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Review

Sleep EEG pattern in childhood: from newborn through adolescent

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Abstract Sleep is as important for good health as diet and exercise. It is particularly important for children and adolescents, as they need to grow and develop. Quality sleep is necessary to ensure the plasticity of the brain, growth and maturation, and development and improvement of mental abilities, to prevent some chronic diseases. Younger the child, the more is the time he sleeps. It is well known that each behavioral state, characterized by certain physiological parameters, electroencephalography (EEG) activity and phenomena, is changed across the life span. An advanced EEG monitoring can improve our understanding of the brain neuronal activity through studying the sleep patterns. In age-related neurophysiology, importance is attached to the formation and temporary changes in such EEG patterns such as slow-wave activity (SWA), sleep spindles (SSs), vertex waves (V-waves), K-complexes (KKs) and positive occipital sharp transients (POSTs). The features of the formation of bioelectrical activity during sleep and various sleep EEG pattern in newborns depending on the conceptual age and the further maturation of the sleep EEG as the child grows and develops are widely discussed. However, one of the main sleep EEG phenomena which occur in non-rapid eye movement (non-REM) sleep 2, associated with a wide range of brain functions, such as memory and neuroplasticity, general intelligence and cognitive performance, and undergo changes throughout life, are SSs. This review attempts to summarize the available literature data on the formation of the main EEG sleep patterns in childhood and adolescence, especially SSs, and also identifies some studies conducted at the our Scientific center on age-related neurophysiology and EEG sleep characteristics and their associations with some diseases in middle adolescence. Modern methods of sleep assessment and its EEG patterns are the next step in understanding the neurophysiological ontogenetic aspects of the sleep–wake cycle. All this will open up perspectives and «windows of opportunity» in predicting postnatal maturation, understanding the mechanisms of brain neuroplasticity and memory consolidation in sleep, which is one of the tasks of modern somnology and neurophysiology.

1 Introduction

In human life, there are three primary behavioral states characterized by certain physiological parameters: wakefulness, as an activated electroencephalogram (EEG) presented by alpha waves, and characterized by high muscle tone and gross body movements; non-REM sleep (quiet sleep in newborns), as inactivated EEG tetha rhythm, sigma and delta waves (slow waves), characterized by decreased muscle tone and behavioral quiescence; and REM sleep (active sleep in newborns) as an activated EEG by alpha waves and characterized by the absence of muscle tone and the presence of rapid eyes movements (REMs) [1]. In neonates, sleep time dominates over wakefulness, and active and quiet sleep each occupies 8 h of the day. As active sleep decreases with age, the amount of time spent awake increases (Fig. 1).

In 1966, the ontogenetic hypothesis of sleep has been put forward by Howard Roffwarg et al. It was the first attempt to explain the great quantities of REM sleep during early development. Researchers looked into the brainstem activity that drives REM sleep, including the forebrain activation and motor events. They believe that a high percentage of active sleep in both neonatal period and early childhood is important for autostimulation, because in these ages «waking life is limited in time and scope and offers little occasion for stimulation» [2],

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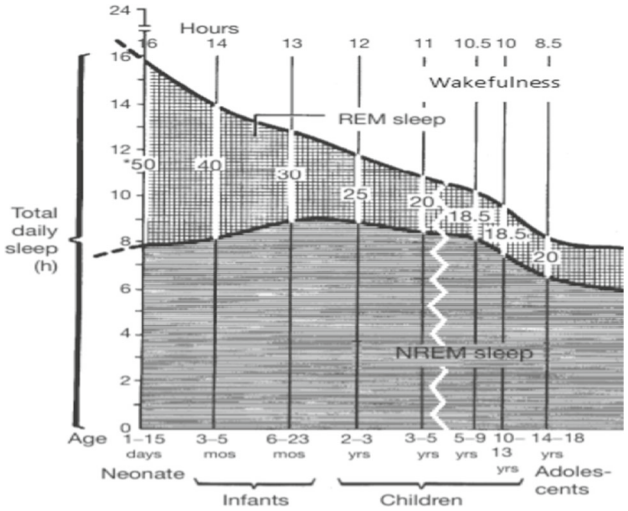

Fig. 1 Relative rates of wakefulness, non-REM sleep, and REM sleep from neonates to adolescents (revised from [2]). *REM* rapid eye movement

and active neural stimulation arising from the brainstem during REM sleep substitutes for the lack of wakefulness stimulation [3]. They have also suggested that it assists in a variety of developmental processes, as well as functional brain maturation, namely, neuronal differentiation and myelinization in higher nervous centers and the development and improvement of cognitive abilities.

It should be noted that synaptogenesis, apoptosis, and myelination are critical in the maturation of functional neuronal circuit from neonate stage to adolescence. These processes determine the physiological neuroplasticity, which is reflected in the formation and development of various neurophysiological patterns in different behavioral states. It is known that slow-wave activity (SWA, 1–4.5 Hz) during non-REM sleep reflects cortical maturation and reaches a maximum during the middle of childhood and exponentially declines during adolescence. Some investigations in children indicate that myelin is an integrative component of the propagation dynamics of SWA across the scalp. Thus, overall evidence is increasing that SWA is connected to myelin growth [4]. There is posteroanterior trajectory (from back to front) of SWA maturation across childhood (Fig. 2) [5].

