

Abdominal Compartment Syndrome

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Abstract

Compartment syndrome is a pathophysiological term, comprising a variety of tissues and organ alterations, due to a higher than normal pressure in an anatomically detached space (compartment). In the human body, areas denoted as compartments include the orbital globe, the sub and epidural space, the abdomen, pleura, pericardium, and others. Compartment syndrome was described initially in limbs. Abdominal compartment syndrome is defined as an intra-abdominal pressure above 20 mmHg with evidence of organ failure. Abdominal compartment syndrome develops when the intra-abdominal pressure rapidly reaches certain pathological values, within several hours (intra-abdominal hypertension is observed), and lasts for 6 or more hours. The key to recognizing abdominal compartment syndrome is the demonstration of elevated intra-abdominal pressure which is performed most often via the urinary bladder, and it is considered to be the “gold standard.” Multiorgan failure includes damage to the cardiac, pulmonary, renal, neurological, gastrointestinal, abdominal wall, and ophthalmic systems. The gut is the most sensitive to intra-abdominal hypertension, and it develops evidence of end-organ damage before alterations are observed in other systems. The surgical decompression of the abdomen remains the treatment of choice of abdominal compartment syndrome; this usually improves the organ changes, and is followed by one of the temporary abdominal closure techniques in order to prevent secondary intra-abdominal hypertension.

Key words Abdominal compartment syndrome · Intra-abdominal hypertension · Intravesical pressure · Temporary abdominal closure

Introduction

Compartment syndrome (CS) is a condition in which increased pressure in a confined anatomical space adversely affects the function and viability of the tissues therein. The confined anatomical spaces mostly associated with compartment syndromes are the spaces of the extremities, the orbital globe (glaucoma), the cranial cavity (epidural or subdural hematoma), the pleura, pericardium, abdominal cavity, etc. Abdominal compartment syndrome (ACS) is defined as a constellation of pathological alterations of intra and extra-abdominal organs leading to a certain group of symptoms, all of which are caused by a sustained increased pressure within the abdominal cavity (compartment). Abdominal compartment syndrome is observed when the intra-abdominal hypertension (IAH) develops quickly within several hours and lasts for 6 or more hours. In 2006 an International Conference of Experts on IAH and ACS defined the latter as an intra-abdominal pressure (IAP) >20 mmHg, with clinical evidence of multiorgan failure (MOF).¹ The systems, affected by IAH are the cardiac, pulmonary, neurological, renal, gastrointestinal, abdominal wall, and ophthalmic. The gut is the organ most sensitive to IAH, and it develops evidence of end-organ damage before the development of the classic renal, pulmonary, and cardiovascular signs.² Severe intestinal ischemia is more important than the cardiac, pulmonary and renal changes.^{3,4} Recent data suggest that some of the adverse effects of elevated intra-abdominal pressure occur at lower levels than previously thought and are manifested prior to the development of a fulminating ACS. Therefore, ACS should be viewed as an end-result of a progressive, unchecked rise in IAP resulting from a myriad of disorders that eventually leads to MOF. The incidence of ACS is highly variable according to different trials but the severity of scores is a common factor. The measurement of IAP is pivotal for recognizing this condition, and mortality is still very high, especially

when MOF is already observed. The surgical decompression of the abdomen is a method of choice for treatment of ACS and usually improves the multi-organ disorder.^{5,6} A temporary abdominal closure method (TAC) should be considered in most cases following a defense laparotomy, so that secondary IAH is prevented.

History

The compartment syndrome was described for the first time in 1881 by Volkmann—limb compartment syndrome (LCS), a condition in which raised pressure within a closed fascial space reduces the blood perfusion of the muscles and leads to a contracture. The treatment of LCS was reported 7 years later by Petersen and it was experimentally shown in 1926.

Marey and Burt reported for the first time in 1863 the relationship between the level of IAP and respiratory function. The relationship between intra-abdominal hypertension and oliguria was recognized in 1876.⁷ In 1890, Heinricius showed that intra-abdominal hypertension of 27 to 46 cmH₂O was fatal in cats and guinea pigs. He attributed the deaths to the suppression of respiration by interference with thoracic expansion. In the first half of the 20th century some reports appeared giving details on the adverse effects of intra-abdominal hypertension on cardiovascular, renal, and pulmonary function. This became possible after the development of crude ventilatory support. When surgeons started treating peritonitis operatively, they were concerned about the “enormous pressure increase that often precludes abdominal closure.” In 1911 Emerson stated that “pressure conditions which exist within the peritoneal cavity had received insufficient attention.” In 1951 an anesthetist from Dublin suggested that postoperative intra-abdominal hypertension might be associated with an adverse outcome. He described the deleterious effects of forcing a distended bowel back into the abdominal cavity and attempting abdominal wound closure under tension after a dehiscence of “abdominal blow-up.” Later on, many different authors emphasized the occurrence of various changes in some systems in patients with IAH. Apart from these isolated reports it was not until the 1980s that clinicians began fully to appreciate the significance of increased IAP. For the first time the term abdominal compartment syndrome was used in 1984 by Kron et al., who described 11 oliguric patients undergoing an operation for ruptured abdominal aortic aneurism with intra-abdominal pressure >25 mmHg in the postoperative period. Following abdominal decompression seven of the patients recovered. The other four patients not operated on developed acute renal failure and died. The authors suggested that if the intra-

abdominal pressure exceeds 25 mmHg in the postoperative period and is associated with renal dysfunction despite adequate cardiac output and circulating blood volume, then decompression of the abdomen should thus be considered. The same author applied the universal intravesical (within the urinary bladder) method of indirect measurement of IAP.⁷ It was only in the past decade, that the pathophysiological repercussions of the increased intra-abdominal pressure and ACS were recognized in a wide spectrum of surgical patients and treated aggressively. The most recent work by Ivatury et al. and Surgue et al. should revise the classic definition of ACS to include isolated impairment of gut perfusion inasmuch as it may adversely affect the outcome, independent of either cardiopulmonary or renal dysfunction.^{8,9}

Normal Intra-abdominal Pressure

Compliance of the abdominal wall together with the abdominal contents determines the intra-abdominal pressure. The normal pressure in the intra-abdominal cavity (intra-abdominal pressure, IAP) is a state of steady pressure within the abdominal cavity and is individual—from sub-atmospheric to 5–6 mmHg, but less than 10 mmHg in supine position.¹⁰ According to some data from 2001, the mean normal IAP is 6.5 mmHg (range 0.2–16.2 mmHg), and body mass index (BMI) is positively related to intra-abdominal pressure and is influenced by recent abdominal surgery. The increased IAP in morbid obesity is not a true IAH and ACS, but represents a direct mass effect of the visceral obesity. Intra-abdominal hypertension is observed in 18% of elective laparotomies (exploratory laparotomies, upper and lower gastrointestinal, and aortic operations.) and in up to 40% of emergency laparotomies. Gender, age, and medical and surgical histories do not significantly affect IAP. It is not a constant value and varies within the respiratory cycle. Patients whose lungs were mechanically ventilated showed a slight increase in intra-abdominal pressure due to the transmission of pleural pressure across the diaphragm. Although an intra-abdominal pressure of >20 mmHg was clinically significant in nearly all patients, recent studies demonstrated that even at the relatively low intra-abdominal pressure of 10–15 mmHg significant alterations in organ functions can be seen.^{11,12} In postoperative patients IAP was 3.5–10.5 mmHg in males and 3.3–8.8 mmHg in females. To evaluate the factors affecting IAP some authors compared two groups of patients—one with hernia and a second one with obesity. The patients were at rest, straining, supine, erect, and before and after anesthesia. The authors concluded that in the hernia group the position made no difference in the IAP and the pressure

