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CURRENT CONCEPTS IN CLINICAL SURGERY

Current insights in intra-abdominal hypertension and abdominal compartment syndrome: open the abdomen and keep it open!

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Abstract
Background and aims The abdominal compartment syndrome (ACS) is associated with organ dysfunction and mortality in critically ill patients. Furthermore, the deleterious effects of increased IAP have been shown to occur at levels of intra-abdominal pressure (IAP) previously deemed to be safe. The aim of this article is to provide an overview of all aspects of this underrecognized pathological syndrome for surgeons.
Methods and contents This review article will focus primarily on the recent literature on ACS as well as the definitions and recommendations published by the World Society for the Abdominal Compartment Syndrome. The definitions regarding increased IAP will be listed, followed by a brief but comprehensive overview of the different mechanisms of organ dysfunction associated with intra-abdominal hypertension (IAH). Measurement techniques for IAP will be discussed, as well as recommendations for organ function support in patients with IAH. Finally, surgical treatment and management of the open abdomen are briefly discussed, as well as some minimally invasive techniques to decrease IAP.
Conclusions The ACS was first described in surgical patients with abdominal trauma, bleeding, or infection, but in recent years ACS has also been described in patients with other pathologies such as burn injury and sepsis. Some of these so-called nonsurgical patients will require surgery to treat their ACS. This review article is intended to provide surgeons with a clear insight into the current state of knowledge regarding IAH, ACS, and the impact of IAP on the critically ill patient.

Keywords Abdominal pressure · Abdominal hypertension · Abdominal compartment syndrome · Diagnosis · Pathophysiology · Treatment

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Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are increasingly described, first in trauma patients and later in several populations of critically ill or injured patients, and have been independently associated with organ dysfunction and mortality [1, 2]. Although this syndrome was first described in the Middle Ages, scientific interest has remained low during history until the last decade, which has brought a renewed interest, exemplified by an exponential increase in publications on this subject [3]. In an attempt to bring all physicians and other health care workers who are confronted on a regular basis with the adverse effects of IAH together, the World Society for the Abdominal Compartment Syndrome (WSACS; website: www.wsacs.org) was founded. This society has held three congresses so far to discuss the results of the latest advances in this field. This article intends

to give a state-of-the-art update on different aspects of IAH and ACS such as definitions, epidemiology and etiology, intra-abdominal pressure (IAP) measurement, IAH and organ dysfunction, and therapeutic options for IAH and ACS.

Definitions

The results of the 2004 consensus conference of the WSACS held in Noosa, Australia, were published in 2006, and contain a set of definitions related to IAH and ACS [4]. The definitions are listed in Table 1. These definitions are based on the best available scientific data today, but they are likely to undergo some minor changes in the future.

IAH is defined by a sustained or repeated pathologic elevation of IAP≥12 mmHg and ACS is defined as a sustained IAP>20 mmHg that is associated with new organ dysfunction or failure. ACS can be classified into primary, secondary, and recurrent ACS according to the presence of an intra-abdominal cause of elevated IAP. "Normal" IAP is variable. In the strict sense, it is lower than 5 mmHg in adults under resting conditions [5]. However, in obese persons [6–8], in pregnant women, or in patients with chronic ascites, it can be higher, up to 10 mmHg or even 15 mmHg, without causing significant adverse effects, probably due to the chronic nature of the IAP increase with adaptation of the individual's physiology. In children, normal IAP is generally lower [9]. In general, IAP values must be interpreted relative to the individual patient's physiologic state.

Epidemiology and etiology

While first described in trauma patients or after abdominal surgery, epidemiologic studies have demonstrated that IAH–ACS is a frequent occurrence in the intensive care unit (ICU), even in medical ICU patients. Malbrain et al. [2] showed in a multicenter prevalence study including 59% medical and 41% surgical patients that IAH was present in 24% to 59% of patients and 4% to 8% of patients had ACS (respectively for mean and maximal IAP values). A second study by Malbrain et al. describing the incidence of IAH–ACS during the first 7 days after admission found that IAH [1] occurred in 26% to 57% and ACS in 3% to 15% of patients (respectively for mean and maximal IAP values). Nonsurvivors had a higher IAP on admission than survivors and the development of IAH during ICU stay was an independent predictor of mortality. The occurrence of IAH was also associated with higher degrees of organ dysfunction described by the Sequential Organ Function

Table 1 Consensus definitions regarding IAH and ACS (adapted from Malbrain et al. [4])

	Definition
Definition 1	IAP is the steady-state pressure concealed within the abdominal cavity
Definition 2	APP = MAP − IAP
Definition 3	FG = GFP − PTP = MAP – 2 × IAP
Definition 4	IAP should be expressed in millimeter of mercury and measured at end expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line
Definition 5	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline
Definition 6	Normal IAP is approximately 5–7 mmHg in critically ill adults
Definition 7	IAH is defined by a sustained or repeated pathologic elevation of IAP≥12 mmHg
Definition 8	IAH is graded as follows Grade I: IAP 12–15 mmHg Grade II: IAP 16–20 mmHg Grade III: IAP 21–25 mmHg Grade IV: IAP>25 mmHg
Definition 9	ACS is defined as a sustained IAP>20 mmHg (with or without an APP<60 mmHg) that is associated with new organ dysfunction–failure
Definition 10	Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention
Definition 11	Secondary ACS refers to conditions that do not originate from the abdominopelvic region
Definition 12	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

ACS Abdominal compartment syndrome, *APP* abdominal perfusion pressure, *FG* filtration gradient, *GFP* glomerular filtration pressure, *IAH* intra-abdominal hypertension, *IAP* intra-abdominal pressure, *MAP* mean arterial pressure, *PTP* proximal tubular pressure

Assessment score. In these and other studies, IAH–ACS has been associated with trauma, burn injury, sepsis, acute lung injury, and many other pathologic conditions.