Recent researches have defined this phenomenon as the ratio of frontal/occipital SWA (F/O ratio) [6]. It has been proven that development of SWA parallels major changes in cortical maturation (e.g., the formation and subsequent elimination of synapses) and SWA distribution is increased in scalp regions showing structural and behavioral maturation. Interestingly, the posteroanterior maturation of SWA topography precedes the maturation of gray matter thickness as well as the maturation of motor skills by ∼3.7 years [5].

It should be noted that another important sleep pattern associated with brain maturation and neuroplasticity includes sleep spindles (SSs), which undergo various changes across the life span [7]. SSs for the first time were described by Loomis et al., in 1935 during a scalp surface EEG [8], as the "flash" on the sleep EEG of spindle-shape, which appears increases and disappears within about 1 s during non-REM sleep. The SSs has a frequency of 9–16 Hz (most commonly 12–14 Hz) [9]. In fact, several studies have revealed that there are two types of SSs with differences in frequency: slow (about 10 Hz) and fast (about 13 Hz), with different topographical distributions [10]. Recently, it has been shown that EEG sleep activity at 11.50 Hz exhibits specific regional changes during the first 48 months of life [11]. This EEG activity has a positive relation between age and the EEG rhythm on the

Fig. 2 Posteroanterior maturation of slow-wave activity topography across childhood and youth (revised from [5])

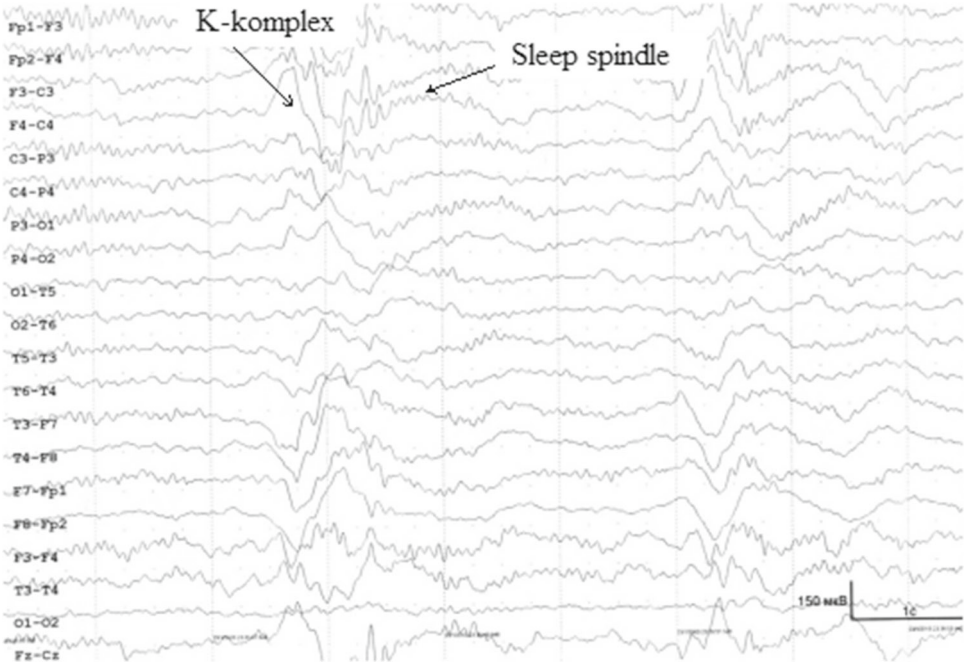

Fig. 3 K-complexes are high-amplitude biphasic waves, followed by sleep spindles

frontal cortex. It is known that SSs characterized by the following quantitative characteristics: density (number of SSs per 1 min non-REM sleep), amplitude (voltage difference between SSs peaks, in microvolts, μV), duration (in seconds), and frequency (number of SSs per 1 s. non-REM, in hertz, Hz). SSs characteristics are assessed in total for NREM sleep across the night and for each sleep period separately [12]. The SSs are generated in the reticular thalamus nucleus by inhibitory neurons (i.e., GABA), transmitted to the thalamic relay nuclei, and then expressed in the cerebral cortex through a network of reciprocal thalamo-cortical pathways. Despite the fact that SSs are generated in the thalamus, their expression and synchronization are fully coordinated by thalamo-cortical neural networks [13]. The results of recent studies indicate that the cerebral cortex can initiate the expression of SSs through feedback from the thalamus, thereby causing their generation [14].

In sleep studies, other phasic events such as vertex waves (V-waves) and K-complexes (KKs) are also usually evaluated as the hallmark graphic elements of non-REM sleep [15]. V-waves are triphasic sharp waves with 50–200 ms duration, localized over the precentral area and observed of non-REM 1 and 2. KKs consist most commonly of a large-amplitude diphasic slow-wave frequently associated with SSs (Fig. 3).

These phasic events can occur spontaneously or in response to a sudden sensory stimulus during sleep [16].

