

GENERAL PATHOLOGY AND PATHOLOGICAL PHYSIOLOGY

Reaction of Bulbar Cardiovascular and Respiratory Neurons to Intra-Atrial Laser Irradiation

S. D. Mikhailova, A. V. Kudinova,
T. M. Semushkina, and G. I. Storozhakov

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Unilateral changes in activity of afferent and intercalary cardiovascular neurons were observed during intra-atrial laser irradiation. Most respiratory neurons shortened the burst duration and the number of spikes in it. These changes in neuronal activity seem to play an important role in the antiarrhythmic effect of laser irradiation, preventing discoordination of activity of the bulbar cardiovascular center.

Key Words: *laser irradiation; impulse activity; cardiovascular neurons; ischemic cardiac arrhythmias*

Intra-atrial laser irradiation (IALI) has a protective effect during myocardial ischemia. It decreases the probability of severe cardiac rhythm disturbances and preserves cardiac innervation. The cold blockade of vagal afferent fibers antagonizes this effect and provokes ventricular fibrillation [6]. In addition, the development of ventricular fibrillation in ischemic myocardium is preceded by discoordination of impulse activity (IA) of the afferent and intercalary neurons of the bulbar cardiovascular center as well as by changes in IA of neurons in the bulbar respiratory center primarily due to prolongation of bursts and an increase in the number of impulses in them [4,6].

These premises set a problem of whether IALI affects neuron functioning in the bulbar centers via the cardiac afferent systems. We investigated IA of cardiovascular and respiratory neurons during IALI.

MATERIALS AND METHODS

Experiments were carried out under Nembutal anesthesia (40 mg/kg intraperitoneally) on 49 artificially ventilated cats of both sexes weighing 2.5-4.0 kg.

Irradiation of the right atrium lasted 20 min (an ULF-01 He-Ne laser, output power $N=3-5$ mW, wavelength $\lambda=632.8$ nm). Electrical activity of neurons was recorded in the region of medulla oblongata solitary tract nuclei (2 mm in rostral and caudal direction from the obex) using extracellular glass microelectrodes filled with 2.5 M KCl (resistance 3-5 M Ω). Functional identification of the neurons was performed on the basis of character of initial IA in accordance to the criteria described elsewhere [5,10]. Dynamics of IA in the cardiovascular neurons was assessed by changes in the rate of discharge and by transformation of its character (variations in the burst duration and in the pulse distribution within a train). The character of changes in the activity of the respiratory neurons was determined by the number of impulses in a burst, its duration, and by the mean discharge frequency [9]. These parameters were analyzed for the minutes 1, 5, 10, 15, and 20 of irradiation. Changes in neuronal activity were assessed by comparing it with the preceding measurements. A change in a single analyzed parameter was sufficient to document the reaction. In addition to neurograms, the following data were recorded with the help of an SR-41 recorder (Nihon Kohden): electrocardiogram

in standard leads I and II, blood pressure in the femoral artery, and the pneumogram. Data were processed with an original digitizer and the program "Multisignal". The results were statistically analyzed using Student's *t* test.

RESULTS

In the first part of the study we evaluated IA of 40 neurons in the bulbar cardiovascular center during IALI: 5 of them were afferent, and 35 were intercalary neurons. Two of 5 recorded afferent neurons demonstrated a change in IA in the first minute of irradiation. However, all the recorded neurons modified their IA to the minute 5 of irradiation: 4 of them attenuated the discharge rate while one neuron incremented it. The same number of increments and decrements in IA frequency was observed on minute 10th, 15th, and 20th of irradiation.

Similar to the afferent neurons, the intercalary ones also responded with changes in IA from the first minute of irradiation (54%). More than a half of the responding neurons increase the discharge frequency. By contrast, on minute 5, when 83% neurons produced a response, the prevailing reaction was a decrease in IA frequency. The intercalary and afferent neurons demonstrated both increase and decrease of the IA frequency on minutes 10, 15, and 20. To the end of irradiation, IA in 31% neurons did not differ from the background activity.

The described reactions of the cardiovascular neurons were observed against the background of stable arterial pressure and heart rate. During the entire period of irradiation there were no significant changes of these parameters relative to the initial level (arterial pressure: systolic, 111.6 ± 7.1 mm Hg;

diastolic, 66.4 ± 5.6 mm Hg, heart rate 157.8 ± 8.7 beats/min).

Thus, both afferent and intercalary neurons of the bulbar cardiovascular center responded to IALI, and the trend of their reactions was uniform (Fig. 1).

In the second part of the study we investigated IA of 18 bulbar respiratory neurons during IALI: 8 of them were inspiratory, 6 inspirator-expiratory, 3 expirator-inspiratory, and 1 expiratory. 94% respiratory neurons responded to IALI by a change in IA. There were no dependence between the type of initial IA and the character of neuron response during entire period of irradiation. Changes in IA occurred mostly due to a shortening of bursts and decrease in the number of impulses in them. On the first minute of IALI, 39% neurons demonstrated a response, while the respective percentage was 56% for minute 5 and 94% for minute 20. These data indicate that IALI evokes a response not only in the bulbar cardiovascular neurons, but also in neurons of the respiratory center.

