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THE ROLE OF BRONCHOSCOPY IN THE DIAGNOSIS AND TREATMENT OF INHALATION INJURY.

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Abstract: The history of studying the problem of inhalation injury has several decades. I.I. Dzhanelidze wrote about changes in the lungs of victims of a fire. Janelidze back in 1941. Already in the 60s, it became clear to researchers that damage to the respiratory tract is one of the main causes of death in burn victims. The most severe lesions of the respiratory tract develop under the action of chemical compounds inhaled along with smoke. It is undeniable that the prognosis for life with a combination of skin burns with inhalation injury is unfavorable. According to various authors, mortality in combined thermal injury ranges from 45 to 78%. These data indicate that, despite the advances in the treatment of burn patients achieved in recent decades, which have significantly reduced mortality in extensive skin burns associated with hypovolemic disorders during the period of burn shock, with infectious complications, as well as wound exhaustion, inhalation lesions airways still remain a serious problem in combustiology. The existing therapeutic and tactical schemes for managing the victims need further development, taking into account the severity of the respiratory tract injury and prognostic assessment of the outcome of combined trauma.

Keywords: inhalation injury, toxic combustion products, hypoxia, fiberoptic bronchoscopy, oxygenation

Introduction.

Inhalation injury syndrome from a modern point of view is explained by the generalization of thermal and chemical damage to the respiratory tract and lung tissue, which is often accompanied by systemic intoxication [1].

Inhalation injury The history of studying the problems includes several decades. In 1941, I.I. Dzhanelidze wrote about the changes in the lungs of fire victims [2]. By the 1960, researche found that respiratory tract injuries are one of the main causes of death in burned people [3].

But [4] in his data states that about 80% of deaths are caused by hypoxia developed as a result of poisoning of the respiratory organs with fire products or toxic gases.

For a long time, there were opinions that damage to the upper respiratory tract is local, because the vocal cords reflexively close under the influence of hot air [5].

However, after the advent of fibrobronchoscopy, there were reports that fire products play a leading role in damage to the tracheobronchial tree in more severe and widespread damage to the respiratory system [6].

Later, a number of studies noted the combined effect of harmful factors (hot air, smoke and its components, toxic, chemical compounds, steam, barotrauma) [7].

GMHead and co-dependent According to (1980), during a fire, the temperature of inhaled air can reach 300-400 degrees. Air is cooled to 40 degrees in "natural air conditioners" in the upper respiratory tract. Long-term exposure to flame, inhalation of water vapor, gas explosions, the temperature of the breathing air reaches 2000 degrees. At this time, thermal damage spreads to the respiratory tract and tracheobronchial tree. The frequency of such injuries is about 20% [e most serious damage to the respiratory

tract develops under the influence of chemical compounds that enter with smoke during breathing. It is known that the composition of smoke is not the same, it consists of solid particles of the body, liquid resins, poisonous gases. Modern construction modules and equipment consist of various polymeric synthetic materials, the combustion of which produces complex smoke gases (Table 1).

GAS	Materials _	Effect
СО	All organic matter	T read hypoxia, coma
CO2 _	All organic matter	Acidosis, narcosis
NO2	Popcorn, wood, celluloid	Bronchospasm, pulmonary edema
		, sopor
COCl, HCl	Cable insulation	Tracheobronchitis, bronchiolitis
HCN	wool, silk, Polyurethane	T read hypoxia, coma
Benzenes _	They are petro - chemical plastics	Bronchospasm, pneumonia or
		coma
Aldehydes _	cotton, sawdust	Tracheobronchitis, bronchiolitis,
		pneumonia

 Table 1.

 Description of fire toxic products (R. Voeltz, 1995)

Develops as a cellular reaction to the effects of chemical products of the flame reaching the surface of the alveoli. An increase in lymph flow, an increase in vascular permeability under the influence of free cytokines, accumulation of extravascular fluid, as well as alveolar collapse lead to a sharp decrease in the surfactant function, which then disrupts the ventilation - perfusion ratio. All this can lead to the development of terrible complications such as acute lung injury syndrome [9].

due to impaired clearance of bronchial epithelial cells, cough reflex and immune component cell activity decline. Function decreases, which leads to the addition of secondary infection and the development of purulent complications in the respiratory organs and generalization of the infection.