An EEG allows us to observe any neurophysiological phenomenon during wakefulness as well as in sleep. It is one of the safest, most accessible and adequate methods for assessing the functional brain state, its maturation and the formation of bioelectrical activity across the childhood. The sleep EEG objectively quantifies the need for recovery of neuronal networks.

Further, we presented the results of an analysis of existing literature regarding the formation and development of various neurophysiological sleep phenomena in children of different age groups, their features in ontogenesis, as well as some data of our own research.

2 Specific sleep EEG patterns in newborns

It is interesting that each period of childhood has specific features and phenomena of sleep EEG, which are associated with brain maturation. The number of research among newborns using surface EEG has allowed us to re-evaluate the features of the generation of neuronal oscillations on early brain development [17]. EEG curves during this period of childhood are characterized by intermittent temporal organization with both activity and no activity periods. It should be noted that normal developmental landmarks on a newborn's EEG provide information regarding the functional maturity and reflect the conceptual age (CA) of the baby. Developmental landmarks are best categorized into three phases: < 30 weeks, 30–37 weeks, and > 37 weeks. Synchrony of the bursts and amplitude symmetry in the three behavioral states (wakefulness, non-REM sleep, and REM sleep), as burst of

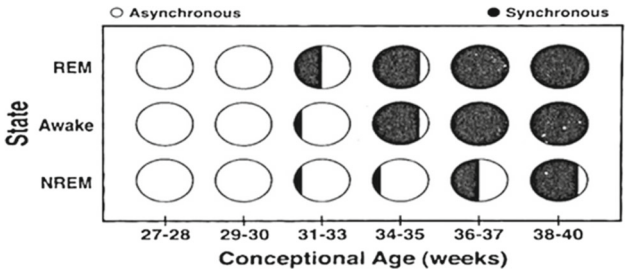

Fig. 4 Synchrony features of neonatal EEG according to CA [18]. *EEG* electroencephalogram, *CA* conceptual age

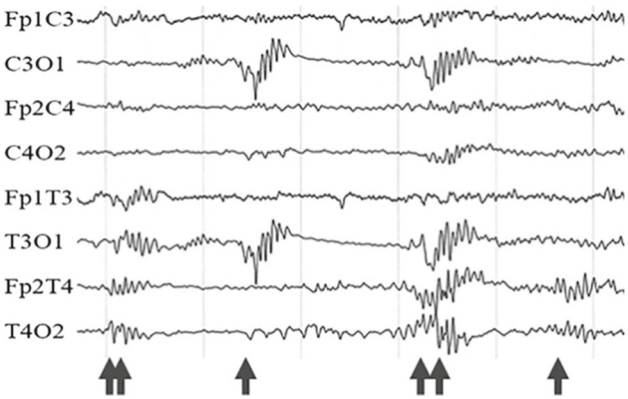

Fig. 5 Delta brushes in a 32-week CA neonate during quiet sleep [21]

morphologically similar activity in the homologous head regions, is established only after 37–38 weeks of CA (Fig. 4).

The normal EEG pattern of premature neonates is a discontinuous pattern, alternating between high-amplitude and low-amplitude activity periods [19], with superimposed fast oscillations (8–25 Hz) [20] form, so-called beta–delta complexes or «delta brushes» (DBs) (Fig. 5).

DBs can be present from 27 weeks CA and are the dominant rhythm of the premature neonatal EEG [21]. The presence of DBs reflects the development and maturation of the cerebral cortex in a premature neonate: formation of bioelectric activity and neuronal plasticity, and their absence indicating pathology of the central nervous system [17, 22]. The amplitude of the superimposed fast activity in DBs is usually 20–50 μV, which, in combination with the delta wave's amplitude, can reach 200 μV. DBs are located asynchronously in the ipsilateral regions of both hemispheres [23]. DBs topographies across CA are interesting: they appear pericentrally and then shift to the temporal–occipital regions from 30 to 34 weeks CA. If children were born after 28 weeks of pregnancy, DBs can be detected in the central, occipital and parietal regions [24]. Scientists have proven that spontaneous twitching of the arms and legs, as well as their direct stimulation, causes the appearance of DBs in the corresponding areas of the cerebral cortex [25]. Up to 33–34 weeks CA, these patterns can occur both during sleep and wakefulness; however, as the brain matures, they are limited by the period of quiet sleep. The maximum activity of DBs is observed between 32 and 34 weeks CA, but then the amplitude and density decrease by 40 weeks CA [26]. The temporal and spatial development of SSs and their timely extinction are important indicators of the brain maturation both in premature and full-term newborns. These phylogenetically immature EEG patterns, reminiscent of the sigma rhythm of quiet sleep in older children, are the «precursors» of SSs.

EEG activity becomes more continuous, leading to a «tracé alternant», with the establishment of functional interactions within the cortical neuronal networks at term [27]. It is known that between 37 and 40 weeks CA, the EEG is continuous and similar during awake and active sleep states. During quiet sleep, there is tracé alternant with some periods of continuous slow-wave sleep. EEG is completely synchronous and reactive to internal or external stimuli. Between CA 40 and 44 weeks, the EEG is continuous during the awake, active sleep and continuous slow-wave sleep portion of quiet sleep. EEG is reactive in all states and synchronous. Between CA 44 and 46 weeks, the EEG is continuous in all behavioral states [28].

