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Influence of Low-Intensive Red Light on the Myocardium in Experimental Asphyxia

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We studied the effects of low-intensity broadband red light on electrical activity of the heart and oxidative modification of proteins in the myocardium of rats after asphyxia. It was shown that low-intensity red light reduced the content of oxidatively modified proteins in rat heart after oxidative stress caused by asphyxia. Exposure to low-intensity red light normalized ECG parameters in rats after asphyxia.

Key Words: *red light; oxidative modification of proteins; ECG; asphyxia*

According to WHO data, deaths from cardiovascular diseases occupy the leading place in the structure of mortality and loss of working capacity in economically developed countries, including Russia [1]. The leading pathogenetic role in cardiovascular diseases is played by external and internal hypoxia with inadequate compensatory reaction of the body [12]. It is known that asphyxia accelerates initiation of protein oxidation chain processes, because hypoxia is accompanied by oxidative stress [7,9,11,13]. Exposure of the myocardium to low-intensity light effectively modifies the function of the cardiovascular system [5], and broadband red light positively influences the content of free radical oxidation products in the damaged rat tissues [3,4].

We studied the effect of low-intensity broadband red light on electrical activity of the heart and content of oxidatively modified proteins (OMP) in the myocardium in experimental asphyxia.

MATERIALS AND METHODS

The experiment was conducted on 3-4-month-old male white rats weighing 180-250 g. For asphyxia modeling, a soft conic sealed tube (diameter corresponded to the diameter of rat trachea) was inserted through the mouth to block airflow through the trachea and nasopharynx. The projection of the heart and lungs was irradiated with broadband red light (intensity 5 mW/cm^2; peak width at half height 70 nm; peak wavelength 640 nm) through a light guide inserted in the trachea. Three groups of animals were maintained under identical conditions: group 1 (n=23) consisted of intact rats, group 2 rats (n=25; control) were subjected to 2-min asphyxia, group 3 rats (n=25; test group) were subjected to 2-min asphyxia and exposed to broadband red light for 10 min.

ECG was recorded before asphyxia (intact group), during and immediately after asphyxia, and then every 5 min over 30 min (control). Exposure to broadband red light in the experimental group was started immediately after asphyxia; ECG was recorded at the same terms as in the control group. All manipulations with the experimental animals were conducted under general anesthesia with Zoletil 100. Samples in the experimental and control groups were taken 1 h after asphyxia. The animals were sacrificed by decapitation.

ECG was recorded with a Poly-Spectr-8/B veterinary electrocardiograph and processed using Poly-Spectr Analysis/B module (Neurosoft). The following ECG parameters were studied: *PR*, *QT*, and standardized *QTc* by using Bazett formula.

The content of OMP in myocardial homogenate was assessed by measuring the content of carbonyl derivatives as described elsewhere [6]. After protein precipitation with 20% trichloracetic acid, 2,4-dini-

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trophenylhydrazine (2,4-DPH) solution in 2 M hydrochloric acid was added, incubated at room temperature for 1 h, and centrifuged for 20 min at 3000*g*. The pellet was washed with ethanol-ethyl acetate mixture to remove free dye and lipids, dried to remove solvents, and dissolved in 8 M urea in a water bath. Optical density was measured at 356 and 363 nm (to measure neutral aliphatic aldehyde-dinitrophenylhydrazones), 370 nm (neutral aliphatic ketone-dinitrophenylhydrazones), and 430 and 530 nm (basic aliphatic aldehyde and ketone-dinitrophenylhydrazones) against control sample (without 2,4-DPH). The results were expressed in optical density units/g protein; to this end, the total protein content in each sample was measured by the biuret method.

The results were processed statistically using Microsoft Excel and SPSS Statistics 21.0. The significance of differences between the groups was assessed by the Student's *t* test. Normal data distribution was verified using the Shapiro—Wilk test, correspondence of the data distribution in all groups to the same distribution law was determined using the Kolmogorov—Smirnov test.

RESULTS

Analysis of ECG recorded during asphyxia modeling showed that *QT*, *QTc*, and *PR* significantly differed ($p \leq 0.05$) from the corresponding parameters in the control group. The duration of *QT* and *QTc* after asphyxia gradually and steadily increased and reached a maximum in 15-20 min after its termination, which attested to lengthening of the electric systole (Fig. 1, *a*, *b*). It should be noted that ventricular function disturbances were irreversible. This effect was determined by hypoxia and attested to disturbances in heart function. After asphyxia *PR* interval characterizing propagation of the excitation wave in the atria rapidly returned to practically normal values, *i.e.* stable lengthening of the atrial systole did not develop (Fig. 1, *c*). Exposure to low-intensity red light led to significant recovery of the parameters of the electrical activity of the ventricles and atria.

It is known that the tissues less adapted to the anaerobic energy production, including the myocardium, are most sensitive to oxygen deficiency [10]. In light of this, we studied the dynamics of OMP content in the heart of rats subjected to asphyxia and followed by exposure to low-intensity red light.