The most obvious cause of intra-abdominal hypertension is increased volume in the abdominal space, either within the peritoneal cavity or in the retroperitoneum, but abdominal wall compliance is equally important. Similar to the situation in the brain, there are essentially two parts in the abdominal pressure–volume curve. When the abdominal wall is very compliant and at low intra-abdominal volumes, relatively large increases in volume will lead to minor changes in IAP only [10]. However, at higher volumes, the abdominal wall compliance decreases and small volume changes can lead to important increases in IAP. This means that a small increase in intra-abdominal volume can lead to clinically important effects on organ function but also that relatively small decreases in volume can lower IAP substantially, which offers options for treatment. This abdominal pressure–volume curve is shifted to the left in situations where the abdominal wall compliance is decreased due to hematoma, voluntary muscle activity, edema, or other factors. The occurrence of IAH is usually associated with a situation that causes increased abdominal volume, decreased abdominal compliance, and often a combination of both these factors. The WSACS published a list of risk factors associated with these situations [4]. They are summarized in Table 2.

IAH and ACS were first described in situations where the intra-abdominal content was acutely increased, namely after emergent surgery for abdominal trauma or ruptured aortic aneurysm. Increased volume of any intra-abdominal organ or structure can lead to IAH (e.g., liver hematoma, distended bowel or stomach, retroperitoneal hematoma or edema of the bowel wall) as well as peritoneal fluid collections such as ascites or postoperative bleeding. The abdominal wall compliance can be compromised, e.g., after hernia repair, in case of abdominal burns or eschars, or due to voluntary muscle contraction. These are often quite straightforward situations, but sometimes both mechanisms are involved, such as in patients after massive fluid resuscitation who develop both ascites and abdominal wall edema.

The mechanisms that link IAH with organ dysfunction are not yet completely understood. There is certainly a direct mechanical effect of the increased IAP on the blood supply of the intra-abdominal organs, which is most convincingly seen in the kidney [11, 12]. Some of the deleterious effects may be associated with direct compression of the organ involved and hormonal changes have been implicated as well [13]. The adverse effect of IAH on thoracic organs may be related to the cephalad displacement of the diaphragm. However, IAH also has an impact on distant organ function. Ischemia–reperfusion injury may be involved in this complex pathophysiology as a "second-hit" phenomenon after shock resuscitation [14, 15].

Table 2 Risk factors for the development of IAH and ACS [4]

Risk factors
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 or the presence of auto-PEEP
Basal pleuropneumonia
High body mass index
Pneumoperitoneum
Abdominal (vascular) surgery, especially with tight abdominal closures
Pneumatic antishock garments
Prone and other body positioning
Abdominal wall bleeding or rectus sheath hematomas
Correction of large hernias, gastroschisis, or omphalocele
Burns with abdominal eschars
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
Related to abdominal collections of fluid, air, or blood
Liver dysfunction with ascites
Abdominal infection (pancreatitis, peritonitis, abscess,...)
Hemoperitoneum
Pneumoperitoneum
Laparoscopy with excessive inflation pressures
Major trauma
Peritoneal dialysis
Related to capillary leak and fluid resuscitation
Acidosis* (pH below 7.2)
Hypothermia* (core temperature below 33°C)
Coagulopathy* (platelet count below 50,000 per cubic meter *or* an activated partial thromboplastin time more than two times normal OR a prothrombin time below 50% *or* an international standardized ratio more than 1.5)
Polytransfusion–trauma (>10 units of packed red cells per 24 h)
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 h with capillary leak and positive fluid balance)
Major burns

*In the literature, the combination of acidosis, hypothermia and coagulopathy have been referred to as the deadly triad leading to abdominal compartment syndrome

The mechanisms involved will be discussed in the section on the effect of IAH on the different organ systems.

IAP measurement

Surveys among clinicians show that many of them use clinical examination for the diagnosis of ACS, a practice which has repeatedly been shown to be unreliable with a sensitivity and positive predictive value of around 40–60% [16, 17]. The use of abdominal perimeter is equally inaccurate. Radiologic investigation with plain radiography of the chest or abdomen, abdominal ultrasound, or CT scan is also insensitive to the presence of increased IAP. However, they can be indicated to illustrate the cause of IAH (bleeding, hematoma, ascites, abscess...) and may offer clues for management (paracentesis, drainage of collections...).

The most important tool in establishing the diagnosis of IAH or ACS is IAP measurement [18]. Since the abdominal contents are primarily noncompressive in nature and predominantly fluid-based, they can be assumed to behave according to Pascal's law. Therefore, the IAP measured at one point can be assumed to be the pressure throughout the abdominal cavity. IAP increases with inspiration (due to downward displacement of the diaphragm) and decreases with expiration (due to diaphragmatic relaxation). IAP can be measured directly or indirectly, either intermittently or continuously. The most frequently used routes for indirect IAP measurement are the bladder and the stomach.