Normally, some transient EEG patterns are recorded in full-term newborns. There are continuous activity (CSWS) and «tracé alternant» (TA) [29]. CSWS is a trace with a steady amplitude, low- and high-amplitude theta and delta waves (Fig. 6a), TA is a specific EEG pattern during quiet sleep characterized by alternating periods of high-voltage burst intervals and low-amplitude interburst interval (Fig. 6b).

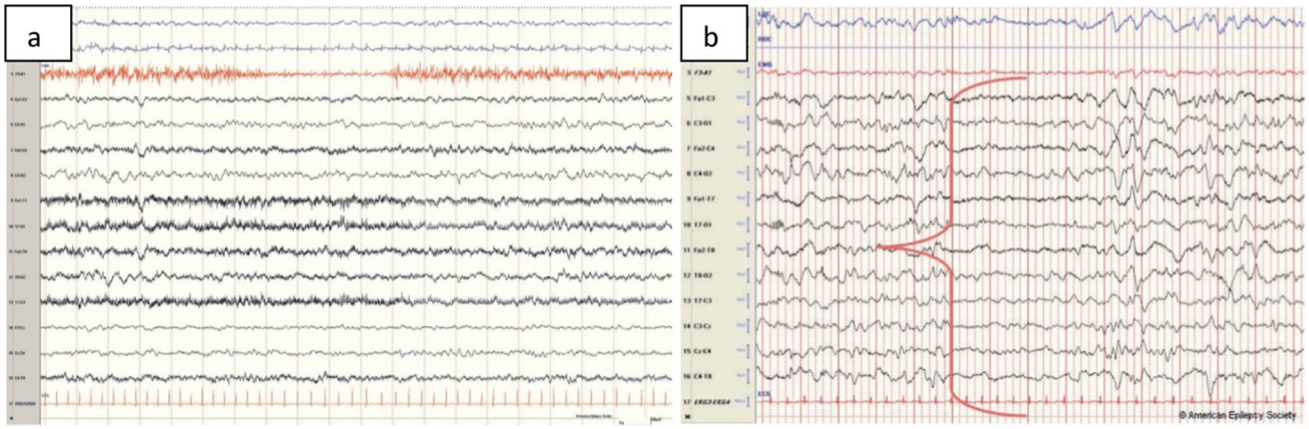

Fig. 6 Transient EEG patterns in term babies: continuous activity (**a**) and «tracé alternant» (**b**) [28]

At 39–41 weeks CA, DBs are only observed during quiet sleep, with an amplitude < 200 μV. The mature frontal transient appearance is a good maturational index. They are clearly differentiated in frontal areas during active sleep 1 and the onset of quiet sleep. Slow anterior dysrhythmia, as short bursts of monomorphic or polymorphic slow waves of 1–3 Hz with an amplitude of 50–100 μV, are registered in the frontal areas during active sleep 1. The appearance of clearly differentiated short-burst theta waves in the centrotemporal area of brain, which are sometimes sharp and of high amplitude, are observed during quiet sleep.

Another sleep EEG pattern of mature infants is slow continuous tracing («tracé lent continu») which is present during quiet sleep. It is characterized by continuous delta-wave activity (1–3 Hz), with occipital predominance and a variable amplitude of 50–200 μV. If the «tracé alternant» and «tracé lent continu» occur in the same newborn, the last one precedes the first one.

It should be noted that abnormal features or EEG activity disturbances should warn physicians of possible neurological complications in newborns and infants. Further researches are nevertheless required to define the neurodevelopmental prognostic value of possible abnormalities of young child.

3 First 3 years of life: what happens with sleep EEG?

Many further maturational changes unfold during the first 3 years of life. This is a critical period during which the thalamocortical brain connections and gray matter of the cerebral cortex develop and mature [30]. It is known that synaptogenesis, apoptosis, and myelination determine the physiological neuroplasticity characteristic of this period of ontogenesis, which is reflected in the formation and development of various neurophysiological patterns, including during sleep [31]. Multiple changes in the brain morphology and functioning during child's development reveal an occipital–frontal trajectory of cerebral cortical maturation, accompanied by regional modifications of the non-REM sleep.

One of the characteristic EEG patterns during non-REM sleep and one of the most prominent markers of it regional modifications are SSs [32]. According to a number of researches, these EEG patterns serve as indicators of neuronal integration, their development depends on the number of interneuron connections, and their maturation is characterized by a certain trajectory. SSs appear for the first time in early infancy [33]. SSs first appear at 4–7 weeks in 50% and at 12 weeks in about 100% of babies. They have a particular morphology (a spiky negative component and a rounded positive component), are often asynchronous, short, low voltage (20 μV peak-to-peak) and infrequent ($<$ 3–4 per hour of quiet sleep). By 3–4 months of life, SSs increase in amplitude (up to 30–50 μV), duration and density (up to 3–4 per min. of quiet sleep). At 2–6 months of age, frequency peaks of SSs at13–13.5 Hz are noted (Fig. 7) [34].

