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PRECIPITATION OF ABSENCE EPILEPSY IN THE COURSE OF ELECTRICAL STIMULATION OF PALEOCEREBELLAR CORTEX IN CATS WITH SIMPLE PARTIAL EPILEPSY MODEL

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SUMMARY

The effects of paleocerebellar cortex (nodulus, uvula) electrical stimulation (ES) (100-300 Hz, 0,25 ms, 150-250 μA, duration of trial- 3-7 s, interstimuli intervals- 3,0-4,0 min) in artificially ventilated cats upon benzilpenicillin- induced epileptic foci in sensorimotor cortex were investigated. ES caused a suppression of power of epileptic foci and this effect was not reproduced after electrocoagulation of cerebellar tissue neighboring the stimulative electrodes. Along with the suppressive effects upon the activity of the epileptic foci, ES induced the formation of pronounced primarily positive component (PPC) of a spike discharge along with the induction of spike-wave discharges (SWD) (3/sec). Both phenomena were restricted to the zone of penicillin application. SWD activity substituted spikes and disappeared after renewal of epileptogen application. It was concluded that PPC and SWD are characteristic for the antiseizure effects of cerebellum on penicillin-induced foci in cats.

KEY WORDS*:* simple partial epilepsy, absence epilepsy, penicillin, paleocerebellum, spike-wave discharges, electrical stimulation

INTRODUCTION

Cerebellar aspiration as well as damage of it's cortex or nuclei are followed by an increase of brain excitability along with an intensification of epileptogenic phenomena [4, 7, 13]. Electrical stimulation (ES) of cerebellar structures is followed by both intensification or suppression of epileptic manifestations, the outcome depends on the characteristics of the epilepsy model, the functional state of irritated structure and parameters and regimens of ES [4, 10, 13].

Effects of cerebellar ES are most pronounced in the generalized forms of epilepsies in fact it was most frequently the target for clinician implementation of cerebellar-ES treatment of epilepsy. Effects of cerebellar ES upon absence seizures, however, was not in the scope of systematic investigations. Meanwhile, the main assumption, based on the opposite character of the GABAergic inhibition in generalized and absence forms of epilepsy [8], is that cerebellar ES activates this form of epilepsy. Besides, as far as primary positive component (PPC) of spike discharge is developed due to the activation of neighboring (surrounding) inhibition [8, 13], it is quite expected that PPC might be specifically changed under conditions of paleocerebellar ES.

In the present work the effects of paleocerebellar stimulation on penicillin induced epilepsy is investigated in cats. Taking into consideration that penicillin is able to induce all possible range of electrographic epileptic manifestations (interictal, ictal and absence-like spike-wave activity), the changes over time of the EEG characteristics of simple partial epilepsy (foci induced in cortex by local application of epileptogen) in the course of ES of paleocerebellar cortex will be Hence, the aim of the present investigation was confined to the elucidation of the possibility of converting of simple partial epilepsy into absence one. All these, that is planned to perform on the basis of the analysis of dynamics of form of epileptic discharges investigations.

MATERIALS AND METHODS

a) Animals

The experiments were performed on 15 male cats, weighting 2,5-3,5 kg under acute experimental conditions. Procedures involving animals and their care were conducted according to University guidelines that comply with international laws and policies [European Community Council Directive 86/609, OJ L 358, I, December 12, 1987; National Institute of Health *Guide for Care and Use of Laboratory Animals*, US National Research Council, 1996].

b) General surgery

Tracheostomy and skull trepanation was performed in all animals under ether anesthesia. Besides, all points of pressure as well as all zones of soft tissues dissection were infiltrated with 0,5% of novocaine solution and this procedure was repeated every 2,0 h. Tubocurarine ("Orion", Finlandia, 0,2 mg/kg, i.v.) was injected and thereafter the cats were artificially ventilated. Nichrome bipolar electrodes (outer diameter 0,12 mm, interelectrode distance 0,2 mm) were inserted to caudal part of paleocerebellum (uvula, nodulus) under visual control after separation of neck muscles. Stimulation electrodes were fixed to the skull with quick-drying dental cement.

c) Registration of EEG and model of epileptogenesis

The dura mater was dissected 2,0-2,5 h from the moment of the cessation of ether anesthesia, and filter paper (2,0x 2,0 mm) soaked

with *ex tempore* prepared sodium benzilpenicillin solution (16,000 IU/ml) was applied to the posterior sigmoidal gyrus. The indifferent electrode was than placed in nasal bones.

