The results of this investigation confirm those of experiments *in vitro*, which revealed a reduction in the effectiveness of vasoconstrictor stimuli when the intravascular pressure rises, and considering the conditions of intravital microscopy of the muscle, they suggest that in this case limitation of the tissue blood flow, evoked by sympathetic vasoconstriction, also is inhibited under these circumstances. This may indicate that the rise of intravascular pressure in the arteriolar part of the microvascular bed may play a protective role. Elevation of the intravascular pressure in the arterioles modifies the action of vascular smooth muscles, in the same way as is observed in perfusion experiments [8], transforming it to isometric operation, when vasoconstrictor stimuli can only potentiate the tonic contraction of the vascular smooth muscles, without changing the lumen of the microvessel in so doing. It can be tentatively suggested that a fall of microvascular pressure acts in the opposite way, considerably strengthening the likelihood of vascular occlusion during potentiation of vasoconstrictor influences.

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# MOTOR RESPONSES IN RATS DURING CRITICAL GROWTH PERIODS IN EARLY POSTNATAL LIFE


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UDC 612.76:612.648.014.49

KEY WORDS: postnatal ontogeny; motor response; critical growth periods.


During adaptation to environmental factors and physical exercise in the early periods of postnatal ontogeny (a state of stress) the functions of the body return to those typical of previous stages of ontogeny [8, 9]. The question arises whether this return takes place only during adaptation to new environmental conditions (conditions of development) or whether similar changes also take place at critical periods of growth. Several critical growth periods in rats are distinguished in the literature [3, 4].

To study this problem an attempt was made to evaluate the physiological features and, in particular, the character of motor responses (MR) of jerking type, specific for the early age, in rats during critical growth periods, and also to determine the character of the physiological mechanisms of the return of functions to forms typical of these animals at previous stages of development.


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(Presented by Academician of the Academy of Medical Sciences of the USSR V. S. Rusinov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 9, pp. 261-264, September, 1985. Original article submitted April 12, 1983.




0007-4888/85/0009-1150$09.50

TABLE 1. Changes in FMR of Jerk Type, Body Weight, and Velocity Constant of Growth (K) in Rats during First Month of Postnatal Development (M ± m)

| Parameter tested | Age of animals, days | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 5 | 7 | 9 | 11 | 13 | 16 | 18 | 23 | 25 | 28 | 30 |
| FMR per hours | 375±22 | 374±51 | 626±64 | 647±66 | 832±59 | 623±85 | 285±46 | 85±22 | 132±12 | 163±14 | 349±21 | 88±9 | 76±8 |
| Body weight, g | 6,1±0,1 | 6,9±0,1 | 8,7±0,22 | 12,3±0,34 | 15,2±0,4 | 16,8±0,4 | 18,2±0,62 | 20,9±0,61 | 22,4±0,9 | 30,2±0,96 | 43,4±0,72 | 45,8±0,84 | 46,8±0,84 |
| K | — | 0,48 | 0,86 | 0,98 | 1,3 | 0,91 | 0,48 | 0,39 | 0,38 | 0,31 | 1,31 | 0,42 | 0,36 |

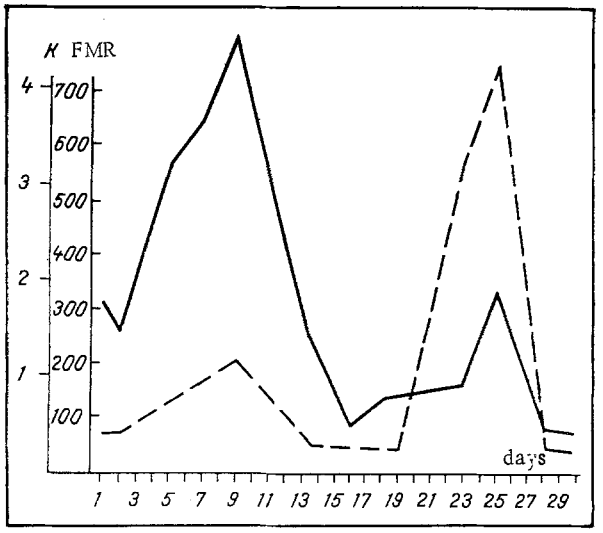


Fig. 1. Changes in FMR of jerk type (continuous line) and velocity constant of growth (broken line) in rats during first month of life. Horizontal axis — age (in days), vertical axis — FMR and velocity constant of growth of animals (K).

