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Abdominal compartment syndrome

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Abstract

The abdominal compartment syndrome (ACS) was first described in surgical patients with abdominal aortic aneurysm repair, trauma, bleeding, or infection, but in recent years it has also been described in patients with other pathologies such as burn injury and sepsis and in medical patients. This F1000 Medicine Report is intended to provide critical care physicians a clear insight into the current state of knowledge regarding intra-abdominal hypertension (IAH) and ACS, and will focus primarily on the recent literature as well as on the definitions and recommendations published by the World Society of the Abdominal Compartment Syndrome. The definitions regarding increased intra-abdominal pressure (IAP) will be listed, followed by a brief but comprehensive overview of the different mechanisms of organ dysfunction associated with IAH. The gold standard measurement technique for IAP as well as recommendations for organ function support in patients with IAH and options for medical and surgical treatment of IAH and ACS will be discussed.

Introduction and context

A compartment syndrome exists when the increased pressure in a closed anatomic space threatens the viability of surrounding tissue. When this occurs in the abdomen, the impact on end-organ function within and outside the cavity can be devastating. The abdominal compartment syndrome (ACS) is not a disease; as such, it can have many causes and can develop within many disease processes. It is only recently that ACS has received heightened attention [1]. The prevention of intra-abdominal hypertension (IAH) and ACS is of tremendous importance in the care of critically ill, surgical, and trauma patients. Serial intra-abdominal pressure (IAP) measurements are essential to the diagnosis, management, and fluid resuscitation of these patients. Clinical examination is not an accurate method to estimate IAP [2]. Intravesicular pressure is a good estimate for IAP, is easily measured, and should be monitored in all patients believed to be at risk for significant elevations in IAP [3-6].

The management of IAH is based on four principles [3-6] (Figure 1): (a) serial monitoring of IAP; (b) optimization of systemic perfusion and organ function in the patient with elevated IAP; (c) institution of specific medical procedures to reduce IAP and the end-organ consequences of IAH/ACS; (d) prompt surgical decompression for refractory IAH.