SSs between ages 8 and 12 months often have a "comb-like" shape. Some scientists have shown that between 4 and 12 months of age, the frequency and density of fast SSs (12–14 Hz) increase during quiet sleep (Fig. 8), and they are recorded in the EEG over most parts of the cerebral cortex, except for the occipital ones, with a maximum in the central and frontal areas [34].

This may occur due to the maturation of the deep brain structures, responsible for providing activation processes of the cerebral cortex, followed by an "avalanche-like" increase in the number of cortical synapses, so-called enhanced synaptogenesis [35].

Between 13 and 24 months of life, the activity of fast SSs decreases, they become more synchronous, and their "sharpness" disappears, possibly due to an increase in the white and gray matter of the cerebral cortex by 88%

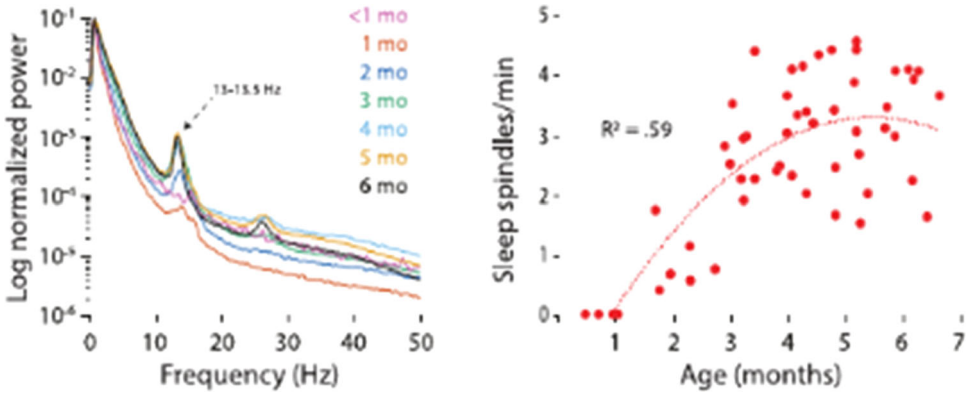

Fig. 7 Increase in the rate and power of sleep spindles over a child's development (revised from [34])

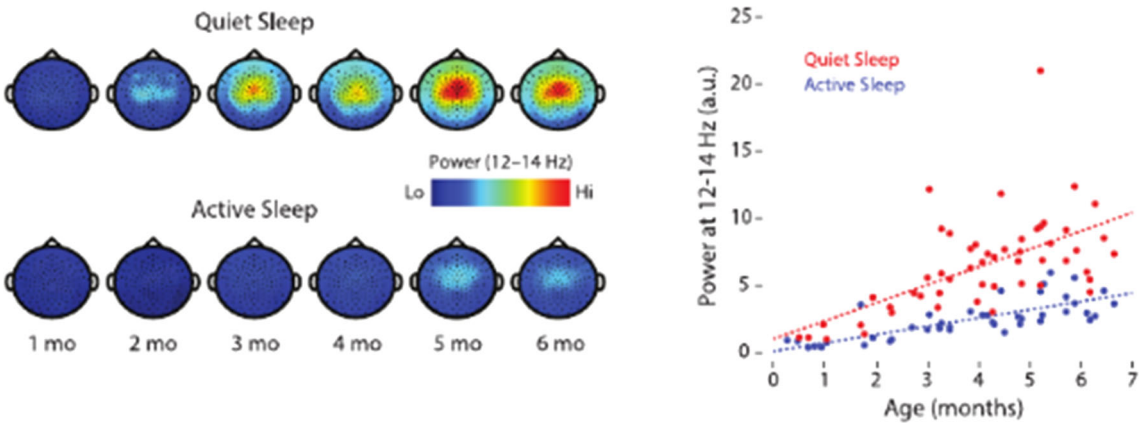

Fig. 8 Topoplots showing sleep-spindle power (12–14 Hz) during quiet sleep and active sleep in infants (revised from [34])

and the degree of its maturation [36], followed by the formation of full-fledged SSs. From 2 years of age, the density of slow SSs (11 Hz, a frequency of the fast alpha rhythm) begins to progressively increase. They begin to dominate in the prefrontal and frontal cortex areas and reach a maximum by 4 years of life that corresponds to the posteroanterior trajectory (from the occipital to the frontal regions) of the appearance of slow SSs [10]. Knowledge of these SSs features in the early childhood is necessary when EEG is performed (the lowest limit of the frequency filter for the sigma range should be 11 Hz). The duration and density of SSs also indicate the degree of brain maturation. By 18 months of age, the duration of SSs decreases and remains minimal until 3 years, then increases sharply. At the same time, the interval between SSs during this period is the largest in comparison with younger and older age groups of children [37]. Thus, the SSs undergo certain topographical and morphological changes during the child's maturation.

Other sleep EEG patterns, as V-waves and KKs, develop between 3 and 6 months, and a slower frequency background of 1–3 Hz predominates during sleep. They configuration changes from blunt to sharp by 12 month of life. REM sleep begins to diminish from approximately 50% level of active (REM) sleep seen in newborns, to about 40% of sleep time by age 3–5 months, reaching 30% by 12–24 months of age [38].