Direct IAP measurement

The use of abdominal drains for postoperative direct IAP measurement has recently been reported by Risin et al. [19]. In this study, 14 Fr polyvinyl chloride drains were connected to a pressure transducer, and IAP values obtained directly correlated well with transvesical-measured IAP. Unfortunately, no Bland and Altman statistics were performed. In another study, the same authors reported good correlation in patients undergoing laparoscopic procedures between the directly measured IAP and pressure obtained from the insufflator [20].

Indirect IAP measurement

Several routes have been proposed for indirect IAP measurement. All these methods are based on the principle that the abdominal cavity can be considered to be a closed box [4]. Therefore, the pressure measured at one point within this cavity is supposed to reflect the pressure throughout the cavity, as its contents behave according to Pascal's law. From this, it is assumed that IAP can be measured indirectly in all cavities within the abdomen.

Transvesical IAP measurement

The bladder has been studied and used most extensively to measure IAP. The technique described by Kron et al. [21] has been adopted over the years by Cheatham and Safcsak [22] and served as model for commercially available devices such as the Abviser (WolfeTory Medical, Salt Lake City, USA).

A manometer technique can also be used, which has first been described by Harrahill [23] in 1998. The patient's own urine is used as a transducing medium, and the height of the fluid column in the catheter reflects the IAP. Based on this technique, a commercially available device has been developed (FoleyManometer, Holtech, Copenhagen, Denmark) which offers the advantage that it can be used without a pressure transducer and monitor, i.e., outside the ICU [24, 25].

Using this technique, an IAP can be obtained at regular intervals, but it remains labor intensive, especially when hourly IAP measurements are needed.

Therefore, continuous IAP measurement techniques have been investigated. Balogh et al. [26] introduced a method for continuous IAP measurement using a three-way Foley catheter, which was found to perform excellent in ICU patients.

Pitfalls in transvesical IAP measurement

Instillation volume

Kron et al. used 50–100 mL for transvesical IAP measurement, and similar amounts have been used in clinical practice. Already in 1999, Johna et al. [27] reported a systematic overestimation of IAP using the transvesical route. Although IAP behaved differently in individual patients in this study, intrinsic detrusor muscle activity was suggested as a possible explanation for this observation. The same was also reported in animal experiments by Gudmundsson et al. [28], who also found that this effect was more prominent when IAP was high. These findings were confirmed in two studies in critically ill patients that demonstrated that increasing instillation volumes cause a progressive increase in measured transvesical pressure [29, 30]. In these studies, volumes as low as 10 and 25 mL of saline respectively resulted in reliable IAP measurement. Instillation volume should be no more than 25 mL and, probably, lower volumes can be used as was recently described [29, 30].

Intrapelvic mass effect

Hematomas or other intrapelvic masses may cause erroneous elevation of intrabladder pressure, in which case the intrabladder pressure does not reflect the IAP. The transvesical route should not be used in patients with known

intrapelvic masses, and in case erroneous IAP values are obtained from a transvesical IAP measurement method, an intrapelvic mass effect should be considered.

Transgastric IAP measurement

Transgastric measurement of IAP has been reported but, up to now, is not used frequently in clinical practice. Colllee et al. [31] used a fluid column in the nasogastric tube to measure IAP, but this technique has been replaced by the use of a balloon-tipped catheter [10, 32], which can be used in a continuous or semicontinuous fashion (Pulsion Medical Systems, Munich, Germany and Spiegelberg, Hamburg, Germany). However, experience in critically ill patients is limited, and the influence of intestinal peristalsis and enteral nutrition to name a few has not been studied so far. The transgastric route remains mainly an alternative when the introduction of a bladder catheter is contraindicated.

APP measurement

Analogous to the widely accepted and clinically utilized concept of cerebral perfusion pressure, calculated as mean arterial pressure (MAP) minus intracranial pressure (ICP), abdominal perfusion pressure (APP), calculated as MAP minus IAP, has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation [33–36]. APP, by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP), has been demonstrated to be statistically superior to either parameter alone in predicting patient survival from IAH and ACS [36]. A target APP of at least 60 mmHg has been demonstrated to correlate with improved survival from IAH and ACS.

Recommendations for IAP monitoring

Should I measure IAP in all patients?

Although the incidence of IAH in critically ill patients is considerable [1], routine IAP measurement in all patients admitted to the ICU is currently rarely performed and probably not indicated. The WSACS has provided a list with risk factors associated with IAH and ACS (Table 2) [4]; in patients with two or more risk factors, routine IAP monitoring is advised.

What technique should I use?

According to the WSACS consensus guidelines, IAP should be expressed in millimeter of mercury and measured at end expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line.

The technique used should be decided based on the indication (e.g., APP monitoring or screening) and the condition of the patient, the available monitoring equipment, and the experience of the nursing staff with regards to possible pitfalls related to the technique used.

In some patients, a continuous technique may be preferable, e.g., when the abdominal perfusion pressure is used as a resuscitation endpoint or in patients with impending ACS requiring urgent abdominal decompression. For most patients, however, an intermittent technique may be adequate.