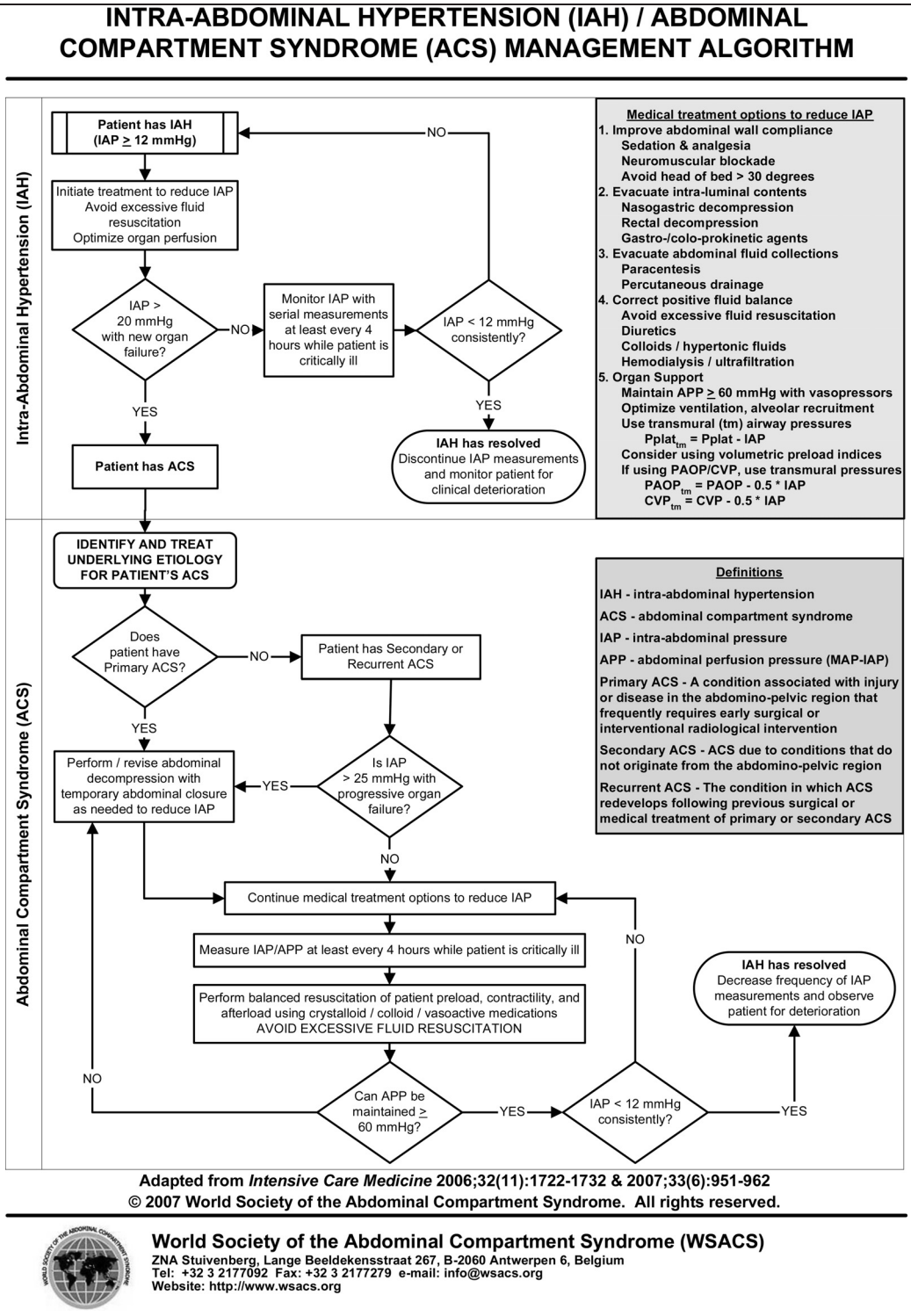
Recent advances

Recently, the World Society of the Abdominal Compartment Syndrome (WSACS) [7] published the consensus definitions and recommendations regarding the diagnosis and management of IAH and ACS [4,5].

Consensus definitions

Intra-abdominal pressure: IAP is the steady-state pressure concealed within the abdominal cavity. It is directly affected by the volume of the solid organs or hollow viscera (which may be either empty or filled with air, liquid, or fecal matter), the presence of ascites, blood or other space-occupying lesions (such as tumors or a gravid uterus), and the presence of conditions that limit expansion of the abdominal wall (such as burn eschars or third-space edema). The respiratory variation seen in the IAP tracing is an indirect measurement of abdominal wall compliance [8].

Figure 1. Intra-abdominal hypertension/abdominal compartment syndrome management algorithm of the World Society of the Abdominal Compartment Syndrome



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ACS, abdominal compartment syndrome; APP, abdominal perfusion pressure; CVP, central venous pressure; CVP_{tm}, transmural central venous pressure; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; MAP, mean arterial pressure; PAOP, pulmonary artery occlusion pressure; $PAOP_{tm}$, transmural pulmonary artery occlusion pressure; Pplat, plateau pressure; $Pplat_{tm}$, transmural plateau pressure. Adapted from [4,5]. Copyright 2006, 2007, Springer Berlin/Heidelberg.

Perfusion: Analogous to the widely accepted and clinically used concept of cerebral perfusion pressure [calculated as mean arterial pressure (MAP) minus intracranial pressure (ICP)], abdominal perfusion pressure (calculated as MAP minus IAP) has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation [9]. Although patients with IAH need to be well resuscitated, there is a lot of debate regarding futile crystalloid over-resuscitation and the development of secondary IAH [10].