In general, in response to the multifactorial effects of smoke, the drainage function is disturbed with local destructive changes such as damage to the ciliated epithelium of the bronchi and the active inflammation of monocytes, macrophages, and polymorphonuclear neutrophils. It develops with systemic inflammatory reactions caused by cytokines and neuropeptides released from the cells [10]. Also, other authors emphasize the leading role of polymorphonuclear leukocytes in the pathogenesis of complications of inhalation injuries [11,12].

In inhalation injury the pathogenesis of respiratory failure varies depending on the nature of the respiratory tract damage, the time interval and the severity of the adjacent thermal injury. The main cause of shortness of breath in the first hours is swelling of the larynx. Such complications are mainly characteristic of thermoinhalation damage of the respiratory tract [13]. Acute lung injury syndrome also causes respiratory failure in the early stages [14,15].

SO, NSN, SO 2 cause systemic intoxication of the body. V.S. According to A. Ilichkina (1993), the affinity of CO for hemoglobin is 200-300 times higher than that of oxygen.

Inhalation with SO 0.2-1% concentration mixture for 3-6 minutes leads to death. CO causes tissue hypoxia by blocking oxygen transport. A coma develops at a concentration of NbCO 50%. NSN is also very toxic [16].

Thus, the cause of hypoxia in the first hours after the injury (hypoxic, tissue, circulatory) is carbon monoxide, cyanide poisoning, burn shock, respiratory failure against the background of laryngeal swelling, airway obstruction caused by fire products, damage to fibrin and bronchial epithelium, acute lung injury is a syndrome. Later, the cause of hypoxia is purulent complications of the respiratory system, respiratory failure developed against the background of sepsis.

There is no doubt that inhalation injury with skin burns the combination is lifethreatening for the patient. According to various authors, the mortality rate in combined thermal injury is 45 to 78% [17].

According to P. Vaeltz (1995), the average index of severity of thermal injury is 65 conditional size (sh.o'.) - the mortality rate is 15%, when it is accompanied by inhalation injury, it is 53% [18]. Pulmonary complications (pneumonia, acute lung injury syndrome) served as the cause of death in 77% [19].

Thus, the presented data show that although the progress in the treatment of burns in recent decades has been achieved to significantly reduce mortality rates in large-scale skin burns associated with hypovolemic changes during burn shock, infectious complications, in addition to wound complications and inhalation injuries of the respiratory tract, there is still they remain serious problems in the field of combustiology.

The result of inhalation injury depends on timely diagnosis of respiratory tract injury, assessment of its severity and selection of adequate therapeutic tactics.

Inhalation injury can be suspected after a carefully collected anamnesis, that is, it is important to determine the situation of injury, the duration of stay in a smoky room, the mechanism of fire (explosion, flash), the composition of the burned material, the level of consciousness at the time of injury (alcohol intoxication, sleep, loss of consciousness). is considered

The following complaints are taken into account: voice change (dysphonia, aphonia), cough with sputum, wheezing, suffocation.

During the examination, the state of consciousness and the degree of its impairment are also determined, because the clinic of unconsciousness and respiratory failure indicates severe damage to the airways.

According to Karvayal (1990), conjunctival hyperemia, pus in the nasal passages, oropharynx, and sputum are characteristic of smoke inhalation. Hair loss in the nasal passages, dysphonia, inspiratory wheezing, and stridor indicate damage by high temperature or a combination of temperature and fire products [20].

P. Voeatz (1995), has a polymorphic character (slowing of breathing in the lung areas, scattered dry wheezes, wet wheezes). The absence of any changes in auscultation in the first days does not indicate that the airways are not damaged [21]. M.J. Masanes et al. (1994) confirmed this fact in their data, who noted changes in the auscultatory picture in only 10% of patients with inhalation injury [22].

According to laboratory studies, changes in gas content in arterial and venous blood give the most information. However, the identified changes characterize both the damage of the respiratory tract and the severity of the burn injury [23]. Against the background of normal or moderate tension of CO2 with compensated acid-base changes in the blood, a decrease in RO2 and oxygenation index in arterial blood was noted in patients with more severe inhalation injury and was determined in 50% of cases [24].