Monopolar EEG registration was performed using ink-writing electroencephalograph 4-EEG-3 type (FSU).

The power index was used to quantify the amount of epileptic activity; it was calculated via multiplying a 1-min average amplitude of the spikes by 1-min average frequency of their generation. The primary positive component (PPC) of the spike discharges was only taken into consideration when it was not less than 50 mcV.

Electrical stimulations (ES) of cerebellum was done with a universal electrostimulator ESU-1 (FSU) (100- 300 Hz, 0,25 ms, 150-250 μA, duration of trial- 3-7 s). Interval between ES was not less than three minutes. Electrocoagulation of tissue beneath the electrodes was performed using anode of constant current (5,0 mA, 30 sec), and the stereotaxic frame as cathode.

The experimental design consisted of two groups, both groups were given the sodium benzilpenicillin solution, however the experimental animals received intermittent cerebellar ES which started in 1,0 min from the moment of spikes appearance, while animals of the control groups were devoid of ES. Animals with implanted electrodes, connected to stimulator without applying electrical stimuli served as controls. This control group (7 cats) was used again in 3,0 h from the moment of disappearance of spikes for new application of penicillin solution application (16,000 IU/ml). The repeated character of penicillin foci creation was justified by previously gained data on comparable characteristics of foci created with penicillin both in rat's [13] and cat's [4] neocortex. Paleocerebellar ES started in these animals, when the stable level of epileptic activity established in foci (15-20 min from the moment of epileptogen application). New applications of benzilpenicillin solution (16,000 IU/ml) were made in these observations upon zones of SW-activity precipitation. Hence, total duration (life-span) of foci was not determined in this subgroup, while in other observations it was verified as a period from the first to the last spike discharge [13].

d) Histology

At the end of the experiments, the rats were anesthetized with pentobarbital sodium and perfused with paraformaldehyde. All locations of electrodes were controlled visually, and correct places of them were taken for farther consideration.

e) Data analysis

The bandwidth power data were analyzed by a one-way ANOVA, followed by Newman-Keuls test.

RESULTS AND DISCUSSION

Control observations. During the first 10 min from the moment of sodium salt of benzilpenicillin solution application, in only one out of 8 subjects a PPC equal to 2,7% pertained to the total amplitude of discharge was registered (Table 1). During the next 10 min, a 10- fold increase of the average magnitude of PPC was observed and the maximal value of this index was noted at 25-3 5th min. after the beginning of the application (Table 1). At this period of time the maximal magnitude of PPC was equal to 15,9% pertained to the total amplitude of spikes. It should be stressed that this period coincided with the maximal power (25- 35th minutes) of epileptic foci (Table 2). The consequent reduction of power of epileptic foci coincided with the reduction of PPC magnitude and at 90th min in 3 out of 8 rats the PPC was absent, while at this time all animals demonstrated spike discharges. There were no signs of the presence of SWD. The life-span of foci was 121,0±11,3 min (84-122 min).

Table 1

Mean (and SEM) PPC in ten minute blocks of spike discharges induced via benzilpenicillin sodium salt solution (16,000 IU/ml) application to sensorimotor cortex of cats (in % pertained to total amplitude of discharge) under control and experimental (cerebellar electrical stimulation (ES) conditions

№№	Time from the moment of epileptogen application (minutes)					
	10	20	30	40	60	90
Control (n=8)	0,3 ± 0,3	3,5 ± 1,1	7,7 ± 1,8	6,9 ± 1,5	6,4 ± 2,6	4,8 ± 1,9
ES (n=7)	8,0 ± 3,4#	22,1 ± 4,5##	27,3 ± 7,0##	16,6 ± 3,1 #	10,02 ± 3,2	8,9 ± 2,9

