ELECTRICAL STIMULATION OF THE PHRENIC NERVE IN DOGS

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In order to study the influence of electrical stimulation of the phrenic nerve it is essential to have clinical electrical stimulators of respiration designed for patients with spinal cord injuries, diseases of the respiratory system, or postoperative complications. Experimental studies of the effect of direct electrical stimulation of the phrenic nerve [6-8] and also of paraphrenic [9], transvenous [1, 3], and radiofrequency electrical stimulation [2, 4, 5] have been undertaken in this direction. This type of stimulation has been shown to maintain adequate respiration in animals and the method is suitable for use under clinical conditions.

The aim of the present investigation was to determine optimal parameters of electrical stimulation for long-term phrenic nerve stimulation and to establish the principles governing changes in the physiological parameters of respiration with different respiration rates imposed by electrical stimulation and with natural breathing excluded.

EXPERIMENTAL METHOD

Experiments were carried out on 12 adult mongrel dogs weighing 15-25 kg. The animals were anesthetized with pentobarbital sodium (30 mg/kg). Natural breathing was suppressed by barbiturate poisoning, i.e., by injection of pentobarbital sodium in a dose of 50 mg/kg.

The ECG, the respiration rate and spirogram (by means of a META-1-25 oxyspirograph) were recorded, the respiratory minute volume (RMV) was determined, and the coefficient of oxygen utilization (CUO_2) was calculated. The experiments were carried out under closed chest conditions. Electrical stimulation of the proximal part of the phrenic nerve was applied through silver electrodes, contained in a polyethylene sleeve, fixed to the nerve. The distance between the electrodes did not exceed 3 mm. Electrical stimulation was carried out by means of an experimental electrostimulator, providing for changes in pulse duration and shape, in the duration of the pulse bursts, and in their following frequency between 5 and 20 bursts/min. The duration of each burst of pulses varied from 1 to 2 sec. Each burst was followed by a period of "silence." Its duration was determined by changing the duration of the pulse burst. The following frequency of the bursts and periods of "silence" determined the respiration rate, which could be varied from 5 to 20 cycles/min. The phrenic nerve was stimulated for 3-19 h in different animals.

EXPERIMENTAL RESULTS

At the beginning of the investigations the effect of electrical pulses of different shapes (square monopolar, square bipolar, with intervals between pulses) was studied during long-term stimulation of the phrenic nerve. The experiments showed that long-term stimulation of the nerve by monopolar pulses with a following frequency of 60 Hz led after 30-90 min to a decrease in excitability, followed by an increase in the strength of the stimulating current. This was evidently caused by the formation of a constant component, which caused depolarization of the nerve fibers. During phrenic nerve stimulation with bipolar pulses with intervals, the thresholds of the stimulating current were higher than during stimulation by monopolar pulses. Electrical stimulation of the phrenic nerve with bipolar pulses caused no significant changes in the threshold of excitability for a long period of time. It was concluded from the results of the investigation that bipolar electrical stimuli are the most physiological for phrenic nerve stimulation.

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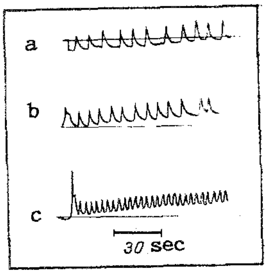

Fig. 1

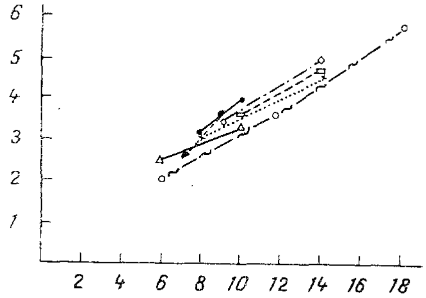

Fig. 2

Fig. 1. Spirograms recorded during electrical stimulation of phrenic nerve at different frequencies, with natural respiration excluded, in dogs. Respiration rate: a) 6, b) 9, c) 18 cycles/min.

Fig. 2. Dependence of change in RMV on RR imposed by electrical stimulation of phrenic nerve with natural respiration blocked in seven dogs (individual curves indicated by different symbols). Abscissa, RR, cycles/min: ordinate, RMV (liters/min).