An increase in blood amylase levels in venous blood biochemical tests in patients with inhalation injuries was noted in 40% of cases [25], which is probably associated

with damage to the salivary glands in burn patients.

M. Ya. Malakhova et. al. (1999) developed biochemical tests to detect lung injury in burn patients. The tests are based on measuring the difference in the concentration of low and medium molecular weight masses and total protein in arterial and venous blood [26].

Lung scintigraphy performed with radioisotope xenon 133 revealed respiratory damage at the alveolar level. The results are evaluated by the uneven absorption of the radioisotope by the lung tissue, which indicates a violation of the ventilation-perfusion ratio [27].

Sh.I. Kurbanov et. al. (1995), S. K. Boenko et al. (1995) studied extrinsic respiratory function in burn patients with airway injury [28]. They determined the change of external respiration depending on the severity of respiratory tract damage: increase of NM H (by 40% of the correct values), decrease of OTS (by 40%), decrease of oxygen utilization coefficient, decrease of expiratory force and forced respiratory volume for 1 second. These changes are characteristic of the obstructive type of external breathing.

Thus, the information presented in the literature shows that the cited instrumental none of the research methods (except for lung scintigraphy) allows to determine diagnostic criteria specific to respiratory tract damage.

Many authors [29] believe that fibrobronchoscopy is the most informative method of diagnosing inhalation injuries in burn patients.

Sh.I. Kurbanov (1995) found in their work that it depends on the type of endobronchitis, the degree of inhalation damage determined by fibrobronchoscopy, and clinical data and the degree of severe respiratory failure determined by the results of external respiratory function studies. As a result of the study, the authors found that the localization and nature of the injury in inhalation injuries is one of the main factors in the pathogenesis of lung complications, which are important factors for the treatment and prognosis of this category of patients.

Endoscopic examination is considered to be the most objective method of assessing damage to the mucous membrane of the tracheobronchial tree. However, the discrepancy observed in some cases between the macroscopic data and the clinical view led to the search for morphological criteria confirming the visual data [30].

Morphological analysis of biopsies of the mucous layer of the tracheobronchial tree in patients showed various degrees of destructive changes of the bronchial epithelium depending on the severity of the damage.

In mild injury, epithelial cells are limited by degenerative changes. In severe cases, the epithelium is completely separated from the optically intact basement membrane. In very severe cases, deep disruptions of the hyalinized basement membrane, cellular inflammatory infiltration of submucous connective tissue are detected [31].

Cytological description of brush-bioptates of tracheobronchial tree mucosa gives enough information about the severity of damage . The method of sampling the material with a brush biopsy does not require additional examination, has no contraindications, and is considered to be a less traumatic procedure than taking a biopsy with a clamp. The method of chromobronchoscopy, which was first used in burn patients, was based on the ability of the dye to penetrate into the cytoplasm of damaged cells. The intensity and area of the mucosal staining areas were visually assessed, and the depth and boundaries of the mucosal lesions were indicated [32].

I.F. Shpakov (1997) describes a different cytological picture depending on the severity of inhalation injury and the time elapsed since the injury. According to his opinion, depending on the severity of the injury, the number of undamaged ciliated cells in the field of vision decreases, the number of basal cells increases, and the number of ciliated cells remains unclear [33].

Also, without studying the cellular composition of the bronchoalveolar lavage, only the cytological examination of the bronchial epithelium, smear brush biopts, does not fully explain the clinical picture of respiratory system damage.

The cytological method of studying the cell composition in the bronchoalveolar fluid is widely used to diagnose chronic inflammatory diseases of the lungs and disseminated processes in the lungs [34].

J. Barth (1990) studied the cellular composition of the lavage in burn patients with respiratory tract injuries [35]. Examination of the cytograms of victims of acute lung injury syndrome and those who died showed a critical accumulation of neutrophilic granulocytes. It is worth noting that lymphocytes were not shown in the cytograms because of their very small number and, accordingly, their significance was not taken into account. Although at the end of the last century, Polish researchers J. Winarski, E. Korozynski (1896) studied pleural fluid and concluded about the prognostic value of the number of lymphocytes in biological fluids [36]. A decrease in the number of lymphocytes in the exudates indicated a tendency to abscess or a tumorous nature of the fluid.