Note: #- P<0,05; ##- P<0,01 (ANOVA+ Newman-Keuls)

Table 2

The dynamic of the mean (and SEM) power of foci induced via benzilpenicillin sodium salt solution (16,000 IU/ml) application to sensorimotor cortex of cats (in % pertained to total amplitude of discharge) under control and experimental (cerebellar electrical stimulation (ES) conditions

№№	Time from the moment of epileptogen application (minutes)					
	10	20	30	40	60	90
Control (n=8)	26,4 ± 1,8	63,2 ± 4,9	86,2 ± 7,1	48,0 ± 3,4	35,3 ± 3,5	12,8 ± 0,9
ES (n=7)	14,6 ± 2,2 #	42,7 ± 5,3 #	53,6 ± 6,1 #	31,4 ± 4,4	15,9 ± 1,8 ##	5,7 ± 0,7

Note: #- P<0,05; ##- P<0,01 (ANOVA+ Newman-Keuls)

Control observations. During the first 10 min from the moment of sodium salt of benzilpenicillin solution application, in only one out of 8 subjects a PPC equal to 2,7% pertained to the total amplitude of discharge was registered (Table 1). During the next 10 min, a 10- fold increase of the average magnitude of PPC was observed and the maximal value of this index was noted at 25-3 5th min. after the beginning of the application (Table 1). At this period of time the maximal magnitude of PPC was equal to 15,9% pertained to the total amplitude of spikes. It should be stressed that this period coincided with the maximal power (25- 35th minutes) of epileptic foci (Table 2). The consequent reduction of power of epileptic foci coincided with the reduction of PPC magnitude and at 90th min in 3 out of 8 rats the PPC was absent, while PPC was registered on 30th min from the moment of epileptogen application. This period of time PPC magnitude was 56,3% pertained to the total amplitude of spike (maximal value of PPC). In this case conventional ES of paleocerebellum resulted in complete suppression of epileptic discharges. The decrease of the PPC amplitude was going on in parallel fashion to a decrease of the power of foci (Table 2). It should be noted that on 60-th min in two animals of experimental group focal activity was completely suppressed while on 90th min epileptogenesis was kept in 4 out of 7 animals. Life-span of foci under condition of cerebellar ES was 84,1$\pm$ 10,1 min (F(1,13)=1,13, P=0,032).

Some peculiarities of discharge dynamics during period of ES are illustrated in Fig. 1. As it was observed earlier [13, 14, 20], relatively high power of epileptogenesis predisposed to elaboration of activating influence of ES of at this time all animals demonstrated spike discharges. There were no signs of the presence of SWD. The life-span of foci was 121,0$\pm$11,3 min (84-122 min).

Cerebellar stimulations. Penicillin solution application (16,000 IU/ml) with the consequent periodic cerebellar ES, started in 1,0 min from the moment of appearance of spikes was followed by the pronounced PPC development in 5 out of 7 animals during the first 10 min (Table 1). In one observation amplitude of PPC was 25,3% pertaining to the total magnitude of discharges (maximal magnitude) during the first 10 minutes. During the next 10 min, all animals displayed PPC and average magnitude of it exceeded that one in control by 6,3 times (P<0,001) (Table 1). Maximal amplitude (one-third of the total spike amplitude, in average) of cerebellar structures upon epileptic foci. Hence, the increase of the frequency of spikes in zone of penicillin solution application was registered in the course of ES of paleocerebellum (Fig. 1, zone 1). Along with this effect a remarkable increase of PPC was observed. In the zone of propagated discharges (free from epileptogen application cortex) the reduction of the amplitude of discharges was registered (by 0,3-0,5 mV in comparison with the amplitude of discharges before ES) (Fig. 1, zone 2). All these effects disappeared after cessation of ES. These mentioned phenomena were observed in 5 out of 7 animals. It should be noted that in one observation, during the first ES, the magnitude of PPC was dramatically increased and was equal to more than half of total spike amplitude; pronounced PPC was preserved during the interstimulus interval.

