01 \); 20 Hz: \( F_{3,69} = 4.51, p < 0.01 \); 21 Hz: \( F_{3,69} = 4.14, p < 0.01 \); 22 Hz: \( F_{3,69} = 3.54, p < 0.05 \); 23 Hz: \( F_{3,69} = 2.14, p < 0.10 \)). The post hoc comparisons showed that, for each bin and derivation, EEG power was higher during the presleep waking period compared to the awakening (all \( p < 0.01 \)).

3.2. Eyes-open

The eyes-open condition (recorded in the 6–10 min after awakening) largely confirms the above-mentioned pattern of results (see Fig. 2).

Here, again the fronto-central and the parieto-occipital sites showed a different behavior in the delta range. In fact, the Recording Time \( \times \) Derivation interaction was significant across the 1–4 Hz range (1 Hz: \( F_{3,69} = 4.48, p < 0.01 \); 2 Hz: \( F_{3,69} = 8.89, p < 0.0001 \); 3 Hz: \( F_{3,69} = 13.67, p < 0.00001 \); 4 Hz: \( F_{3,69} = 11.33, p < 0.00001 \)). Post hoc tests pointed out that EEG power at 1 Hz was higher upon awakening that during presleep on Pz and Oz leads (\( p < 0.01 \)), and only on the occipital derivation at 2–4 Hz (all \( p < 0.01 \)). On the other hand, the fronto-central derivations showed a decrease of power after awakening in the 2–4 Hz range (2 Hz: \( p < 0.05 \); 3–4 Hz: \( p < 0.01 \)).

The main effect for Recording Time in the 7–9 Hz (7 Hz: \( F_{3,69} = 9.16, p < 0.01 \); 8 Hz: \( F_{3,69} = 27.67, p < 0.0001 \); 9 Hz: \( F_{3,69} = 24.52, p < 0.0001 \)) and 11–12 Hz ranges (11 Hz: \( F_{3,69} = 26.85, p < 0.0001 \); 12 Hz: \( F_{3,69} = 20.49, p < 0.0001 \)) confirmed also in the eyes-open condition the increase of upper theta–lower alpha power ranges and the decrease of high-alpha power upon awakening.

**Fig. 2.** Absolute EEG power (± S.E.M.) in the 1–24 Hz range (plotted on a logarithmic scale) in the eyes-open condition of presleep wakefulness (open circles) and of postsleep wakefulness (filled circles, recorded in the 6–10 min after awakening).
Finally, as in the eyes-closed condition, a significant interaction was found in the 19–24 Hz range (19 Hz: $F_{3,69} = 3.07, p < 0.05$; 20 Hz: $F_{3,69} = 5.05, p < 0.01$; 21 Hz: $F_{3,69} = 4.20, p < 0.01$; 22 Hz: $F_{3,69} = 2.16, p < 0.10$; 23 Hz: $F_{3,69} = 3.60, p < 0.05$; 24 Hz: $F_{3,69} = 3.29, p < 0.05$). Post hoc comparisons showed that, for each bin and derivation, EEG power was decreased after awakening compared to the presleep waking period (all $p < 0.01$).

3.3. Relative power

Fig. 3 shows the relative EEG power (postsleep/presleep) across the 1–24 Hz range for each scalp location, respectively, for the eyes-closed (left side) and eyes-open (right side) conditions. The bottom part of the figure shows the ANOVA $F$-values and the corresponding significance levels ($p < 0.05$ and $p < 0.01$).

The relative power data highlight that the differences between presleep and postsleep EEG power are larger on the parieto-occipital derivations in the delta–theta range and on the occipital derivation in the alpha–beta range.

More specifically, during the eyes-closed condition the increase of EEG power upon awakening in the 1–10 Hz range showed a posterior (Pz–Oz) prevalence (see Table 2 for the detailed post hoc comparisons). Moreover, the decrease of power in the alpha–beta range (11–12 and 15–23 Hz) was significantly higher on Oz.

The eyes-open condition showed a similar pattern, characterized by a larger increase in the delta-theta EEG power upon awakening (1–6 Hz) on the more posterior scalp locations (Pz and Oz), and by a larger decrease of beta power (19–24Hz) on the occipital derivation (see Fig. 3, right side and Table 2).

