Effects of Mesodiencephalic Modulation on Electrical Spike Activity in Rat Small Intestine after Massive Blood Loss

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Blood loss-induced ischemia reduced intestinal electrical spike activity and delayed or blocked the migrating myoelectric complex. A single session of mesodiencephalic modulation significantly intensified electrical discharges, stimulated contractions of the small intestine, and restored normal cycles of the migrating myoelectric complex.

Key Words: *small intestine; migrating myoelectric complex; spike activity; rhythms; blood loss*

There is ample evidence that mesodiencephalic modulation (MDM) affects the digestive tract function in diverse pathological conditions [1]. Positive effects of MDM are due to significant elevation of stress-protective activity of opioid peptides (endorphins and enkephalins) [2]. However, there are no data on the effects of MDM on intestinal contractile activity and on MDM potential for restoring electrical spike activity (SA) after massive blood loss.

It is known that SA closely correlates with contractile activity of the small intestine [7]. Periodic bursts of action potentials form two rhythms: minute and fasting (the migrating myoelectrical complex, MMC) periodicity. These rhythms can be used for the assessment of motor function of the gastrointestinal tract.

The purpose of this work was to investigate the effects of MDM on intestinal SA after massive blood loss.

MATERIALS AND METHODS

Experiments were carried out on male Wistar rats weighing 250-300 g anesthesized with 5% hexenal (0.3 ml/100 g, intraperitoneally). Two groups were formed: control (n=9) and experimental (n=8). All rats underwent midline laparotomy and bipolar stainless steel electrodes (0.2 mm in diameter with 2-mm distance between tips) were implanted in the intestine 5 cm distal to the Treitz ligament. Connecting wires were passed through the walls of the peritoneal and pelvic cavities and the leads were brought out at a distance 5-6 cm from the tail tip. A silicone catheter was inserted in *v. cava inferior* and passed under the tail skin together with the electrodes.

In experimental group, two subcutaneous electrodes for MDM were additionally implanted in the frontal area and occipital fossa with their leads brought out in the interscapular region.

Acute blood loss was modeled by draining venous blood (3.5% body weight) at a rate of 0.8 ml/min for approximately 20 min through syringe connected to the intravenous catheter.

Electrical spike activity was recorded before, during, and for 4 h after blood draining. The signals from the recording electrodes were amplified (0.1 mV input sensitivity, 1-200 Hz frequency band), fed to a computer, and analyzed using the nonlinear filtration algorithm [3]. Time characteristics of SA were additionally assessed by visual inspection of the electromyogram.

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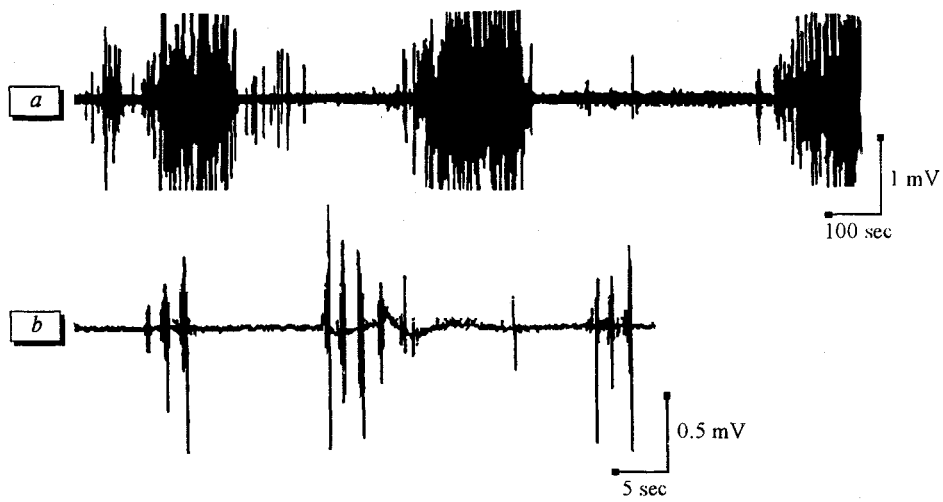

Fig. 1. Normal electrical spike activity of rat intestine (*a*) and a half-minute rhythm 1 h after blood loss (*b*).

Neuroendocrine hypothalamic structures were stimulated 1 h after blood loss with a computer-assisted MDM apparatus (30 min, 0.5 mA current), designed to produce direct and alternating currents at a 1:1 ratio and 4 pulse configurations in a frequency range of 0-0,000 Hz.

