

# ABDOMINAL COMPARTMENT SYNDROME

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**V.A. Ovchinnikov**, D.Med.Sc., Professor, Head of the Department of General Surgery named after A.I. Kozhevnikov;

**V.A. Sokolov**, Resident Medical Practitioner, the Department of General Surgery named after A.I. Kozhevnikov

Nizhny Novgorod State Medical Academy, Minin and Pozharsky Square, 10/1, Nizhny Novgorod,  
Russian Federation, 603005

We considered one of the most complicated problems of surgery and intensive care — abdominal compartment syndrome. It is a severe, and in some cases lethal complication developing in major injuries and pathology of abdominal cavity and retroperitoneal space, as well as in extra-abdominal pathology. In addition, compartment syndrome can be the complication of a number of surgical procedures accompanied primarily by laparotomy wound closure with tissue tension. We demonstrated the classification of the complication taking into consideration the etiology, pathogenesis of intra-abdominal hypertension development, the significance of intra-abdominal pressure level, as well as its role in multi-organ failure development. We considered the methods of intra-abdominal pressure measurement, and the urinary pressure measurement using Unometer™Abdo-Pressure™ device turned out to be the most accurate, simple and safe. The preventive and treatment modalities of abdominal compartment syndrome were described. The monitoring of intra-abdominal pressure level in risk group patients, adequate infusion therapy, timely decompression (nasogastrintestinal intubation, decompressive laparotomy), and optimal respiratory support conditions are of primary importance.

**Key words:** intra-abdominal pressure; abdominal compartment syndrome; intra-abdominal pressure monitoring; decompressive laparotomy.

In the second half of XIX c. Marey and Burt established the relationship between the intensity of respiratory failure and the degree of the abdominal wall stress, though the widespread interest to the problem appeared in the late XX c. By now many experimental and clinical researches have been carried out, and extensive evidence concerning the problem has been collected.

Normal level of intra-abdominal pressure (IAP) is 0–5 mm Hg [1]. In addition, there is significant positive correlation between its value and body mass index. It explains the fact that some conditions such as obesity or pregnancy are accompanied by steady IAP increase up to 10–15 mm Hg, however, a patient manages to adapt to such an increase, and it is of no great importance compared to a sharp IAP rise [2]. Gender, age, concomitant diseases and previous operations are considered to have no significant effect on IAP.

In 2004 the first conciliation conference devoted to the abdominal compartment syndrome (ACS) defined intra-abdominal hypertension (IAH) as the steady IAP increase up to 12 mm Hg and more that is recorded at least in three standard measurements with 4–6-hour intervals [2]. This definition excludes the record of short IAP variations that have no clinical relevance.

In 1996 J.M. Burch et al. developed the IAH classification

[3] based on IAP level. The classification has undergone some changes and now it is as follows:

Degree I is characterized by intravesical pressure of 12–15 mm Hg;

Degree II — 16–20 mm Hg;

Degree III — 21–25 mm Hg;

Degree IV — over 25 mm Hg.

Currently, abdominal compartment syndrome (synonyms: intra-abdominal hypertension, high intra-abdominal pressure syndrome, abdominal syndrome) is defined as persistent IAP increase up to the level of over 20 mm Hg and associated with the manifestations of multiorgan failure/dysfunction [2]. The most important thing is that compared with IAH phenomenon, ACS needs no classification by IAP level due to the fact that this syndrome in modern literature is presented as “all or nothing” phenomenon (i.e. if ACS develops in some IAP degree, further IAP increase is of no importance).

ACS is one of severe complications of a variety of acute and chronic abdominal and retroperitoneal diseases, as well as extra-abdominal pathology and requires timely corrective and preventive measures.