Measurement: The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 20-25 mL of sterile saline. The IAP should be expressed in millimeters of mercury (mm Hg) and measured at end-expiration in the complete supine position after the clinician ensures that abdominal muscle contractions are absent and that the transducer is zeroed at the level of the midaxillary line. Recent studies have examined the effect of different zero reference and head-of-bed positions on IAP [11,12].

Normal values: Normal IAP is approximately 5-7 mm Hg in critically ill adults. IAP may be increased to 12-15 mm Hg in postoperative patients. Chronic IAP elevations can be seen in liver cirrhosis with ascites, large ovarian tumors, pregnancy, chronic ambulatory peritoneal dialysis (CAPD), or obesity.

Intra-abdominal hypertension: IAH is defined as a sustained or repeated pathologic elevation of IAP of greater than 12 mm Hg [4]. IAH is graded as follows: grade I, IAH of 12-15 mm Hg; grade II, IAH of 16-20 mm Hg; grade III, IAH of 21-25 mm Hg; and grade IV, IAH of greater than 25 mm Hg. It should be noted that the IAP ranges associated with these grades have been revised downward in recent years as the detrimental impact of elevated IAP on end-organ function has been recognized. Table 1 lists some risk factors for the development of IAH.

Abdominal compartment syndrome: ACS is defined as a sustained IAP of at least 20 mm Hg that is associated with new organ dysfunction/failure.

The WSACS suggests the following classification for IAH: Primary IAH is a condition associated with injury or disease in the abdomino-pelvic region which frequently requires early surgical or radiological intervention. Secondary IAH is a condition that does not originate from the abdomino-pelvic region. Recurrent IAH is a condition in which IAH/ACS redevelops following previous surgical or medical treatment of primary or secondary IAH/ACS.

Table 1. Risk factors for the development of intra-abdominal hypertension and abdominal compartment syndrome

A. Related to diminished abdominal wall compliance
- Mechanical ventilation, especially fighting with the ventilator and the use of accessory muscles
- Use of positive end-expiratory pressure (PEEP) or the presence of auto-PEEP
- Basal pleuropneumonia
- High body mass index
- Pneumoperitoneum
- Abdominal (vascular) surgery, especially with tight abdominal closures
- Pneumatic anti-shock garments
- Prone and other body positioning
- Abdominal wall bleeding or rectus sheath hematomas
- Correction of large hernias, gastroschisis, or omphalocoele
- Burns with abdominal eschars
B. Related to increased intra-abdominal contents
- Gastroparesis
- Gastric distention
- Ileus
- Volvulus
- Colonic pseudo-obstruction
- Abdominal tumor
- Retroperitoneal/abdominal wall hematoma
- Enteral feeding
- Intra-abdominal or retroperitoneal tumor
- Damage control laparotomy
C. Related to abdominal collections of fluid, air, or blood
- Liver dysfunction with ascites
- Abdominal infection (pancreatitis, peritonitis, abscess, and so on)
- Hemoperitoneum
- Pneumoperitoneum
- Laparoscopy with excessive inflation pressures
- Major trauma
- Peritoneal dialysis
D. Related to capillary leak and fluid resuscitation
- Acidosis[a] (pH <7.2)
- Hypothermia[a] (core temperature <33°C)
- Coagulopathy[a] (platelet count <50,000/mm^3 OR an activated partial thromboplastin time more than two times normal OR a prothrombin time <50% OR an international standardised ratio >1.5)
- Polytransfusion/trauma (>10 units of packed red cells per 24 hours)
- Sepsis (as defined by the American-European Consensus Conference definitions)
- Severe sepsis or bacteremia
- Septic shock
- Massive fluid resuscitation (>5 L of colloid or >10 L of crystalloid per 24 hours with capillary leak and positive fluid balance)
- Major burns

[a] The combination of acidosis, hypothermia, and coagulopathy has been forwarded in the literature as the deadly triad leading to abdominal compartment syndrome.