was generally lower in this group. In the obese group IAP was higher in general, and it was higher in straining. There was a significant drop in IAP after anesthesia in both groups. When the IAP values were elevated for a certain prolonged period of time, we define this condition as intra-abdominal hypertension. According to different authors the limit between normal IAP and IAH varies—12 mmHg, 15 mmHg, 25 cmH₂O.¹³ During laparoscopic procedures, the inflation pressure should not be higher than 15 mmHg. It must be taken into consideration that 1 mmHg = 1.36 cmH₂O = 0.13 kPa. Intra-abdominal hypertension does not include physiological acts leading to short-term elevation of IAP that does not influence organs and systems, such as Valsalva maneuver, coughing, defecation, straining, sneezing, some sports (sumo, judo etc.), though some authors call this hyperacute IAH. In such circumstances, the peak pressure can reach 100–200 mmHg.

Causes of IAH and ACS

Intra-abdominal hypertension is defined as an intra-abdominal pressure of 12 mmHg, or more.¹ Intra-abdominal hypertension does not necessarily lead to abdominal compartment syndrome. In a cohort of 706 patients with trauma, 15 were with IAH, and ACS developed in 6 of them. According to some authors IAH, which is present for longer than 6 h, leads to ACS. The exact value of pressure in the abdomen that can trigger the ACS varies in different individuals and is affected by both external factors and the abilities of the compensatory mechanisms of each patient. The combination of acidosis, hypothermia, and coagulopathy has been proposed as a triad leading to IAH and ACS. Intra-abdominal hypertension has been found to vary within the range 2%–30% in the surgical ICU population, and in emergency and major surgery patients the percentage has been even higher, whereas ACS has been observed in only 1%–16%. The risk of developing ACS increases as the Injury Severity Score (ISS) increases.

To understand the processes in the abdomen when the IAP changes, the abdominal cavity is compared with a tank, formed out of hard components (ribs, spine, pelvis), and extensible to a certain limited part (diaphragm, abdominal wall, viscera).¹⁴ This system follows Pascal's hydrostatic law: "If a pressure is applied to a non-flowing fluid in a container, then the pressure is transmitted equally in all directions within the container." So in cases when different causes raise the pressure in an arbitrary locus within the abdomen, the hydrostatic law leads to equilibrium of identical level of pressure within the whole system and its walls. The above-mentioned partially flexible part of the abdominal compartment wall reacts compensatorily by expand-

Table 1. Grading system for intra-abdominal hypertension

1.	10–15 cmH ₂ O	Grade I
2.	16–25 cmH ₂ O	Grade II
3.	26–35 cmH ₂ O	Grade III
4.	>35 cmH ₂ O	Grade IV

ing to its possible maximum. Subsequently, the pressure in the abdomen rises quickly, affecting different organs and systems.

Some authors propose a grading system for evaluation of IAH (see Table 1).^{5,6,10}

According to others, for practical purposes one should differentiate between three intensity grades of increased IAP¹⁵:

1. Mild abdominal hypertension—sustained acute elevation of 10–20 mmHg: Physiologic effects are generally well compensated and thus clinically non-significant. Nonoperative therapy may be required
2. Moderate abdominal hypertension—sustained acute elevation of 21–35 mmHg: Therapy is generally necessary. Intervention such as operative abdominal decompression may be crucial
3. Severe abdominal hypertension—sustained acute elevation >35 mmHg: Operative abdominal decompression is always indicated. (ACS)

Types of ACS According to Causes for IAH

1. Primary ACS—when there is a direct intra-abdominal organ injury—ileus, peritonitis, acute bleeding (rupture of liver, spleen, abdominal aorta), etc.
 2. Secondary ACS—due to third space edema, resuscitation, or as a result of forced closure of the abdominal wall after surgery
 3. Recurrent ACS—a patient has recovered from ACS, and something triggers the pathological cycle again
- Some data indicate the different etiological factors, leading to IAH and ACS.¹⁵

Acute

1. Spontaneous—peritonitis, intra-abdominal abscess, ileus, ruptured abdominal aortic aneurysm, tension pneumoperitoneum, acute pancreatitis, mesenteric venous thrombosis.
2. Postoperative—peritonitis, abscesses, ileus, acute gastric dilatation, intra-peritoneal hemorrhage.
3. Post—traumatic—intra or retroperitoneal bleeding, post-resuscitation visceral edema.
4. Iatrogenic—laparoscopic procedures, abdominal packing, reduction of massive hernia, abdominal closure under excessive tension, military anti-shock garments.

One study described a “hyperacute” form of secondary ACS (HACS) that developed intra-operatively while repair of extra-abdominal injuries was being carried out simultaneously with massive resuscitation for shock caused by those injuries.

Chronic

Ascites, large abdominal cysts and tumors, pregnancy, morbid obesity, and chronic ambulatory peritoneal dialysis can cause IAH. The chronic causes of IAH lead to a slower elevation of IAP as the abdominal wall accommodates and becomes more compliant over time, a phenomenon known as “stress-relaxation.” Consequently, the quick and high-grade deterioration seen in acute ACS does not occur in these patients. The chronic IAH is not benign and eclampsia is likely to be due to some extent to chronically elevated IAP.

A single case of development of IAH and ACS was reported after gastric overdistention due to aerophagia, one as a consequence of a penetrating heart injury; one case of IAH and late ACS secondary to traumatic gallbladder rupture, and one due to a rectus sheath hematoma. The development of IAH and ACS was observed once during colonoscopy, and in a case report after diaphragmatic plication.

The causes and the clinical manifestation of IAH and ACS in children differ from those in adults, the most important one being the low threshold of IAP, leading to relative IAH in this age and secondary triggering ACS. In this group a congenital problem could cause IAH, and respiratory pressure monitoring is likely to predict the IAH in neonates with gastroschisis.

Indications for the Measurement of IAP

The II World Congress of Abdominal Compartment Syndrome, Noosa, Queensland, Australia, December 6–8, 2004, proposed the following indications for measuring of IAP.^{16,17}

1. Postoperative abdominal surgery patient with a distended abdomen
2. Patient with open or blunt abdominal trauma
3. Mechanically ventilated ICU patients with other organ dysfunctions as assessed by daily Sequential Organ Failure Assessment (SOFA) score
4. Patients with a distended abdomen and signs and symptoms consistent with ACS:
 - a. Oliguria
 - b. Hypoxia
 - c. Hypotension
 - d. Unexplained acidosis
 - e. Mesenteric ischemia
 - f. Elevated intracranial pressure (ICP)

5. Patients with abdominal packing after temporary abdominal closure for multiple trauma or liver transplantation
6. Patients with open abdomens, especially if they have a Bogota bag closure and are in the early post-operative period (such patients may develop “recurrent” ACS despite their “open” abdomen)
7. Patients who have not undergone operation, but have received large volume fluid resuscitation in the context of an underlying capillary leak (peritonitis, septic shock, trauma etc)

Measurement of IAP

The key to recognizing ACS in critically ill patients is the demonstration of elevated IAP.¹⁸ On the other hand, the values of IAP are the most important data indicating the exact time for surgical decompression in primary ACS, re-exploration in secondary ACS, and definitive abdominal closure when the TAC technique is used.

