



INFLUENCE OF HERNIA IMPINGEMENT ON THE DEVELOPMENT OF ACUTE ENTERAL INSUFFICIENCY SYNDROME AND INCREASED INTRA-ABDOMINAL PRESSURE

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Abstract A review of the literature has shown that enteral insufficiency syndrome and intra-abdominal pressure are two common pathological conditions that cause a vicious circle of mutually exacerbating changes, regardless of the aetiology of intra-abdominal hypertension. This also showed the basic steps that should be taken to prevent increased intra-abdominal pressure.

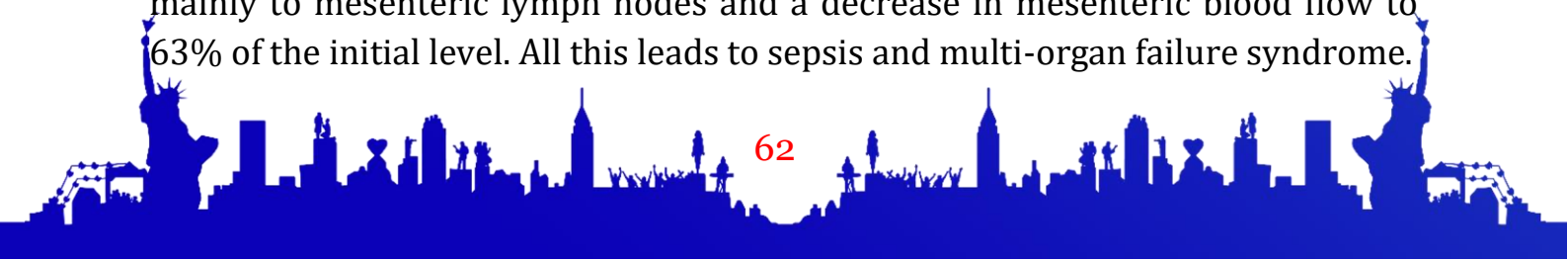
Key words. Usthmic hernia, intra-abdominal hypertension, methods of prevention

Introduction. Surgical treatment of patients with pinched postoperative ventral hernias of large and giant sizes represents an additional problem. One-stage repositioning of the hernial contents into the abdominal cavity is very dangerous because it can cause permanent intestinal paresis, increased intra-abdominal pressure and subsequent cardiac and respiratory problems. Krön I.L. suggested the name of such multi-organ failure developing as a result of UBH as 'abdominal split syndrome'.

Burch et al. first proposed a classification of the level of intra-abdominal hypertension. In 2006, the World Organisation for Abdominal Compartment Syndrome (WSACS) proposed new classifications of IAP: Grade I - 12-15 mmHg, Grade II - 16-20 mmHg, Grade III - 21-25 mmHg and Grade IV - greater than 25 mmHg. ACS is defined as IAP elevation of more than

The nature of abdominal organ disorders and mortality rate directly correlate with the level of intra-abdominal hypertension. Increase in intra-abdominal pressure above 10 mm. Hg. for 1-2 days leads to lethality in 3-7% of cases, and with an increase of more than 35 mm. Hg. for 6-7 hours leads to 100% of death.

In experimental work Diebel L.N. found that IAP 25 mmHg for 60 minutes leads to a violation of the intestinal barrier function, bacterial translocation mainly to mesenteric lymph nodes and a decrease in mesenteric blood flow to 63% of the initial level. All this leads to sepsis and multi-organ failure syndrome.





Kingsnorth A.N. believed that when modelling and performing abdominal wall plasty there should be a marginal increase in IAP to 15-20 mmHg, because disorders of cardiovascular and respiratory systems can be easily compensated. It is proposed to solve the problem of performing 'non-tension' hernioplasty in case of IAP preservation at the level of II degree (16-20 mm Hg).

In an animal experiment conducted by Ahman U., it was found that an increase in intra-intestinal pressure in intestinal obstruction from 20 to 100 mm leads to a decrease in vascular blood flow, capillary filtration and a reduction in blood loss of up to 70%. The vertebral column, diaphragm, pelvic bones, ribs and abdominal wall make up an enclosed space called the abdominal cavity. Physical laws state that the pressure in the enclosed cavity spreads evenly, compressing the intestine from the outside until it equals the pressure of the intraluminal space. The compression of the intestinal wall between the intraluminal pressure from the inside and IAP from the outside leads to microcirculation and thrombus formation in small vessels, ischaemia and oedema of the intestinal wall. As a result, fluid transudation and exudation worsens, resulting in a vicious circle .

Conclusions: Thus, regardless of the aetiology of IAP occurrence, the focus should be on the prevention of increased intra-abdominal pressure, because the relationship between IAP and enteral insufficiency syndrome represents a mutually aggravating relationship of pathological processes

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