The manometer techniques can be used without the need for additional electronic equipment, which also allows for IAP measurement in the general ward when IAH or ACS is suspected.

Preferably, a protocol describing a preferred method of IAP measurement, with details regarding the conditions in which it should be obtained, should be available in every ICU.

What frequency?

When an intermittent method is used, measurements should be obtained at least every 4 h, and in patients with evolving organ dysfunction, this frequency should be increased up to hourly measurements.

When should I stop IAP measurement?

IAP measurement can be discontinued when the patient has no signs of acute organ dysfunction, and IAP values have been below 10 mmHg for 24–48 h. In case of recurrent organ dysfunction, IAP measurement should be reconsidered.

IAH and organ dysfunction

The abdominal compartment syndrome is diagnosed when the IAP is greater than 20 mmHg along with evidence of new end-organ dysfunction [37]. Intra-abdominal hypertension is diagnosed at lower levels of IAP when the patient is at risk, but there is no evidence of organ dysfunction yet, although subtle forms of organ dysfunction may be present at levels of IAP previously deemed to be safe [4]. There probably is a “dose-dependent” association between IAP and organ dysfunction. IAH has been shown to have deleterious effects on organ function, both within and outside of the abdominal cavity. It is beyond the scope of this paper to give a complete overview of all pathophysiologic mechanisms involved. We have focused on those pathologic observations that

have direct implications on the clinical management of critically ill or injured patients.

Effect on the cardiovascular system

Multiple factors influence the complex interaction between IAP and the cardiovascular system [38]. First of all, due to the cranial movement of the diaphragm during IAH, the intrathoracic pressure increases during IAH. Animal and human experiments have shown that 20–80% of the IAP is transmitted to the thorax. This leads to compression of the heart and reduction of end diastolic volume. Secondly, the cardiac preload decreases due to decreased venous return from the abdomen and the systemic afterload is initially increased due to direct compression of vascular beds and activation of the renin–angiotensin–aldosterone pathway [39–42]. This leads to decreased cardiac output. Mean arterial blood pressure may initially rise due to shunting of blood away from the abdominal cavity but thereafter normalizes or decreases [36, 38]. The cardiovascular effects are aggravated by hypovolemia and the application of positive end expiratory pressure (PEEP) [43–47], whereas hypervolemia has a temporary protective effect [48].

A very important issue in the management of patients with IAH is the interpretation of hemodynamic monitoring parameters. Due to the abdominothoracic transmission of pressure, traditional filling pressures (central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP)) are falsely elevated in the presence of IAH and do not reflect true cardiac preload. Therefore, it may be more useful to use volumetric monitoring parameters such as right ventricular end diastolic volume index or global end diastolic volume index. They are especially useful because of the changing ventricular compliance and elevated intrathoracic pressure (ITP) [42, 49-52]. Preload responsiveness can be better evaluated using dynamic parameters such as pulse pressure variation (PPV) or stroke volume variation (SVV) [53–55]. However, they are only reliable in completely sedated patients who do not exhibit spontaneous breathing movements nor have cardiac arrhythmias. Also, Duperret et al. showed that SVV and PPV are (falsely?) increased in euvolemic pigs with IAH, which may compromise the use of these parameters in clinical practice. Since the authors did not subject the animals to a fluid challenge, they were unable to ascertain whether SVV and PPV still represented fluid responsiveness although the pigs were euvolemic prior to the IAH, since IAH in itself may induce hypovolemia and lead to increased SVV and PPV.

If volumetric or dynamic parameters are not available and filling pressures have to be used for hemodynamic monitoring, they should be corrected for intrathoracic pressure. This means that transmural CVP (CVP_{tm}) is equal to CVP minus ITP and $PAOP_{tm}$ = PAOP − ITP. Since the abdominothoracic transmission amounts to 20–80%, ITP can be assumed to be IAP/2.

This finding has important implications. The Surviving Sepsis Campaign guidelines targeting initial and ongoing resuscitation towards a CVP of 8 to 12 mmHg [56] should be interpreted and adjusted according to these findings. Moreover, targeting APP>60 mmHg rather than a MAP of 65 mmHg [57] may influence prognosis.

Effects of IAH on the respiratory system

The transmission of IAP to the thorax also has an impact on the respiratory system. Patients with primary ACS will often develop a secondary acute respiratory disease syndrome (ARDS) and may require a different ventilatory strategy and more specific treatment than a patient with primary ARDS [58, 59]. The major problem lays in the reduction of the functional residual capacity. Together with the alterations caused by ARDS, this will lead to the “baby lungs”. IAH decreases total respiratory system compliance by a decrease in chest wall compliance, while lung compliance remains virtually unchanged [60, 61]. Some recommendations can be made in terms of ventilation strategy for patients with IAH:

- Best PEEP should be set to counteract IAP while in the same time avoiding over-inflation of already well-aerated lung regions

  Best PEEP = IAP

- During lung protective ventilation, the plateau pressures should be limited to transmural plateau pressures below 35 cmH2O

  $Pplat_{tm}$ = Pplat − IAP/2

- Monitoring of extravascular lung water index seems warranted in risk patients since IAH is associated with increased risk of lung edema [62].

The effect of IAH on renal function

Renal dysfunction is one of the most consistently described organ dysfunctions associated with IAH. The etiology is multifactorial and offers a unique insight into the deleterious and sometimes cumulative effects of IAH on organ function.