Simultaneous changes in sleep EEG pattern and cerebral cortex areas in early childhood may indicate the presence of certain causal relationships, which is a "window of opportunity" for diagnosing and predicting the adequate maturation of the higher cortical functions in children and/or its disorders.

4 Sleep EEG maturation in preschoolers and schoolchildren

The study of the EEG phenomena changes, especially during periods of active socialization and processing a lot of information, constant intellectual loads, and their intensification during learning, as well as in puberty, is an integral part of neurophysiological research of sleep in the ontogenetic aspect.

It should be noted that other EEG patterns such as positive occipital sharp transients (POSTs) appear in the non-REM 1–2 sleep between 3 and 5 years of age. They become more frequent from 6 to 12 years of age [34]. Earlier it has been found that the incidence of POSTs as a normal component of the human sleep EEG is fewer in number among young children than adults [39].

Common non-REM sleep EEG features including POSTs, V-waves, KKs, and SSs become fully developed in school-aged children (Fig. 9).

The most closely related to dependent of memory consolidation, the development of general cognitive abilities, intelligence in the process of active cognition of the around world, and neuroplasticity in childhood and adolescence are EEG sleep patterns such as SSs [40], which allowed us to focus only on them further in this review.

The results of the study of individuals aged 4–24 years, conducted in 1999 by a group of Japanese scientists, showed for the first time two types of SSs activity with different maturation trajectories (Fig. 10) [41].

In children aged 4–6 years, frontal SSs in EEG are organized in the form of "flares" (11–12 Hz) of high power with significant increase up to 12 years of age. During the first decade of life, central–parietal SSs are also in relatively low frequency range (12.5–13.5 Hz) [41]. Later, Tarokh and Carskadon (2010) also revealed a linear dependence of central SSs frequency on age [42]. Studies also showed changes in other terms of SSs activity with maturation. For example, Scholle et al. [37] showed that the interspindle interval has an inverse relationship with the child's age and progressively decreases from 4 to 16 years of life. At the same time, the changes in SSs duration are indicated by a U-shaped curve: a rapid increase from 3 to 7 years old, a plateau period, and a sharp decrease at age 9 years.

It is known that in puberty, the "sleep–wake" cycle undergoes significant changes due to both biological (delayed sleep phase and a decrease in homeostatic sleep pressure in the evening) and socio-behavioral factors (etc., using gadgets before bedtime) compared with pre-pubertal children [43, 44]. In addition to changes in sleep hygiene, adolescents show a significant transformation of sleep architecture associated with the ongoing brain reorganization [45]. The most obvious change in the sleep structure, described by a number of authors, is a sharp decrease in slow-wave activity [46] between 12 and 18 years of age. It is believed that this phenomenon reflects the processes of brain maturation [47], especially in the frontal and parietal cortical areas. According to scientists, brain reorganization during this period of ontogenesis is the final stage of the postnatal trajectory of the nervous system development. This process includes both constructive (active synaptogenesis in children of the first year of life) and regressive changes (synaptic elimination, or pruning—a process of brain development that reduces the number of synaptic contacts). In puberty active synaptic elimination occurs, but the remaining synapses become more complex and efficient [48].

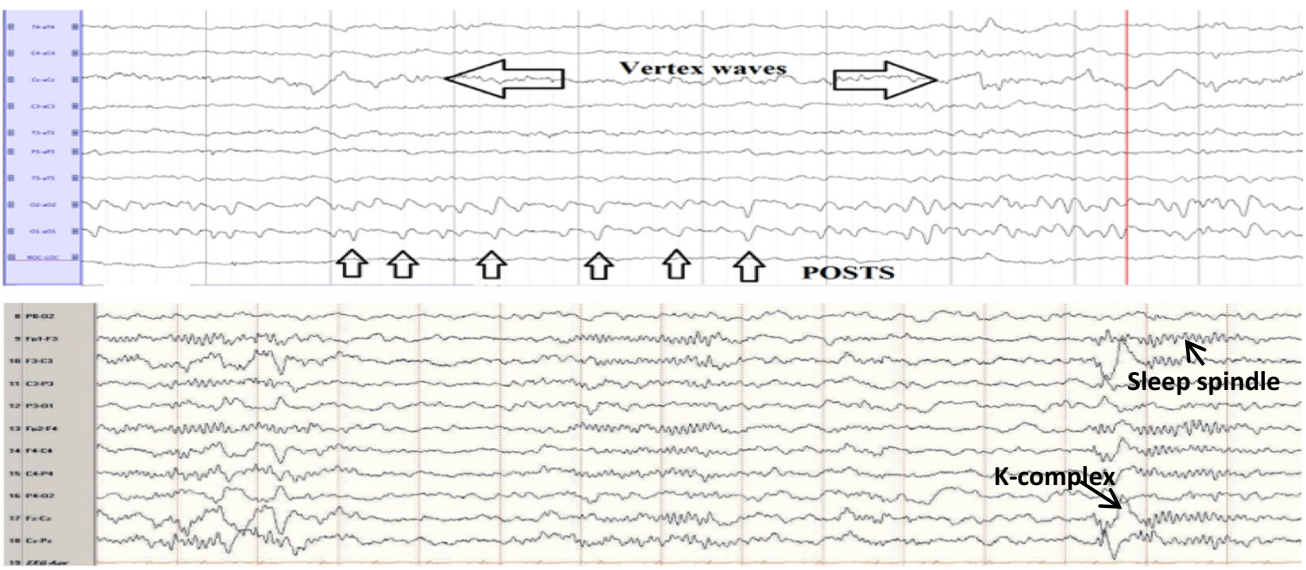

Fig. 9 «Adult» sleep EEG phenomena in children (revised from [34]). *POSTs* positive occipital sharp transients