Therefore, even on the first minute of IALI, the bulbar cardiovascular and respiratory neurons change IA against the background of stable arterial pressure. Both afferent and intercalary bulbar cardiovascular neurons can either diminish or enhance IA, preserving the uniform trend of the response during entire period of IALI. The changes in IA of all respiratory neurons were manifested in shortening of bursts and in decrease of the number of impulses in them against the background of artificial ventilation. The observed neuronal responses could be evoked by sensitivity changes of the cardiac receptive apparatus under the effect of laser irradiation. This hypothesis is corroborated by the data on variation of functional state of the receptive devices in the acupuncture points [2].

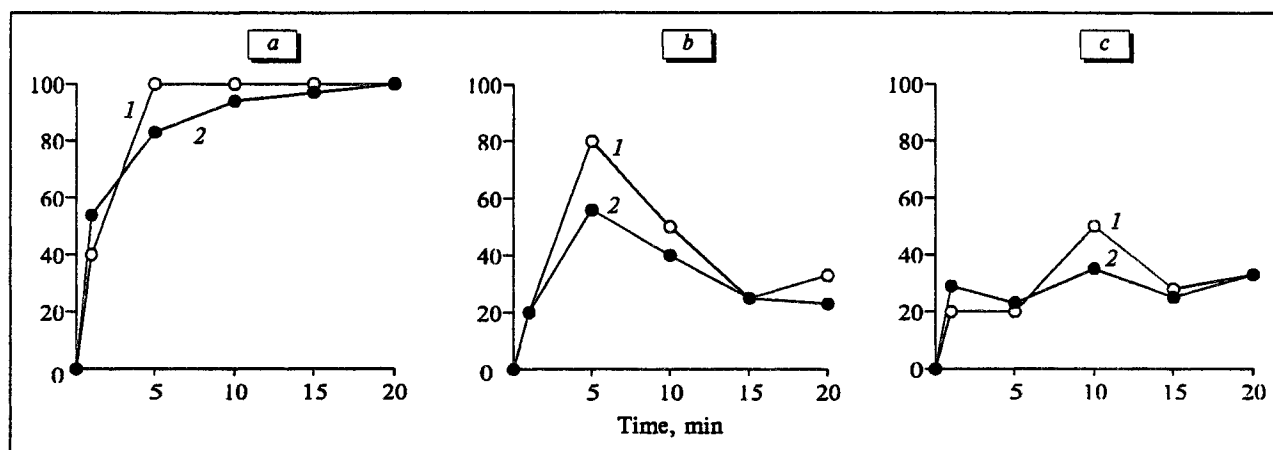


Fig. 1. Trends in the response of the cardiovascular neurons to intra-atrium laser irradiation: a) existence of reaction; b) decrease and c) increase in frequency of impulse activity. The numbers refer to: 1) afferent and 2) intercalary neurons. Ordinate: number of neurons in percent.

Previously we showed that myocardial ischemia accompanied at different stages of its development by multidirectional changes in IA of afferent and intercalary bulbar cardiovascular neurons is further aggravated by ventricular fibrillation. One can suppose that by changing the sensitivity of cardiac receptive systems, preliminary laser irradiation contributes to rearrangement of activity of cardiovascular and respiratory neurons. During the development of myocardial ischemia such a rearrangement prevents discoordination of neural activity in the bulbar cardiovascular center. As was previously shown, the unidirectional reaction of afferent and intercalary neurons is not accompanied by ventricular fibrillation during the development of myocardial ischemia [4].

Thus, changes in the activity of the bulbar centers during IALI may play a key role in preventing severe ischemic arrhythmias in addition to inhibition of lipid peroxidation, increase in blood microcirculation, etc. [1,3,8].

REFERENCES

1. E. B. Gel'fgat, R. I. Samedov, Z. N. Kurbanova, and G. G. Gadzhiev, *Cardiology*, **33**, No. 2, 22-23 (1993).
 2. V. I. Kozlov, V. A. Builin, N. G. Samoilov, and I. I. Markov, *The Basis of Laser Physio- and Reflexotherapy* [in Russian], Samara (1993).
 3. I. M. Korochkin, O. L. Barbash, and I. N. Chukaeva, *Sov. Med.*, No. 5, 36-39 (1990).
 4. G. I. Kositskii, S. D. Mikhailova, S. V. Putygin, *et al.*, *Patol. Fiziol.*, No. 6, 31 - 35 (1980).
 5. G. I. Kositskii, S. D. Mikhailova, S. V. Pytygin, *et al.*, in: *Actual Problems on Experimental Cardiology* [in Russian], Moscow (1987), pp. 12-20.
 6. S. D. Mikhailova, T. M. Semushkina, P. Kol', *et al.*, *Byull. Eksp. Biol. Med.*, **105**, No. 6, 657-660 (1988).
 7. S. D. Mikhailova, G. I. Storozhakov, S. Yu. Gukova, *et al.*, *Ibid.*, **113**, No. 5, 460-462 (1992).
 8. V. I. Ruzov and K. K. Dauksha, *Vopr. Kurortolog.*, No. 3, 19-23 (1993).
 9. R. Baumgarten, A. Baumgarten, and K. P. Schaefer, *Pflugers Arch.*, **264**, 217-227 (1957).
 10. M. Stroh-Werz, P. Langherst, and H. Camerer, *Brain Res.*, **106**, No. 2, 293-305 (1976).
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