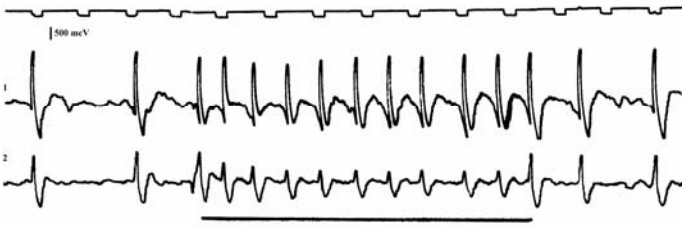

Fig. 1. Effect of ES of paleocerebellum on PPC. The epileptic focus was induced in posterior sigmoidal gyrus by penicillin solution application.
Notes: 1- posterior, and 2- anterior sigmoidal gyri
Parameters of ES: 100 Hz, 0,25 ms, 220 μA (period of ES is marked with solid line).

SWD appearance. 15-20 min after the moment of epileptogen application upon cortex (posterior sigmoidal gyrus), spikes with an amplitude of 1,5-2,2 mV and frequency of discharges of 30 to 50 per min (Fig 2, A) were noticed in cats previously composed in control. Propagated discharges with amplitude from 0,45 up to 0,6 mV were simultaneously registered in nearby localized zone devoid of epileptogen application (anterior sygmoidal gyrus) (Fig 2, A, zone 2). This period of time was characteristic for the most pronounced and stable picture of electroencephalographic phenomena. The development of pronounced (up to 50% of total

magnitude of discharge) PPC of discharges was induced in 1-2 ES (Fig. 2, B, zone 1). This time the amplitude of the discharge was diminished and was equal to 1,2-1,7 mV, while frequency of generation of discharges was reduced until 18 to 36 per min. Also a decrease of the amplitude of propagated discharge up to 0,35-0,5 mV was observed as well (Fig 2, B, zone 2).

Next 2-4 ES-s of cerebellar cortex were followed by a decrease of the frequency of spike discharges while typical sharp-wave of spike-wave discharges (SWD) (3/sec) were present during stimulation (Fig 2, C, D, zone 1). It should be noted that under condition of precipitation of SWD propagated spike-induced discharges were almost abolished while amplitude of spikes in primarily focus remained between 1,2 and 1,8 mV (Fig.2, D). Consequent 2- 4 ES were enough to suppress all spike activity. Such an effect was observed in 5 observations out of 7, and in all of them spikes were substituted by regular SWD (3/sec) with an amplitude from 0,4 up to 1,2 mV (Fig. 2, E, F, zone 1). Similar effect of precipitation of SWD was registered in 3 out of 7 observations in first experimental trial.

New application of penicillin solution (16,000 IU/ml), which was made under condition of the presence of regular spike-wave activity in zone of application (posterior sigmoidal gyrus), was followed by an initial decrease of SWD amplitude (Fig. 2, G, zone 1) with the appearance of spikes in 1,0-3,0 min (Fig.2, H, zone 1). In the next 1,0-4,5 min, the amplitude of spikes reached their maximal value (Fig. 2, I), and SWD completely disappeared. Such an effect was achieved in all five cases when SWD was precipitated in the course of paleocerebellar ES.

Hence, the data obtained showed that ES of paleocerebellar cortex resulted in a decrease of the excitability of penicillin-induced foci in cat brain cortex. Antiepileptic effects were registered both in the form of suppression of power of epileptic activity, and shortening of the life span of foci. The absence of antiepileptic effects of ES after coagulation of tissue neighboring to electrodes was in favor of an active, tonic antiepileptic nature of influences from stimulated structure. These data are in correspondence with data from other authors [4, 10] who also showed that paleocerebellum ES in different animal species induced suppression of penicillin-induced foci in bra in cortex.