The accurate incidence of this complication is unknown. According to different sources, IAH incidence in patients after laparotomy performed for abdominal injuries in

For contacts: Sokolov Victor Alexeevich, phone +7 908-736-24-01; e-mail: bolshevik-88@mail.ru

postoperative period is from 2 to 30% [3–6]. Among ICU patients admitted in critical condition, IAH phenomenon occurs in 54.4% of therapeutic cases, and in 65% of surgical patients. Moreover, ACS develops in about 8.2% of IAH cases [7–10].

When decompression laparotomy/relaparotomy is performed 3–6 h after the first ACS sings, the lethality is on the average 20%, in late period — from 43 to 65.5%, and without surgical decompression the case fatality reaches 100% [9].

## Etiology

Anatomical background of IAH and ASC development is as follows: the abdominal cavity and retroperitoneal space are limited from different sides by muscles, spine, diaphragm and pelvic bones [11]. Due to the extensibility of soft tissues, the abdominal cavity volume can increase up to some final values.

A variety of contributory causes of ACS development have been distinguished. They can be divided into four groups [12]:

*Factors contributing to the reduction of the anterior abdomen wall compliance:* a marked pain syndrome and anterior abdominal wall; artificial lung ventilation, especially in respiratory resistance, the mismatch of positive end expiratory pressure, pleuropneumonia, giant ventral hernia tension repair, the anterior abdominal wall scarring.

*Factors contributing to the increase of abdominal cavity content:* gastroparesis, bowel obstruction, abdominal tumors, abdominal aortic aneurysm, retroperitoneal edema or hematoma.

*Factors contributing to fluid or gas accumulation in abdominal cavity:* ascites, pancreatitis, peritonitis, hemo- and pneumoperitoneum.

*Factors contributing to “capillary leak” syndrome development:* acidosis (pH is under 7.2), hypothermia, polytransfusion (over 10 units of packed red blood cells per day), coagulopathy, sepsis, bacteriemia, massive infusion therapy (over 5 L of colloids or crystalloids within 8–10 h), major burns (over 25% of body surface).

These factors explain a wide range of contributory causes of ASC development; the most frequent ones are: major injuries of abdominal cavity organs [6, 13–15], fracture of the pelvis [14], abdominal [6, 16, 17] and retroperitoneal [6, 18] hemorrhages, rupture of abdominal aortic aneurysm [19], peritonitis [17, 20], destructive pancreatitis [21], burns of over 60% of body surface [12, 17, 22–24], massive infusion therapy of shock [14, 17]. Less frequent ASC causes are: bowel obstruction [16, 17], laparotomy wound closure with tissue tension [17, 25], herniotomy [20, 26], peritoneal dialysis [16]. The following causes are referred to as rarer ones: ACS development in patients with isolated craniocerebral injuries and isolated extremity trauma [27–29] accompanied by soft tissue compression syndrom [29]. V. Fraipont et al. [30] described ACS development after transcuteaneous dilated tracheostomy complicated by pneumoperitoneum, V. von Gruenigen et al. [31] reported on two observations of ACS after panhysterectomy [32]. There have also been described the cases of ACS

developed after esophagogastroduodenoscopy [33], after endarterectomy from superior mesenteric artery [34, 35], and during laparoscopy [16, 17, 36, 37].

Etiological ASC classification is the following:

*Primary ACS* (surgical or abdominal) is the consequence of pathological processes developing in abdominal cavity as a result of abdominal injury, rupture of abdominal aortic aneurysm, hemoperitoneum, acute pancreatitis, widespread peritonitis, retroperitoneal hematoma, etc.

*Secondary ACS* (therapeutic, extra-abdominal) is characterized by the presence of subacute or acute chronic IAH caused by extra-abdominal pathology, e.g. sepsis, major burns, or other conditions requiring massive infusion therapy [2, 14, 38–42].

*Recurrent ACS (tertiary)* is recurrent onset of symptoms typical for ACS against the background of resolving presentation of the primary or secondary ACS that occurred earlier. This condition is frequently called “the second stroke” phenomenon. Recurrent ACS can develop against the background of “open abdomen” (open abdomen compartment syndrome) or after the complete closure of the abdominal cavity (laparostoma elimination) [21]. Tertiary ACS is characterized by reliably high lethality rate [43].

