Effects of Red Light on Postischemic Myocardium during Reperfusion

O. V. Drugova, V. A. Monich, and O. V. Zhitnikova

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 131, No. 4, pp. 386-387, April, 2001 Original article submitted December 26, 2000

Effects of low-intensity red light on lipid peroxidation in isolated rat heart in the postischemic period were studied. It was established that both laser and wideband luminescent irradiation applied during reperfusion reduced the content of lipid peroxidation products in tissues to a near-control level. This effect is possibly associated with reactivation of antioxidant enzymes.

Key words: ischemia; isolated heart; light; laser

Lipid peroxidation affecting all plasma membrane components plays an important role in ischemic damage to cardiomyocytes [4]. Reactivation of lipid peroxidation in the early reperfusion period is the leading pathogenic factor responsible for reperfusion-induced impairment of myocardial contractile activity [1]. In light of this, inhibition of lipid peroxidation remains an important clinical problem. Published data suggest that functional activity of antioxidant enzymes decreased during ischemia and, especially, during reperfusion [1]. The evidence on reactivation of superoxide dismutase (SOD) by helium-neon (He-Ne) laser irradiation shows a principle way of normalization of free radical oxidation [2].

Our aim was to evaluate the possibility of preventing postischemic damage to the heart with low-intensity incoherent light and to compare the phototherapeutic effects of laser with those produced by incoherent light of the same spectral range and intensity.

MATERIALS AND METHODS

Experiments were carried out on isolated Langendorf-Fallen perfused hearts from adult inbred albino male rats. The hearts were perfused with oxygenated Krebs—Henseleit solution at 37°C [7]. After 15 min the per-

Department of Medical Physics and Informatics, State Medical Academy, Nizhii Novgorod. *Address for correspondence:* vam@n-nov.mednet.com. Monich V. A.

fusion was stopped to reproduce ischemia and resumed after 30 min. During 15-min reperfusion the hearts were irradiated with a He-Ne laser (experimental group 1) or luminescent lightguide source (group 2), or reperfused without irradiation (control). The lightguide tip was placed at a distance of 1 mm from the myocardium over the sinoatrial node (SN) or the left ventricle (LV). Illuminated area was a circle 1.5 mm in diameter. The radiation powers used for He-Ne laser and incoherent light source were 0.2 mW/cm² and 1.5 mW/ cm², respectively. The spectrum of luminescent light had a maximum about 630 nm with half-height width of 70 nm. Tissue samples isolated after 15-min perfusion, 30-min ischemia, and 15-min reperfusion were stored in liquid nitrogen. The intensity of lipid peroxidation processes was evaluated by the content of diene and triene conjugates [3], and Schiff bases [8]. The state of antioxidant defense system was assessed by superoxide dismutase (SOD) and catalase activities measured by conventional methods [6,9]. To this end, the hearts were homogenized in sucrose solution (pH 7.4) and centrifuged for 15 min at 1000 rpm in a PC-6 centrifuge.

RESULTS

Myocardial ischemia was accompanied by considerable accumulation of primary lipid peroxidation products (Table 1). Thus, the contents of diene and triene conjugates considerably increased compared to baseline

Lipid peroxidation product	15-min perfusion (baseline)	30-min ischemia	15-min reperfusion				
			without irradiation (control)	applied to SN		applied to ventricle	
				NI	LI	NI	LI
Diene conjugate	100.00±22.92	206.83±21.22	325.00±15.43	173.17±12.39	117.56±21.57	278.05±8.59	124.88±17.96
Triene conjugate	100.00±37.75	174.17±19.01	217.88±11.24	115.23±14.36	113.91±14.53	123.18±26.34	164.24±12.90
Schiff bases	100.00±13.75	100.69±19.95	140.97±12.98	87.56±9.93	98.39±8.45	_	_

TABLE 1. Effects of Laser (LI) and Incoherent Irradiation (NI) on Content of Lipid Peroxidation Products (%) in Isolated Hearts During Reperfusion ($M\pm m$)

levels. Postischemic reperfusion for 15 min led to further accumulation of lipid peroxidation products. Irradiation during reperfusion markedly reduced the content of lipid peroxidation products in the myocardium (Table 1). Changes produced by laser and incoherent red light were similar. The observed effects are probably related to photoreactivation of antioxidant enzymes in cardiomyocytes. During ischemia ATP synthesis is switched to the anaerobic way, which accompanied by tissue acidosis due to activation of glycolysis and lactate accumulation. SOD activity decreases at low pH [2]. Red light promotes SOD reactivation in living tissue, which can explain the observed normalization of lipid peroxidation in cardiomyocytes in our experiments.

It should be noted that irradiation of the SN region was more effective than irradiation of the LF regions (Table 1), which was probably due to stimulation of contractile of ischemic myocardium. It should be also emphasized that the intensity of incoherent light was one order of magnitude higher than that of the laser. At intensities of 1 mW/cm² and higher, laser light produced arrhythmic contractions and fibrillation of the myocardium. Only when the intensity of the laser beam was reduced to few tenths of mW per cm²,

the effects were comparable in both experimental groups. It is likely that this phenomenon relates to optical interference of the coherent light in myocardial tissue and concentration of luminous energy of high intensity at local maximums.

REFERENCES

- 1. M. V. Bilenko, *Ischemic and Reperfusion Damages in Organs* [in Russian], Moscow (1989).
- E. A. Gorbatenkova, O. A. Azimova, and Yu. A. Vladimirov, Bofizika, No. 4, 717-723 (1988).
- V. Z. Lankin, E. N. Gerasimova, and L. B. Kasatkin, *Kardiologia*, No. 6, 71-75 (1979).
- F. Z. Meyerson, Pathogenesis and Prevention of Stress- and Ischemia-Induced Cardiac Damages [in Russian], Moscow (1984).
- 5. V. A. Monich and S. L. Malinovskaya, *Medtekhnika*, No. 5, 33-36 (1991).
- 6. H. Aebi, Biochemistry, 2, 636-647 (1970).
- E. T. Fallen, W. G. Elliot, and R. Gorlin, *J. Appl. Physiol.*, 22, No. 4, 836-839 (1967).
- 8. B. Z. Fletcher, C.J. Dillared, and A. V. Tappel, *Anal. Biochem.*, **52**, No. 3, 497-499 (1973).
- 9. M. Nishicimi, A. Roo, and K. Xadi, *Biochem. Biophys. Res. Commun.*, **146**, No. 2, 849-854 (1972).