# The Results of Gastric pH-metry in Patients with Extensive Burns

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AIM OF STUDY To study of the role of hydrochloric acid in the genesis of stress-induced gastric ulceration in patients with extensive burns.

MATERIAL AND METHODS The results of gastroscopy and endoscopic pH-metry of 30 young patients with extensive burns (19 male patients and 11 female patients). The acidity of the gastric contents was measured on day 1,7 and 14 after the trauma.

RESULTS It was found that the early period of burn disease was followed by the pH growth in the acid-producing area of the stomach to 3.8–4.2 (p=0.002–0.020). This indicated that extensive burns provoked a significant decrease of hydrochloric acid production by parietal cells of the stomach. The restoration of the acid production occurred only 14 days after thermal injury and was not followed by the development of a hyperacid state. The frequency of erosive-ulcerative lesions detection in the gastric mucosa by the end of the first day after receiving burns was 83%, 87% on day 7, and 96% on day 14. In total, stress-induced lesions of the gastrointestinal mucosa were diagnosed in 26 of 30 patients with a shock-induced thermal injury. Consequently, in patients with burn shock the protective factors of the gastric mucosa were depressed so that a minimum amount of hydrochloric acid was sufficient to damage it.

CONCLUSION Inhibition of acidogenic function is typical for patients with burn disease. Hydrochloric acid does not play a leading role in the genesis of stress-induced ulceration in patients with severe thermal trauma.

Keywords: gastroscopy, pH-metry, acute ulceration, gastrointestinal bleeding, burns

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#### BACKGROUND

Stress-induced damages to the mucous membrane of the upper digestive tract often leads to the development of life-threatening gastroduodenal bleeding. Critical conditions, such as severe mechanical or thermal injury, sepsis, an early period after extensive surgical interventions, etc., are one of the causes of acute lesions of the stomach and duodenum. [1–6].

The pathogenesis of damage to the gastroduodenal area in critical conditions is an imbalance between the factors of aggression and the factors of protection of the mucous membrane. The main mechanisms are activation of factors of aggression (increased production of hydrochloric acid) and the suppression of protection factors (impaired microcirculation in the mucous membrane). Thus, according to laboratory studies, severe thermal injury, which is one of the reasons for the development of a critical condition, leads to a significant growth of cortisol secretion, which level remains increased up to 14 days from the time of the burn [7, 8]. The released cortisol stimulates the production of histamine by argyrophil cells, which, in response to stimulation of H<sub>2</sub> receptors of gastric parietal cells, leads to an increase in the discharge of hydrochloric acid [7, 9–11]. Despite adequate anesthesia, critically ill patients are regularly exposed to stressful factors, such as multiple surgical interventions and prolonged painful dressings. Accordingly, all this time, the production of hydrochloric acid remains elevated, which is confirmed by the results of clinical studies [9, 12]. On the other hand, there is a point of view that since the stomach primarily suffers from hypoperfusion, the production of hydrochloric acid, on the contrary, should decrease on the background of a critical state [13, 14].

**The aim and objectives of the study.** The aim of our study was to assess the production of hydrochloric acid in victims with extensive burns. During the study, the state of the acid-producing function of the stomach at different

periods of burn disease and its impact on the incidence of erosive and ulcerative lesions of the mucous membrane of the upper digestive tract was assessed.

#### **MATERIAL AND METHODS**

The study included 30 patients aged from 18 to 60 years with thermal burns of the skin over an area of more than 20% of the body surface, hospitalized in the Burn Resuscitation Department in January-November, 2015. The exclusion criteria were chronic diseases of the gastrointestinal tract. Among the patients included into the study, there were 19 men (63.3%) and 11 women (36.7%), the average age of patients was 42.2±13.2 years. The total area of thermal damage to the skin was 35.8±15.1% of the body surface.

In all patients, the state of the acid-forming function of the stomach was evaluated 24±4 hours after the moment of injury (burn shock stage), as well as on day 7 (burn toxemia) and 14 (burn septicoxemia) using endoscopic pH-metry. A primary gastroenterological converter (G1-D-E) with a skin electrode and RJ-45 connector were used for pH-metry. Gastroenteromontor computer wearable Gastroskan-GEM, manufactured by Istok-Sistema, JSC (Russia) was used as a recording unit. The meal was given up 4 hours prior to the study, and enteral nutrition was replaced with a glucose-electrolyte mixture. Immediately before the examination, the stomach content was evacuated through a nasogastric tube. To obtain the true value of acidity before the first pH measurement, antisecretory preparations were not administered, they were abolished in subsequent measurements 36 hours before the study [16, 17]. In accordance with the manufacturer's recommendations, immediately prior to the study, technological check and calibration of the pH probe were performed [15].

During endoscopic examination of the stomach and duodenum, a visual assessment of macromorphological changes of the mucous membrane was performed. After the examination, the reference electrode was fixed on the intact area of the patient's skin, and the working part of the pH probe was inserted into the endoscope biopsy channel. After the contact of the measuring electrode with the mucous membrane of the stomach and stabilization of the indications on the recording unit, the obtained values were recorded. The study used seven standard points recommended for pH measurement (Fig. 1) [16, 17].

The obtained pH values were assessed according to functional areas: an acid-forming zone and an alkaline secretion area. The function of acid release was regarded as an anacid if pH in the gastric body and fundus was > 5.0, as hypoacid if pH was 2.0-5.0, normal if pH was 1.2-2.0 and hyperacid if pH was less than 1.2 [16, 18]. The alkalizing function was estimated as compensated at pH> 5 in the antrum, subcompensated at pH 2.0-4.9, and decompensated at pH less than 2.0 [19]. Data processing was performed by IBM SPSS 20.0 and Microsoft Office Exel 2007 using the Shapiro-Wilk and Friedman test.

