Comparative Assessment of Conduction Anesthesia Produced by Procaine, Marcaine, Richlocaine, and RU-353

A. P. Galenko-Yaroshevskii, Yu. P. Sheikh-Zade*, and I. L. Cherednik*

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Conduction anesthesia of the vagus nerve produced by procaine (0.5%), marcaine (0.25%), richlocaine (0.5%), and imidazobenzoimidazole derivative RU-353 (0.25%) was compared in acute experiments on cats. RU-353 produced the most long-term local anesthesia (assessed by inhibition of cardiochronotropic effect of vagus nerve).

Key Words: *conduction anesthesia; procaine; marcaine; richlocaine; imidazobenzoimidazole derivative RU-353*

The search for new and modernization of known local anesthetics is an actual problem of pharmacology. Of particular importance is screening for agents for conduction anesthesia. Our aim was to compare the degree and duration of local anesthesia produced by procaine [2,4], marcaine [2,4], richlocaine [1,2], and an imidazobenzoimidazole derivative RU-353 (laboratory code) [5,6].

MATERIALS AND METHODS

Experiments were carried out on artificially ventilated temperature-controlled (37°C) cats (n=26) narcotized with intraperitoneal chloralose and urethane (75+15 mg/kg). In all cats, the cervical fragment of the right vagus nerve was isolated and cut. The peripheral stump was placed into a special bath containing the test substance. The nerve was stimulated with single trains of electrical pulses applied above the anesthetized segment synchronously to *P*-wave of intracardiac ECG. The negative chronotropic effect was assessed before and during application of local anesthetic and during the recovery period after washout.

The degree of conduction anesthesia (DCA, %) was calculated by the formula: DCA=100×(ICE-TCE), where ICE and TCE are the initial and tested chronotropic effects of stimulated vagus nerve, respectively. The chronotropic effect (CE, %) was calculated by the formula $CE=100\times(T_{n+1}-T_n)/T_n$, where T_n is duration (msec) of the last cardiac cycle before stimulation, while T_{n+1} is duration of the first cycle immediately after stimulation.

The data were processed statistically. The experimental methods are described elsewhere [3].

RESULTS

In all experimental series the maximum degree of conduction anesthesia (DCA_{max}) was 100% and the rate of local anesthesia development was similar. Probably, this fact reflects similar conditions for diffusion of different anesthetic molecules into the nerve. However, the duration of anesthesia after washout from the anesthetic and the rate of recovery of ICE drastically differ in all series (Table 1). This difference was most likely caused by individual degree of binding of local anesthetic to neural plasmalemma. The maximum ac-

Krasnodar Regional Research Medical Center; *Kuban State Medical Academy, Krasnodar. ***Address for correspondence:*** yurh@rambler.ru. Yu. P. Sheikh-Zade

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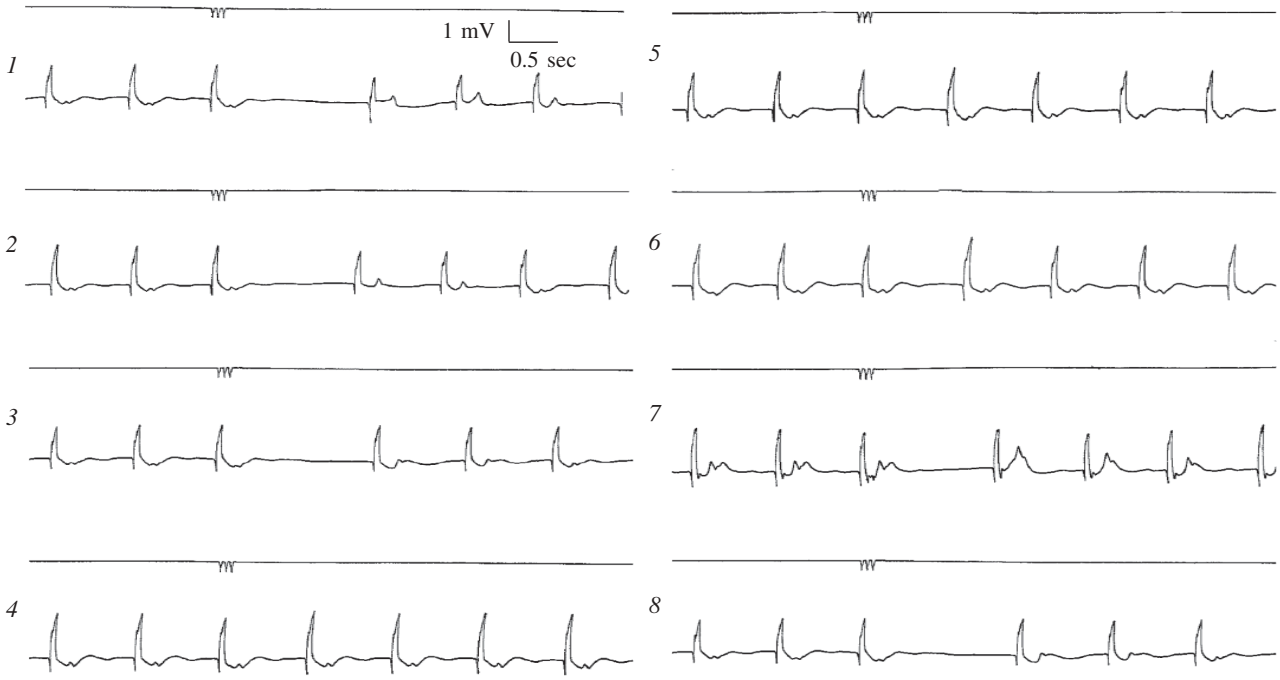

Fig. 1. Effect of local application of RU-353 on cardiochronotropic effect of vagus nerve. In each panel, the upper record is stimulation mark and the lower trace is intracardiac ECG with dominating *P*-wave. *1*) chronotropic effect of the vagus nerve before application of local anesthetic; *2-4*) vagal chronotropic effect 2, 4, and 6 min after application of 0.25% RU-353; *5-8*) the chronotropic effect 200, 220, 250, and 300 min after removal of RU-353 from the bath.

TABLE 1. Local Anesthesia Effects of Examined Agents (*M*±*m*)

Substance	*n*	Time to attaining DCA_{max}, min	Duration of DCA_{max} after removal of the agent, min	Duration of DCA_{max} drop to zero, min
Procaine, 0.5%	7	9.0±0.7	0.6±0.6^{+}	23.0±2.1^{+}
Marcaine, 0.25%	6	10.0±0.9	56.0±5.8*	211.2±14.7*
Richlocaine, 0.5%	7	11.8±3.4	0.5±0.8^{+}	182.2±16.9*
RU-353, 0.25%	6	5.7±0.2*$^{+}$	222.6±12.8*$^{+}$	105.9±17.2*$^{+}$

Note. $p<0.05$ compared to *procaine and $^{+}$marcaine.

tivity was observed in experiments with RU-353 (Fig. 1), which strongly correlates with the data on this agent obtained in other experimental models [5,6].

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