The most important effect of IAH on the kidney is related to renal blood flow. IAH has been shown to lead to renal venous compression and increased renal venous pressure [63, 64]. Also, renal arterial blood flow and microcirculatory flow in the renal cortex are decreased. Direct compression of the renal cortex may be a contrib-

uting factor [63, 65]. The changes in renal blood flow lead to activation of the renin–angiotensin–aldosterone pathway and, also, ADH secretion is increased in IAH [66, 67]. The clinical importance of these hormonal changes is still unclear.

Biancofiore and Sugrue and their colleagues [11, 12, 68-70] showed that renal dysfunction is rather common in IAH. Ulyatt suggested that filtration gradient (FG) may be an important factor in explaining renal failure associated with IAH [71]. The FG is the mechanical force across the glomerulus and is equal to the difference between glomerular filtration pressure and the proximal tubular pressure. Glomerular filtration pressure is equal to renal plasma flow and thus to MAP − IAP. In the presence of IAH, proximal tubular pressure can be equated with IAP. The FG can therefore be calculated as $FG = MAP - (2 \times IAP)$. This may explain why the kidney seems to be more vulnerable to IAH than other surrounding organs and maybe one of the key factors in the development of IAH-induced renal failure [12, 36].

The effect of IAH on the central nervous system

A direct relationship between IAP and ICP has been observed in both animal and human studies [35, 48, 72-74]. Several authors hypothesized that the increase in ICP secondary to IAH was caused by increased ITP, leading to increased CVP and decreased venous return from the brain and thus, venous congestion and brain edema. This hypothesis gained acceptance when Bloomfield et al. [48] demonstrated that the association between IAP and ICP could be abolished by performing a sternotomy and bilateral pleuropericardiotomy in pigs. The reduced systemic blood pressure associated with decreased cardiac preload and the increase in ICP will lead to a decrease in cerebral perfusion pressure. Some authors have even demonstrated successful treatment of refractory intracranial hypertension with abdominal decompression or neuromuscular blockers [35, 72].

Some recommendations:

- IAP monitoring is essential for all traumatic or non-traumatic patients at risk for ICH or IAH (according to the risk factors published by the World Society of the Abdominal Compartment Syndrome)
- In all patients with ICH, preventive measures should be undertaken to avoid increase in IAP
- Neurologic status should be frequently monitored in patients with IAH
- Avoid hypervolemia in patients with IAH to prevent further increase in ICP
- Provide adequate treatment for IAH, especially if ICH is also present
- Avoid laparoscopy in patients at risk for ICH. The pneumoperitoneum used for laparoscopy creates a situation analogous to experimental settings of IAH and ICH in which detrimental effects on ICP have been observed. This is especially important in trauma patients with associated brain and abdominal injuries.

The effect of IAH on bowel function

Where the digestive tract is concerned, intra-abdominal hypertension causes diminished perfusion and mucosal acidosis and sets the stage for multiple organ failure (MOF) [75]. The pathologic changes are more pronounced after sequential insults of ischemia–reperfusion and IAH. [76, 77]. Recent clinical studies have demonstrated a temporal relationship between ACS and subsequent MOF [15, 75, 78]. In animals, ACS provokes cytokine release and neutrophil migration resulting in remote organ failure. In humans, ACS results in splanchnic hypoperfusion that may occur in the absence of hypotension or decreased cardiac output. This ischemia and reperfusion injury to the gut serves as a second insult in a two-hit model of MOF where the lymph flow conducts gut-derived proinflammatory cytokines to remote organs [15, 75]. IAH has also been associated with increased bacterial translocation in animal experiments, especially when combined with ischemia–reperfusion injury [76, 79, 80].

Therapeutic options for IAH and ACS

In analogy to other compartment syndromes in the human body, decompressive laparotomy (DL) seems the most logical treatment option. It is also the most widely used and best described treatment modality today. However, DL leaves the patient with an open abdomen which can lead to extensive fluid losses, infection, enterocutaneous fistulae, ventral hernia, and cosmetic dysfunction. Therefore, DL is mostly used today as a rescue therapy for patients with overt ACS, who have not responded to medical treatment. Indications and results for different treatment modalities will be discussed here. Figure 1 incorporates the different treatment options for IAH in a multimodal strategy to manage IAH patients.

Nonsurgical management

Most nonsurgical treatment strategies are aimed at either decreasing abdominal volume or increasing wall compliance. An overview of possible treatment strategies is given in Table 3. Some of these will be highlighted here in detail.

