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CHANGES IN THE MORPHOLOGICAL PARAMETERS OF THE SMALL INTESTINE IN SEVERE TRAUMATIC BRAIN INJURIES

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Annotation.
The proven relationship between a critical condition and changes in intestinal morphology in neurosurgical patients is a justification for the need to continue research aimed at developing a strategy for their treatment, including the prevention of secondary damage to the brain and other body systems. The results of the analysis of literature data on changes in the morphological parameters of the small intestine in severe traumatic brain injuries are presented.
Key words: morphology, small intestine, traumatic brain injury, white rats.

Relevance. For many years, hypotheses have been put forward about the important role of the intestine in the development of systemic inflammation and multiple organ failure in critically ill patients, which over time have been confirmed using modern diagnostic and treatment methods in studies performed by specialists in various fields [1]. Every year, more than 5.7 million patients in the United States are hospitalized in intensive care units (ICUs) for critical illness, representing up to 10% of all adult admissions, accounting for a fifth of all hospital expenses, and the mortality rate among these patients is higher than from all other causes [2]. The prognosis of patients in whom a critical condition acquires the status of chronic is mainly unfavorable, with a one-year survival rate of about 25% [3]. One of the leading causes of the development of a critical condition is brain damage of various etiologies. According to the literature, traumatic brain injury is the main cause of disability and mortality among all types of injuries in people under 45 years of age. In the United States, up to 5.3 million people with the consequences of injuries live not only with physical, cognitive, emotional and behavioral disorders, but also with dysfunction of the gastrointestinal tract (GIT), which annually costs the national economy 60 billion dollars [4-6]. In patients in critical condition in the intensive care unit, the main vital signs are constantly monitored, while changes in the intestines are often not taken into account, since intensive care is usually aimed at maintaining the function of other organs and systems that are more accessible for monitoring, in while the intestine, at first glance, is switched off from the digestion process and does not create life-threatening situations [7, 8]. Nevertheless, during the stay in the ICU, 59.1% of patients have at least one symptom of a gastrointestinal disorder, which can serve as a predictor or initial manifestation of changes at deeper levels of regulation of its function and morphological structure [9]. In clinical practice, critically ill patients often develop bacteremia, sepsis, or multiple organ failure syndrome in the absence of an apparent source of infection; this indicates that bacteria and their metabolic products contained in the gastrointestinal tract are potentially able to migrate from its lumen to the lymph nodes, systemic circulation, and internal organs [1, 10, 11]. Underestimation of the state of the gastrointestinal tract in critically ill patients can lead to worse clinical outcomes and the development of secondary complications.

The centralization of blood supply that develops as a result of a critical state and persists for a long time leads to hypoperfusion of various organs, in particular, the gastrointestinal tract. In an experimental study on rats and mice, which reproduced traumatic injury to the brain and spinal cord as a model of a critical condition, typical changes in intestinal morphology caused by circulatory hypoxia against a background of reduced cardiac output were revealed. First of all, this is a narrowing of the crypts, an increase in the number of mast cells [12], the phenomena of prestasis and stasis in various structures and sections of the intestine, an increase in the depth of damage to the mucous membrane and thickening of the smooth muscle layer [13], diapedetic hemorrhages, erosions, ulcers, as well as the development of gastrointestinal bleeding. At the same time, morphological manifestations of microcirculation disorders are recorded unevenly throughout the entire intestine and are more pronounced in areas of the greatest accumulation of vessels [14]. It has been established that it is the mucous membrane of the small intestine that is most vulnerable to circulatory hypoxia due to the phenomena of arterio-venous shunting through the anastomoses of the submucosal layer, in which detachment and desquamation of the epithelium occur, early destruction of enterocyte membranes and impaired transcapillary metabolism, followed by necrosis of the intestinal mucosa [12 , 14]. In particular, the study of microcirculation disorders of the intestinal mucosa in studies on rodents with a model of mesenteric thrombosis demonstrated the absence of significant damage to the small intestine, but revealed severe circulatory disorders after reperfusion of its sections located opposite the mesenteric edge, which increased over time and manifested hemorrhagic changes later. 6 hours [15]. The mucous membrane of the duodenum is most vulnerable to hypoxia, while only minimal disturbances are noted in the ileum, which is associated with the activation of reserve zones of parietal digestion, characteristic of the digestive system of rats [16]. In addition, as a result of hypoxia, a decrease in the number of T- and B-lymphocytes in Peyer's patches is observed [17]. Circulatory hypoxia also affects the structure of the intestinal villi. Due to the peculiarities of their microcirculation, the centralization of blood circulation already in the early stages causes oxygen starvation in them and malnutrition, which normally is provided by 50–80% directly from the intestinal lumen [18]. Arterial and venous blood supply to the intestinal villi occurs in the opposite direction, which leads to “shunting” of blood in the main part of the villus, leaving its apical end in a state of hypoperfusion, which, in turn, leads to ischemia of the gastrointestinal mucosa, impaired parietal digestion and destruction of the villi [7, 19-21]. Hypoxia causes an increase in the production of pro-inflammatory cytokines and other inflammatory mediators (leukotrienes, prostaglandins, etc.), which, along with an increase in the tone of the sympathetic nervous system, lead to disruption of the integrity of tight junctions between enterocytes, a decrease in the motor-evacuation function of the gastrointestinal tract, and bacterial translocation [22–24] .

The proven relationship between a critical condition and changes in intestinal morphology in neurosurgical patients is a justification for the need to continue research aimed at developing a strategy for their treatment, including the prevention of secondary damage to the brain and other body systems.

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