## Pathogenesis

By now the most studies are devoted to pathological physiology of ACS development.

IAP level increase up to 15 mm Hg and higher is considered to be a trigger mechanism. Experimental data are of interest [44, 45], and according to them, multi-organ failure rapidly develops if IAP increases up to 30 mm Hg (animal studies), though another similar experiment showed the IAP increase up to 15 mm Hg persisting within 24 h to result in ACS development as well. IAH phenomenon triggers a number of pathological changes in organs and systems that can be successively described as follows.

**The changes occurring in the organs of the abdominal cavity and retroperitoneal space.** IAP increase up to 15 mm Hg significantly impairs the blood supply of abdominal and retroperitoneal organs. The blood flow along the superior mesenteric artery and the blood supply of gastrointestinal tract mucosa reduce, gastric wall oxygenation decreases that results in marked pH reduction in mucosa [46]. In jejunum these pathological changes are expressed in a lesser degree [47], however, histological examination reveals mucous damage of all parts of gastrointestinal tract [44], up to the necrosis of all intestinal wall layers and peritonitis [48]. These changes are likely to develop till cardiac output significantly decreases [49], and therefore, can serve as early precursory symptom of ACS [50, 51]. Perfusion pressure of abdominal cavity (abdominal perfusion pressure) calculated as the difference of mean arterial pressure and intra-abdominal pressure, decreases earlier and more intensely than the pressure in the superior mesenteric artery and cardiac output, and can be the most important prognostic parameter [52]. The homeostasis changes damage barrier function of gastrointestinal mucosa, it leading to bacterial translocation into lymph nodes [53], spleen and portal vein [46]. Blood flow along

the hepatic artery slows down in IAP over 11 mm Hg, along the portal vein — in IAP over 20 mm Hg. It results in necrosis of up to 12% of hepatocytes [25], and it manifests both morphologically [44], and by laboratory tests: by the increase of ALT level [44] and alkaline phosphatase [49].

#### **Changes in cardiovascular and respiratory organs.**

Cardiorespiratory changes develop in 57% of ACS patients. IAP increase results in diaphragm upward displacement, the increase of pressure in pleural cavities, thoracic compliance decrease, and a number of changes of ventilation function and gas exchange. The researchers [38, 44] think that IAP up to 25–30 mm Hg leads to the increase of maximal inspiratory pressure nearly twofold (from 18 to 34–40 cm of water column).  $p\text{CO}_2$  value also grows [53] and  $p\text{O}_2$  drops. Respiratory distress-syndrome gradually develops [54]. Rapid rise of respiratory insufficiency causes a patient to be transferred to artificial lung ventilation with stringent parameters [32]. Elevated IAP results in the increase of intrathoracic pressure due to inferior vena cava compression [55] that reduces venous return to the heart [56]. Some researchers indicate the reduction of cardiac output [44] and cardiac index, while others consider that it changes insignificantly [38, 48]. There have been recorded the increase of total peripheral resistance and central venous pressure [38]. The pressure in femoral vein, inferior vena cava [38] and internal jugular vein [57] also elevates. Systolic arterial pressure can remain unchanged [53], grow [38, 57] due to peripheral vasoconstriction, or drop in case of unsuccessful or inadequate therapy. Respiratory acidosis is associated with metabolic acidosis, and it is manifested by the increased lactate concentration and poor basis [46], reduced blood pH [46, 53]. There also decreases muscular tissue oxygenation that directly depends on oxygen content in pulmonary artery [47].