Pressure is a force applied uniformly over a surface. Nowadays the SI system proposes the Pascal unit as a standard for measuring pressure. In spite of this, in clinical practice, experimental medicine, and animal models mmHg or cmH₂O or kPa are used. The sensitivity of the individual feel of the hand of the examiner on abdomen tenseness is about 40% according to some new studies, and simply putting the hand on the abdomen does not permit discrimination between pressures of 10 or 20 mmHg. As a result, other, more precise methods are used for more exact measuring of IAP.

Direct Methods for Measuring IAP

In many earlier experiments the IAP was measured directly through a metal cannula or a wide bore needle inserted into the abdominal cavity and attached to a saline manometer. Similar methods are still used in animal models. In a porcine model some investigators measured directly IAP, pulmonary artery pressure, and superior mesenteric artery pressure. An inflatable bag was placed into the abdominal cavity of the experimental animal to produce and measure elevated pressure. Parallel direct measuring of IAP was reported via a Verres needle connected to a pressure transducer and indirect trans-rectal method. No significant difference between intra-rectal pressure and directly measured IAP was noted. Recently, a method has been described in an animal model for direct measurement of IAP using modified piezoresistive technique and water-capsule technique, which is quite precise.¹⁹ When laparoscopic surgery is performed an electronic gas insufflator provides a continuous monitoring of IAP. Some authors used 14-F PVC round drain, inserted via

one of the laparoscopic ports in the abdominal cavity, connected to the invasive blood pressure measurement system. The correlation coefficient between this drain and the laparoscopic insufflator was 0.996.

Indirect Methods for Measuring IAP

This could be done through the inferior vena cava, stomach, rectum, vagina, or in the urinary bladder. According to some investigations, all these methods compared to each other, and to the direct measurement of IAP have an acceptable correlation.

Intragastric Pressure

Intragastric pressure can be measured by water manometry via a nasogastric or gastrostomy tube.²⁰ Gastric pressure is determined by infusing 50–100ml water through a nasogastric tube into the lumen of the stomach. The proximal end of the open tube is held perpendicular to the floor. The distance from the water level to the mid-axillary line is taken as IAP in cmH₂O. The method can be used in patients after either a cystectomy or a damaged urinary bladder. Another technique is the transgastric tonometry when a gastric tonometer with an air filled balloon can be installed in the stomach via the oro- or nasopharynx. The intragastric position is confirmed by aspiration of gastric juice and by the rise in IAP after external epigastric pressure. Instillation of up to 3ml of air allows the balloon to act as a pressure transducer. This technique allows for continuous measurements. A water manometer or pressure transducer is attached to one of these devices and the midaxillary line is considered “0,” equal to atmospheric pressure. The tonometric balloon on the tube is usually employed for tonometry, a technique of indirect measurement of gastric pH. A similar technique for fully automated continuous measurement of IAP in the stomach has been used in some recent investigations.¹⁷ In human studies an acceptable correlation of gastric pressure with urinary bladder pressure is found, while in animal models this correlation is poor.

Inferior Vena Caval Pressure

This method includes an infradiaphragmatic femoral venous catheter, the data for the pressure within the vein correlates directly with IAP. The pressure changes in the supradiaphragmatic vena cava are less pronounced.²⁰

Urinary Bladder Pressure (UBP)

This method is called also measurement of intravesical pressure (IVP), and was applied for a first time by Kron et al. in 1984;⁷ the technique was described more precisely in 1998. The method, which is used most often now for UBPs measuring includes inserting a Foley cath-

eter into the urinary bladder. In the supine patient (the nonsupine position may not provide valid interpretation for IAP, even more so in cases of increased body mass index), and after emptying the bladder, it is filled with 50–100ml of sterile saline. The newest investigations from 2006 recommend only 25ml to be infused in the urinary bladder for indirect measuring of IAP. Fifty or more milliliters may cause clinically relevant overestimation of IAP.²¹ Nowadays, a three-way Foley catheter is used with an adapter for the connection. The tubing then can be attached to a water manometer or pressure transducer, using the symphysis pubis as a “zero” point. This technique is considered to be the “gold standard” for indirect measurement of the IAP in clinical practice. The method is minimally invasive and easy to apply, and in animal models it has shown a high degree of correlation with directly measured IAP. Urinary bladder pressure measurement is the technique most often used to prove or reject the existence of IAH, and according to many new investigations with its help excellent results have been obtained for clinical practice.^{22–24} A simple U-tube method has been described for measuring IVP. Some authors have measured the intra-abdominal pressure simultaneously with UBPs in humans and reported a reliable degree of correlation. The routine measurements of IAP by means of bladder pressure have not been associated with an increased rate of urinary tract infections.

According to some investigators the intermittent measurements of IAP are just “snapshots” that poorly illustrate the “moving picture” of the patient’s response to injury and resuscitation.^{25,26} To avoid these pitfalls some authors use a continuous IVP monitoring technique—continuous intra-abdominal pressure measurement technique (CIAP).²⁷ Inferior vena caval pressure is continuously monitored via the irrigation port of a three-way urinary catheter in which a continuous normal saline perfusion 4ml/h is connected via two-way stopcocks and pressure tubing.

Some leading investigators of ACS state that the IVP as a gold standard for IAP has become a matter of debate, thus suggesting new revised methods of IVP measurement less prone to error, such as manometry screening techniques and microchip transducers.²⁸

Vaginal Pressure

In a canine model, the intra-vaginal indirect measurement of IAP in comparison to concomitant trans-rectal measurements did not show reliable results.

Intrarectal Pressure

No significant difference has been noted in the data from intrarectal and directly measured IAP in humans. Some recent investigations in the UK have shown that despite widespread awareness of IAH and the ACS,

many intensive care units never measure the IAP, and those who do always use the intravesical route. Some authors validate two novel methods in vitro for intra-abdominal pressure monitoring, using an abdominal compartment model.

Computed Tomography (CT) Findings

The following CT findings can confirm the presence of elevated IAP and pending or manifested ACS:

1. Tense infiltration of the retroperitoneum out of proportion for peritoneal disease
2. Extrinsic compression of the inferior vena cava due to retroperitoneal hemorrhage or exudate
3. Massive abdominal distension with an increased ratio of antero-posterior (AP), also called sagittal abdominal diameter (SAD), to transverse abdominal diameter (AP-T)—the so-called round belly sign
4. Direct renal compression or displacement
5. Bowel wall thickening with enhancement
6. Bilateral inguinal herniation²⁹

Pathophysiology of Increased IAP

The intra-abdominal hypertension causes a variety of dysfunctions in many systems in and outside the abdomen in a graded fashion especially when it jumps over the level of 20 mmHg. To better understand the clinical presentation and management of disorders of IAH, one must understand the physiologic derangements within each organ system separately. The usual evaluation of all dysfunctions caused by IAH is by sepsis-related organ failure assessment (SOFA) score, using the worst values of the day. The SOFA score for each organ ranges from 0 (normal) to 4 (most abnormal). Organ failure is defined as a SOFA organ subscore equal or above 3. Intra-abdominal hypertension provokes the release of pro-inflammatory cytokines which may serve as a second insult for the induction of multi-organ failure.