RESULTS

In control rats the background SA recorded for 1 h after 18-h food deprivation consisted of regular MMC including 3 phases: resting (I), irregular (II), and regular (rhythmic) activity (III) (Fig. 1, *a*). The MMC period was 585-625 sec, phase III lasted approximately 210 sec, activity time was 40% of the period. The minute rhythm with a period of 45±5 sec was observed during phase II, the SA intensity was 3.92±1.51 arb. units.

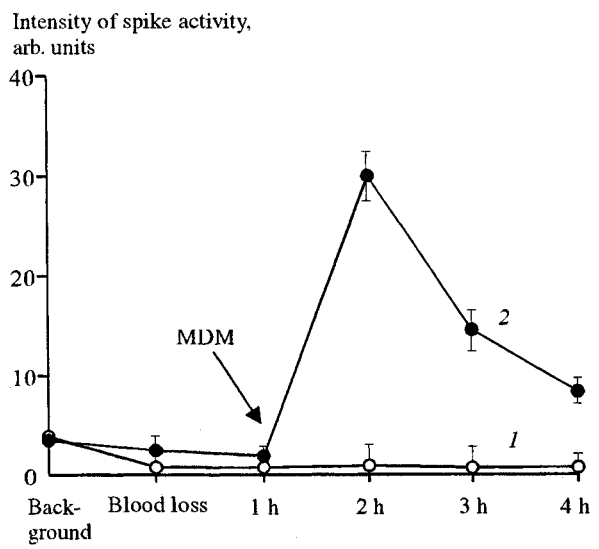

Fig. 2. Intensity of spike activity at different times after blood loss in the control (*1*) and after mesodiencephalic modulation (MDM, *2*).

The start of blood letting was accompanied by a short (5-6 min) period of rhythmic activity followed by a long rest period (about 15 min) when the SA intensity was lowered 5-fold (0.76±0.13 arb. units).

No MMC occurred within 1 h after blood loss when long periods of rest alternating with periods of irregular activity including a pathological half-minute rhythm with a period of 25±5 sec were recorded (Fig. 1, *b*). The intensity of SA remained very low.

The migrating complexes appeared during the second hour after blood loss, but their periods 2.5-fold surpassed the normal value. The duration of phase III did not significantly differ from normal (270±30 sec). The half-minute rhythm with a period of 25±5 sec was observed during phase II. The SA intensity remained far below the initial value (0.91±0.33 arb. units).

In 4 animals, MMC appeared 3 and 4 h after blood loss, but characterized by a 2-fold longer period in comparison with the initial MMC. Duration of phase III remained within the normal range (270±30 sec). No MMC occurred in the remaining 5 animals, SA of these animals consisted primarily of periods of irregular activity. The regular minute rhythm with a period of 45±5 sec could alternate with a pathological half-minute rhythm with a period of 25±5 sec. Spike activity was below normal even by the end of 4-h monitoring.

In the experimental group, the parameters of SA before, during, and 1h after the blood loss were the same as in the control group. A sharp (more than 5-fold) increase in SA intensity was observed after a 30-min MDM session with simultaneous increase in both the amplitude and duration of SA (Fig. 2). The MMC were absent, but isolated rhythmic phases of normal duration were recorded. Irregular SA without clear-cut periods of rest and a regular minute rhythm with a period of 45±5 sec were recorded throughout the observation period.

Although SA gradually decreased 3 and 4 h after blood loss, SA intensity remained above normal (8.39±2.64 arb. units) to the end of monitoring. The regular MMC with a period of 607±14 sec and normal duration of phase III was recorded in a half of animals. Four animals without MMC exhibited irregular SA of high intensity and a regular minute rhythm with a period of 45±5 sec.

These data indicate that the intensity of SA and intestinal contractile activity sharply decreased during acute ischemia. A single session of MDM considerably stimulates generation of action potentials so that SA intensity 4 h after blood loss exceeds the initial value. The presence of regular MMC indicates normalization of intestinal SA [4,5]. Ischemic episodes prolong or abolish MMC cycles [6], which was observed in our experiments. In addition, we recorded pathological half-minute rhythms soon after blood loss. MDM restored the regular minute rhythm and MMC, which indicate recovery of coordinated motor activity of the small intestine.

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