**Changes in the kidneys.** Some researchers [40] believe that cardiopulmonary insufficiency and renal failure are the most frequent and threatening signs of ACS. Elevated IAP impairs macro- and microcirculation, urine secretion, and glomerular filtration [6]. Histology reveals necroses of glomerular epithelium [44, 49]. Due to this, renal blood flow and glomerular filtration decrease, and there is the increase of renin, aldosterone [58] and antidiuretic hormone [38] in plasma, proteinuria occurs [59]. The rate of hourly diuresis decreases more than twice if IAP elevates up to 15 mm Hg and persists within 24 h [49]. However, system arterial pressure can be not over 90 mm Hg. The increase of IAP up to 20–30 mm Hg in most observations results in anuria that is not corrected by the administration of diuretics [58]. According to D. Meldrum et al. [60], renal dysfunction develops in 33% of ACS patients.

**The increase of intrathoracic pressure.** This factor causes **intracranial hypertension** [61] due to the functional blockage of outflow from brain through jugular veins [38, 57]. There intensify cerebral edema and dysfunction [48] that in its turn increases the risk of secondary ischemic neuronal damage [62]. The patients with the combination of craniocerebral trauma and abdominal injuries have the syndrome of mutual aggravation that significantly worsens the prognosis. In addition, W. Ertel et al. [39] report on two patients with critically evaluated intracranial pressure

against the background of ACS. Moreover, G. Citerio et al. [57] recommend being cautious when performing laparoscopy in patients with craniocerebral traumas.

Elevated IAP **worsens the healing of laparotomy wounds and can cause ischemia and necrosis of abdominal wall** [48].

Thus, ACS has a variety of pathogenic mechanisms affecting all organs and systems. And the most significant are respiratory disorders, oliguria, proteinuria, reduced abdominal perfusion pressure, necrotic changes in intestinal wall, and in later period — the decrease of cardiac output, hepatic ischemia signs, respiratory and metabolic acidosis. All this determines the necessity to continuous monitoring of IAP level in risk patients.

#### **Clinical presentation**

Clinical presentation of ACS is nonspecific [63]. According to some authors [64], ACS is clinically manifested by the abdomen volume expansion and rigidity of abdominal muscles combined with oliguria and respiratory disorders. Other reports [41] indicate the growth of maximal inspiratory pressure and decrease of diuresis rate to be the earliest manifestations compared to other symptoms. The formula of early ACS diagnostics is high suspicion of a physician and regular (every 6 h [65]) IAP measurements in patients with high risk of elevated IAP [66, 67].

#### **IAP measurement techniques**

Direct method of measurement was suggested by R. Overholt in 1931 [68]. It consists in direct IAP measurement in case of open abdomen (laparostoma), during laparoscopy, peritoneal dialysis, or through the drainage in abdominal cavity. These techniques are considered accurate, though they have not come into use due to their technical complexity and possible complications that is why a number of researches have been carried out to search an indirect method of pressure measurement.

Pascal law says that pressure delivered on liquid or gas spreads unchanged on all particles of liquid or gas, therefore the pressure is equal in all parts of the abdomen cavity and retroperitoneal space. The law says it is possible to measure IAP in the urinary bladder, stomach, inferior vena cava.

There has been demonstrated [69] that IAP monitoring using pressure measurement in urinary bladder is an accurate and save technique. Elastic wall of the urinary bladder functions as a passive membrane and passes accurately the pressure level in the abdominal cavity. I.L. Kron et al. were the first to offer this method in 1984 [70].

Currently, there have been developed sterile closed systems to measure intravesical pressure, the most preferable is Unometer™Abdo-Pressure™ (Unomedical, Denmark). These systems include urimeter of the last generation, and testing section with built-in hydrophobic antibacterial air filter. When measuring intravesical pressure, we are to pay attention to the volume and rate of administering saline solution, and its temperature.