There is a single report in the literature concerning a decreased blood loss and improved hemodynamics in a porcine model when an iatrogenic abdominal hypertension was produced. According to the authors, this could be used for prehospital stabilization of abdominal injury patients and have the same autotransfusion effects as those observed with military antishock trousers.

Cardiovascular Effects

The first report on the impact of IAP on cardiac function (in an animal model) was made in 1911. The increase in the IAP results in a decrease to cardiac output (CO) and left and right ventricular stroke work. The direct com-

pression of the inferior vena cava and portal vein, intra-thoracic compression of superior vena cava and heart decrease the end-diastolic ventricular volumes and increase the central venous pressure (CVP), pulmonary artery wedge pressure, and pulmonary vascular resistance. By inducing venous stasis, IAH puts the patient at risk for venous thromboembolism. In patients with normovolemia, IAP even lower than 15 mmHg leads to a redistribution of abdominal blood volume towards the thoracic compartment, whereas in hypovolemia there is no thoracic compartment gain. The afterload is higher by the increase of systematic vascular resistance due to mechanical compression of the capillary beds. All these reduce the stroke volume by compensatory tachycardia.^{20,21,26,30-32} Despite the very high left and right atrial pressure, radionuclide-gated pool scans and echocardiography showed an adequate ejection fraction averaging 55% and ventricular volumes that were small or normal. If ACS is not treated, patients die due to left ventricular failure. After decompression an immediate decrease of IAP is observed, and improvement of the hemodynamic changes within 48 h of surgery.

Respiratory Effects

Recently, secondary acute respiratory distress syndrome (ARDS) in patients with IAH and ACS has been recognized as a separate entity. Increased IAP can lead to respiratory failure. The movement of the diaphragm cranially declines both static and dynamic compliance of the lungs and decreases lung volume and cardiac output. Compression atelectasis is mainly observed in the caudal parts of the lungs where IAP tends to squeeze the lower lobes. Pulmonary infection such as pneumonia is a typical early complication in abdominal hypertension from diffuse peritonitis. The peak inspiratory pressure and the pulmonary vascular resistance both increased. Ventilation-perfusion mismatches induced hypoxemia and respiratory acidosis and required mechanical lung ventilation.^{4,14,20,21} Some authors do not advocate high positive end-expiratory pressure (PEEP) for all patients with hypoxemia and ACS, especially considering that many of the conditions associated with ACS can also precipitate acute lung injury and ARDS. On the other hand, a poor lung function requiring ventilatory support and PEEP causes intra-thoracic pressure to be transmitted into the abdomen further increasing IAP.

Renal Effects

Since 1876 it has been known that IAH causes renal dysfunction, and its decrease leads to reversal of renal impairment.³³ Oliguria and increased blood creatinine was observed, at intra-abdominal pressures ranging

from 15 to 20 mmHg, which turns to anuria when pressure exceeds 30 mmHg. The pathogenesis of this malfunction is multifactorial,³⁴ and restoration of the CO to normal or supranormal values by blood volume expansion does not appear to prevent it reliably.²⁰ Changes in CO, direct compression of the abdominal aorta and the renal arteries, compression of the renal vein causing outflow obstruction, and compression of renal parenchyma with diminished renal blood flow, an increase in the renal vascular resistance and a redistribution of blood from the renal cortex to the medulla are the basic causes for the decrease in glomerular filtration rate (GFR). Furthermore, the direct compression of the kidneys increases cortical pressure, thus leading to a "renal compartment syndrome."³⁴ Ureteral compression can be excluded as the cause of diminished urine production with elevated IAP since oliguria has not been prevented by placing ureteral stents. In some animal models reduced GFR mediated by an increase of plasma renin, angiotensin, and aldosterone levels has been reported, when artificial IAH was produced.³⁵ Similar data in humans have been reported by some urologic investigations. The above-mentioned hormones turned normal with decompression of the abdomen, and significant improvement of urinary output, creatinine clearance, and osmolar clearance has been observed.²⁰ A single exception to this rule was reported, when laparotomy did not improve renal functions, although 10 patients out of 49 consecutive patients with IAH experienced brisk diuresis. The same authors showed IAP, together with age, hypotension, and sepsis to be independent risk factors for renal impairment in a sample of 263 patients after abdominal surgery.

Gastrointestinal Effects

When pressure in the abdominal compartment overcomes the pressure inside the capillaries perfusing the organs of the abdomen, ischemia and infarction of those organs occur. Intra-abdominal hypertension affects the splanchnic hemodynamics with decreased blood flow and perfusion of intra-abdominal arteries, veins and lymphatics, thus resulting in tissue hypoxia and intestinal swelling.^{5,14,36,37} Some authors conclude that the severe intestinal ischemia may be more important than the cardiac, pulmonary, and renal changes usually described.^{3,4} The gut is the organ most sensitive to IAH, and it develops evidence of end-organ damage before the development of the classic renal, pulmonary, and cardiovascular signs. In an experimental model with dogs the modest elevations in IAP were found to significantly affect the mesenteric lymph flow and lead to development of gut edema. An elongation of the mesenteric veins due to intestinal swelling causes tissue hypoxia and triggers a vicious circle generating further

intestinal swelling and subsequent hypoxia. This causes increase in intra-abdominal visceral and vascular volumes, consequently raising IAP. Animal experiments using radioactive microspheres to measure intra-abdominal blood flow have demonstrated these changes in all intra-abdominal organs with exception of adrenal glands, when IAP increases to >20 mmHg. According to others, the microcirculation is impaired even with normal global perfusion indices and without clinical signs and symptoms of overt ACS. This impairment takes place at relatively low levels of IAP (around 15 mmHg) and within a short time interval (60 min). Abdominal perfusion pressure (APP), the difference between the mean arterial blood pressure (MAP) and IAP, may thus be a superior prognostic parameter. The data obtained from animal models showed that IAP of 30 mmHg causes reduced blood flow in the portal vein, gastric mucosa, small bowel mucosa, pancreas, spleen, and liver. This result was confirmed when laser Doppler flowmetry was used to assess changes in hepatic microvascular blood flow. Some other investigators conclude that IAH leads to a decreased cardiac index and splanchnic flow, while the hepatic artery flow is selectively preserved and may provide protection for liver function during sustained elevations in IAP. The hypoperfusion of the intestines and other peritoneal and retroperitoneal structures leads to release of cytokines, formation of oxygen free radicals, and decrease of cellular production of adenosine triphosphate. Finally, this leads to higher permeability of the intestinal wall and bacterial translocation (BT).^{3,38} The bacterial translocation and growth starts at IAP > 14 mmHg and increased significantly above 20 mmHg. The translocated bacterial species from the bowel lumen into the free peritoneal cavity act synergistically, that is to say, together they bring about a severe disease that cannot be caused by a single organism alone. Others have observed bacterial translocation in the ileocecal lymph nodes, the spleen, and the liver, as confirmed by multiple biopsies in rabbits. A gradual increase in the phenomenon was noted as IAP increased from 10 to 15 mmHg, BT was overt at 15 mmHg, and significant at 20 mmHg. *Klebsiella pneumoniae*, *Serratia marcescens*, and *Escherichia coli* were the predominant pathogens identified by culture. Some other studies did not find a significant increase in bacterial translocation when artificial IAH was applied in an animal model, although in their study of hemorrhage and reperfusion followed by IAH they observed intestinal mucosal acidosis and hypoperfusion. Other authors have found an increased endotoxin content in the portal vein during IAH in a rabbit model. This was associated with bacterial translocation to intestinal mesenteric lymph nodes and liver. They conclude that this might be an important factor in the progress from IAH to ACS and MOF. According to some data