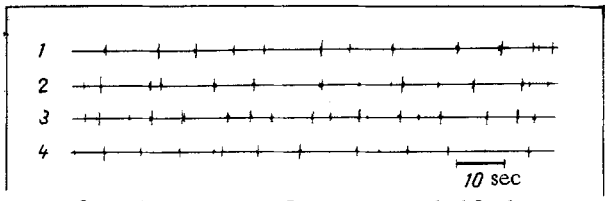


Fig. 2. Actogram of rats aged 12 days: 1) original stage, 2) after injection of physiological saline, 3) immediately after injection of iproniazid (A) and rausedil (B), and 4) 30 min after injection.

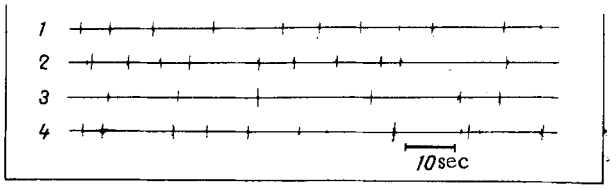


Fig. 3. Actogram of rats aged 25 days. Legend as to Fig. 2.

## EXPERIMENTAL METHOD

Noninbred rats aged from 1 day to 1 month were used. Specific MR of jerking type specific for the first days of life [6-8] were studied. For this purpose an actogram of the rats was recorded on the 4ÉÉGP4-02 electroencephalograph during performance of MR of jerking type during sleep, which occupies the greater part of the 24-h period for these animals. The actogram was recorded as follows. The animals were placed in a transparent plastic box. To the floor of the box on the outer side were glued piezoelectric crystals, which served as transducers for recording MR. The velocity constant (intensity) of growth was calculated by the equation [3]:

$$K = \frac{\log m_2 - \log m_1}{\log t_2 - \log t_1},$$

where $m_1$ and $m_2$ denote the body weight of the animals and $t_1$ and $t_2$ the ages of the rats. The rats were weighed daily in the morning. During the first days of life of immaturely born mammals MR of jerking type of high frequency (FMR) correspond to high blood catecholamine levels [5, 10], respiration rate, heart rate, and oxygen consumption [1, 11]. Later, a reduction in FMR of jerking type is accompanied by a decrease in the blood catecholamine concentrations, respiration and heart rates, and oxygen consumption. During the critical growth period at the age of 25 days these parameters increase once again (the catecholamine level was not determined at this time), and decrease toward the end of the month.

The increase in FMR of jerking type during a critical growth period is connected with elevation of the blood catecholamine level. It was therefore decided to study to what extent the artificial creation of the pattern of neurohumoral homeostasis existing at an earlier postnatal age (i.e., raising the catecholamine level) at later stages of development can induce an increase in FMR of jerking type and a return of the functions to the pattern charac-

teristic of the first days of life. If an increase in FMR of jerking type is due to a rise of the blood catecholamine level, administration of drugs which change blood catecholamine levels during critical periods of growth ought therefore to give rise to corresponding changes in the frequency of MR. Adrenergic and adrenolytic drugs were administered to the experimental animals and FMR of jerking type were assessed in them. Iproniazid, a monoamine oxidase inhibitor [2], was used as the adrenergic drug, and rausedil, which flushes catecholamines out of the tissues [10], was used as the adrenolytic drug. The drugs were dissolved in physiological saline and injected intraperitoneally into rats aged 12 days, when MR of jerking type, specific for the first days of life, were lost, and at the age of 25 days, during a critical growth period. MR were recorded in the original state, after a single injection of physiological saline, immediately after injection of the drug, and 30 min later. Iproniazid was injected in a dose of 15 mg/kg and rausedil in a dose of 0.5 mg/kg. Altogether 54 rats were used in the experiment.

## EXPERIMENTAL RESULTS

As the previous investigations showed, three forms of MR of jerking type are characteristic of rats: fibrillary motor activity, MR of jerking type, and sustained motor activity. It will be clear from Table 1 that during the first days of life of the rats high FMR of jerking type was observed. With age this parameter decreased from 542 ± 51 MR/h at the age of 10 days to 118 ± 10 MR/h at the age of 19 days ($P < 0.001$). However, during a critical growth period at the age of 25 days, there was an increase in FMR of jerking type, namely of those MR which are found most frequently during the first few days of life. During a critical growth period there is therefore an increase in FMR of jerking type and a return of the functions to the physiological pattern existing in the earliest stages of postnatal ontogeny.

