



## MORPHOLOGICAL CHANGES IN THE SMALL INTESTINE AFTER LOCAL ANESTHESIA IN EXPERIMENTAL MECHANICAL TRAUMA

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### ABSTRACT

*This literature review presents the pharmacokinetic properties of local anesthetics responsible for the effectiveness of regional anesthesia methods, and also examines in detail the gastrointestinal effects of local anesthetics, allowing a broader look at the possibilities of their use in intensive care.*

**Relevance.** Today, several modern types of analgesics and drugs have been developed. It is very useful for performing invasive procedures in surgery, dentistry and traumatology. The methods used in anesthesia and the effects of drugs on all organs have been studied by scientists for many years, but the morphological changes of anesthetics in the intestine have not been fully studied.

The effect of local anesthetics on the rate of gastrointestinal paresis resolution as a manifestation of systemic anti-inflammatory action. The pathophysiology of postoperative intestinal paresis is complex. Factors such as surgical stress response, sympathetic hyperactivity, intestinal manipulations, visceral pain stimulate nociceptive afferent and sympathetic efferent nerve fibers. The stress response is accompanied by increased release of endogenous opioids, which inhibit the propulsive activity of the gastrointestinal tract.

The use of opioid analgesics in the postoperative period also prevents the restoration of normal gastrointestinal function due to the effect on opiate receptors of the small intestine. Increased formation of inflammatory mediators during tissue damage sensitizes peripheral nociceptors. Persistent activation of C-fibers contributes to central sensitization with expansion of receptor fields. Morphine and other opioid analgesics suppress the release of acetylcholine from the mesenteric plexus, which increases the tone of intestinal smooth muscles and reduces the propulsive activity of the intestinal tract. Intravenous infusion of MA, in particular lidocaine, seems very promising in light of the recent trends of reducing the length of patients' stay in the clinic, as well as increasing their satisfaction with the quality of treatment in general.

The severity of postoperative paresis depends on: a) the intensity of the inflammatory process, b) increased sympathetic tone, c) the total dose of prescribed opioids. It is believed that the positive effect of MA on this process mainly depends on their systemic anti-inflammatory effect [8], although one cannot ignore the decrease in the doses of opioid analgesics noted in a number of studies [9, 10]. Intravenous administration of lidocaine can

also have a certain effect on sympathetic tone, although limited by its low concentration in plasma.

Harvey K. et al. (2019) believe that systemic administration of MA has both peripheral and central mechanisms of action [11]. At the peripheral level, MA limit the synthesis of inflammatory mediators, at the central level, they modulate neuronal responses at the level of the posterior horns of the spinal cord. MA have a direct stimulating effect on intestinal smooth muscles, which is possibly the result of blockade of inhibitory reflexes originating from the mesenteric plexus. In addition, spinal reflexes, as well as peripheral reflexes conducted through the prevertebral ganglia, have an inhibitory effect on gastrointestinal motility. Intravenous infusion of lidocaine can limit the duration of postoperative intestinal paresis by reducing postoperative irritation of the peritoneum, as well as inhibition of inhibitory gastrointestinal reflexes.

Some authors believe that amide MA, in addition to a powerful anti-inflammatory effect, are also capable of significantly reducing sympathetic activity, which, in particular, is manifested by a decrease in the concentration of catecholamines in the urine for 48 hours after surgery [12, 13]. Some studies have shown that intravenous prolonged administration of lidocaine allows not only to shorten the period of inpatient treatment, but also reduces the intensity of postoperative pain syndrome [9, 14]. The reduction in the length of stay in the hospital is mainly due to faster recovery of gastrointestinal functions.

Three independent studies [8, 9, 14] showed that patients (open and endoscopic colorectal surgery, as well as radical prostatectomy) who underwent intravenous lidocaine infusion were discharged from the clinic 24 hours earlier than the control group, which is associated with faster recovery of gas passage and independent stool. The end of the infusion time varied from 1 to 24 hours after the end of the surgery. In all cases, the plasma concentration of lidocaine did not reach toxic levels.

In the already mentioned study by Harvey K. et al. (2019), patients who underwent elective colon surgery received patient-controlled intravenous analgesia (PCA) with morphine in the postoperative period [11]. In addition, patients in the study group received an intravenous infusion of lidocaine 1 mg/min for 24 hours after surgery. Against the background of lidocaine infusion, a decrease in the average intensity of postoperative pain was noted, in particular, 24 hours after surgery it was  $26.1 \pm 8.2$  on a 100-point VAS, and in the comparison group -  $45.4 \pm 6.4$ . The appearance of the first peristaltic noises in the lidocaine group was noted after  $88.3 \pm 6.8$  hours, in patients receiving placebo - after  $116 \pm 10.1$  hours (28 hours later). It is interesting to note that against the background of the introduction of lidocaine, no decrease in the average doses of morphine was noted, compared with the placebo group, i.e., the earlier restoration of gastrointestinal motility in this study cannot be explained by the opioid-sparing effect.

Lidocaine infusion allowed patients to be discharged from the hospital on average one day earlier. Intestinal paresis is a serious enough problem in the acute phase of spinal cord injury. In the study by Baumann A. et al. (2019), it was shown that intravenous lidocaine infusion at a rate of 2–3 mg/min allowed to eliminate intestinal paresis within 10–20 hours in patients resistant to neostigmine therapy [15]. No signs of systemic toxicity of lidocaine were noted in any of the studies. It is known to be dose-dependent and usually manifests itself when the infusion rate is  $>3$  mg/min and the drug concentration in plasma reaches  $>5$   $\mu\text{g/ml}$  [16].

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