Further classification can be done in relation to the time course of the events: chronic IAH (occurring over the course of months to years) as seen with morbid obesity, intra-abdominal tumor (large ovarian cyst, fibroma, and so on), chronic ascites (liver cirrhosis or CAPD), or pregnancy; in those cases, the abdominal wall adapts progressively during months or years to the increase in IAP and allows time for the body to adapt. Acute IAH is due mainly to trauma or intra-abdominal hemorrhage of any cause and leads to ACS within hours. Subacute IAH is seen with most medical cases in the intensive care unit that lead to IAH within days and results from a

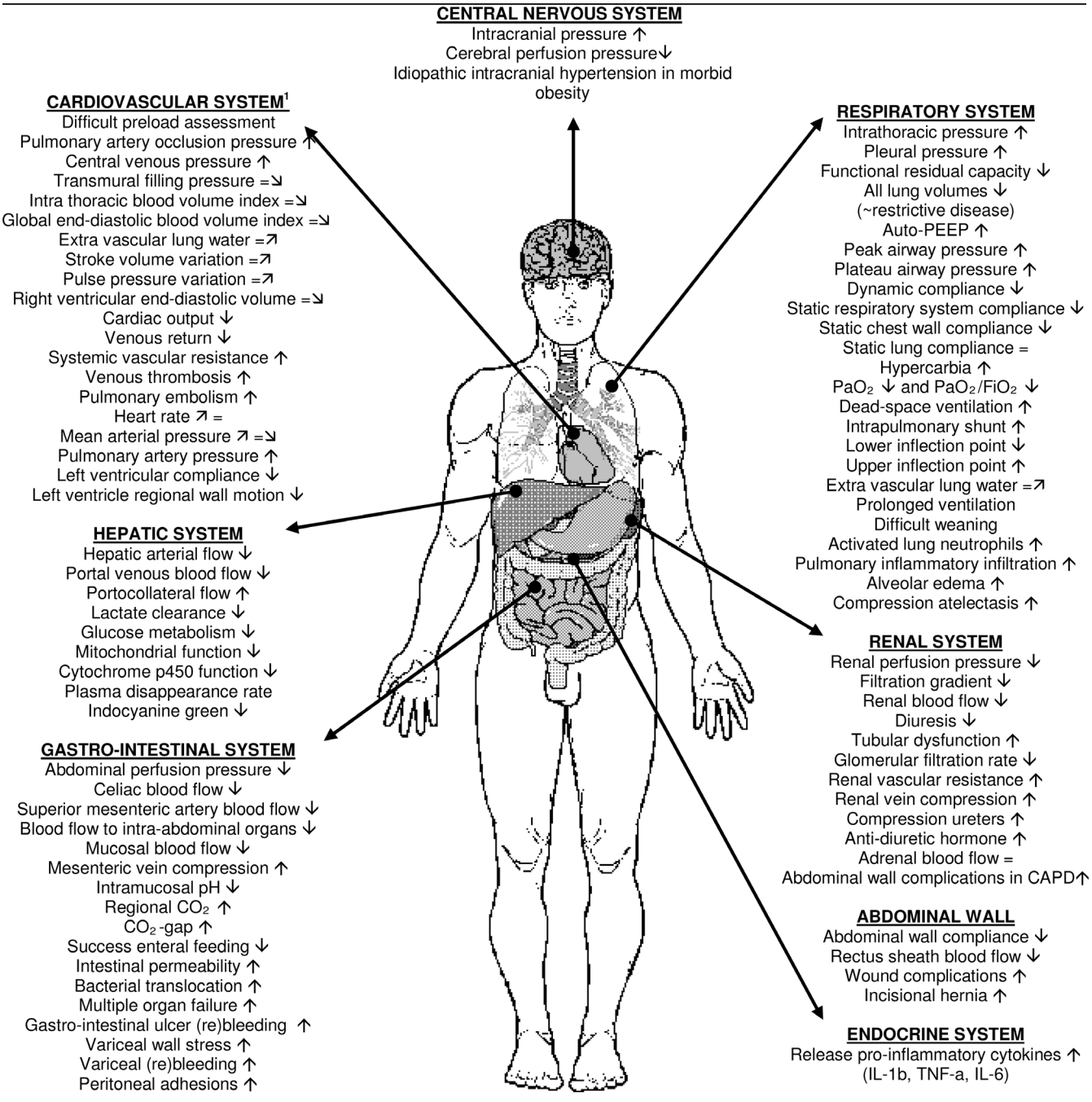
combination of etiologic factors and predisposing conditions. Hyperacute IAH lasts for only a very short period (seconds or minutes) as with laughing, straining, coughing, sneezing, defecating, or performing physical activity and has no clinical implications.