Rapid administration of cold solution can result in reflex contraction of the urinary bladder and elevated intravesical, and therefore, intra-abdominal pressure. The volume of the administered liquid is the object of discussion, and now equals to 20–25 ml. A patient is in supine position, horizontally, pubic symphysis being taken as a zero mark. When measuring IAP in postoperative period, the patients need adequate pain relief, which due to the relaxation of anterior abdominal wall muscles enables to receive the most exact values [8, 10]. The contradiction to the technique is the urinary bladder damage or compression by a hematoma or a tumor. In such a situation IAH is assessed using intra gastric pressure measurement. A tube is inserted into the stomach, and the height of liquid column is measured in a clear tube connected to the gastric tube. The anterior axillary line level is taken as a zero mark [71]. Less frequently IAP is measured in inferior vena cava using a catheter inserted through a femoral vein [72].

### Principles of ACS prevention and management

The basic principles of ACS prevention are the adequate infusion therapy, a correct adjustment of respiratory support parameters, timely decompression of the abdominal cavity [73, 74].

Currently, there are two approaches to IAP and ACS prevention. Some authors [25, 54, 75] for the preventive purpose suggest not to suture aponeurosis in patients with high risk of ACS who undergo laparotomy (primarily it concerns the patients with abdominal injury). Unfortunately, this technique does not always prevent IAP increase and ACS development [76]. Other authors [64, 66, 69, 77] in such patients recommend to monitor IAP after primary laparotomy wound closure, and in case of pressure increase — to perform decompression laparotomy.

**Surgical technique of abdominal cavity decompression.** The most researchers [64, 78–80] consider this technique to be the basic one. However, indications for conservative and surgical ACS treatment are still the matter under discussion, and have not been determined yet.

D. Meldrum et al. [60] suggest the following management algorithm: in degree I of IAH — adequate infusion therapy and monitoring; degree II — the continuation of therapy and observation, decompression laparotomy in case of ACS clinical presentation; degree III — decompression laparotomy and the continuation of intensive therapy; degree IV — immediate decompression and resuscitation measures. In patients with free fluid in abdominal cavity, particularly in secondary ACS, some researchers suggest as the first stage to perform peritoneal tap [75], abdominal paracentesis [24, 26], or laparoscopy with fluid evacuation and drainage of the abdominal cavity [13], and laparotomy is performed in case no effect follows.

Decompression laparotomy is the operation of choice now, and enables to reduce significantly the lethality in ACS patients. It is recommended to be performed not only in an operating room, but also in ICU [23, 50]. C. Toens et al. [44] demonstrated in their study that decompression laparotomy leads to IAP decrease from  $31 \pm 4$  to  $17 \pm 4$  cm of water column. W. Ertel et al. [81] after the surgery

recorded a significant increase of cardiac index, respiratory volume and diuresis rate, central venous pressure, the decrease of pulmonary artery pressure, maximal inspiratory pressure,  $p\text{CO}_2$  in arterial blood, and plasma lactate level. In 2 patients with accompanying severe craniocerebral injury decompression laparotomy resulted in the decrease of critically high intracranial pressure.

Laparotomy wound is closed after the edema of abdominal and retroperitoneal organs decreases and ACS arresting that usually occurs 1–8 days after decompression laparotomy [25, 39]. The reconstruction and final closure of the laparotomy wound in early period was found [75] to be possible only in 18% of cases.

Typical complications of decompression laparotomy are intestinal fistulas [82] and postoperative hernias (up to 90% of cases), which frequently develop after using meshes and absorbable materials [83]. Less severe complication is the formation of numerous suture sinuses (frequency rate of the complication is about 22% [84]).

It should be noted that decompression laparotomy can be complicated by the increase of acute cardiovascular insufficiency and asystolia due to sharp decrease of IAP, total peripheral resistance, hypovolemia exacerbation [85] and reperfusion of extensive ischemic zone [19], there are data [85] on the increase of the number of thromboembolic complications, including the increased risk of pulmonary artery thromboembolism.