IAP increased by 10 mmHg results in significant increase in variceal pressure, radius, volume, and wall tension in patients with esophageal varices. The authors conclude that IAH may contribute to variceal rupture in cirrhotic patients. Intra-abdominal hypertension affects the liver hemodynamics and some degree of hyperbilirubinemia occurs, while the liver functions are not much affected. In patients with ascites, the reduction of IAP by paracentesis results in a significant reduction in gastroesophageal reflux.

In the postoperative period the secondary IAH and ACS can compromise the intra-abdominal anastomosis, which triggers a heavy bacterial contamination of the peritoneum and leads to an extreme rise in IAP; in this way a vicious circle is thus observed.

Abdominal Wall Effects

Patients with IAH often have wound complications such as dehiscence, infection, and herniation. Decreased abdominal wall compliance is caused by a direct compressive effect on the microvasculature and the inferior epigastric vessels, which leads to fascial ischemia and edema. The blood flow to the rectus is reduced by 60% at an IAP of 10 mmHg or more.³⁹ As collagen deposits and resistance to infection are directly proportional to tissue perfusion and oxygenation, elevated IAP adversely affects the wound healing. Some investigators have confirmed in animal models, that the rectus sheath blood flow is significantly reduced at IAP levels of 10, 20, 30, and 40 mmHg. All this leads to a further increase of IAP, and a vicious cycle develops. An additional risk factor is the long-term ambulatory peritoneal dialysis. The authors suggest that automated long-term ambulatory peritoneal dialysis with low daytime fill volume and pressure and high body mass index should be considered in all patients at risk for hernias and/or leakage. Others also note a strong correlation between body mass index and IAP in children on peritoneal dialysis.⁴⁰

Neurological Effects

Intra-abdominal hypertension significantly increases intracranial pressure, which normalizes after decompressive laparotomy.⁴¹ The mean intracranial pressure at baseline is 13.41 Torr, it rises to 18.715 Torr during pneumoperitoneum of 10–15 mmHg.²⁰ According to the modified Monro–Kellie approach, the intracranial contents are divided into four compartments: osseous, vascular, cerebrospinal fluid, and parenchymal. Intracranial pressure reflects the relationship between these volumes and intracranial compliance. In the physiological range a small volume increase does not cause substantial pressure increases up to a decompensation point, after which

each small increase in volume results in a relatively large increase in intracranial pressure and reduction of intracranial compliance.⁴² Even a small increase in the cerebral blood flow leads to a marked increase in ICP. Since intracranial compliance differs between patients, and over time within the same patient, we cannot expect that a rise in IAP will always cause the same rise in ICP. Some investigations confirm that the elevated IAP significantly decreases the cerebral perfusion pressure (CPP). This decrease did not become statistically significant until IAP reached 25 mmHg. The CPP drops secondary to impaired venous drainage due to increased intrathoracic pressure (ITP), via the jugular venous system, from the cranial vault. Some studies found a variable pressure transmission (from 20% to 80%) from the abdominal to the intrathoracic compartment. Another proposed mechanism is that the decrease or restriction of lumbar venous plexus outflow causes an increase in spinal cord and cerebral spinal fluid pressure as well as a secondary increase in intracranial pressure. Because the spinal canal and the intracranial vault are continuous, a sudden rise in intraspinal pressure causes a concomitant increase in intracranial pressure. In an animal model study an artificial IAH was produced, which caused a significant increase in ICP. A subsequent sternotomy and pleuro-pericardiotomy to decrease pleural pressure led to an immediate decrease in intracranial pressure, thus suggesting that intracranial hypertension was secondary to obstruction of cerebral venous outflow due to increased intra-thoracic and central venous pressure.³⁶ Some authors describe a decrease in IAP accompanied by a decrease in ICP after a bolus administration of neuromuscular blocking agent.^{25,43,44} Fentanyl, on the contrary, may acutely increase IAP and ICP by stimulation of active phasic respiratory activity. Some data do not confirm these results, in a group of investigated patients the CPP did not change after elevation of IAP. The association between the IAH and ICP explains the existence of idiopathic intracranial hypertension (pseudotumor cerebri) in patients with abdominal trauma without cranial trauma, in obese patients and pregnant women. In up to 8% of pregnancies hypertension, renal dysfunction, proteinuria, headaches, photophobia, seizures, and coma develop. The etiology of pre-eclampsia/eclampsia is unknown and treatment is aimed at ameliorating the end-organ effects by supportive care. Since pregnancy is associated with an increased IAP, some of the pathophysiological changes could be attributed to IAH. Abdominal trauma in head-injured patients contributes to intracranial hypertension. Data support the notion that it is better to have a low threshold for abdominal decompression in patients with combined injuries.^{20,45} Diagnostic laparoscopy may increase intracranial pressure and must not be used in evaluating patients with severe head

injuries. The high ICP and low CPP affect morbidity and mortality in head injured patients.

Ophthalmic Effects

Intra-abdominal hypertension causes a rupture of retinal capillaries, resulting in the sudden onset of decreased central vision (Valsalva retinopathy).⁴⁶ This phenomenon has been described in a number of settings in which a sudden increase of IAP and/or ITP has occurred. The retinal hemorrhage usually resolves within days to months without any treatment. If a patient with ACS develops visual changes, Valsalva retinopathy should be considered and an appropriate ophthalmic examination should be performed.

Lower Limb Effects

Another adverse effect of IAH is the impaired distal extremity circulation secondary to pressure on the aorta.

Prevention and Treatment of Abdominal Compartment Syndrome

The threat of the development of secondary ACS and the treatment of clinically manifested primary ACS must be clearly distinguished. In the first case preventive measures should be taken, while in the second one an aggressive treatment must be applied. In 2003 a study involving 188 patients with major torso trauma found 6% with primary ACS, and 8% with secondary ACS. The authors conclude that primary and secondary ACS have similar pre-decompression physiology, demographics, injury severity, time from hospital admission to decompression, treatment, and poor outcome. With appropriate monitoring, both could be accurately predicted upon ICU admission.⁴⁷ In another study the same authors conclude that primary and secondary ACS differ only in their different predictors. The primary ACS predictors upon intensive care unit (ICU) admission are a low temperature, low hemoglobin concentration, and a high base deficit, which are all indicators of the “bloody vicious cycle” physiology, and such factors are the reason why damage control surgery is selected. The secondary ACS predictors are high crystalloid infusion volume and

impaired renal function compared to non-ACS resuscitation patients.⁴⁸ They conclude that because the predictors of ACS include both physiological measurements and resuscitative interventions, this model had better be performed in a clinical situation during ongoing resuscitation rather than only relying on arbitrary IAP and organ dysfunction thresholds.⁴⁷ Another indicator for increased IAP before organ changes occur is the serum D-lactate level. Some data suggest that neither a single factor nor a group of factors can predict with sufficient accuracy which patients are likely to develop IAH.