Thus, data from this literature show that, despite certain achievements in the development of technologies for the diagnosis of respiratory tract injuries in burned patients, some issues related to their objectivity, in particular, parametric evaluation of endoscopic signs of respiratory trauma, determination of the prognostic role of cytological examination data in the development of lung complications, etc. cases will remain the subject of further research.

Attempts to systematize the clinical, endoscopic, morphological and cytological signs of respiratory tract damage detected during the examination of burned patients led to the creation of many classifications of inhalation injuries.

A.N. The first endoscopic classifications proposed by Orlov (1964) included three levels of severity:

* mild degree: moderate swelling and inconspicuous hyperemia of the mucous membrane of the trachea, the bifurcation area and the main bronchi, the dream is acute, mobile, fibrinous coatings are few or absent;

* moderate level: obvious hyperemia and edema with large fibrinous coverings ;

* hard level: obvious swelling , hyperemia, many fibrinous coverings .

The classification of Touama (1972) suggests that the damage of the respiratory tract is divided into three levels of severity depending on the functional disorders in the respiratory organs :

I degree - without respiratory disorders;

II degree - respiratory disorder after 6-12 hours;

III - degree of injury and breathing disorders in q.

I.F. Inhalation in patients prescribed by Shpakov (1997) proposed to classify the fracture as follows:

I. Burns of the upper respiratory tract (from the external nasal passages to the vocal cords), hyperemia, edema of the mucus layer, hemorrhages in the submucosa, and desquamated epithelial areas in the form of erosion are manifested.

II . Damage to the respiratory tract by fire products:

A. Mild degree (individual accumulation of pus up to 1 cm, moderate hyperemia and swelling of the mucous membrane; a small amount of discharge ; during chromobronchoscopy, the damaged mucous membrane does not stain; the number of undamaged cells in a brush biopsy : 60-70 cyprus , 10-15 goblet , 5-7 in the basal field of vision; bacterial contamination of bronchial smear - 10^{3} in 1 ml cells and 2 types of bacteria .

Moderate (obvious swelling and hyperemia of the mucous layer; accumulation of pus up to 2 cm²; large amount of discharge; light blue and isolated reddish blue staining of the mucous layer on x -ray bronchoscopy; the number of unbroken cells in smears : 40-50 kip, 20-25 cups of cement , 10-20 in the basal field of view , bronchial smear , microbial contamination 10⁵ cells/ml and 3-4 types of bacteria grow).

B. Severe degree (total solid layer, after aspiration, a fluid "dry" mucous membrane with several petechial hemorrhages is visible; in chromobronchoscopy - several areas of intense blue color; the number of intact cells in smears: 20-25 kip r - shaped, 1 0-1 5 goblet - shaped, 25-3 0 in the basal field of vision; complete accumulation of nuclear detritus; microbial contamination of bronchial smear 10⁷ cells/ml and 5 types of bacteria vegetate) [37].

III . A combination of burns of the respiratory tract and damage from fire products (all the above signs correspond to the severity of the damage).

The large scale of this classification, as well as the lack of gradation of descriptive signs according to leading principles (etiology, localization, etc.), cause some inconvenience in working with this classification.

the classifications provided by V.P. Tsurikov's classification fully reflects not only the factors influencing the damage of the respiratory tract, but also takes into account the degree of dysfunction of the damaged organ. However, the lack of prognostic assessment of the severity of these symptoms does not fully satisfy the clinicians and causes attempts to further improve and improve the existing classifications [38].

In the last quarter of a century, much attention has been paid to the problem of forecasting in medicine. The development of prognostic methods was initially carried out in relation to severe mechanical damage. Currently, there are more than 50 classifications and scales for assessing the severity of mechanical damage. However, most of them were not widely used for many reasons (large scale, impossibility of obtaining necessary data, etc.). They can be divided into three types depending on the tasks set when creating a specific classification.

The first type of classification is aimed at sorting out the victims at the scene, the second is a retrospective assessment of the severity of the injury for further analysis and planning, and the third is a prognostic assessment of the results of intensive care treatment. In addition to the intended use, one or another scale should be based on anatomical information about the injury , changes in physiological parameters, or a combination thereof. According to the general opinion, the requirements for prognostic methods are ease of use, availability of data, use of prognostic equations as an argument , agreement of predicted results with actual results in a sufficient percentage (at least 75 %) , and the possibility of use in both isolated and multiple lesions.

