consequent changes in the physicochemical parameters of the membrane, and these may lead to realization of the physiological effects [2].

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EFFECT OF SODIUM HYPOCHLORITE ON OXYGEN BALANCE AND FUNCTIONAL STATE OF THE SMALL INTESTINE IN EXPERIMENTAL PERITONITIS

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The known bactericidal agent sodium hypochlorite (NaOCl) has an anticoagulant [4] action and a marked antiaggregating and disaggregating action on platelets [6]. NaOCl is effectively used in surgery to sterilize the peritoneal cavity in the case of spreading peritonitis and in the treatment of septic wounds [2, 3].

In the investigation described below the effect of NaOCl was studied on the oxygen balance and the acid—base state (ABS) of the blood and functional activity of the small intestine in spreading peritonitis, when administered intravenously and intraperitoneally.

EXPERIMENTAL METHOD

Experiments were carried out on 121 albino rats weighing 180-230 g, in three series, under combined anesthesia (5 mg diazepam + 600 mg sodium hydroxybutyrate/kg body weight intramuscularly). Parameters of the electromyogram (EMG), partial pressure of oxygen in the intestinal wall (pO_2), gas composition and ABS of the blood in intact animals, after experimental spreading peritonitis for 24 h, and also 30-40 min and 24 h after intravenous or intraperitoneal injection of NaOCl. The same parameters but when a 0.9% solution of sodium chloride (physiological saline — PS) was used served as the control. The NaOCl used in the work was obtained electrochemically [3]. A 0.1% solution of NaOCl in a dose of 10 mg/kg body weight was injected intravenously, and 3-4 ml of the 0.12% solution was injected intraperitoneally. Series I consisted of 47 intact rats (of which 19 received NaOCl intravenously and 16 intraperitoneally). Twelve animals served as the control. In series II, on 22 animals (16 experimental and six control) the effect of intraperitoneal injection of NaOCl on the course of spreading peritonitis was studied. In series III, on 52 rats with spreading peritonitis, changes in values of pO_2

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TABLE 1. Changes in Gas Composition and ABS of Arterial and Venous Blood of Rats with Spreading Peritonitis Following Intravenous Injection of NaOCl (M ± m)

Parameter	Initial data	Peritonitis	NaOCl	PS
pH:				
a	7,33±0,03	7,44±0,02*	7,44±0,05	7,62±0,04**
v	7,30±0,03	7,35±0,03	7,36±0,02	7,46±0,01**
pCO_2, mm Hg				
a	30,0±4,96	28,5±5,13	26,4±3,94	16,2±3,13
v	45,6±3,90	41,7±2,12	42,5±2,06	32,1±1,15**
pO_2, mm Hg				
a	103,0±8,01	92,4±7,90	85,6±5,17	85,0±1,65
v	40,2±2,90	31,1±3,64	36,6±4,16	39,6±3,31
HCO_3, mmoles/liter				
a	15,1±1,90	18,6±2,98	17,1±1,01	16,0±1,60
v	21,9±0,93	23,4±2,01	24,0±0,75	22,8±0,79
BE, mmoles/liter				
a	—8,7±1,08	—3,1±2,04*	—4,8±1,48	—0,7±0,57
v	—4,4±0,96	—1,9±2,36	—1,0±0,95	0,65±0,76
O_2SAT, %:				
a	97,1±0,47	96,9±0,78	95,8±1,60	97,9±0,26
v	67,9±5,01	53,1±6,02	64,6±7,30	76,0±3,62**

Legend. *p < 0.05-0.01) Difference from initial data, **p < 0.05-0.001) difference from parameter in peritonitis; a) arterial, v) venous blood.

and the EMG parameters (20 rats) and in the blood gas composition and ABS of arterial and venous blood (32 rats) were determined in animals receiving intravenous infusion of NaOCl. Experimental peritonitis was produced by a modified method in [5]. Bioelectrical activity (BEA) was recorded by means of silver clip electrodes from the side of the serous membrane of the intestine, on a Mingograf-82 apparatus, for a constant time of 1 sec and at a constant speed of 2.5 cm/sec. During analysis of the EMG the amplitude (in millivolts) and the following frequency of the potentials (the number of cycles per minute) were estimated. The value of pO_2 was determined in the muscular layer of the small intestine by the polarographic method in [1], and the blood gas composition and ABS were studied on a Corning-178 blood gas analyzer.

EXPERIMENTAL RESULTS

In the experiments of series I rhythmic slow electrical waves (SEW) were recorded on the EMG in the initial state, with a following frequency of 21.8 ± 0.4 cycles/min and an amplitude of 0.28 ± 0.01 mV; pO_2 in the wall of the small intestine was 27.1 ± 1.1 mm Hg (Fig. 1b). An increase of pO_2 to 30.8 ± 2.5 mm Hg and a decrease in the amplitude of the potentials to 0.27 ± 0.02 mV and in their frequency to 20.6 ± 0.6 cycles/min were observed 30 min after intravenous infusion of NaOCl. Meanwhile, with both methods of administration of PS, BEA and the oxygen balance of the small intestine did not change significantly, although there was a tendency for pO_2 to rise and for the amplitude of the potentials of the intestinal wall to fall. Thus, intravenous and, in particular, intraperitoneal injection of NaOCl in intact animals led to an increase in oxygenation of the tissues of the small intestine and to an increase in its metabolism.