Implications for clinical practice

Organ support

Figure 2 lists the effects of IAH/ACS on end-organ function [13]. In the paragraphs below, some key points to remember are listed [14].

Figure 2. Impact of increased intra-abdominal pressure on end-organ function



[1]Cardiovascular effects are exacerbated in case of hypovolemia, hemorrhage, ischemia, and ventilation with high positive end-expiratory pressure (PEEP). CAPD, continuous ambulatory peritoneal dialysis; FiO_2, fraction of inspired oxygen; IL, interleukin; PaO_2, arterial partial pressure of oxygen; TNF-a, tumor necrosis factor-alpha.

For the brain [15-17]: IAH/ACS will increase ICP and decrease cerebral perfusion pressure. Because of the interactions between IAP, intrathoracic pressure (ITP), and ICP, accurate monitoring of IAP in head trauma victims with associated abdominal lesions is worthwhile. The presence of increased IAP can be an additional 'extracranial' cause of intracranial hypertension (ICH) in patients with abdominal trauma without overt craniocerebral lesions. Laparoscopy should be avoided in patients with ICH. Obesity causes idiopathic ICH, and weight loss after bariatric surgery is associated with improvements in ICP and symptoms. The direct effects of IAH on neurologic function can be ablated by sternotomy, pericardiotomy, or bilateral pleurotomy [18]. Recently, IAH has been related to ventriculoperitoneal shunt dysfunction [19].

For the heart [20-23]: The clinician must be aware of the polycompartment syndrome and the interactions between ITP, IAP, positive end-expiratory pressure (PEEP), and intracardiac filling pressures. Use transmural filling pressures as a preload estimate, calculated as the end-expiration value minus the ITP: transmural central venous pressure (CVP_{tm}) = end-expiration central venous pressure (CVP_{ee}) – ITP, or transmural pulmonary artery occlusion pressure ($PAOP_{tm}$) = end-expiration pulmonary artery occlusion pressure ($PAOP_{ee}$) – ITP. The average abdomino-thoracic transmission is around 50%. A quick estimate of transmural filling pressures can also be obtained by subtracting half of the IAP from the end-expiratory filling pressure, or $CVP_{tm} = CVP_{ee} – ½$ IAP and $PAOP_{tm} = PAOP_{ee} – ½$ IAP. Maybe the Surviving Sepsis Campaign guidelines for initial and ongoing resuscitation should be guided toward a CVP_{tm} of 8-12 mm Hg and a transmural MAP of 65 mm Hg to avoid unnecessary over- and under-resuscitation. Use volumetric estimates of preload status to take into account the changing ventricular compliance and elevated ITP: right ventricular end-diastolic volume index or global end-diastolic volume index. Functional hemodynamic parameters such as stroke volume variation or pulse pressure variation, but not systolic pressure variation, should be used to assess volume responsiveness.