Burn injury depends on the area and depth of skin damage, N. Frank (1960) proposed the use of a prognostic indicator of burn injury severity based on the evaluation of these two parameters based on the anatomical principle. Frank the index is expressed in conditional units, the percentage of each burning surface is equal to 1 unit, and the depth is equal to 3 units [39]. Frank according to the index, together with clinical and laboratory data, the development of burn shock, its severity and the time of recovery

from this condition are predicted.

When skin burns are accompanied by respiratory injuries, the severity of burn shock increases, the prognosis of the victim's life worsens.

Frank the prediction of the outcome of a combined burn wound according to the index is carried out as follows. In particular, the Frank index is less than 30 - the prognosis is positive, 31-60 - relatively positive, 61-90 - doubtful, more than 90 - negative.

As mentioned above, the search for efficient prognostic algorithms is not ideal. The interest of many researchers in this problem is related to the possibility of optimizing treatment tactics depending on the results of prognostic assessment of the course and results of pathological processes associated with injury.

First of all, they are related to issues such as volume and content of infusion therapy, indicators of artificial lung ventilation, prevention of possible complications.

The composition of solutions in infusion therapy should be selected taking into account water-electrolyte disturbances, shifts in acid-base status, and increased energy needs. In order to improve the rheological properties of blood and microcirculation, it is recommended to use colloidal solutions in the amount of 5-7 ml/kg per day. The use of fresh frozen plasma, human albumin is appropriate, but at least 8 hours after the time of injury [40].

Timely initiation and adequate implementation of adequate respiratory therapy in patients with multifactorial failure has a positive effect on the course and outcome of heartburn disease (Klimov A.G., 1998). Indications for tracheal intubation and various types of respiratory support (assisted, high-frequency, controlled USV) in critically ill patients:

* symptoms of respiratory failure,

* lack of consciousness,

* severe thermal injury of the upper respiratory tract and damage to the entire respiratory tract from fire products.

When analyzing the literature, it is worth noting that high-frequency pulmonary ventilation (HFV) is widely used in patients with inhalation injuries.

Intubation method (transoral, nasotracheal or installed tracheostoma through) importance occupation because it does n't purulent of complications frequency, T. Lund et al. (1985) intubation duration with depend. That's it with together, some the authors lungs artificial ventilation long time during when applied too tracheostoma to put recommendation not enough for that reason as of the tracheostomy infection, necrotic tracheobronchitis, pneumonia, bed wound, trachea stricture like serious complications development probability high to be is cited [41].

Breath gets of the ways heavy damage conductivity recovery and his mucus from the floor fire toxic products elimination for tracheobronchial the tree Sanitation to do need (Bingham UG et al., 1987).

Pallua N et al. (1997) distress syndrome with complicated inhalation in damaged ones exogenous surfactant successful used confession enough [42].

Yu. L. Shevchenko and co-authors in 1999 reperfusion syndrome in the background come out of the lungs sharp damage syndrome in treatment exogenous from surfactant (Surfactant BL) long term inhalation successful use about information print they did

It should be emphasized that adequate intensive therapy during burn shock reduces the risk of generalization of the infection.

The issue of using rational antibiotic therapy is also important. Many authors recommend the appointment of antibacterial drugs only after bacteriological examination and after determining the sensitivity of the microflora in the culture.

References:

1.Miller A.C., Fernada P.A., Kadri S.S. et.al. High- Frequency Ventilation Modalities as Savage Therapy for Smoke Inhalation -Associated Acute Lung Injury: A Systematic Review. J. Intensive Care Med. 2018 June; 33(6): 335-345.

2.Alekseev A.A., Tyurnikov Yu.I. Analysis of the work of burn hospitals in the Russian Federation for 2016. // All-Russian public organization "Association of combustiologists" World without burns ", Moscow, Russia, 2017. - p. 5-8.