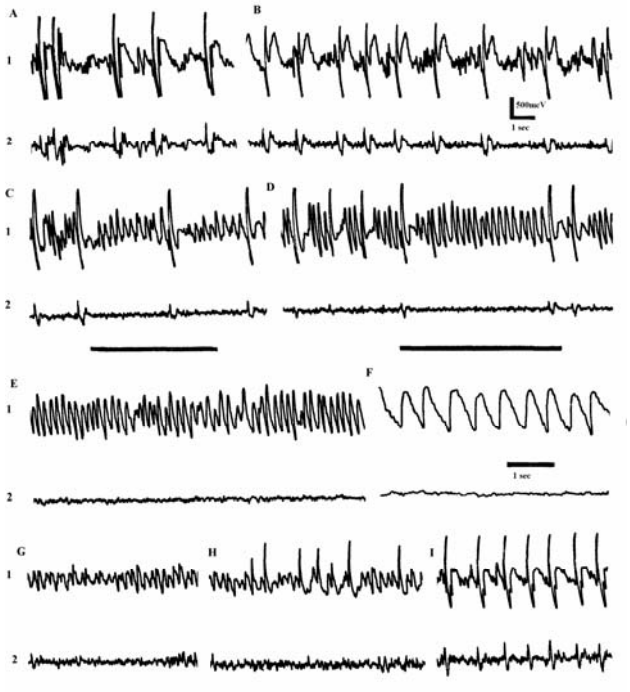

Fig 2. SWD precipitation in the course of paleocerebellar ES in cats with focal penicillin- induced activity in brain cortex.
A- 23 min from the moment of application of benzilpenicillin sodium salt (16,000 IU/ml) to posterior sigmoidal gyrus (zone 1);
B-1,0 min from the moment of cessation of 2-d ES; C-3th ES; D-5th ES; E, F- 1,0 min from the moment of 7th ES;
G- 0,5 min, H- 2,5 min, and I- 5,5 min from the moment of new application of benzilpenicillin salt solution (16,000 IU/min) to posterior sigmoidal gyrus.
Parameters of ES: 200 Hz, 0,25 ms, 250 μA (period of ES is marked with solid line).

Along with the suppression of focal epileptic activity, a substantial increase of PPC was noted: this is indicative for the intensification of surrounding inhibitory control [13]. This intensification was marked both during ES per se with the disappearance of the effect immediately after cessation of stimulation and during interictal periods. Hence, it might be supposed

that the activation of cerebellum was followed by heightening of the level of functional activity of inhibitory interneurons in the brain cortex, which in turn resulted in more powerful restriction of the zone of epileptisized neurons. The decrease of the amplitude of propagated discharges observed during paleocerebellar ES was also in favor for the worsening or decrease of excitation spreading from the primarily zone of spikes generation.

Alternatively, migration of primarily zone of discharges generation, as a result of ES-induced "reconstruction" of surrounding inhibitory barrier, also could not be excluded as a cause of observed changes in amplitude dynamic. In this case the decrease of propagated discharge amplitude as well as "increase" of PPC might be explained by moving of the active zone of foci somewhere aside from the electrode localized in the zone of primarily focus. It should be noted that during ES the increase of that was continuously present during slow wave sleep, was indeed restricted to the parietal cortex, surgical resection completely abolished SWD [12].

As far as spike-wave activity induced in cats is developed as a result of enhancement of recurrent inhibition [13], it might be supposed

Hence, proepileptogenic role of cerebellum with regard to absence model of epilepsy might be assumed. This assumption corresponds to the known neurophysiological mechanism of absence epilepsy development, which was confined to strengthening of the brain inhibitory mechanisms [4, 8, 13]. Later on support of spike-wave activity in rodents by neuronal cerebellar elements was shown by [6]. Hence, our data are in line with mentioned authors and suppression of spikes without affecting spike-wave discharges was a most impressive fact.

It should be noted that for SWD in WAG/rij rats and GAERS, two commonly accepted rat models for generalized absence epilepsy [3], most often a frequency of 8/sec was observed [2]. Hence, that rhythm, which was registered in our work, might be regarded as two-times dividing of the frequency of that rhythm which is inherited and which is in charge for the absence epilepsy manifestations in the rat models such as WAG/rij strain. Such "splitting" of main rhythmicity is known for rhythmic evoked responses in brain cortex and might be explained by modifications in inhibitory mechanisms in cortical neuronal chains [5]. However, 3-5 Hz SWD were described by [11] after chemical lesions of the reticular thalamic nucleus. Finally, [8] noticed that after application of penicillin in the feline model the frequency of the slowly in frequency decreasing sleep spindles in the process of changing into SWD also ended frequency of spike discharges was pronounced, which might be explained by relatively high epileptogenicity of neurons affected by penicillin. Such neurons are prone to react on afferent stimuli mainly in the form of intensification of their own activity [13].