There are reports on open management of the abdominal cavity (laparostoma in its proper sense), and absolute indications for this procedure are determined if there are the signs of anaerobic infection of the abdominal cavity or retroperitoneal space, ACS progressing (particularly against the background of treatment measures), as well as if it is impossible to close the laparotomy wound edges, particularly as the result of a marked suppurative necrotic inflammation of the wound or phlegmons of the anterior abdominal wall [86, 87].

**Nasointestinal intubation.** The necessity of intestinal decompression in ACS treatment raises no doubts. The use of nasointestinal tubes of various designs is preferable for this purpose. One can make sure that the position of a tube is correct, i.e. the tube enables in such a situation (the intestine condition, the characteristics of the tube) to perform its functions, only intraoperatively, therefore, in peritonitis the evacuation efficiency control of the intestinal content and lavage are to be started immediately after the tube is inserted (regular filling of the intestine in intubation and free evacuation) and control in any sanitation in case of staging treatment. The contradictions to nasointestinal intubation are marked infiltration of the intestinal wall (intestinal injure exceeds the intubation effect), critical condition of a patient if step therapy of peritonitis has been chosen. The use of unloading stomas for gastrointestinal tract decompression under widespread peritonitis attracts bitter criticism and is considered undesirable [86–89].

**Respiratory support.** The patients with developed ACS are certain to require artificial lung ventilation. Mismatched ventilation parameters not only eliminate but also exacerbate respiratory failure. Respiratory support is to be performed according to protective ventilation concept

in order to prevent ventilator-associated pulmonary injury. It is necessary to choose optimal positive end expiratory pressure in order to increase functionally active alveoli due to collapsed basal segments. An insignificant level of such pressure does not prevent alveoli collapse in expiration due to a high position of the cupula of the diaphragm contributing to the development of atelectatic injuries, and a very high level exacerbates hemodynamic disorders due to the increase of intrathoracic pressure, therefore, this parameters should be chosen according to the scheme “pressure-volume” [90–92]. Under IAH there decreases the compliance of the thoracic wall rather than the compliance of the lungs, therefore, the use of small respiratory volume (5–6 ml/kg) is optimal to prevent baro- and volutrauma [19, 76]. Inspiratory oxygen fraction should be sufficient minimum to normalize the saturation of mixed venous blood. The decrease of the saturation can be related to oxygen delivery decrease under impaired hemodynamics. The use of aggressive parameters of artificial lung ventilation against the background of ACS can be followed by the development of acute respiratory distress-syndrome [92–95].

**Infusion therapy.** Clinical manifestations and pathophysiological disorders, which develop in ACS are more expressed in patients with hypovolemia. Organ disorders in this case occur in earlier stage and more severe [88]. The presence and degree of hypovolemia intensity in IAH are cannot be found using conventional methods; therefore, infusion therapy should be performed taking into consideration possible edema of ischemic intestine and IAP increase [85, 93–95]. Crystalloid infusion is recommended for patients being prepared for surgical decompression to prevent hypovolemia. It should be remembered that after decompression laparotomy the patients with open abdominal cavity need more fluid administered — sometimes up to 10–20 L a day [41, 96–99]. Even after the decompression the diuresis rate in contrast to hemodynamic and respiratory indices does not recover immediately, it may take a long time. In this period it is reasonable to use extracorporeal detoxication methods including monitoring of electrolytes, urea, and creatinine [8].

**Conclusion.** The findings indicate abdominal compartment syndrome to be a severe complication of injuries and diseases of the abdominal cavity and retroperitoneal space. It is triggered by a significant increase of intra-abdominal pressure. The development of this syndrome is certain to result in pathophysiological changes, and multi-organ failure development with high lethality rate. If this complication occurs, the possibility to save a patient sharply decrease due to rampant development of organ disorders, therefore the patients with high risk of abdominal compartment syndrome development need monitoring of intra-abdominal pressure, adequate infusion therapy, timely decompression, the choice of optimal respiratory support.

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