For the lungs [24-26]: As a rule of thumb, the best PEEP may be set to counteract IAP whilst at the same time avoiding overinflation of already well-aerated lung regions. The best PEEP (cm H_2O) = IAP (mm Hg) to a maximum value of 20. This holds especially true in the setting of secondary acute respiratory distress syndrome related to abdominal problems (with IAH). In case of ACS with an IAP of greater than 20 mm Hg, all medical and surgical options to lower IAP and restore end-organ perfusion should be performed. Plateau pressures (Pplat) should be limited to transmural plateau pressures ($Pplat_{tm}$) of below 35 cm H_2O: $Pplat_{tm}$ = Pplat – ½ IAP. IAH will lead to pulmonary hypertension and increases lung edema in case of capillary leak and fluid overload. Therefore, monitoring extravascular lung water index may be worthwhile.

For the liver [27,28]: Monitor noninvasive hepatosplanchnic perfusion during IAH. Indocyanine green plasma disappearance rate decreases during IAH [29].

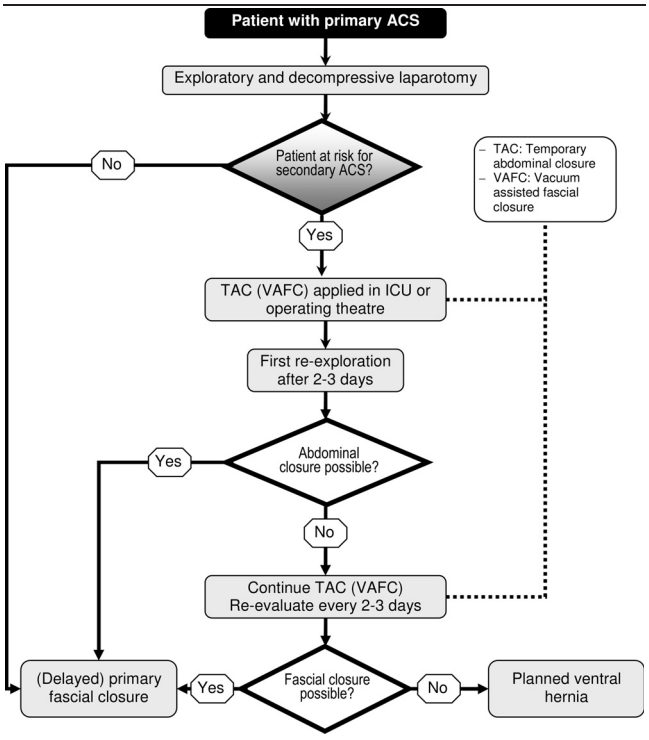
For the kidneys [30-32]: Elevated IAP significantly decreases renal artery blood flow and compresses the renal vein, leading to renal dysfunction and failure [33]. Oliguria develops at an IAP of 15 mm Hg and anuria at 30 mm Hg in the presence of normovolemia and at lower levels of IAP in the patient with hypovolemia or sepsis. Renal perfusion pressure (RPP) and renal filtration gradient have been proposed as key factors in the development of IAP-induced renal failure (RPP = MAP – IAP).

Changes in IAP have a greater impact upon renal function and urine production than do changes in

Table 2. Medical treatment options for abdominal compartment syndrome

1. Improvement of abdominal wall compliance
- Sedation and pain relief (not fentanyl)
- Neuromuscular blockade
- Body positioning (avoid upright, use anti-Trendelenburg)
- Negative fluid balance
- Skin pressure decreasing interfaces
- Weight loss
- Percutaneous/endoscopic abdominal wall component separation
2. Evacuation of intraluminal contents
- Gastric tube and suctioning
- Gastroprokinetics (erythromycin, cisapride, metoclopramide)
- Rectal tube and enemas
- Colonoprokinetics (neostygmine, prostygmine bolus, or infusion)
- Endoscopic decompression of large bowel
- Colostomy or ileostomy
3. Evacuation of peri-intestinal and abdominal fluids
- Paracenthesis or ascites evacuation
- Computed tomography (CT)- or ultrasound-guided aspiration of abscess
- CT- or ultrasound-guided aspiration of hematoma
- Percutaneous drainage of (blood/fluid) collections
4. Correction of capillary leak and positive fluid balance
- Albumin 20% in combination with diuretics (furosemide)
- Correction of capillary leak (antibiotics, source control, and so on)
- Colloids instead of cristalloids
- Dobutamine (not dopamine)
- Dialysis or continuous venovenous hemofiltration with ultrafiltration
- Ascorbinic acid in burn patients
5. Specific therapeutic interventions
- Continuous negative abdominal pressure
- Negative external abdominal pressure
- Targeted perfusion pressure
• Target abdominal perfusion pressure >60 mm Hg