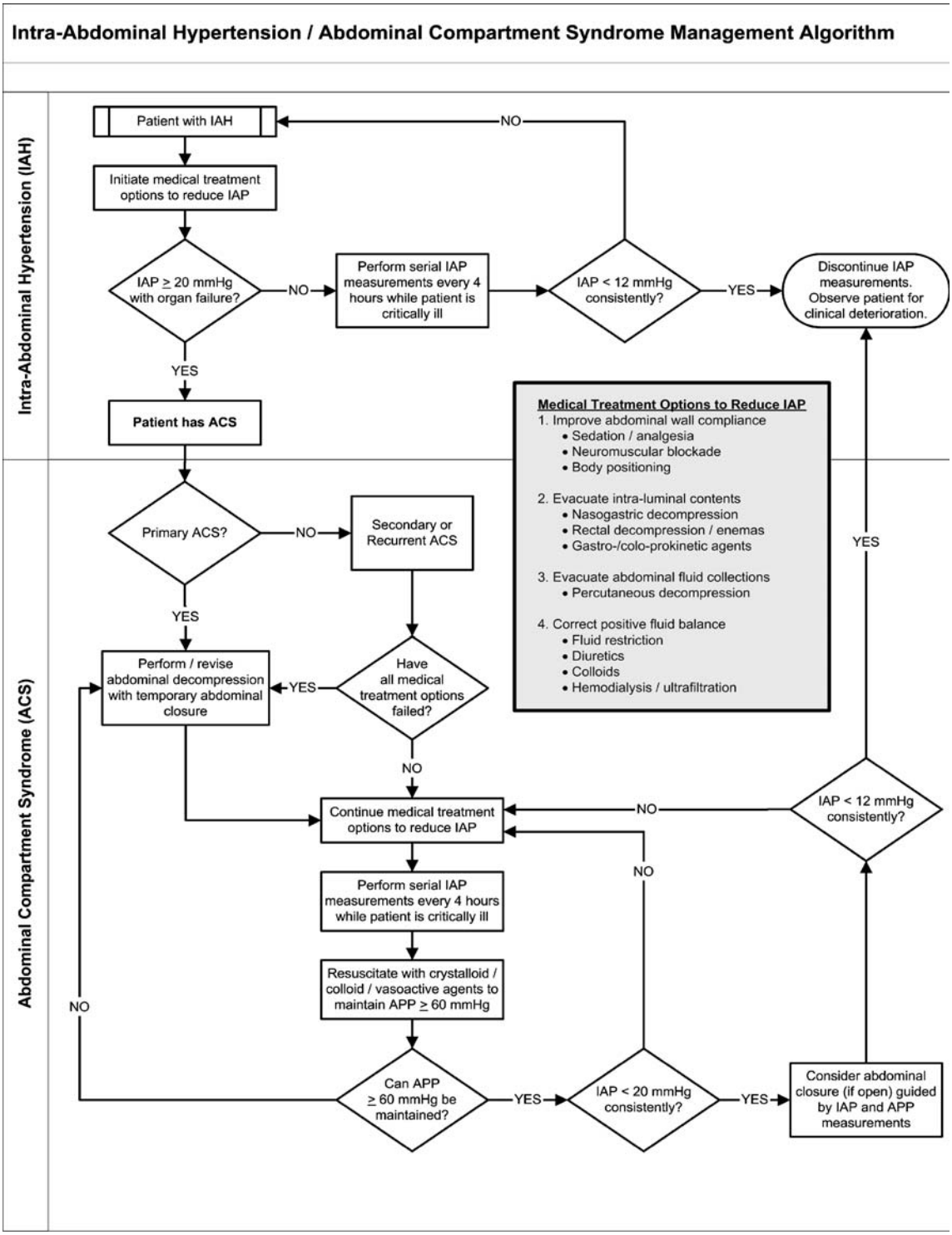

Fig. 1 Treatment algorithm for IAH–ACS as proposed by the WSACS (adapted from [128])

Evacuation of intraluminal contents

Ileus is a frequent phenomenon in critically ill patients. Noninvasive removal of intraluminal contents by gastric tube placement and suctioning, rectal tube placement, enemas and, if indicated, endoscopic decompression should be attempted [81–83].

Also, gastroprokinetics (such as metoclopramide or erythromycin) and/or colonoprokinetics (neostigmine or prostigmine) may be used [84–88]. In patients with gross dilatation of the stomach or the colon, this alone may be sufficient to lower IAP to harmless levels, but in most general ICU patients, other measures will have to be considered.

Table 3 Nonsurgical treatment options for IAH–ACS

Nonsurgical treatment
Improvement of abdominal wall compliance
Sedation
Pain relief (not fentanyl) [110]
Neuromuscular blockade [35, 98, 111-113]
Body positioning [114–116]
Negative fluid balance
Weight loss
Evacuation of intraluminal contents
Gastric tube and suctioning [81-53, 117]
Gastroprokinetics (erythromycin, cisapride, metoclopramide) [85, 87, 118]
Rectal tube and enemas [81-83, 117]
Colonoprokinetics (neostigmine, prostigmine bolus, or infusion) [84, 86, 88]
Endoscopic decompression of large bowel
Colostomy
Ileostomy
Evacuation of peri-intestinal and abdominal fluids
Ascites evacuation in cirrhosis [89-93, 119]
Percutaneous drainage of abscess or hematoma
Removal of free intraperitoneal blood
Correction of capillary leak and positive fluid balance
Albumin in combination with diuretics (furosemide) [43, 99, 120]
Correction of capillary leak (antibiotics, source control,...)
Colloids instead of crystalloids [101, 121]
Dialysis or CVVH with ultrafiltration [102, 103, 122]
Specific therapeutic interventions
Continuous negative abdominal pressure [123, 124].
Targeted abdominal perfusion pressure
Negative external abdominal pressure [125–127].