Prevention of Secondary ACS

Recent reports have described resuscitation-induced, secondary ACS in trauma or nontrauma patients.⁴⁹ Some data from the World Congress of the Abdominal Compartment Syndrome in December 2004 strongly confirm this statement.¹⁶ Secondary ACS has been observed after burns of the thoracic and abdominal area, postoperative hemorrhage, or orthotopic liver transplantation, rarely after laparoscopic procedures.⁵⁰ Some data define the condition as a highly lethal—38% in trauma and 100% in nontrauma patients with secondary ACS. Secondary ACS can develop without abdominal injury,⁵¹ after severe hemorrhagic shock with an incidence of 0.5% in this cohort. Conventional preload directed resuscitation to enhance cardiac function is not effective in patients with impending ACS, and this traditional resuscitation strategy is detrimental in this subgroup of patients. Massive fluid resuscitation could cause secondary ACS and respiratory failure during surgery for a ruptured descending thoracic aortic aneurysm (rTAA).⁵² Supranormal resuscitation, in comparison to normal resuscitation, is associated with increased incidence of IAH, ACS, MOF, and death because of decreased intestinal perfusion.⁵³ Routine bladder pressure monitoring is recommended for patients with ongoing resuscitation greater than 500cc/h. According to some authors the peak airway pressure and net 24-h fluid gradient are the only predictors of secondary ACS.

In some recent works the authors recommend different therapeutic approaches according to the IAP level (Table 2). In severe abdominal trauma and inflammation, which requires massive fluid replacement, the development of ACS can be foreseen due to massive

Table 2. Recommended therapeutic approaches differing according to the intra-abdominal pressure level

1.	Grade I (10–15 cmH ₂ O)	To maintain normovolemia
2.	Grade II (16–25 cmH ₂ O)	Hypervolemic resuscitation
3.	Grade III (26–35 cmH ₂ O)	Decompression
4.	Grade IV (>35 cmH ₂ O)	Decompression and re-exploration

bowel, abdominal wall, retroperitoneal, and solid organ edema. These changes are usually well visualized on CT scanning, and confirmed during laparotomy. Tight abdominal closure in these circumstances can worsen the tissue injury and can result in a fatal outcome. When looking at the open abdomen horizontally, the guts can be seen above the level of the wound (rule of thumb) – 10. In these cases the abdomen should be left open (laparostomy) and temporary abdominal closure utilized.⁵⁴ It is better to anticipate the development of ACS and to take due measures after surgery.

The opportunity to prevent secondary ACS in the at-risk patients usually presents after laparotomy. However, there is disagreement between surgeons as to whether it is more beneficial to prevent ACS by performing some method of temporary abdominal closure or whether one should perform primary fascial closure and adopt a “wait and see” approach, only intervening when signs of ACS develop. Some opinions do not support liberal use of an open abdomen technique to prevent ACS,⁵⁴ while others propose the use of a temporary laparostomy in all circumstances.^{13,56,57}

Patients identified as being at risk for developing IAH and ACS should therefore undergo close monitoring of IAP, usually via the urinary bladder. As the development of IAH is usually gradual, measurement of IAP every 8h is generally adequate unless there is major intra-abdominal hemorrhage, when more frequent measurement is warranted. The development of secondary ACS is a particular risk in those with severe abdominal trauma, especially when a staged or “damage control” laparotomy is performed. This focused surgery with a short operating time minimizes the vicious cycle of hypothermia, coagulopathy, and acidosis. Bleeding is rapidly controlled, and a minimal necessary bowel resection is performed to prevent gross contamination. If surgery is followed by primary fascial closure, the continued intra-abdominal bleeding, bowel edema secondary to massive crystalloid and colloid resuscitation, and bowel ileus conspire to increase IAP and lead to ACS. In one series, 100% of patients who underwent a damage-control laparotomy and primary fascial closure for severe abdominal trauma developed severe IAH after surgery.

Many methods of TAC have therefore been described that allow the definitive closure of the abdominal wall to be deferred until the factors responsible for the development of IAH have resolved. Postoperative monitoring of intra-abdominal pressure can be used to determine the optimal time for definitive abdominal closure. These data could help also to anticipate the threat of relapsing ACS. The necessary prerequisites are good systemic oxygenation, normovolemia, and correction of coagulopathy. The most frequently used techniques have been open drainage (laparostomy), lately substituted by semi-

open methods, and staged abdominal repair (STAR) operation. Unfortunately, fascial closure may not be always possible, especially when there is a long period of time until the second operation.

Treatment of Primary ACS

Surgical decompression, also called a defensive laparotomy, remains the treatment of choice in primary ACS.⁵⁸ The decompression laparotomy followed by TAC for treatment of primary ACS is undisputed.⁵⁹ Recently, a few reports have been published describing ACS in patients with severe acute pancreatitis requiring decompressive laparotomy. In a porcine model of ACS subcutaneous endoscopic fasciotomy was successfully applied, in one patient with ACS due to acute lymphocytic leukemia and peripancreatic fluid an ultrasound-guided drain was successfully applied, and in cases with ruptured abdominal aortic aneurysm (rAAA) a trans-lumbar extraperitoneal decompression was performed.

The timing indications and threshold for decompression are controversial, with very few large trials available to give firm guidance.⁶⁰ The different sources propose a variety of tactics, based on the data of IVP—15–19 mmHg, >20 mmHg, 25 mmHg or higher, ≥20 mmHg, or when the pressure is raised parallel to organ dysfunction, indicated by increased airway pressure, reduced cardiac output, and oliguria. In these circumstances, generous fluid administration is required, because cardiovascular collapse may occur during the operation. Most of the organ system changes appear to be reversible after the IAP returns to normal values following a laparotomy. Some authors advocate tactics of treatment based on the values of IAP-IVP, proposing a grading system to aid decision making and recommending abdominal decompression when intra-abdominal pressure is >35 cmH₂O (25 mmHg), as abdominal pressures exceeding this value are invariably accompanied by severe physiological dysfunction and clinical deterioration.⁶ In 1997 a therapeutic approach according to the level of IAP was recommended (Table 3).⁶¹

Most other authors advocate the use of markers of physiological deterioration such as oliguria, hypotension, and acidosis in conjunction with abdominal pressure >25 mmHg to determine the optimal time for intervention. Worsening hypercapnia, deteriorating pulmonary compliance, and excessively increased airway pressure often warrant surgical decompression. Persistent splanchnic hypoperfusion as measured by gastric tonometry may also be a useful aid in the decision making process.⁶² The best management of these complex patients therefore requires very close cooperation between surgeon, intensivist, interventional radiologist, and several other specialties. In all cases the