The exposure of the heart projection to low-intensity red light after asphyxia restored the OMP content in the cardiac tissue to normal values (Table 1). Positive dynamics in OMP content after exposure of the heart to broadband red light indicates the possibility of correction of asphyxia-related disorders by low-inten-

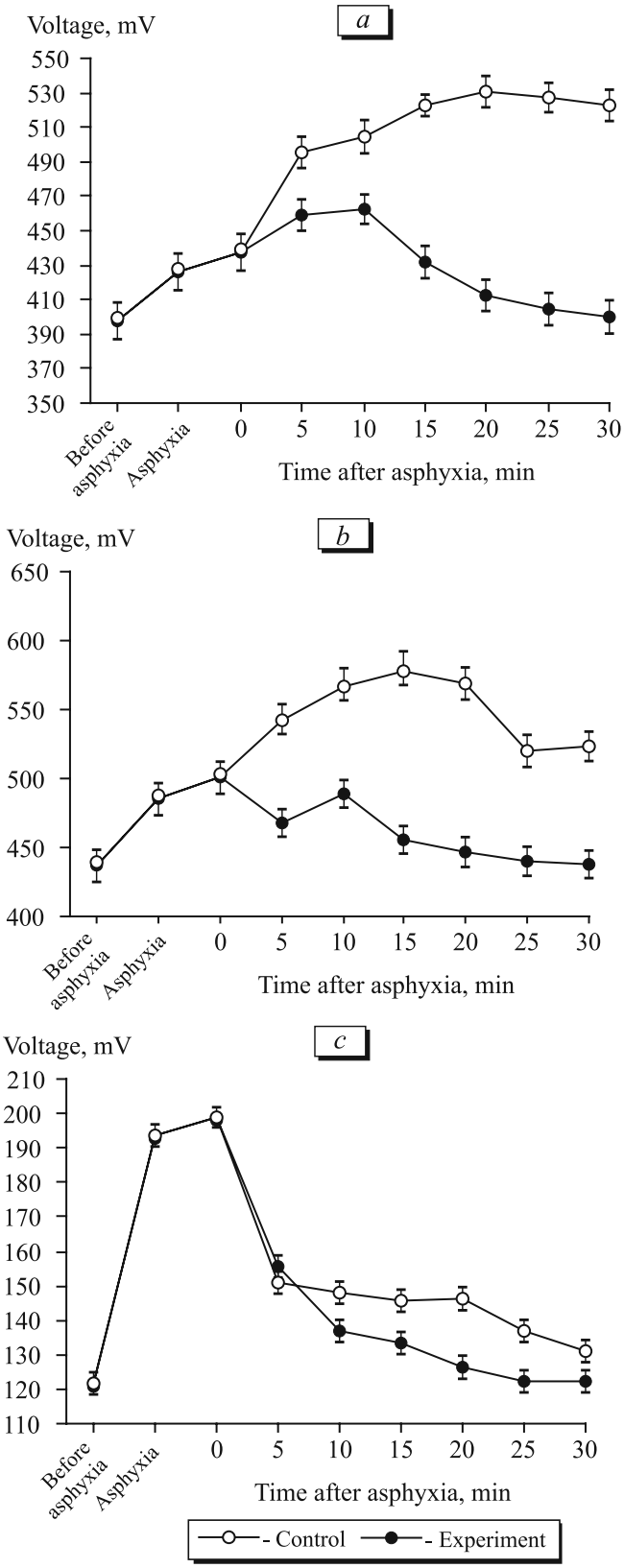

Fig. 1. Parameters of electrical activity of the heart (msec): *QT* (*a*), *QTc* (*b*) and *PR* (*c*). The results of the intact group correspond to the values before asphyxia. Averaged values for three standard leads are presented.

TABLE 1. Content of OMP, Neutral and Basic Aliphatic Aldehyde- and Ketone-Dinitrophenylhydrazones (optical density units/g protein) in the Myocardium of Rats with Asphyxia (*M*±*m*)

Wavelength	Intact group	Control group	Experimental group
356 nm	0.048±0.002	0.087±0.004*	0.045±0.006**
363 nm	0.049±0.001	0.089±0.005*	0.047±0.001**
370 nm	0.055±0.001	0.091±0.001*	0.058±0.009**
430 nm	0.028±0.001	0.052±0.002*	0.030±0.004*,**
530 nm	0.011±0.001	0.035±0.001*	0.012±0.004**

Note. $p \leq 0.05$ in comparison with *intact group, **control group.

sity electromagnetic radiation of this spectral range. In the myocardium of the experimental group, the content of oxidation products returned to normal, for most of the parameters there were no significant differences between the experimental and intact groups. In the control group, the content of the final products of protein oxidation in the myocardium significantly increased: the content of all OMP increased by 1.8 times in comparison with the corresponding parameter in intact animals. The exception was products measured at λ=530 nm, *i.e.*, basic aliphatic ketone-dinitrophenylhydrazones, whose content was 3.3 times higher than in the intact group.

The observed recovery of physiological properties of the myocardium can be explained by strengthening of the antioxidant defense in cardiomyocytes, which can be due to normalizing effect of red light energy. Our previous studies demonstrated an increase in antioxidant enzyme activity, *e.g.* superoxide dismutase and glutathione transferase [2,8]. Increased activity of antioxidant enzymes reduced the content of OMP in the myocardium. In addition, low-intensity red light can contribute to decomposition of molecular nitrosyl complexes and stimulation of the mitochondrial respiratory chain, which results in activation of ATP synthesis [14].

Thus, the content of aliphatic dephynitrophenylhydrazones in rat heart increased after asphyxia, but significantly decreased after exposure to low-intensity red light. This fact indicates the possibility of correction of heart dysfunction after asphyxia by irradiating myocardium with low-intensity broadband red light.

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