In the course of ES- induced declining of epileptic activity, the appearance of 3/sec waves (spike- wave rhythm) was clearly noted. It should be stressed that such effect was absent in control observations. Hence, ES of paleocerebellum induced typical absence-like EEG activity in cat's sensorimotor cortex but this was restricted to the zone of epileptogen application. SWD did not encompass intact from epileptogen cortical zone. Hence, in strict sense, our phenomenon can be regarded as some form of model of absence type of epilepsy lacking synchronous generalized type of activity, which is quite characteristic for this form of epilepsy [8]. Recently a case was described in which SWD, that restoration of these mechanisms, which had been broken down in the zone of penicillin application, might be among targets for cerebellar influences upon epileptisized neuronal populations. This is supported by the observed fact of pronounced increase of PPC in epileptic foci induced by paleocerebellar ES [4].

up with a frequency half of the naturally occurring sleep spindles.

It should be also noted that registered in our observations more prolonged duration of periods of SWD represents by itself a clear difference with those bursts of SWD registered in typical form of experimental absence epilepsy in WAG/rij rats, in which the mean length of an episode is about 5 sec [4]. The nature of such differences is obscure and cannot be easily explained at present.

Considering the possible role of cerebellum in absence epilepsy development it should be noted that an increase of the cerebellar activity might be in charge for the arrest of locomotor activity, tremor precipitation and staring as well [5]. The timely developed locking out of cortical function might be induced by hyperfunctional state of cerebellar structures and precipitation of short-time extraordinary inhibitory influences upon sensorimotor cortex.

It is of worth to mention about some neuromeditor/neurochemical background of the observed effects in our rat study. Hence, it was shown that GABA receptor agonist muscimol enhanced the number, while the antagonist bicuculline reduced the number of SWD [9]. In line with this is that tiagabine, a GABA- reuptake inhibitor with strong anticonvulsant properties, enhances the number of SWD [9]. It is in good correspondence with the assumption that activation of SWD might be expected from ES

of cerebellar cortical structures as far as elaboration of GABA is documented [4]. Besides, appearance of agonists of opiate receptors in CSF of animals with stimulated cerebellum [13] might be also suspected as the mechanisms of intensification of SWD-activation since SWD was reported under conditions of stimulation of μ-opiate receptors [9].

Meanwhile, activation of norepinephrine system of the brain, induced by cerebellar ES [10], presumably might lead to decreasing of mechanisms of SWD generation, which are suppressed under conditions of activation of catecholaminergic mechanisms [1]. Hence, this effect is not in favor for the general scheme of cerebellar determined components of absence-epilepsy behavior. Besides, some discrepancies on the contribution of cerebellum to rhythmic movements observed during spike-wave pattern of activity were described [6]. Authors mention a correlation of unit activity of neuronal elements of both cortex and nuclei of cerebellum with SWD in rodents while rhythmic movements (tremor) were absent in half of the observations. As an explanation of this discrepancy, it should be taken in mind that motor effects from cerebellum are dependent upon the state of inhibition of the output in involved neuronal structures. Moreover, the large inhibition of spinal motor neurons might be in charge of ameliorating of final motor effect [4].

The apparent antagonism between spikes and SWD revealed after renovation of penicillin application is similar to known fact of antagonism between spikes and generalized ictal discharges [13]. It might be that three levels of epileptogenicity are characteristic for penicillin epileptogen action upon brain cortex (absence-the lowest one, simple partial- middle and ictal-highest). The absence of SWD in control observation of spikes evolution is in favor for the necessity of participation of cerebellar influences upon reconstruction of neuronal cortico-thalamic chains in such a fashion that it became prone to demonstrate SWD. In this context pronounced inhibitory influences from cortical epileptic focus upon transferring information from cerebellar structures to cortex via thalamic nuclei are of interest [13]. It might be that cerebellar-dependent peculiarities of "gate control" of processing of informational streams to cortex [2] under conditions of simple partial epilepsy contribute to the facilitation of conversion of simple partial epilepsy to absence type of epilepsy.