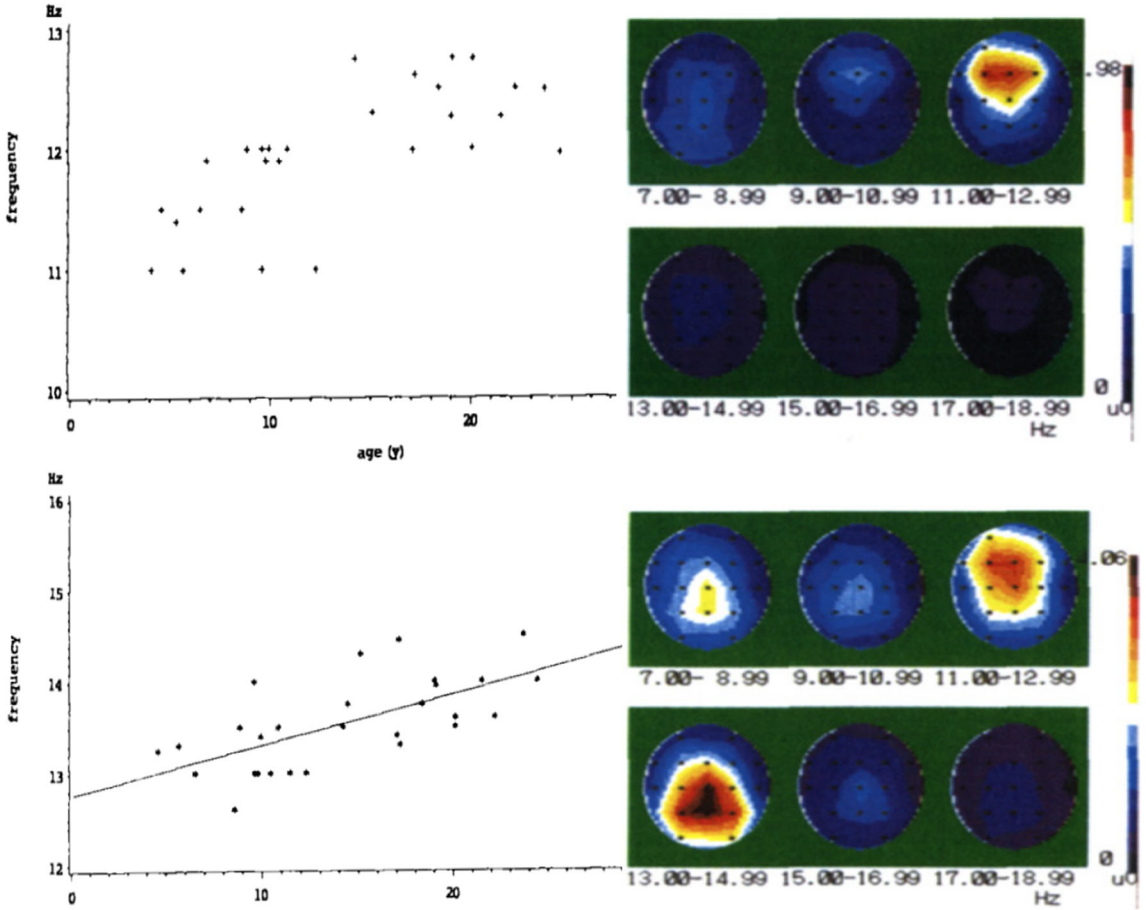

Fig. 10 Sleep spindles' maturation trajectory across childhood. Peak frequency of frontal SSs is in the range of 11–12 Hz during the first decade and changes in early adolescence in the range of 12–14 Hz. Peak frequency of centroparietal SSs becomes higher linearly with increasing age. *SSs* sleep spindles

Studies showed longitudinal SSs activity changes in terms of density, duration, amplitude, frequency, and power in adolescents [46]. So, the SSs density increases non-linearly up to a peak of 4.9 u/min. at the age of 14.5 years (for frontal SSs) and 15 years (for central SSs), then it begins to gradually decrease [49]. The SSs duration decreases linearly throughout puberty, starting from 9 years of age, as noted earlier [37], with an average rate of 6.5 ms/year for central SSs, and 7.5 ms/year for frontal SSs. The maximum SSs amplitude under consideration in adolescents rapidly decreases, starting from age 13.5 years, simultaneously in the central, parietal and frontal cortical anterior areas. However, it was found that the age onset of the central SSs amplitude decreasing in girls was 1.4 years less on average than in boys [50]. It should be emphasized that the change in the density and amplitude of SSs, including gender differences, corresponds to the maturation trajectory of the slow-wave activity pattern in adolescence, which has been mentioned above.