Figure 3. Surgical treatment algorithm for the patient with abdominal compartment syndrome (ACS)



ICU, intensive care unit; TAC, temporary abdominal closure; VAFC, vacuum-assisted fascial closure.

MAP. It should not be surprising, therefore, that decreased renal function, as evidenced by the development of oliguria, is one of the first visible signs of IAH. Therefore, it behooves us as clinicians to be cognizant of the elevated IAP, and its effect on renal function is often the first sign of impending ACS. Recently, Dalfino and colleagues [32] became the first to report a relation between RIFLE (Risk, Injury, Failure, Loss, and End-stage kidney disease) criteria for acute kidney injury and IAH.

Nonsurgical treatment

Before surgical decompression is considered, less invasive medical treatment options should be optimized. The relation between abdominal contents and IAP is not linear but exponential, and this curve is shifted to the left and upwards when abdominal wall compliance is decreased. Therefore, IAH can be treated by improving abdominal wall compliance and decreasing intra-abdominal volume or both. Different medical treatment options have been suggested to decrease IAP; however, it must be noted that, as of today, the evidence regarding these recommendations is scarce and mostly based on case reports and small cohort studies, whereas some evidence is based on speculation and non-evidence-based methods used in ileus and bowel obstruction.

Medical treatment is based on five different mechanisms: (a) improvement of abdominal wall compliance; (b) evacuation of intraluminal contents; (c) evacuation of abdominal fluid collections; (d) correction of capillary leak and positive fluid balance; and (e) specific treatments (see Table 2 for more details).

Surgical treatment

Decompressive laparotomy is the definitive treatment for ACS [34]. The technique can be invasive (midline laparotomy) or minimally invasive (endoscopic techniques based on subcutaneous anterior abdominal fasciotomy). This intervention results in a laparostomy or open abdomen so that a temporary abdominal closure is needed (moist gauze, towel clip closure, Bogota bag, Wittman patch or zipper, home-made system, or vacuum-assisted closure). Figure 3 shows a surgical treatment algorithm.

Abbreviations

ACS, abdominal compartment syndrome; CAPD, chronic ambulatory peritoneal dialysis; CVP_{ee}, end-expiration central venous pressure; CVP_{tm}, transmural central venous pressure; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; ICH, intracranial hypertension; ICP, intracranial pressure; ITP, intrathoracic pressure; MAP, mean arterial pressure; $PAOP_{ee}$, end-expiration pulmonary artery occlusion pressure; $PAOP_{tm}$, transmural pulmonary artery occlusion pressure; PEEP, positive end-expiratory pressure; Pplat, plateau pressures; $Pplat_{tm}$, transmural plateau pressure; RIFLE, Risk, Injury, Failure, Loss, and End-stage kidney disease; RPP, renal perfusion pressure; WSACS, World Society of the Abdominal Compartment Syndrome.

Competing interests

MM is a past president of the World Society of the Abdominal Compartment Syndrome, is a member of the medical advisory boards of Holtech Medical (Charlottenlund, Denmark), Spiegelberg (Hamburg, Germany), and Pulsion Medical Systems (Munich, Germany), has received royalties from Holtech Medical and Spiegelberg, and holds a patent with Pulsion Medical Systems.

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