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РОЗВИТОК АБСАНСНОЇ ЕПІЛЕПСІЇ В РЕЗУЛЬТАТІ ЕЛЕКТРИЧНОГО ПОДРАЗНЕННЯ ПАЛЕОЦЕРЕБЕЛЛУМУ У КОТІВ З МОДЕЛЬОВАНОЮ ПРОСТОЮ ПАРЦІАЛЬНОЮ ЕПІЛЕПСІЄЮ

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РЕЗЮМЕ

В роботі досліджували ефекти електричного подразнення (ЕП) кори палеоцеребелуму (вузлик та язичок) (100- 300 Гц, 0,25 мс, 150- 250 мкА, тривалість ЕП- 3-7 с, інтервал між подразненнями- 3,0- 4,0 хв) у котів, з пеницилін- викликаними епілептичними вогнищами в сенсомоторній корі. ЕП викликали пригнічення потужності епілептичних вогнищ і цей ефект не відтворювався після електрокоагуляції піделектродної тканини мозочку. Впродовж розвитку ефекту пригнічення вогнищ ЕП викликали розвиток виразного первинного позитивного компоненту спайкового потенціалу з розвитком спайк-

хвильових розрядів (СХР) (3 в секунду). Обидва феномена спостерягались лише в зоні аплікації пеніциліну на кору мозку.

СХР пригнічувались і заміщувались спайковими потенціалами після повторної аплікації рочину пеніциліну на кору мозку. Автори дійшли висновку, що первинний позитивний компонент та СХР є характерними для виникнення протисудомних ефектів під час ЕП палеоцеребелярної кори у котів з пеніцилін-викликаною формою епілептогенезу.

***КЛЮЧОВІ СЛОВА**:* проста парціальні епілепсія, абсансна епілепсія, пеніцилін, палеоцеребелум, спайк-хвильові розряди, електричне подразнення

РАЗВИТИЕ АБСАНСНОЙ ЭПИЛЕПСИИ В РЕЗУЛЬТАТЕ ЭЛЕКТРИЧЕСКОГО РАЗДРАЖЕНИЯ ПАЛЕОЦЕРЕБЕЛЛУМА КОШЕК С МОДЕЛИРОВАННОЙ ПРОСТОЙ ПАРЦИАЛЬНОЙ ЭПИЛЕПСИЕЙ

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РЕЗЮМЕ

В работе исследовали эффекты электрического раздражения (ЭР) коры палеоцеребеллума (узелок и язычок) (100- 300 Гц, 0,25 мс, 150- 250 мкА, длительность ЭР- 3-7 с, интервал между ЭР- 3,0- 4,0 мин) у кошек с пенициллин-вызванными эпилептическими очагами в сенсомоторной коре. ЭР вызывало уменьшение мощности эпилептических очагов и этот эффект не воспроизводился после электрокоагуляции подэлектродной ткани мозжечка. На протяжении периода подавления очаговой активности ЭР вызывало развитие выраженного первичного положительного компонента спайкового потенциала с развитием спайк-волновых потенциалов (СВП) (3 в секунду). Оба феномена отмечались только в зоне аппликации пенициллина на кору мозга. СВР подавлялись и замещались спайковыми разрядами после повторной аппликации раствора пенициллина на кору мозга. Авторы пришли к выводу, что первичный положительный компонент и СВР являются характерными для развития противосудорожных эффектов в течение ЭР палеоцеребеллярной коры у кошек с пенициллин-вызванной формой эпилептогенеза.

***КЛЮЧЕВЫЕ СЛОВА**:* простая парциальная эпилепсия, абсансная эпилепсия, пенициллин, палеоцеребеллум, спайк-волновые разряды, электрическое раздражение