In children and adolescents, the SSs frequency also changes during the night and becomes faster in subsequent sleep cycles [51]. A shift in the SSs frequency range occurs around age 9–11 years and becomesfaster (up to 13 Hz). By 12 years of age, slow patterns completely disappear and are replaced by fast ones (13.5–14.5 Hz). These changes can be associated with active synaptic pruning at this age. The SSs frequency continues to progressively increase throughout puberty by 0.119 Hz/year in the central cortical areas and by 0.099 Hz/year in the frontal regions. It was noted that the rate of increase of frontal SSs frequency in girls is much higher than in boys [50]. The EEG power at a frequency of 11–12.8 Hz demonstrates decreasing trajectory (from 12 years old), while the power at a frequency of 13.4–14.4 Hz shows an increasing one. Two types of SSs and the difference in their development may indicate the existence of different generators and topographical differences in maturation of the thalamo-cortical network. The activity of the frontal SSs could indicate brain maturation [52]. We can evaluate these age-related changes of SSs terms as indicator of maturation of the thalamo-cortical network and changes in sleep depth as the child matures. At the same time, the peaks of these transformations in most parameters coincide with the age of the active synapse elimination [53]. At late adolescence, regressive shifts in the EEG disappear. By this time, according to morphological and functional criteria, all parts of the brain are mature.

Thus, in late adolescence, the SSs power shifts toward higher frequencies. This suggests that the synaptic elimination led to the formation of "strong" complexly organized neuronal contacts for faster and more efficient cortical circuits. Understanding the features of SSs development from infancy to adolescence can facilitate the search for their cognitive–behavioral correlates and become the basis for further fundamental studies of the neurophysiological mechanisms of postnatal brain maturation and neuroplasticity.

5 A short review of own past and present work in childhood neurophysiology

Views and ideas about sleep as the organization and functioning of individual areas, or somnogenic structures of the brain, arose in the second half of the twentieth century. However, it should be noted that modern somnology and sleep medicines are based on the origins of neurophysiological research. Thus, a huge amount of scientific research at the Scientific Centre for Family Health and Human Reproduction Problems (SC FHHRP) was devoted to aspects of age-related neurophysiology. For many years, the most promising area of research was the study of the formation of brain electrogenesis in children aged 1–7 years, carried out by a group of researchers led by academician of the Russian Academy of Sciences S.I. Kolesnikov. The use of systems neurophysiology methods has significantly expanded the understanding of the mechanisms of ontogenetic development of the central nervous system in practically healthy children and those with learning problems in the preschool period and in primary school. Scientists have written a manual that discusses the key components of an encephalographic study and EEG characteristics in normal conditions and in various functional states. Age-related changes in the EEG and individual pathological manifestations are shown [54, 55]. It was also suggested that the state of the biorhythms of intellectual activity depends on the daily rhythm of interhemispheric relations in children and adolescents [56].

It is now known that sleep characteristics change throughout a person's life and are especially active in childhood and adolescence. Modern approaches to the study of sleep are based on the capabilities of somnology and neurophysiology, which make it possible to assess its macro- and microstructural organization. Polysomnographic and neurophysiological sleep studies carried out at the SC FHHRP made it possible to reveal new mechanisms of sleep disorders and formulate conceptual, pathogenetically based approaches to both early diagnosis and correction of sleep disorders in various age groups of the population. The relevance of this approach is confirmed by a large volume of publications and research. Thus, the results are interesting, revealing the sleep characteristics and its adaptive role in schoolchildren with high intellectual abilities [57]. Recent studies have shown changes in the main characteristics of SSs and the presence of their associations with cognitive domains [58–60] in adolescents with obstructive sleep apnea/hypopnea syndrome. These studies will continue to search for significant predictors and markers of sleep-associated disorders of cognition, maturation and development in childhood and adolescence.

Modern methods of sleep assessment and its EEG patterns are the next step in understanding the neurophysiological ontogenetic aspects of the sleep–wake cycle**.** All this will open up perspectives and «windows of opportunity» in predicting postnatal maturation, understanding the mechanisms of brain neuroplasticity and memory consolidation in sleep, which is one of the tasks of modern somnology and neurophysiology.

6 Conclusion

In ontogenesis, dynamic changes in the characteristics of a child's sleep are noted, which must be taken into account for the correct interpretation of clinical data and optimization of its development and learning. Simultaneous changes in sleep EEG and cerebral cortex areas across childhood may indicate the presence of certain causal relationships, which is a «window of opportunity» for diagnosing and predicting the adequate maturation of the higher cortical functions and/or its disorders. Finally, understanding the features of sleep EEG pattern development from birth to adolescence can facilitate the search for their cognitive–behavioral correlates and become the basis for further fundamental studies of the neurophysiological mechanisms of postnatal brain maturation and neuroplasticity.

Author contributions

All authors contributed to the idea and conception. Material collecting and material processing were performed by OB, IM and LR. The first draft of the manuscript was written by OB and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Data availability Not applicable.

Declarations

Conflict of interest No financial or non-financial interests and funding.

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