CVVH continuous venovenous hemofiltration

Evacuation of extraluminal contents

Drainage of tense ascites may result in a decrease in IAP [89–93]. Paracentesis is the treatment of choice in burn patients with secondary ACS [94–96] or any other patients who develop ascites after massive (usually crystalloid) fluid resuscitation. If intra-abdominal abscesses, hematomas, or fluid collections are present, they should be drained also.

Use of sedation and neuromuscular blockers

Increased muscle tone in the rectus abdominis muscle and other abdominal wall muscles due to voluntary muscle contraction, pain, or agitation causes decreased abdominal wall compliance and thus IAH. Therefore, it is important to titrate analgesia and sedation to allow for maximal relaxation of the abdominal wall muscles. However, in critically ill patients with capillary leak and abdominal wall edema, control of pain and agitation are often not sufficient and the use of neuromuscular blockers has to be considered. In a study on the use of neuromuscular blockers, De laet et al. [97] demonstrated that IAP can significantly be reduced, albeit that IAP was not completely normalized in patients with IAH. Other authors have confirmed these findings [35, 98]. However, neuromuscular blockers have been associated with increased incidence of ventilator-associated pneumonia and ICU muscular weakness and their use has been restricted in the last few years to avoid these and other complications. The possible benefit of reducing IAP has to be balanced against the risk of complications at the individual patient level.

Correction of capillary leak and positive fluid balance

Most patients with IAH, due to the nature of their illness or trauma, present with capillary leak syndrome. In the early stages of their illness, it is important to resuscitate these patients towards euvolemia and adequate intravascular fluid status, both in terms of their general condition and in terms of their IAH, since hypovolemia in patients with IAH can lead to splanchnic hypoperfusion and aggravation of the organ dysfunction [43, 99]. Dobutamine may help to counteract this splanchnic hypoperfusion [100].

However, fluid resuscitation will lead also to increased edema formation, third spacing, and possibly to a vicious cycle of ongoing IAH. After hemodynamic stabilization, correction of the fluid balance and decreasing edema formation becomes important. If renal function is only minimally to mildly compromised and the patient is hemodynamically stable, mobilization of edema by administration of albumin (to increase colloid osmotic pressure) and diuretics can be attempted. Also, in some patients, the use of colloids for fluid resuscitation may be preferable to crystalloids [101]. However, as renal function deteriorates further, patients often no longer respond to diuretic therapy. Fluid removal by means of ultrafiltration has been demonstrated to have a beneficial effect on IAP and possibly on organ function, e.g., compliance of the respiratory system [102, 103]. The institution of renal replacement therapy with fluid removal, if hemodynamically tolerated, should not be delayed. In patients with borderline hemodynamic status, continuous venovenous hemofiltration may be preferred over intermittent renal replacement therapy to avoid hemodynamic instability.

Decompressive laparotomy

A recent systematic review on decompressive laparotomy, based on 18 studies, was published by De Waele et al. [104]. This review illustrated that DL is successful in lowering IAP in all studies. Concerning the results on organ function, results are variable. Regarding the cardiovascular function, heart rate and MAP remained unchanged in most

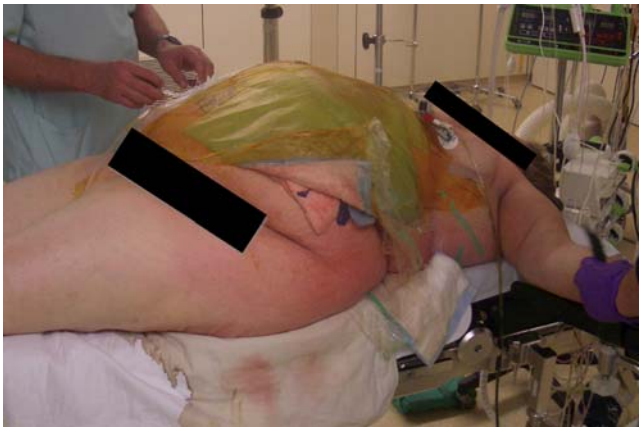

Fig. 2 Patient with cellulitis due to "chronic" fluid loss from a TAC procedure

studies. CVP and PAOP decreased significantly, which is to be expected in view of the abdominothoracic transmission. This probably does not reflect a true improvement in cardiac function. However, cardiac index was also improved. In analogy, peak inspiratory pressures decreased after decompression, but PaO2–FiO2 also improved. The effect on renal function is less clear. In most studies, urine output was significantly improved after DL but, interestingly, in the two largest series [105, 106] urine output was not affected. Sugrue suggests that acute tubular necrosis might be involved which takes longer to recuperate and does not appear in short-term outcome analyses. In general, DL seems to have a beneficial effect on organ function. Overall, mortality remains high (49.2%), but since most of the reported studies do not include data on APACHE II scores (and thus predicted mortality) and none of them include control groups, it is impossible to determine what the outcome would have been without DL or if DL causes a survival benefit.

Although most authors agree that DL should be performed in patients with an IAP>20 mmHg and new or progressive organ failure, there is some reluctance to perform DL because of the practical consequences in terms of fluid loss through the open abdomen, difficult wound dressings, risk of infection or fistula, reinterventions, cost, and longer hospital stay. However, a well-performed study by Cheatham et al. [107] demonstrated that physical, social, and mental health after DL is restored to the level of the general population after abdominal wall reconstruction and DL does not lead to permanent disability or unemployment. Delaying DL when indicated may worsen organ dysfunction and increase mortality.