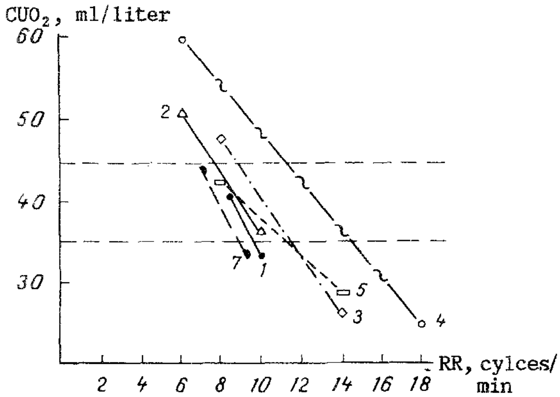

Fig. 3. Dependence of changes in CUO_2 on RR imposed by electrical stimulation of phrenic nerve with natural respiration blocked. 1-7) Nos. of dogs in experiment. Below 35 ml/liter: zone of hyperventilation, above 45 ml/liter: zone of hypoventilation. Normoventilation zone indicated by broken lines. Abscissa, RR, cycles/ min; ordinate, CUO_2 (in ml/liter).

The study of the effect of the pulse bursts showed that the most physiological form of stimulation of the phrenic nerve was to increase the number of pulses per burst up to a maximum in the course of two-thirds of the duration of the burst. This induced smooth contraction of the diaphragm.

The subsequent investigation was aimed at studying changes in parameters of respiration imposed by electrical stimulation of the phrenic nerve. Spirograms recorded in a dog with different frequencies of electrical stimulation of the phrenic nerve are given in Fig. 1. During phrenic nerve stimulation by bursts of pulses with frequencies of 6, 10, and 18/min the respiration rate (RR) was 6, 10, and 18 cycles/min respectively. With an increase in RR the amplitude of the spirograms was reduced, accompanied by a corresponding decrease in the depth of respiration. Special investigations of the relationship between changes in RMV and RR imposed by electrical stimulation are illustrated in Fig. 2. With an increase in RR there was a proportionate increase in the value of RMV, and with a decrease in RR, RMV also was reduced. At the normal RR (8-9 cycles/min) RMV was 3-3.5 liters/min. With an increase in RR to 14 and 18 cycles/min RMV increased to 4.5-5 and 6 liters/min respectively. Reducing RR to 6 cycles/min led to a decrease in RMV to 2-2.5 liters/min.

Spirograms recorded in dogs with different imposed respiration rates revealed changes in another parameter of respiration, namely CUO_2. The graph in Fig. 3 shows how CUO_2 depends on RR imposed by electrical stimulation. Three zones are visible on the graph, associated with different values of RR: hyperventilation below 35 ml/liter, normoventilation from 35 to 45 ml/liter, and hypoventilation over 45 ml/liter. As RR falls from 9 to 6 cycles/min CUO_2 rises to 47-48 ml/liter, which corresponds to hypoventilation. At this frequency the amplitude of the spirogram rises (Fig. 1a). An increase in RR to 10, 14, and 18 cycles/min causes a decrease in CUO_2 to 30, 20, and 15 ml/liter respectively. Changes in these parameters with an increase in RR indicate the development of hyperventilation. The amplitude of the spirogram under these circumstances is reduced and respiration becomes superficial (Fig. 1c). This indicates a reduction in oxygen utilization. These comparisons show that electrical stimulation of the phrenic nerve can ensure artificial ventilation of the lungs in cases when natural breathing ceases in animals. This conclusion having been drawn from analysis of the data, it was possible to construct a miniature electrostimulator with independent power supply, for long-term stimulation of the phrenic nerve and to maintain the respiratory function when natural respiration is blocked.

Miniature electrostimulators of the phrenic nerve of this kind were implanted in two dogs. They were fixed beneath the animal's skin in the neck by means of a thick silk ligature. The electrodes were contained in a sleeve which was fixed to the phrenic nerve. The apparatus was switched on while the animal was on the operating table; it stimulated respiration with a frequency of 8 cycles/min. Artificial respiration maintained the vital functions of the animal for many hours. Both electrostimulators implanted in dogs kept the animals alive, one dog for 16 h, the other for 19 h. The respiration rate corresponded to the assigned frequency throughout the experiment.

Artificial respiration applied by electrical stimulation of the phrenic nerve can thus maintain life of an animal for many hours without any significant deviations of the principal physiological parameters.

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