Table 3. Recommended therapeutic approach according to the level of intra-abdominal pressure

Grade	IVP cmH ₂ O	Recommendation
I	10–15	Maintain adequate intravascular volume
II	16–25	Maintain adequate intravascular volume and closely monitoring
III	26–35	Consider decompression
IV	>35	Perform surgical decompression

IVP, intravesicle pressure

existing functional or mechanical ileus must be taken in consideration and evacuation of the toxic bowel content performed, using one of the open or closed methods. This is usually followed by temporary abdominal closure.¹³

Providing anesthesia for the critically ill patient with ACS undergoing decompressive laparotomy is extremely challenging. The patient should be well resuscitated before opening the abdomen. The ACS is an example of ischemia/reperfusion injury, known as reperfusion syndrome, or systemic reperfusion injury.¹⁰ Rapid decompression of the abdomen results in acute hypovolemia as a result of the decompression of the mesenteric vascular bed and release of lactic acid into the blood stream. This is also a sequel of an abrupt drop in central filling pressure and systematic vascular resistance. Adverse cardiovascular events including malignant arrhythmias, asystole, acidosis, and vasodilation have been described in abdominal decompression; they are the result of starting reperfusion of the abdominal viscera and lower extremities, resulting in a sudden increase in the blood levels of products associated with anaerobic metabolism such as lactic acid and potassium.⁶² During reperfusion, gut injury may be amplified by increased production of free oxygen radicals and exhaustion of endogenous antioxidant defense mechanism. The translocation of bacteria and toxins through the leaky gut mucosa once again amplify or perpetuate systemic inflammation, thus leading to MOF syndrome and death in critically ill patients. Asystole developed in 4 of 34 patients with primary ACS after abdominal decompression. A hyperventilation occurs because of the increase of pulmonary and chest wall compliance if appropriate measures are not taken. The central venous pressure, pulmonary artery occlusion pressure, mean arterial pressure, and systematic vascular resistance decrease. Diuresis is usually observed after decompression, and the CO is increased.

The current recommendation is to preload the patient with volume before surgery by infusing 2l of 0.45% saline with mannitol and bicarbonate added and to continue infusing during surgery. Some encouraging reports point out the role of octreotide (OCT), a synthetic

somatostatin analog, for improving reperfusion-induced damage.

Some authors propose noninvasive continuous negative extra-abdominal pressure (NEXAP) for decreasing IAH in ACS. In patients with cirrhosis, ascites and chronic IAH laparocentesis is successfully applied. The removal of ascites is associated with a dramatic improvement in renal function, cardiac performance, and hepatic perfusion.²⁰ Unfortunately, a significant percentage of intensivists may be unaware of current approaches to ACS management, including monitoring bladder pressure and decompression laparotomy.

There are basically four different types of advanced treatment techniques for the management of ACS after the decompression operation:

1. Laparostomy
2. On-demand relaparotomy (wait-and-see tactics)
3. Planned relaparotomy (etappenlavage)
4. Stage abdominal repair (STAR)

Laparostomy

Laparostomy or open abdominostomy is generally defined as a laparotomy without approximation and suturing of the abdominal fascia. The method allows regular inspections of the bowel and drainage of intra-abdominal collections. It is used for prevention of pending secondary, and treatment of developed primary ACS.⁶³ Classically the laparostomies at the initial operation are left open and packed with saline-soaked packs. These are changed at regular intervals either in the ICU, the operating theatre, or the wards. The wounds are left open to heal by secondary intention, and the complete closure can take several months.

The principles of leaving the abdominal wall open are threefold. Firstly, it helps when making a re-entry into the abdomen. Re-opening a closed abdomen under these circumstances can be difficult and can cause iatrogenic bowel injuries, which can lead to further complications. Secondly, leaving the entire abdominal cavity open allows effective drainage of the intra-abdominal sepsis. More importantly, it helps in inspecting the abdominal cavity for any new collections that can be

effectively drained. It also gives an opportunity to inspect the anastomotic sites for any leaks. The aim is to eradicate intra-abdominal infection and to minimize or prevent systematic inflammatory response syndrome (SIRS). Lastly, the risk of development of raised intra-abdominal pressure and secondary or recurrent ACS is virtually eliminated. According to some authors the disadvantages of a laparostomy are the following¹⁰:

1. Massive fluid losses that cannot be quantified. This can make fluid management challenging
2. Evisceration of intra-abdominal contents
3. Contamination with exogenous organisms
4. Fistula formation
5. Postoperative abdominal wall hernias
6. Small bowel obstruction. This latter complication can occur if the bowel loop is caught in the healing cicatrix
7. Temperature losses (hypothermia)

Some new studies emphasize the fact that the open abdomen represents a significant source of protein/nitrogen loss, so 2 g of nitrogen per liter of abdominal fluid output should be included in the nitrogen balance calculation of any patient with open abdomen.

Abdominal decompression and delayed fascial closure (laparostomy) initially decreases patient perception of physical, social, and emotional health, but subsequent abdominal wall reconstruction restores physical and mental health.⁵⁸

On-Demand (Wait-and-See) Tactics

A relaparotomy is dictated by the patient's clinical condition, and is the technique by which most patients are managed. The mortality of the method is 30%–76%. A shortcoming of the on-demand tactics is that the fascial closure after the decompressive operation causes a significant elevation of IAP and risk of secondary ACS. Also, waiting for proofs for developed ACS and MOF, the surgeon can miss the right moment for revision of the abdomen.

Planned Relaparotomy (Etappenlavage)

A planned relaparotomy constitutes the cornerstone of aggressive surgical management of patients with severe secondary peritonitis and ACS. It involves making a conscious decision by the surgeon at the time of primary operation to carry out a re-exploration 24–72 h later, irrespective of the patient's clinical condition. In Germany the same method is called etappenlavage and the purpose is to evacuate all septic collections, to debride or even resect, until the surgeon decides to discontinue the re-exploration because the peritoneal cavity has become macroscopically clean. This also provides an opportunity to detect and treat any intra-

abdominal complications (collections, anastomotic leaks) at an early stage.

Staged Abdominal Repair (STAR)

Staged abdominal repair combines the principles of planned re-laparotomy and laparostomy. The technique consists of a chain of operations, planned either before or during the first index operation, performed every 24–48 h, regardless of the patient's clinical condition, with temporary abdominal closure, and the last operation being repair of the obligatory abdominal wall hernia. In theory it allows superior control of peritoneal contamination and earlier detection of anastomotic leaks. During the course of treatment, a controlled tension is exerted on the margins, thereby avoiding a secondary ACS. For the prevention and therapy of ACS, STAR operation is gaining greater popularity because of its lower complication rate.

According to some authors STAR appears to reduce mortality by 50% over the standard operations and the patients operated at 24-h intervals seem to do better than those with wider intervals of time. Staged abdominal repair facilitates second look, stabilization of the patient's general condition, decompresses the abdomen, and helps in organ recompensation. Some authors do not find any difference in the results when comparing a cohort of patients treated with a laparostomy with another that underwent STAR operations.