A cloudy effusion with floccules of fibrin, hyperemia of the peritoneum, fibrinous deposits, and swollen loops of intestine were found in all parts of the peritoneal cavity of the surviving animals 24 h after experimental production of spreading peritonitis. Data on ABS in the blood revealed metabolic alkalosis, the extraction of oxygen from the blood was considerably increased, as shown by an increase in the arteriovenous difference for hemoglobin oxygen saturation (AVD $SATO_2$) (Table 1), and pO_2 in the wall of the small intestine increased by 11.4%. Meanwhile signs of failure of the mechanisms for compensation of hypoxia were observed, as shown by a characteristic fall of pO_2 and AVD for pCO_2, as a result of which the functional state of the small intestine was disturbed: the amplitude of the biopotentials was reduced, and their shape and the rhythm of their following frequency were altered. Thus in experimental peritonitis a disparity developed between oxygen transport to the tissues and oxygen consumption, leading to tissue hypoxia.

In the experiments of series II washing out the peritoneal cavity with NaOCl solution led after 30-40 min to a significant rise of pO_2 in the wall of the small intestine to 41.4 ± 2.7 mm Hg and to a moderate increase in the amplitude of SEW to 0.21 ± 0.02 mV. The frequency of the potentials did not change significantly. The important features here were restoration of the cyclic pattern of alternation of the potentials and of their shape, evidence of recovery of intestinal motor activity. Meanwhile, in the control experiments, after rinsing out the peritoneal cavity with ES, a further reduction of pO_2 and, in particular of the amplitude of SEW to 0.1 ± 0.09 mV were observed. The rhythm of the potentials and their shape did not change significantly. All the animals were still alive 24 h after the peritoneal cavity had been rinsed out with NaOCl. On repetition of the laparotomy the signs of peritonitis were observed to have disappeared: there was no effusion and no fibrin in the peritoneal cavity, only moderate hyperemia of the peritoneum. By this time pO_2 in the intestinal wall had fallen somewhat (to 23.7 ± 3.0 mm Hg) compared with data obtained during peritonitis, and it was close to the original value. BEA of the small intestine did not differ from the initial level and consisted of regularly repeated polyphasic two-component complexes, with an amplitude and frequency indistinguishable from the normal values. After rinsing out the peritoneal cavity with PS, three rats died the following day (50%). In this case reduction of pO_2 to 17.7 ± 6.6 mm Hg and some increase in the amplitude of SEW were observed, but the initial value was not reached.

In the experiments of series III intravenous injection of NaOCl caused no significant changes in ABS. The compensated metabolic alkalosis was still present although it showed a tendency to diminish. Meanwhile normalization of the oxygen balance and of the functional state of the small intestine took place, as shown by the values of the tissue pO_2, the amplitude of SEW, AVD $SATO_2$, and pO_2 (Table 1). On intravenous infusion of PS, on the other hand, decompensation of the alkalosis, which in this case was mixed in character took place. Under these circumstances extraction of O_2 from the blood was significantly reduced, as shown by a decrease of 30% in AVD $SATO_2$. Meanwhile the parameters of the oxygen balance (p_vO_2, tissue pO_2) increased. This opposite character of the changes probably indicates a decrease in the oxygen consumption of the tissues, which did not lead to abolition of the hypoxia against the background of marked alkalosis. The absence of recovery of small intestinal function is indirect evidence in support of this conclusion.

Recovery of BEA of the small intestine was observed 24 h after intravenous injection of NaOCl, as shown by a significant increase in the amplitude of SEW to normal values and restoration of the shape of the curve. There was virtually no change in the tissue pO_2, which was 23.8 ± 2.5 mm Hg (Fig. 1d). Meanwhile, 24 h after infusion of PS an increase in the amplitude of SEW and a decrease in tissue pO_2 were observed compared with changes during peritonitis. Changes of this kind are evidence of the more intensive and rapid recovery of the oxygen balance of the small intestine during infusion of NaOCl.

Thus besides its antibacterial action, NaOCl when given by both intraperitoneal and intravenous injection, restores the normal oxygen balance and metabolism of the small intestine and metabolism of the small intestine and also restores its motor function. The antihypoxic effect of NaOCl is most probably realized through an increase in the oxygen concentration in the blood and an increase in its utilization by the tissues. The results indicate the vale of NaOCl in spreading peritonitis, when given with the aim of antibacterial therapy and the control of tissue hypoxia. Under these circumstances NaOCl has no side effects on the functional state of the intestine.

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