TISSUE LIPID PEROXIDATION DURING SUBLIMINAL ELECTRICAL STIMULATION OF THE LIMBIC SYSTEM OF THE BRAIN

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KEY WORDS: lipid peroxidation; limbic system of the brain; electrical stimulation.

In the modern view, structures of the limbic system constitute the key substrate of emotions [5, 6]. Emotional stress (ES) is known to play a role in the pathogenesis of cardiovascular diseases [9, 10, 13]. One of the most important stages in stress-induced tissue damage is activation of lipid peroxidation (LPO) [3, 8, 9]. This suggests that intensification of LPO arising during ES also participates in the mechanism of development of tissue damage arising in response to stimulation of limbic brain structures.

To test this hypophysis LPO activity was studied during prolonged stimulation of limbic brain structures: the ventromedial hypothalamic nucleus, the central amygdaloid nucleus, and the ventral hippocampus.

EXPERIMENTAL METHOD

Acute experiment were carried out on 17 mature male rats weighing 240-300 g. Nichrome electrodes were implanted in the limbic structures of the brain by means of a stereotaxic apparatus. The coodinates of the brain structures for study were determined from the atlas [12]. Electrical stimulation was applied to unrestrained animals with the following parameters: frequency 30 hZ, pulse duration 1 msec, amplitude 10% below the threshold value (130-210 μA). Each structure was stimulated for 1 h in the following order: hypothalamus, amygdaloid complex, hippocampus. The rats were decapitated 2 h after electrical stimulation of the hippocampus.

After the end of the experiments the location of the electrode was verified histologically by electrolytic tagging.

LPO activity in the blood serum was estimated as levels of acyl hydroperoxides in β- and pre-β-lipoproteins (LPO), obtained by measuring absorption at 232 nm, and in other tissues by determining the concentration of malonic dialdehyde (MDA) [7] and the rate of its accumulation during incubation of samples for 1, 2, and 3 h. Activity of superoxide dismutase (SOD) also was determined in the brain and liver tissues [4].

TABLE 1. Effect of Prolonged Stimulation of Limbic Structures of Rat Brain on LPO Parameters (M ± m)

Experimental conditions	Serum hydroperoxides, optical density units/mg LP	SOD, conventional units		MDA in periodontium, optical density units/g tissue
		Brain	Liver	
Control	3,57±0,40	0,79±0,083	3,12±0,20	7,7±0,83
(implantation of electrodes without electrical stimulation)	(7)	(5)	(5)	(7)
Electrical stimulation	6,68±0,71*	0,44±0,029*	2,36±0,29	11,6±1,15*
	(5)	(4)	(4)	(5)

Legend. Number of animals given in parentheses. *P < 0.05 compared with control.

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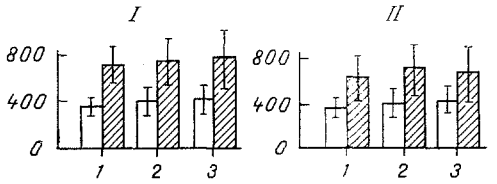

Fig. 1. Accumulation of MDA in tissues during incubation: I) heart, II) liver. Unshaded columns — control; shaded — electrical stimulation. Abscissa, duration of incubation (in h); ordinate, elevation of MDA level (in % of initial value).

EXPERIMENTAL RESULTS

The level of acyl hydroperoxides in β- and pre-β-LP was found to be raised in the blood serum of rats after stimulation of the limbic structures of their brain (Table 1). During incubation of homogenates of myocardium and liver the MDA level in the tissues of the experimental animals by the end of the first hour was raised by a greater degree than in the control rats (Fig. 1).

The MDA concentration in the periodontium also was increased after electrical stimulation. SOD activity in the brain was reduced, whereas in the liver it was virtually unchanged (Table 1).

Consequently, during stimulation of the limbic structures of the brain the intensity of LPO increased in the blood and other tissues. This character of the effect of stimulation of the limbic system is evidently realized by two mechanisms: activation of the sympathico-adrenal system and activation of the pituitary-adrenocortical system [1, 2, 10].

An increase in functional activity of the adrenergic systems of the brain during hypothalamic stimulation was discovered previously [1]. Stimulation of the limbic zones of the brain causes excitation of the pituitary-adrenocortical system [2, 10]. Dependence of corticosteroid production on the state of the limbic structures has been demonstrated in investigations by other workers [10, 11].

Intensification of LPO processes observed in tissues of the myocardium, liver, and periodontium and in the blood is evidently the result of failure of the phsyiological antioxidant system, due to the arrival of an excess of free fatty acids and oxygen in the blood and tissues under the influence of corticosteroids and catecholamines.

Prolonged subliminal stimulation of the limbic system thus potentiates LPO processes, which have a damaging effect on the tissues.

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EFFECT OF PLACENTAL INSUFFICIENCY ON STATE AND RESPONSE TO ANOXIA OF RAT FETUSES

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KEY WORDS: fetus; placental insufficiency; delay in development; anoxia.

Complications observed in the newborn infant frequently arise as a result of pathological labor. The importance of an enfeebled state of the fetus before birth and its resistance to the effects of birth stress has received less study. This is true mild disturbances of the state and, in particular, moderate delay of its development, which is frequently observed and may be difficult to diagnose under clinical conditions.

The course of acute asphyxia in rat fetuses, with normal and delayed development, was studied.

EXPERIMENTAL METHOD

Operative reduction of the utero-placental circulation was performed surgically in 35 Wistar rats under aspetic conditions under ether anesthesia on the 16th day of pregnancy, by ligation of about 40% of the placental vessels at each implantation site in one uterine cornu. The other uterine cornu was left intact, and the fetuses contained in it served as the control. On the last (21st) day of pregnancy the animals were fixed to a frame, laparotomy performed under local procaine anesthesia, the umbilical cords of the fetuses were ligated successively through small incisions in the uterus, and divided, and the fetuses also were consecutively removed from the uterus and immediately (before the first inspiration) placed in physiological saline with a constant temperature of 37°C. The cardiac activity and asphyxial respiratory movements of the fetuses were recorded in the course of asphyxia by impedance rheography. The following parameters were determined by analysis of the traces: heart rate, time of appearance of marked cardiac arrhythmia, duration of survival until the last cardiac contraction, time of appearance of the first inspiration, number of respiratory movements, and duration of asphyxial breathing. Altogether these parameters characterized the resistance of the fetus to anoxia — a factor which often complicates the transition to extrauterine life. After the experiment the fetuses and placentas were weighed, and kept at constant temperature of 56°C to dry them to constant weight. Comparison of the wet and dry weights enabled the degree of hydration to be determined. Altogether 106 fetuses with delayed development and 99 control fetuses were investigated.

EXPERIMENTAL RESULTS

Judging by their weight, the experimental fetuses were significantly but not drastically delayed in development compared with the control fetuses remaining in the intact uterine cornua of the same animals (Table 1). They showed a higher degree of hydration than the control fetuses. We know that in rats, just as in many other species of animals with multiple

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