Effect of Low-Intensity Laser Irradiation and Wideband Red Light on Experimentally Ischemized Myocardium

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The effect of helium-neon laser irradiation and wideband red light on electrical activity of open rat heart was examined after experimentally produced ischemia. The red light applied with the help of optical fibers modified parameters of cardiac electrical activity and intensity of lipid peroxidation in the myocardium. The effects of laser and red light irradiation differed significantly.

Key Words: *ischemia; light; lipid peroxidation*

Low-intensity red light can affect the contractile activity of isolated heart after ischemia [9]. The effects of wideband light and irradiation of the same intensity produced by a helium-neon (He-Ne) laser were comparable. The key mechanism underlying these effects is light-induced (electromagnetic) regulation of lipid peroxidation (LPO). Coronary heart disease (CHD) is accompanied by up-regulation of LPO processes in myocardial tissues [6,7], which provokes damages to cardiomyocytic membranes and aggravates the CHD symptoms. Accumulation of molecular LPO products is an indicator of these processes [1,5,11]. Experiments on isolated rat hearts subjected to acute total ischemia revealed a 2-fold decrease in the content of diene conjugates and malonic dialdehyde (MDA) in the myocardial tissues caused by exposure of the isolated heart to He-Ne laser or wideband red light [10].

Our aim was to study intensity of LPO in myocardial tissues and electrical activity of open rat heart during experimental myocardial ischemia.

MATERIALS AND METHODS

Experiments were carried out on male random-bred albino rats (n=91) weighing 250-280 g. The rats were anesthetized with intramuscular sodium etaminal (50 mg/kg). After tracheotomy, the rats were artificially ventilated. The skin was cut along the median sternal line, the thorax and pericardium were opened. Ischemia was modeled by occlusion of the left coronary artery. To this end, a ligature placed on the left branch of the coronary artery under the left auricle of the heart was tightened for 5 minutes.

The rats were randomized into 2 control and 2 experimental groups. In experimental group 1 (n=21), the sinoatrial node was irradiated with laser, while in the experimental group 2 (n=25) the same region was irradiated with wideband (luminescent) red light. In both experimental groups, irradiation started immediately after removal of the ligature and lasted for 10 min. In both control groups, the thorax was opened as described above. In the control group 1 (n=23), sham irradiation was performed without exposure of the heart to ischemia, while in control group 2 (n=22) sham irradiation was simulated after experimental ischemia modeling. ECG was recorded using a Polygraph cardiograph coupled to PC. The data were analyzed using a Polispektr software. The sources of light radiation were an LG-13 He-Ne laser and original luminescent optical fiber apparatus [8]. The wavelength at the peak of luminescent spectrum was 640 nm with bandwidth of 70 nm. The diameter of the light spot was 3 mm, and the intensity of radiation in the illuminated area was 5 mW/cm^2.

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TABLE 1. Dynamics of HR Recovery during Reperfusion

Group	HR (min^{-1})				
	before experimental ischemia	ischemia minute 5	reperfusion minute 3	reperfusion minute 5	reperfusi minute 10
Experimental 1	263.2±15.4	246.7±15.6	256.0±13.8	275.0±16.5	154.3±17.2
Experimental 2	285.8±11.4	269.5±11.7	249.7±11.1	259.7±12.6	280.3±11.7

Note. Here and in Table 2: the confidence limits were determined at $*p<0.05$.

The intensity of LPO was assessed by the content of its products in the myocardial tissues: diene and triene conjugates and MDA [4]. The state of antioxidant protection in cells was assessed by SOD activity [12]. The level of LPO and enzyme activity (SOD) were measured in myocardium homogenate: 1 g tissue in 3 ml phosphate buffer (pH 7.5) for LPO and 100 mg tissue in 1 ml Tris buffer (pH 7.8-8.0) for SOD.

RESULTS

High mortality was noted in control group 2 (100%) and in experimental group 1 (63%), while in experimental group 2 it was only 11%.

In experimental group 1, HR varied in a wide range, pronounced bradycardia was observed on reperfusion minute 10 (Table 1). Application of the wideband red light restored HR to reperfusion minute 10.

The exposure of the myocardium to laser or luminescent (wideband) light decreased the level of LPO products in the myocardium (Table 2). The only exclusion was the content of triene conjugates in experimental group 1. The decrease in LPO primary products was more pronounced in experimental group 2 and 1 (26 and 16% from the control, respectively).

The drop in the level of LPO products can be caused by photochemical processes related to SOD activation. SOD is a very important enzyme components of the antioxidant defense system in the cell. It is important that SOD absorption band lies within the red part of the light spectrum [2,3]. Table 2 shows that the wideband red light enhanced SOD activity in myocardial tissues, while laser irradiation exerted an opposite effect.

Thus, our data demonstrated the possibility of modifying functional activity of open heart after ischemia period by irradiation of the myocardium with low-intensity red light. The peculiarities of the effects of laser irradiation can be related to optical effects of coherent light in control myocardial structures, redistribution of radiation intensity, and local overdoses in the regions of interference peaks.

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TABLE 2. Content of LPO Products in Rat Myocardium after Experimental Ischemia and 10-min Reperfusion

Group	SOD activity (U/g tissue/min	DC, opt. dens. units/TL	TC, opt. dens. units/TL	MDA, nmol/g tissue
Control 1	21.86±0.71	0.49±0.05	0.19±0.03	2.40±0.12
Control 2	16.75±0.93	0.58±0.08	0.18±0.03	3.09±0.13
Experimental 1	8.96±0.62	0.43±0.02	0.17±0.02	2.67±0.20
Experimental 2	13.06±0.81	0.49±0.04	0.21±0.03	2.43±0.09

Note. TL, total lipids; DC, diene conjugates; TC, triene conjugates; MDA, malonic dialdehyde.

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