

Effect of Low-Intensity Luminescent Radiation on Recovery of Heart Function in Postischemic Period

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 128, No. 9, 302-304, September, 1999
Original article submitted March 1, 1999

We studied the effect of low-intensity red light on the contractile function of isolated heart in the postischemic period. The sources of luminescent radiation used in the study produced visible light of different spectral bands and allowed to illuminate local regions of the myocardium.

Key Words: *ischemia; isolated heart; light*

A low-intensity red laser is highly efficient in the treatment of hypoxia, ischemia, and their consequences [1,3-5]. However, it was shown that the effects of noncoherent luminescent radiation on biological objects sometimes exceeded those of laser light [2, 6]. We have previously found that illumination of the sinus node exerts a bradycardiac effect on isolated heart.

This study was aimed at investigation of the effect of low-intensity luminescent red light on the contractile function of isolated heart in the postischemic period.

MATERIALS AND METHODS

Experiments were carried out on outbred adult male albino rats weighing 250-300 g. The hearts were isolated by the method of Langendorff-Fallen [7]. Chest opening, isolation of the aorta, and dissection of the heart were performed in heparinized animals under Nembutal anesthesia (35 mg/kg, intraperitoneally). The open perfusion of the coronary arteries with a Krebs-Henseleit solution (pH 7.4 after saturation with 95% O₂:5% CO₂ gas mixture) was performed through an aortal cannula. After 15-min perfusion at 37°C, the aorta was occluded to model total myocardial

ischemia. Reperfusion was started after 30-min ischemia. A latex balloon with a constant volume was inserted into the left ventricle to examine the contractile function of the heart. The curve of ventricular pressure and its first derivative were recorded with an H-338 recorder. From these curves, the developed pressure (DP), end-diastolic pressure (EDP), maximum rates of pressure rise and decay, and heart rate (HR) were calculated after 15 min of perfusion and after 7, 10 and 15 min of reperfusion.

The animals were divided into experimental ($n=70$) and control ($n=50$) groups. In the experimental group, two series of experiments were performed: in series I a low-intensity radiation was delivered to the sinus node, in series II the left ventricle was irradiated. The control group comprised sham-irradiated rats.

Low-intensity illumination with visible light was started simultaneously with the onset of reperfusion. A Lumir system composed of optic fibers with a photoluminescent agent added to a light guide core was used as a source of non-coherent luminescent radiation. The distal end of a monofiber light guide with a diameter of 0.8 mm was brought to the sinoatrial node and fixed at a distance of 1 mm from the surface. The illuminated area represented a circle 1.5 mm in diameter. Illumination intensity was 1.5 mW/cm². Radiation spectrum (580-650 nm) belonged to a red light range. Illumination was performed continuously. The data were analyzed statistically with Student's *t* test.

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TABLE 1. Changes in Myocardial Contractility in Postischemic Period (% , $M \pm m$)

Reperfusion, min	Experimental series	Rate		DP	EDP	HR
		contraction	relaxation			
7	Control	80 \pm 7.68	75 \pm 12.2	79.17 \pm 11.1	112.14 \pm 10.3	93.4 \pm 6.59
	Red light to sinus node	116.67 \pm 8.9	78 \pm 9.8	126.67 \pm 10.2	200 \pm 11.1	61 \pm 7.8
	ventricle	121.3 \pm 7.9	93 \pm 7.02	158.3 \pm 8.4	106.6 \pm 5.9	93.2 \pm 8.5
10	Control	73.55 \pm 7.09	78.57 \pm 10.1	79.5 \pm 8.9	122.86 \pm 14.75	83.29 \pm 11.02
	Red light to sinus node	106.67 \pm 8.9	78 \pm 9.6	141.67 \pm 7.01	200 \pm 9.8	61 \pm 7.8
	ventricle	113 \pm 10.2	93 \pm 9.8	152.3 \pm 9.2	283.4 \pm 14.8	96.6 \pm 7.5
15	Control	78.43 \pm 5.57	78.57 \pm 10.1	80.4 \pm 7.03	117.4 \pm 11.07	90.4 \pm 9.57
	Red light to sinus node	105.4 \pm 9.02	100 \pm 5.6	102.67 \pm 11.1	183.3 \pm 10.2	89 \pm 9.5
	ventricle	110.7 \pm 11.2	70.7 \pm 6.78	134.3 \pm 7.3	220 \pm 13.2	100 \pm 1.4

Note. The values corresponding to the 15th min of perfusion were taken as 100%.

RESULTS

Contractile activity of the isolated heart during reperfusion after 30-min ischemia was not restored. The postischemic period was characterized by impaired contraction and relaxation of the myocardium and reduced DP, especially pronounced at the 15th min of reperfusion (Table 1).

Red light illumination of the sinus node during reperfusion reduced HR and increased the rate and force of contractions. This decrease in HR was due to the influence of red light on pacemaker cells, since the affected area was confined to the sinoatrial node. A decrease in excitation rate can result from prolongation of pacemaker refractory period, while muscle force is proportional to the amount of Ca^{2+} entering myofibrils. Taking into consideration constant energy supply under given conditions, it can be suggested that the positive inotropic effect against the background of bradycardia was due to increased Ca^{2+} entry during the action potential plateau, which was probably prolonged due to light-induced enhancement of slow inward Ca^{2+} current.

Illumination of cardiomyocytes induced even more pronounced inotropic effect without affecting HR. It can be suggested that low-intensity radiation enhances membrane permeability for Ca^{2+} , thereby directly increasing the concentration of intracellular Ca^{2+} and consequently the strength and rate of contractions.

It can be noted, that the positive inotropic effect of the red light and the absence of HR shifts play an essential role in the postischemic recovery of the cardiac function.

Accumulation of calcium in cardiomyocytes under conditions of energy deficit and reduced activity of Ca^{2+} -ATPases in the postischemic period [5] gradually decrease the rate of relaxation and promote myocardial contracture, which is associated with impaired removal of excessive Ca^{2+} during diastole. Because of this, ERD after red light illumination was higher than in the control, attesting to increased reperfusion contracture of the myocardium.

Thus, illumination of the sinus node or myocardium with red light in the postischemic period affects Ca^{2+} membrane permeability and exerts a positive inotropic effect with or without bradycardia depending on the irradiated area. Irradiation can have negative consequences like myocardial contracture caused by Ca^{2+} overload under conditions of energy deficit. The effect of red light on Ca^{2+} membrane permeability needs further investigations.

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