4. Discussion

In the present study, we showed that the EEG substrate of the first 10 min after awakening from sleep is mainly characterized by a general pattern of increased power in the delta-theta and low-alpha EEG range, and of decreased power in the beta range, compared to presleep wakefulness. This pattern of electroencephalographic deactivation could be considered as the spectral EEG signature of the sleep inertia phenomenon. Moreover, we reported for the first time that substantial topographical differences characterize the sleep offset period: in fact, the parieto-occipital scalp locations evidenced stronger EEG signs of hypoarousal upon awakening, showing the largest increases of EEG power in the delta-theta range and the largest decreases of power in the beta range upon awakening compared to the fronto-central sites.

4.1. A possible physiological substratum of the sleep inertia phenomenon

The present detailed, Hz-by-Hz, analysis of the waking EEG recorded immediately after awakening from a whole night of sleep pointed out that the sleep offset period is characterized by a pattern of decreased EEG power in the higher frequencies and...
of increased power in the low-frequency range compared to the presleep waking period.

A higher waking EEG power in the low-frequency range (1–9 Hz) is a well-known effect of total sleep deprivation (e.g., [17]). Accordingly, higher theta and alpha activity is also associated with subjective sleepiness and with errors or failures to respond to external signals in nocturnal workers [2,38]. In other words, these spectral changes can be considered as consistent indexes of increased sleepiness or of decreased vigilance. The fact that we have found the same EEG features upon awakening indicate that sleepiness and sleep inertia could share similar underlying psychophysiological processes.

As may easily be seen in Fig. 1, the increase of alpha power upon awakening is accompanied by a shift of the peak frequencies from 9–10 to 7–8 Hz. Interestingly, a similar slowing of the dominant frequency of the alpha band has recently been reported as one of the EEG correlates of the vigilance decrement in narcolepsy [32]. Clearly, the functional meaning of this slow-alpha activity should be recontextualized to the EEG synchronization process [8,15,18], supporting the idea that the sleep inertia effects may reflect the intrusion of residual EEG characteristics of sleep into the waking state. The decreased power in the beta range upon awakening is not surprising: in fact, beta activity is considered to reflect cortical activation [34] that seems to be generally low upon awakening. However, decreases of beta power as a function of increasing sleepiness have not always been found. As an example, a careful and analytic examination of the EEG power during 36 h of sleep deprivation did not show significant changes in the frequency bins above 17 Hz [17]. On the contrary, others have reported counterintuitive increases in beta power after sleep deprivation, attributing this effect to an effort to maintain adequate levels of alertness [6–7,20,27]. On the other hand, and in agreement with our findings, it has been shown that caffeine significantly antagonizes the detrimental effects of sleep deprivation on EEG and psychomotor performance compared to placebo, producing a decrease in delta and theta power and an increase in alpha and beta power (e.g., [30]). Accordingly, the analysis of resting EEG in a group of untreated narcoleptic patients characterized by excessive daytime sleepiness, by means of low-resolution brain electromagnetic tomography (LORETA), showed a decrease of power in the beta band compared to normal controls [33]. The reasons for these discrepancies are at present unclear.

In the only other study of EEG power upon awakening, Ogilvie and Simons [29] reported a slow decrease of power in the delta, theta and sigma bands. Even though our results are not completely overlapping, the discrepancies may be accounted for by the large methodological differences between the two studies (Hz-by-Hz versus EEG bands analysis; provoked versus spontaneous awakenings; analysis of periods of wakefulness after a whole night of sleep versus intra-sleep wakefulness; longer versus shorter periods of FFT-analyzed wakefulness).

Interestingly, the picture of results was substantially similar both in the eyes-closed and eyes-open condition. First, this outcome indicates that the state of hypoausorality does not subside in the first 10 min after awakening, confirming that the sleep-to-wake transition may take a long time to be completed. The fact that EEG signs of sleep inertia are still present 10 min after awakening is not surprising. In fact, it has been shown that global CBF does not change during the first 20 min of wakefulness [3]. Upon morning awakening, more than 30 min can be required to reach CBF velocity values corresponding to the waking baseline [22]. Also from a cognitive and behavioral point of view, it has been reported that the time course of sleep inertia dissipation ranges between 30 and 75 min [1,14,24].

These results also show that the EEG substratum of sleep inertia is largely the same both in a “sleep-conducive” situation (eyes-closed, just upon awakening) and in a more alerting condition (eyes-open, 6–10 min after awakening). The presence of a persistent EEG pattern of hypoausorality even when subjects are in a more arousing situation (eyes-open) was in part unexpected. In our view, the latter result further underlines the presence of a strong and enduring down-regulation of the brain’s electrical activity upon awakening that may have important practical implications (e.g., [12]).

