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# THE EFFECT OF HYPOXIA ON THE TOTAL ELECTRICAL ACTIVITY OF THE DEVELOPING CEREBRAL CORTEX

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**Hypoxia in the nervous system causes morphofunctional changes, which are reflected in the total activity of the cerebral cortex. Lack of oxygen leads to a change in all EEG indicators of the developing cerebral cortex, as well as the appearance of pathological activity in it. This review article summarizes and analyzes data on the effect of hypoxia in early ontogenesis on the total activity of the cerebral cortex.**

**Keywords:** hypoxia, cerebral cortex, ontogenesis, electrocorticogram

The general pattern of the total EEG activity of the developing cerebral cortex as well as its individual indicators can change under the influence of hypoxia [7, 24, 39, 42, 52, 60]. There is no unified classification of changes in the EEG of newborns under the influence of hypoxia. The EEGs of the children who have suffered perinatal hypoxia may have a normal or slightly different from the norm appearance. Acute oxygen deficiency can lead to the appearance of pathological activity in it [24, 39, 42, 52, 64]. Some authors distinguish 7 patterns in the EEG activity depending on its suppression in mature newborns who have survived oxygen starvation. The classification also refers to the sleep-wake cycle in the EEG [65].

In early ontogenesis, as in adults, the most characteristic change in the EEG under the influence of hypoxia is an increase in the severity of the slow waves of the spectrum. Simultaneous reduction of the number and amplitude of high-frequency spectrum waves "unmasks" slow activity [3, 5, 7, 36, 60]. In newborns after anoxia, very slow frequencies are observed in total activity [60]. In some studies, conducted on developing animals and newborns, on the contrary, under the influence of oxygen starvation, no synchronization but desynchronization of EEG activity occurs [8].

Studies conducted on rabbit fetuses of different ages show that changes in the frequency spectrum of the EEG are associated with its extreme frequencies, with an increase in δ– and a decrease in γ–activity [7]. Hypoxia can also reduce the number of standard waves in the spectrum, and the EEG may include one or more standard waves [32, 51].

Hypoxia experienced in early ontogenesis can also affect the EEG structure of sleep [31, 41, 44, 58]. In particular, sleep-wake cycles are disturbed in newborns, they can elongate up to 48 hours [44], or there may be no complete sleep EEG cycle [41].

Perinatal hypoxia leads to a weakening or strengthening of rhythmic activity in the EEG, as well as the appearance of pathological rhythms [1, 5, 9, 63]. In particular, with cerebral anoxia, a pathological α–rhythm is detected in children [51]. Hypoxia also alters intrahemispheric coherence across all rhythms [8]. It was found that the normal and

pathological rhythms of the EEG have ontogenetic features [1, 9].

The patterns of changes in EEG amplitude under the influence of hypoxia have not been sufficiently investigated. Almost nothing is known about the mechanisms of these changes. Usually, under the influence of hypoxia, the amplitude of electrical potentials decreases. However, in many cases, under conditions of hypoxia, the amplitude of electrical activity does not change or, on the contrary, increases. [2, 19, 27, 43].

It is believed that in early ontogenesis, the younger the age, the more EEG is resistant to oxygen deficiency. However, there is evidence to the contrary [6].

One of the reasons for ambiguous changes in the EEG of different cortical regions under the influence of hypoxia is that the thalamic relays are selectively susceptible to a lack of oxygen and glucose [46]. The sensitivity of subcortical white and gray matter also differs in different structures [14]. Different sensitivity levels of neurons to hypoxia may also play a role in this, depending on their size and other features [26].

The pathological activity in the EEG under hypoxic conditions includes suppression and paroxysmal activity. Various types of intermittent activity are distinguished between them, including burst-suppression [37, 39].

As usual, acute oxygen starvation in developing animals and children strongly suppresses electrical activity [30, 31, 45]. The amplitude of EEG waves in piglets in hypoxia/ischemia may decrease to 5 μV [27]. In premature newborns, the amplitude of potentials may decrease below 10 μV [43] or up to 4 μV [54]. An isoelectric line may also appear. The time of occurrence of the isoelectric line in the EEG depends on the severity of hypoxia [12].