3.Bagnenko S.F., Krylov K.M., Shlyk I.V. Burn Center of the I.I. Dzhanelidze - 65 years // Mat. conf. "Modern aspects of the treatment of thermal injury". St. Petersburg, 2011. - p. 16-

4. Traber D.L., Linares H.A., Hendon D.N., Prien T. The pathology of inhalamation injury-a review // Journal Buns, vol.14, Issue 5, October 1988, p. 357-364.

5.Boenko S.K., Bogachev E.P. Therapeutic fibronscopy in the treatment of patients with burns of the respiratory tract // Journal Vest. Otorinolaringol 1988, Jun; (3): 50-3.

6.Boyer, N. Practical management of burns and inhalation injury // Pulmonology in Combut Medicine. - 2016. - Vol. 5, № 2. - P. 63-69.

7.Demenko V.V., Cheplyaev A.A., Shabanov T.V. "Problems of medical evacuation of patients with burn injury" // Mat. 18th All-Russian Congress dedicated to the 120th anniversary of emergency medical care in Russia. St. Petersburg, May 30-31, 2019 - p. 49.

8.Zhilinsky E.V., Chasnoit A.Ch., Alekseev S.A., Doroshenko G.V. Analysis of mortality, main prognostic factors and complications among patients with burn injury // Medical News, 2014, 11 (242) .

9.Mustafakulov I.B., Khakimov E.A., Karabaev H.K. etc. "Thermoinhalation trauma diagnosis and treatment" // Clinical guide// Samarkand 2018. 146 P.

10.Fayazov A.D., Kamilov U.R., Shukurov S.I., Abdullaev U.Kh. To the problem of treatment of burnt patients with combined and concomitant lesions // Sat. scientific works of the IV Congress of Combustiologists of Russia. Moscow, October 13-16, 2013 - p. 37-38.

11.Khadzhibaev A.M., Shukurov B.I. Emergency medical service in the Republic of Uzbekistan: achievements and ways of development // Mat. IV Congress of the Association of Emergency Doctors of Uzbekistan. Tashkent, 2018. S. 3-5.

12.Khakimov E.A.Karabaev H.K., Mustafakulov I.B.et al."Multiple organ failure in burn disease: problems of diagnosis, prevention and treatment. //Clinical guidelines/ / Samarkand 2018. 234 p.

13.Khakimov E.A., Karabaev H.K., Shakirov B.M. etc. Burn disease in children. / /Monograph// Tashkent 2021 331 p.

14.Alhazzani W., Alezeni F., Jaeschke R. et al. Proton pump inhibitors versus histamine 2 receptors antagonists for stress ulcer prophylaxis in critically ill patients: a systematicreview and meta-analysis // Crit. Care. Med. - 2013. - Vol.41. - №3. - P.693-705.

15.Avendano-Reyes J.M., Jaramillo-Ramirez H. Prophylaxis for stress ulcer bleeding in the intensive care unit // Rev. Gastroenterol Mex. - 2014. - Vol.79. - №1. - P.50-55.

16.Barkun A.N., Bardou M., Pham C.Q., Martel M. Proton pump inhibitors vs. histamine 2 receptor antagonists for stress-related mucosal bleeding prophylaxis in critically ill patients: a meta-analysis // Am. J. Gastroenterol. - 2012. - Vol.107. - $N_{0}4$. - P.507-520.

17.Compare D., Pica L., Rocco A. et al. Effects of long-term PPI treatment on producing bowel symptoms and SIBO // Eur. J. Clin. Invest. - 2011. - Vol.41. - N_{24} . - P.380-386.

18.Crooks C.J., West J., Card T.R. Comorbidities affect risk of nonvariceal upper gastrointestinal bleeding // Gastroenterology. 2013.-Vol.144. - №7. - P.1384-1393.

19.Fohl A.L., Regal R.E. Proton pump inhibitor-associated pneumonia: Not a breath of fresh air after all? // World J. Gastrointest. Pharmacol. Ther. - 2011. - Vol.2. - N2. - P.17-26.

20.Hayek S., Ibrahim A., Abu Sittah G., Atiyeh B. Burn resuscitation: is it straightforward or a challenge?// Ann. Burns Fire Disasters. - 2011. - Vol. 24(1). - P. 17-21.