The continuous postoperative peritoneal lavage and relaparotomy on demand do not seem to prevent residual or recurrent intra-abdominal infections and are associated with high mortality. The planned relaparotomy seems to decrease the rate of residual peritoneal infection but has a high complication rate. It may be concluded that the ideal operative approach for patients with abdominal infections and ACS has not yet been established.

Temporary Abdominal Closure (TAC)

In view of the relatively high complications with laparostomies, some evolutionary tactics were introduced. Temporary abdominal closure devices were used instead of leaving the abdominal wound open. The method is also called semi-open and is considered as a modification of the open method. Different centers use a variety of materials for this purpose. Two types of materials are mainly used for TAC:

Mesh

Mesh has been used to cover the abdominal wound, without increasing the intra-abdominal pressure. Such mesh is made from absorbable and nonabsorbable materials. The problems with nonabsorbable mesh are that they can lead to the development of intestinal fis-

tulas, though the latter occur less frequently compared to the open method. In addition, such mesh sometimes has to be removed due to problems with persisting wound infection. Because of these complications, absorbable mesh has been introduced. The main problems with such type of mesh are that they cannot be used if re-explorations are required, as it is very difficult to remove them. They can also disintegrate which could lead to evisceration.

Plastic Bags

The other common material used for the semi-open method is the 1 or 3 liter urological bag, which is used to cover the abdominal wound. The sterile plastic bag is easily and rapidly fixed using a continuous suture and permits gradually bringing the wound margins closer to make final closure easier. When re-exploration is considered, the bag can just be slit in the middle and closed back instead of removing the bag and putting in a new one. The main advantage of this over the mesh is that it does not stick to the underlying structures. It also prevents heat and fluid losses as it is waterproof. As it is never removed before a definite closure of the abdomen, it does not damage the abdominal wall. In addition, there is no risk of wound dehiscence and enteric fistula, and the final closure of the abdomen, when re-exploration is no longer needed, is thus usually possible. Moreover, such a bag is considerably cheaper than the mesh.

Virtually all materials which are not reactive to the body tissues can be used for temporary abdominal closure. Currently, no prospective studies showing which is the best method or material for TAC have been published. Superiority of one over the other material has not been established. All have the common goal of preventing evisceration, allowing enlargement of the abdominal cavity, keeping the IAP low and preventing recurrence of IAH and ACS. All have the potential problems of secondary bacterial infection of the peritoneal cavity, fluid shifts from the exposed bowel and peritoneum, and injury to the bowel with subsequent formation of fistulae. Skin-only closure might be used as a short-term application (e.g., when damage control closure is needed). The use of nonadhesive plastic foil derived from the irrigation bag (Bogota bag) is quite satisfactory. It is named after its first description by Londoni in Bogota, Colombia.⁶¹ The Bogota bag is cheap and transparent, so that the abdominal content can be viewed through the plastic. This is very useful in cases with ongoing ischemia and bowel necrosis and hemorrhage. Some authors have just inserted a plastic bag (Bogota) between intra-abdominal organs and the abdominal wall, instead of suturing the bag to the edge of the open abdominal wall, and have reported better results. Some have also applied the so-called nonopera-

tive progressive Bogota bag. The Bogota bag silo provides space for protruding bowels in pending or manifest ACS. Other materials and techniques used for TAC are zipper, adhesive sheets, a subcutaneous polyethylene bag which the authors claim to be safer compared with the so-called Bogota bag, fenestrated polyethylene, sandwich vacuum pack technique for the treatment and prevention of post-traumatic ACS, and the similar "Vac Pac," Goretex dual mesh, burr closure consisting of two adherent sheets of knitted synthetic fibers, with excellent results in 200 cases,²⁰ absorbable mesh prosthesis, human acellular dermis and bipedicle flaps, silicone sheeting, corset-type closure for wounds with widely distracted edges in pediatric patients, topical negative pressure therapy with an abdominal dressing in management of laparostomies, vacuum-assisted closure using wall suction devices, which apply subatmospheric pressure, reducing bowel edema, bacterial counts, and inflammatory substances found in open abdominal wounds while eliminating the problem of "wet bed" and the need for frequent dressing changes, maintaining the skin intact, and improving fluid management.

Despite the bowel edema and IAH related to the ACS, early enteral feeding is feasible after definitive abdominal closure, while before that the enteric feeding may prolong closure. Skin closure alone may produce IAP of 50 mmHg or more, depending on intra-abdominal status. The term for definitive closure of the abdomen differs according to various indices—the level of IA, intra-peritoneal findings and also the existence and level of organ failure—and so do the methods, the best results having been gained when a multidisciplinary approach was applied with the participation of a plastic surgeon in the team. According to Canadian sources, the surgeons there have no consensus regarding the definition, functional indications, or management of an open abdomen, though they are familiar with ACS.

Prognosis and Conclusions

The single major event resulting in the reduction of abdominal infection mortality from 90% to 40% was the introduction of surgical treatment 80 years ago. Mortality due to ACS is higher than in non-ACS patients. Despite timely and adequate treatment it remains high, ranging from 38%–71%, 63%–72%, and 61.1%, and it is even 40% in those adequately treated, 47% even after decompression, and 49.2% after laparotomy, the data including English publications in the period 1972–2004;⁶⁴ it exceeds 60%–65%, using a mesh for TAC 51% vs. 70% after a primary abdominal closure after repair of rAAA, and 67% in secondary ACS in the absence of abdominal injury. Some data show 0% mortality in a planned relaparotomy group for ACS vs 64% mortality in the on-

demand (wait-and-see) group. For each increase by 5 points in APACHE II score, the relative risk of dying increases for 24%. Patients with APACHE II of 21 ± 7 showed mortality of 32%. Contributing to these results is not only the ACS itself, but also the main diagnosis, thus leading to ACS-ileus, peritonitis, pancreatitis, trauma, etc.

Despite the early recognition of ACS and quick and adequate management, the incidence of multiorgan dysfunction and mortality remain high. Abdominal compartment syndrome should thus be suspected in all critically ill patients, particularly those on ventilatory support in ICU who are hemodynamically not improving in spite of adequate resuscitation. Abdominal compartment syndrome is a distinct clinical entity developing in the wake of a serious insult to the body affecting multiple organs in a progressive manner. The classical clinical causes remain major trauma, massive fluid resuscitation, and prolonged operation. The gut mucosa is affected first and gut ischemia develops before renal and cardiopulmonary derangements. Abdominal compartment syndrome should therefore be highly suspected in cases with:

1. Abdominal distention, tenderness, lack of peristalsis
2. Oligoanuria
3. Respiratory failure with high inspiratory airway pressure in the mechanically ventilated patient
4. Progressive development of MOF

The diagnosis should be confirmed by IAP measurements. An adequate fluid administration is essential. The supranormal resuscitation aiming at an excessive preload to improve cardiac function is harmful in patients with already elevated IAP and excessive risk for ACS. The method of choice for treatment or prevention of ACS is decompressive laparotomy, finishing with TAC. Monitoring of IAP is necessary in the postoperative period, and planned revisions of the abdominal cavity are required. It is time therefore to accept and promulgate the existence of the ACS.⁶⁶

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