Suppression of EEG activity in oxygen deficiency is associated by many authors with histological brain damage [21, 47, 54]. In this case, the amplitude of the EEG changes is inversely proportional to the damage to nerve structures [54]. It has been shown that in rats, the density of neuronal necrosis correlates with the duration of the isoline in the EEG. The death of neurons takes place after the appearance of an isoline [11].

Some authors believe that in newborns, damage to the subcortical white matter almost does not affect the amplitude of the EEG. However, in premature newborns, it was found that the amplitude of potentials depends on the degree of their damage [34].

Acute lack of oxygen in early ontogenesis may also be accompanied by the appearance in the EEG of various types of paroxysmal activity: epileptic, epileptiform activity, convulsive activity, rolandic acute waves, etc. According to many authors, the developing brain is more susceptible to the appearance of paroxysmal activity in the EEG under the influence of oxygen deficiency [15, 29, 31, 35, 37, 50]. In studies conducted on 5, 7, 10, 12, 15, 17, 25, 27, 50, and 60-day-old rats, it was shown that on the $5^{th}$ and $17^{th}$ days of life, more pronounced paroxysmal activity was observed in the EEG [35].

Many authors believe that hypoxia in early ontogenesis, weakening cortical inhibition, leads to the appearance of various types of paroxysmal activity in the EEG [22, 25, 32]. It is believed that in children, normal periodic activity in the EEG below 1 Hz, which has a cortical origin due to the weakening of reverse excitation controlled by inhibitory processes, can transform into epileptiform [32]. However, the mechanisms of different types of paroxysmal activity and, in particular, the role of inhibitory processes are completely unclear, and perhaps the weakening of cortical inhibition plays a role only in the development of some types of paroxysmal activity [16, 56].

Some mechanisms for the development of paroxysmal activity in the developing brain under the influence of hypoxia have been identified. It has been shown that in rats, hypoxia weakens cation currents activated by hyperpolarization in the neurons of the hippocampal CA1 field. These currents may play a role in some forms of epilepsy by regulating synaptic integration and endogenous activity in limbic neurons, which are of fundamental importance for generating pathological activity [56].

The appearance of paroxysmal activity on the EEG is associated with histological brain damage [17, 31, 50]. Both adults and children, in whom different types of paroxysmal activity are recorded in the EEG, have been found to have severe damage to both the gray and white matter of the brain [16, 49]. In newborns, the appearance of rolandic acute waves is associated with damage to the subcortical white matter [17, 50].

The reticular nuclei of the thalamus can play a key role in generating paroxysmal activity in hypoxia. These structures play a critical role in generating various types of pathological activity, in particular spike waves of activity in absentee seizures [18, 33].

Oxygen starvation can also lead to the appearance of burst–suppression in the EEG activity of the developing and mature cerebral cortex, in which high-amplitude bursts periodically appear against a low-amplitude background [37, 55, 59, 66]. This activity may differ in the duration of burst and suppression periods [40, 66], as well as in cyclicity [66]. According to the duration, there are complete and incomplete, or short-term, types of burst activity [55]. It also differs in amplitude and composition. Burst activity can include both slow and fast waves, as well as pathological components [60].

It should be noted that burst-suppression in the EEG takes place in other dysfunctions as well as during anesthesia. At the same time, they may have different origins [13]. It is also assumed that different types of burst-suppression in hypoxia reflect different neocortical dysfunctions [59].

Many authors believe that the appearance of this activity is associated with increased excitability of the cerebral cortex [10, 23] as a result of the weakening of cortical inhibition [23]. Some authors, on the contrary, believe that burst-suppression is registered in the inactivated part of the cerebral cortex, while continuous activity can be recorded in other parts of it [40]. Presumably, periods of suppression in electrical activity reflect the shutdown of synaptic transmission in neurons [38].