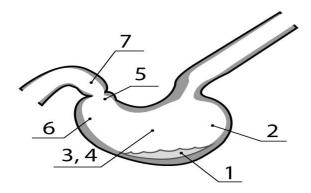


Fig. 1. The location of standard points for pH-metry (www.gastroscan.ru): 1- mucous lake, 2- gastric fundus, 3- gastric middle third of the body along the posterior wall, 4- gastric middle third of the body along the anterior wall, 5- small curvature of the antral section, 6- large curvature of the antral section, 7- duodenal anterior wall

### **RESULTS AND DISCUSSION**

When evaluating the data obtained, it was established that burn shock in the majority of victims is accompanied by an increase in pH in the acid-producing zone of the stomach from 1.2–2.0 to 3.8–4.2 (the Table). Thus, a pronounced decrease in the secretion of hydrochloric acid by the parietal cells of the gastric mucosa is a characteristic of the first period of burn disease. In the future, against the background of complex therapy for burn disease, the recovery of hydrochloric acid secretion gradually occurs, as evidenced by a decrease in pH to 2.4–3.2. The full recovery of the acid-producing function of the stomach (pH 1.5–2.0) in our study was observed on day 14 after the moment of thermal injury, that is, during the period of septicotoxemia. Thus, we couln't confirm the hyperacidity of the gastric juice in severely burned ones. On the contrary, severe burn injury in the majority of the victims was accompanied by hypoacid state followed by reduction towards the normal state. The pH value in the antrum of the stomach throughout the entire study did not have a tendency to change and remained within 2.8–4.8, which indicates subcompensation of the alkalizing function of the stomach in patients with burn disease.

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Measuring point		Research terms		
	Day 1	Day 7	Day 14	
Mucous lake	4.1±2.4	3.1±2.1	1.6±0.6	
Body of the stomach	3.8±2.3	2.4±1.5	1.5±0.7	
Gastric body, posterior wall	4.2±2.4	3.2±2.2	2.0±1.4	
Gastric body, anterior wall	4.0±2.6	3.1±2.2	1.6±0.7	

Erosive and ulcerative changes of the mucous membrane of the stomach and duodenum during the first study were found in 25 victims (83%) of 30. The frequency of stress-induced damage to the upper digestive tract on the day 7 from the moment of injury was 87% (24 injured out of 30). The third study of the pH level was performed only in 26 victims, which is due to a fatal outcome that occurred in 4 patients on day 7-14 after the moment of thermal injury. In this subgroup, signs of lesion of the gastric mucosa and duodenum were found in 96% of victims (25 of 26). Thus, 86 gastroduodenoscopic studies with pH-metry were performed in 30 patients. In 74 cases of them (86%), during a visual examination of the upper GI tract, acute damage to the mucous membrane was detected.

The next stage of the study was the comparison of the acidity level of the gastric juice with the morphological state of the gastric mucosa. In victims of the hypo-and anacid state, the frequency of erosive and ulcerative changes was 24.1%, and in those with hyperacidity, the rate was 100%. An unexpected fact was the frequency of detection of acute ulcers and erosions in 72.4% of injured people in the normal state of acidogenic function. This indirectly indicates that under conditions of hypoperfusion, the protective factors of the mucous membrane are suppressed so that a minimal amount of hydrochloric acid is sufficient to damage it [17].

In the course of statistical analysis using the Shapiro-Wilk test, it was found that the data obtained are characterized by a non-normal distribution. In this regard, the Friedman test was used for further analysis. The significance level of "p" was below the critical value of 0.05 for the following "pH-metric" points: "mucous lake" (Fig. 2), "gastric fundus" (Fig. 3) and the middle third of "the gastric body along the anterior wall" (Fig. 4). Thus, burn shock is accompanied by a decrease in acidity in the acid-producing area of the stomach, followed by recovery to the initial level by day 14 from the moment of injury.

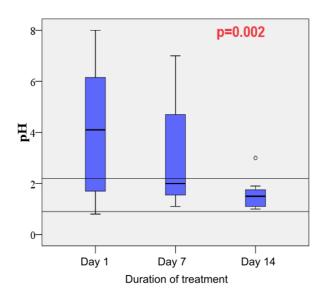


Fig. 2. The dynamics of pH changes in the mucous lake

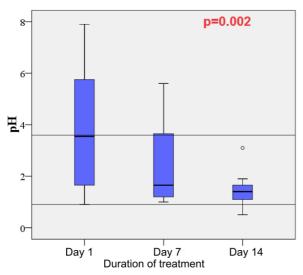


Fig. 3. The dynamics of pH changes in the gastric fundus

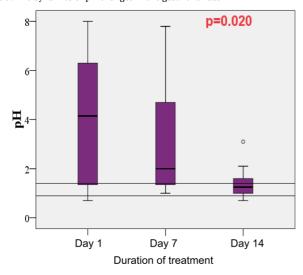


Fig. 4. The dynamics of pH change in the gastric body and anterior wall

### CONCLUSION

The acid-producing function of the stomach is significantly reduced in the majority of patients with extensive burns. Subsequent recovery of the secretion of hydrochloric acid leads to restoration of normal acidity, but not to formation of hyperacidity of gastric juice. However, the frequency of stress-induced damage to the gastroduodenal zone, despite the prevailing hypoacid state, reaches 86%.

Inhibition of the acid-producing function of the stomach is a characteristic of the early period of burn disease. Hydrochloric acid does not play a leading role in the genesis of stress-induced lesions of the stomach and duodenum in patients with severe thermal injury.

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