Let us turn to the analysis of the physiological mechanisms of these developmental features thus revealed. After administration of iproniazid to the rats there was an increase in FMR at the age of 12 days from 628 ± 41 to 682 ± 52 MR/h ($P < 0.001$, Fig. 2), and at the age of 25 days from 349 ± 21 to 396 ± 8 MR/h ($P < 0.001$, Fig. 3). Conversely, administration of rausedil reduced FMR in rats aged 12 days from 612 ± 7.1 to 451 ± 6 MR/h ($P < 0.001$, Fig. 2), whereas in rats aged 25 days it reduced it from 349 ± 21 to 296 ± 11 MR/h ($P < 0.001$, Fig. 3).

Thus, one possible mechanism of the increase in FMR of jerking type in rats aged 12 and 25 days is elevation of their blood catecholamine levels. A natural increase in the frequency of MR of jerking type, specific for the first few days of life, during a critical growth period of rats and the return of their functions to physiological patterns typical of the early postnatal period are evidently due to elevation of their blood catecholamine levels.

Previous investigations showed that the return of physiological functions to patterns characteristic of the early stages of ontogeny is due to blocking of inhibitory processes in the CNS [6, 9] and also to a decrease in the flow of sensory signals into the CNS [8]. Besides these two mechanisms there is also a third, revealed by the present investigation and linked with a high blood catecholamine level.

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# MODULATING EFFECT OF THE SECOND SOMATOSENSORY AREA OF THE CORTEX TO ELECTROACUPUNCTURE EFFECTS IN THE TRIGEMINAL NUCLEI

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UDC 612.826.2+612.819.52]-06: 615.844.4]-06:612.825.1

KEY WORDS: electroacupuncture; evoked potentials; trigeminal nuclei; cortex.

The cerebral cortex plays an important role in the control of conduction of afferent information at different levels of the CNS [1, 3]. This control is effected also on the trigeminal nuclei [7, 8, 10], which play an important role in the formation of pain syndromes and, in particular, in trigeminal neuralgia [5]. As has been shown, electroacupuncture (EAP) modifies the functional state of several of the trigeminal nuclei [2]. For instance, in the caudal trigeminal nucleus (CTN) it leads to inhibition of nociceptive signals, whereas in the oral trigeminal nucleus (OTN) it facilitates conduction of afferent signals. An important role in the formation of the effects of EAP at different levels of the CNS is ascribed to the second somatosensory area of the cortex (area SII) [3, 4, 6]. However, its direct influence on the effectiveness of EAP at the level of the primary relay nuclei has not yet been studied.

In this investigation the effect of reversible functional blocking of area SII on the conduction of afferent signals through OTN and CTN during EAP was studied.

## EXPERIMENTAL METHOD

Acute experiments were carried out on 12 adult cats, anesthetized with hexobarbital (50 mg/kg, intraperitoneally), immobilized with suxamethonium, and artificially ventilated. Afferents of the pulp of the canine tooth (3 msec, up to 20 mA) and of the lip (0.3 msec, 1-2 mA) were stimulated. EAP was applied through steel acupuncture needles, inserted into the lower part of the base of the concha auriculae (where trigeminal nerve endings are located), in the form of square pulses of current 1 and 2 msec in duration, with an intensity of 16 mA, and with a frequency of 1 Hz for 15 min. Reversible functional blocking of contralateral area SII was carried out during EAP by application of cold. The cortical surface temperature in area SII was about 22°C. After the end of EAP and before recording of evoked potentials (EP), the cortex was heated up to the original temperature. Evoked potentials (EP) were recorded by a monopolar method, ipsilaterally in OTN and in the ventromedial border of CTN. The results were averaged by specialized computer on the basis of 10 presentations. The site of recording of EP was verified histologically after microcoagulation.

## EXPERIMENTAL RESULTS

When EAP was carried out with area SII intact, changes in evoked activity were observed in nuclei of the trigeminal complex. In CTN the EP arising to stimulation of the dental pulp were depressed by 50% whereas EP in response to stimulation of the lip in that nucleus were unchanged (Fig. 1). Meanwhile, in OTN, an increase in amplitude of EP in response to stimulation of both dental pulp and lip, by 150-160%, was observed.

One way whereby the cerebral cortex can influence the nuclei of the trigeminal complex is via the pyramidal tract [7, 10]. In the experiments of series I, to discover whether, in

Central Research Institute of Reflex Therapy, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR G. N. Kryzhanovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 9, pp. 264-265, September, 1985. Original article submitted October 10, 1984.

 0007-4888/85/0009-1154$09.50 