There is a lot of data indicating the role of damage to subcortical structures in the genesis of burst-suppression. It is believed that the appearance of this activity reflects a violation of thalamocortical connections [38, 57]. It has been shown that irritations of subthreshold force can cause the generation of burst-suppression when the cortical networks are extremely excited [38].

It is suggested that periods of suppression in burst-suppression are associated with the absence of background activity in neurons, and bursts appear as evoked potentials under the influence of afferent signals [62]. On the other hand, it is known that this pattern of activity is also recorded in the isolated cortex [49]. It was found that in rats, during the restoration of the EEG after cardiac arrest, the reticular nuclei of the thalamus regulate the appearance of burst activity [28].

Some authors have discovered the role of subcortical white matter damage in the genesis of burst-suppression in the EEG. It is assumed that the more acute the damage to this structure, the more intensively this activity is generated [41]. It is possible that burst-suppression generation precedes damage or death of neurons in the nervous system [59].

It should be noted that burst suppression differs from different types of intermittent activity. In the EEG of newborns who suffered hypoxia, burst suppression and intermittent activity with normal voltage can be recorded simultaneously [61]. It is assumed that intermittent EEG in newborns is associated with damage to the basal ganglia and thalamus [17, 20]. In 18-month-old children whose electrical activity of the cerebral cortex has very long interruptions, severe damage to the basal ganglia and thalamus was found [20].

In the EEG of newborns who have undergone hypoxia, different types of pathological activity may be present at the same time. In some newborns, burst suppression and intermittent EEG with normal voltage are recorded [61], while in others, suppression of the background of EEG activity is combined with convulsive activity [17]. There are also cases when the total activity of newborns consists of burst suppression or has an extremely low

voltage [48]. Also, generalized epileptiform complexes can be combined with other patterns of pathological EEG [53].

It was found that the total electrical activity of the cerebral cortex responds ambiguously to hypoxia in different periods of embryogenesis. In 28-day-old fetuses and 10-, 20-, and 30-day-old baby rabbits, hypoxia in different periods of embryogenesis is ambiguously reflected in the total activity of the auditory cortex. In all age groups, after hypoxia in the embryonic period of embryogenesis, the spectral parameters of ECoG slightly deviate from the norm, whereas hypoxia at two later stages of embryogenesis leads to more pronounced but almost similar changes in the spectrum [3, 4].

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## ВЛИЯНИЕ ГИПОКСИИ НА СУММАРНУЮ ЭЛЕКТРИЧЕСКУЮ АКТИВНОСТЬ РАЗВИВАЮЩЕЙСЯ КОРЫ ГОЛОВНОГО МОЗГА

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Гипоксия в нервной системе вызывает морфофункциональные изменения, которые отражаются в суммарной активности коры головного мозга. Недостаток кислорода приводит к изменению всех показателей ЭЭГ развивающейся коры головного мозга, а также появлению в ней патологической активности. В настоящем обзоре обобщены и проанализированы данные о влиянии гипоксии в раннем онтогенезе на суммарную активность коры головного мозга.

**Ключевые слова:** гипоксия, кора головного мозга, онтогенез, электокортикограмма

## HİPOKSİYANIN İNKİŞAFDA OLAN BAŞ BEYİN QABIĞININ YEKUN ELEKTRİK AKTİVLİYİNƏ TƏSİRİ

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Hipoksiya sinir sistemində morfofunksional dəyişikliklər yaradır, bu da ki, baş beyin qabığının yekun aktivliyində əks olunur. Oksigen çatışmazlığı inkişaf edən beyin qabığının bütün EEG göstəricilərinin dəyişməsinə, həmçinin patoloji aktivliyin yaranmasına səbəb olur. Bu icmalda erkən ontogenezdə hipoksiyanın beyin qabığının yekun aktivliyinə təsiri barədə məlumatlar ümumiləşdirilmiş və təhlil edilmişdir.

**Açar sözlər:** hipoksiya, baş beyin qabığı, ontogenez, elektrokortikoqramma

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