Minimally invasive surgical decompression

Because of the complications associated with full DL, surgeons have been looking for less invasive techniques to decompress the abdomen. Endoscopic techniques based on the components separation concept described by Voss and others [108], like the subcutaneous anterior abdominal fasciotomy [109], are being developed and might replace DL in selected cases in the future.

Temporary abdominal closure

Although necessary and possibly life saving, decompressive laparotomy will leave the patient with an open abdomen prone to complications such as bleeding, infection, enterocutaneous fistula, or excessive fluid losses. To avoid these complications, a form of temporary abdominal closure (TAC) has to be used. Any TAC procedure used after DL should first and foremost prevent development of recurrent ACS. Ideally, it should also be cheap and easy, control fluid losses, require minimal dressing changes, and allow for easy re-exploration. There is a large body of literature regarding different TAC techniques after DL, but there are no large randomized clinical trials and the techniques vary between different studies. It is beyond the scope of this text to provide a concise review of TAC procedures, but we will give a short overview of the most frequently used techniques in our department, related to their applicability in situations of IAH–ACS.

Towel clip closure

Towel clip closure has been described mainly in war and disaster surgery where it is sometimes used as part of a damage control surgery approach. Advantages are the low cost and the minimal fluid losses. However, in patients with IAH–ACS, this technique usually does not prevent development of recurrent ACS. Since most patients who would

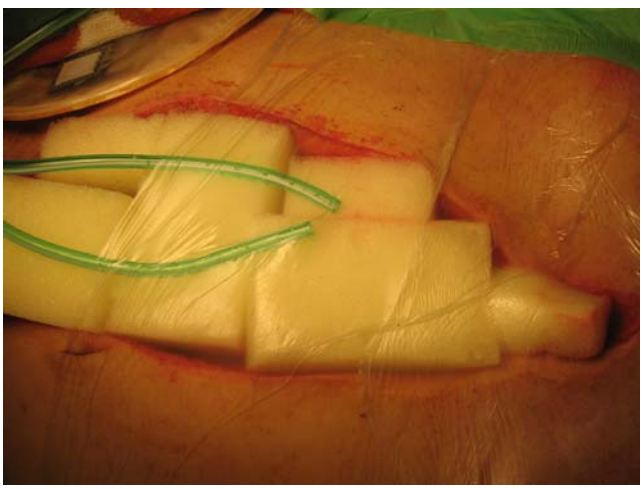

Fig. 3 "Home-made" VAC technique for TAC using scrub sponges over silicone sheath

be eligible for damage control laparotomy are by definition at risk for IAH, we advise against this technique if at all avoidable.

Bogota bag

The Bogota bag technique involves suturing an empty (sterile) infusion bag to the fascia or the skin for TAC. This offers the advantages of low cost, good control over IAP, easy visual inspection of the viscera underneath, and easy re-exploration. It usually does not prevent fluid losses, which leads to frequent dressing changes. In our department, this technique (or a modification thereof) is used mainly during the first 24 h in cases where early re-exploration is likely due to bowel ischemia, ongoing bleeding, or peritonitis.

Zipper–Wittmann patch

The Zipper and the Wittmann patch are commercially available tools for TAC. They mainly facilitate re-exploration and provide good relief of ACS. Theoretically, they should also allow progressive closure of the fascia as edema subsides, meaning that they were designed to remain in place during extended periods of time. The main downside is that they do not control fluid loss and as such, do not constitute a major advantage over the cheaper Bogota bag. The pronounced fluid leakage out of the dressing will cause infection and maceration of the skin if left unsolved for several days (Fig. 2). Therefore, we use these devices in our institution very rarely.

Vacuum-assisted closure

Several techniques for vacuum-assisted closure (VAC) of the abdomen have been described. Essentially, they all include use of a polyethylene sheath draped over the intestines and covered by a type of dressing (either gauze or sponge) with a suction device attached to it. Several authors have described different types of "home-made" VAC (Fig. 3) with good results and in recent years a commercially available abdominal VAC (KCI) has been developed. The advantages of VAC therapy are many: fluid loss is controlled, dressing changes are minimal, angiogenesis is promoted in the wound edges, the fascia edges are approximated gently, and the size of the VAC sponge can be reduced at every dressing change once the edema decreases. There is only one major disadvantage: the commercially available VAC dressing is very expensive and in most countries, not reimbursed. "Home-made" VAC may provide an alternative, but the negative pressure is harder to control and may be excessive, possibly leading to increased bleeding or enterocutaneous fistulae.

Conclusion

Intra-abdominal hypertension and abdominal compartment syndrome occur frequently in ICU patients and are independently associated with mortality. In spite of this, the syndrome is still poorly recognized and thus poorly treated in some cases. The diagnosis relies largely on IAP measurement which is most often performed through a bladder catheter. The effect of IAH on different organ systems has been described, along with recommendations to compensate for these effects. The ultimate goal of treatment is not only to decrease IAP but also to improve organ function and to decrease mortality. Decompressive laparotomy is the only treatment option that has been shown to reach most of these goals today. However, some less invasive techniques and some medical treatment strategies have shown promise in achieving IAP reduction as well as organ function improvement. A complete management algorithm has been proposed although treatment recommendations for IAH are likely to change significantly as more clinical data become available.

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