21.Heidelbaugh J.J., Kim A.H., Chang R., Walker P.C. Overutilization of protonpump inhibitors: what the clinician needs to know // Therap. Adv. Gastroenterol. - 2012. - Vol.5. - $N_{2}4$. - P.219-232.

22.Iida H., Kato S., Sekino Y. et al. Early effects of oral administration of omeprazole and roxatidine on gastric pH // J. Zhejiang. Univ. Sci. B. - 2012. - Vol.13. - N_{21} . - P.29-34.

23.Johnson D.A., Oldfield E.C. Reported side effects and complications of long-term proton pump inhibitor use: dissecting the evidence // Clin. Gastroenterol. Hepatol. - 2013. - Vol.11. - $N_{2}5.$ - P.458-464.

24.Kim Y.J., Koh D.H., Park S.W. et al. Upper gastrointestinal bleeding in severely burned patients: a case-control study to assess risk factors, causes and outcome // Hepatogastroenterology. - 2014. - Vol.61. - N 136. - P.2256-2259.

25.Kirkpatrick A.W., Roberts D.J., De Waele J. et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome//Intensive Care Med. - 2013. - Vol. 39(7). - P. 1190-206.

26.Lanas A., Carrera-Lasfuentes P., Arguedas Y. et al. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents or anticoagulants // Clin.Gastroenterol.Hepatol.- 2015.- Vol.13.- $N_{0}5.P.906-912$.

27.MacLaren R., Reynolds P.M., Allen R.R. Histamine-2 receptor antagonists vs proton pump inhibitors on gastrointestinal tract hemorrhage and infectious complications in the intensive care unit // JAMA Intern. Med. - 2014. - Vol.174. - N_{24} . - P.564-574.

28.Malbrain M.L., De Keulenaer B.L., Oda J. et al. Intraabdominal hypertension and abdominal compartment syndrome in burns, obesity, pregnancy, and general medicine // Anaesthesiol. IntensiveTher. - 2015. - Vol. 47(3). - P. 228-240.

29.Mbiine R., Alenyo R., Kobusingye O. et al. Intra-abdominal hypertension in severe burns: prevalence, incidence and mortality in a sub-Saharan African hospital//Int. J. Burns Trauma. - 2017. - Vol. 7(6). - P. 80-87.

30.McBeth P.B., Sass K., Nickerson D., Ball C.G., Kirkpatrick AW. A necessary evil Intra-abdominal hypertension complicating burn patient resuscitation // J. Trauma Manag. Outcomes. - 2014. - Vol. 8. - 12.

31.Pilkington K.B., Wagstaff M.J., Greenwood J.E. Prevention of gastrointestinal bleeding due to stress ulceration: a review of current literature // Anaesth. Intensive Care. - 2012. - Vol.40. - №2. - P.253-259.

32.Preslaski C.R., Mueller S.W., Kiser T.H. et al. A survey of prescriber perceptions about the prevention of stress-related mucosal bleeding in the intensive care unit // J. Clin. Pharm. Ther. -2014. - Vol.39. - $N_{0}6$. - P.658-662.

33.Ramirez JI, Sen S., Palmieri T.L., Greenhalgh D.G. Timing of Laparotomy and Closure in Burn Patients with Abdominal Compartment Syndrome: Effects on Survival // J. Am. Coll. Surg. - 2018. - Vol. 226(6). - P. 1175-1180.

34. Robert L. Sheridan. Management of Burns. Surgical Clinics of Nourth America

2014 94(4): 721-944.

35.Ruiz-Castilla M., Barret J.P., Sanz D., Aguilera J., Ser- racanta J., Garcia V., Collado J.M. Analysis of intra-abdominal hypertension in severe burned patients: the Valld'Hebron experience //Burns. - 2014. - Vol. 40(4). - P. 719-724.

36.Strang S.G., Van Lieshout E.M., Breederveld R.S., Van Waes O.J. A systematic review on intra-abdominal pressure in severely burned patients//Burns. - 2014. - Vol. 40(1). - P. 9-16.

37.Sun K., Hancock B.J., Logsetty S. Ischemic bowel as a late sequela of abdominal compartment syndrome secondary to severe burn injury// PlastSurg (Oakv). - 2015. - Vol. 23(4). - P. 218-220.