Finally, as a partial limitation of the present results, it has to be born in mind that we did not directly assess sleep inertia by means of a behavioral measure. In fact, based on a large body of evidence (for reviews, see [12,37]), we just assumed the presence of a sleep inertia effect, because its assessment would have prevented the recording of an artifact-free EEG.
4.2. Antero–posterior EEG changes during the sleep offset period

Here, we showed for the first time that the more posterior scalp locations show stronger EEG signs of hypoarousal upon awakening, as indicated by the larger increase of power on the parieto-occipital sites in several bins of the delta and theta range, accompanied by a greater decrease of EEG power in the alpha and especially in the beta range compared to the other derivations. These results are in agreement with another electrophysiological study showing a specific amplitude reduction of the N1–P2 complex of the auditory evoked potential at the parieto-occipital sites during the sleep–wake transition [16]. As far as delta power is specifically concerned, it is of note that the frontal-central and parieto-occipital derivations showed opposite behaviors, both in the eyes-closed and eyes-open conditions. In fact, while the posterior scalp locations evidenced an increased EEG power upon awakening, the more anterior ones showed a higher EEG power during the presleep waking as compared to the awakening. As highlighted by the relative power data (see Fig. 3), even for the 1 Hz bin, the increase of power upon awakening, although present on all the derivations, is larger on the posterior derivations.

The present results seem to indicate that the more anterior areas show strong signs of EEG synchronization already in the presleep period, while the more posterior ones are mostly interested by the sleep inertia effects. They can be interpreted in the light of the frequency-specific and topographical EEG changes observed at sleep onset in humans. In fact, it has been shown that in the presleep period the <7 Hz EEG power is greater at the more anterior scalp locations. This anterior prominenes is paralleled by a prevalence across the >8 Hz frequencies of the EEG activity at the occipital site [9]. These data strongly suggested that sleep does not necessarily begin simultaneously in all cortical areas, the posterior areas being the last one to synchronize their EEG activity [9]. This findings received further support by a study estimating, by means of the directed transfer function (DTF), the direction of the information flow underlying cortico-cortical functional coupling [10]. The presleep period was characterized by posterior-to-anterior functional cortical coupling, while at sleep onset there was an inversion of that direction. The occipitofrontal propagation during the presleep interval has been interpreted as reflecting the information flow of the occipitofrontal and superior longitudinal fasciculi along postero-anterior projections, subserving a passive bottom-up transfer of information from visual sensory to prefrontal association areas [10,25]. On the other hand, a spread of synchronizing signals from associative prefrontal to posterior areas may play a role in the wake–sleep transition.

Accordingly, the present data suggest that wakefulness does not necessarily begin simultaneously in all cortical areas, as indeed showed also by intra-cellular recordings [35]. The picture seems to mirror that shown at sleep onset: the posterior areas could be the last one to desynchronize their EEG activity. In other words, we could speculate that the more posterior brain areas are the last ones to fall asleep, as well as the last ones to properly wake up. The asynchronous development of a normal brain’s arousal state upon awakening may have strong adaptive implications: as a matter of fact, the human brain is characterized by a “hyperfrontality”, that is maintained even during rested wakefulness [21,23]. As reported, the frontal lobes are the first ones to fall asleep; moreover, they typically show an increased local use-dependent sleep intensity (e.g., [18]).

4.3. NREM–REM awakenings

Surprisingly, postawakening EEG did not show any significant difference by comparing REM and stage 2 NREM awakenings. Other studies have already failed to show differences in performance after arousal from stage 2 and REM (e.g., [24,36]). However, we do believe that the lack of any difference in EEG power upon stage 2 and REM awakenings was due to the high variability of individual patterns of EEG power distribution which is, on the other hand, highly stable within individuals [11,19]. Therefore, the current between-subject design comparing REM and NREM awakenings of different groups was not best suited to show the differential effects of stage 2 and REM awakenings on the ensuing EEG power features. Within-subject studies with large groups will possibly confirm that the down-regulation of the brain’s electrical activity at sleep offset is dependent on the EEG features of the sleep stage at awakening.

5. Conclusions

Summarizing, our FFT analysis of the sleep offset period showed that the sleep–wake transition is characterized by an EEG pattern of decreased beta power and of increased power in the delta–theta–lower alpha range. This state of hypoarousal that does not subside in the first 10 min after awakening could be considered the EEG substratum of the sleep inertia phenomenon. With reference to the regional differences of the awakening process, our results demonstrated that the parieto-occipital derivations show stronger EEG signs of sleep inertia compared to the more anterior scalp locations, possibly being the last ones to properly wake up.

References


