RESPIRATORY EFFECTS OF INCREASED INTRA-ABDOMINAL PRESSURE

ASPECTS RESPIRATOIRES DU SYNDROME DU COMPARTIMENT ABDOMINAL

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Abstract There has been an exponentially increasing interest in intra-abdominal hypertension (IAH). Comparison of the published data however is difficult due to the lack of consensus definitions. This review will focus on the available literature from the last years. A Medline and Pubmed search was performed using “intra-abdominal pressure” (IAP), “IAH”, and “abdominal compartment syndrome” (ACS) in combination with “cardiac”, “cardiovascular”, “organ function”, “respiratory” or “pulmonary” as search items. The aim was to find an answer to two questions: first, “Is it not time to pay attention to IAP in the critically ill?” And second, “what is the cardiovascular and respiratory impact of increased IAP?” Although the number of studies published on this topic is steadily increasing and confirms the pathophysiologic implications of IAH on end-organ function within and outside the abdominal cavity it remains difficult to compare the literature data because measurement methods and definitions used are not uniform. Therefore the World Society on Abdominal Compartment Syndrome (WSACS - www.wsacs.org) recently published a consensus definitions report. Provocative data have been published regarding the interactions between the abdominal and thoracic compartments especially in patients with capillary leak and fluid overload. In conclusion we can state that the answer is that it is now time to pay attention to IAP in the critically ill, but it is also time for standardized IAP measurement methods and multicenter randomized interventional studies.

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Introduction

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 it can develop within many disease processes. It is only recently that ACS received a heightened awareness. The development of intra-abdominal hypertension (IAH) and ACS are of tremendous importance in the care of critically ill, surgical and trauma patients. The impact of increased intra-abdominal pressure (IAP) on end-organ function and especially the lungs can no longer be ignored!

Historical background

As was nicely pointed out recently by Schein, the effects of elevated IAP have been known since 1863, when Marey of Paris highlighted that "the effects that respiration produces on the thorax are the inverse of those present in the abdomen" [1]. In 1890, Heinrichius demonstrated that ACS was fatal to animals because of impairment of respiration, decreasing cardiac diastolic distension and hypotension. The term ACS was first used by Fleetsam et al. [2] in the late 1980s to describe the pathophysiologic alterations resulting from IAH secondary to aortic aneurysm surgery. Hence the first definition of ACS was finally coined.

Definitions

The World Society of Abdominal Compartment Syndrome (WSACS - www.wsacs.org) was founded in 2004 to serve as a peer-reviewed forum and educational resource for all healthcare providers as well as industry that have an interest in IAH and ACS. The mission of the society is to foster education, promote research and thereby improve survival of patients with IAH and ACS. Recently the first consensus definitions report of the WSACS has been published [3]. Table 1 summarizes these consensus definitions.

Recognition of ACS

Clinical awareness

Despite an escalation of the medical literature on the subject, there still appears to be an under-recognition of the syndrome. The results of several surveys on the physician’s knowledge of IAH and ACS have recently been published [4]. The bottom line is that there is still a general lack of clinical awareness and many ICUs never measure IAP. No consensus exists on optimal timing of measurement or decompression. In a recent editorial Ivatury [5] states that: “One potential exegesis of this widespread under-appreciation of these syndromes may be related to our rapidly evolving understanding of their patho-physiology. Our knowledge is no longer restricted to experimentally sound (isolated IAH) concepts, but is elevated to a true clinical phenomenon (IAH as a “second-hit” after ischemia-reperfusion).” To fill this void a new survey has been launched. The survey can be accessed via www.wsacs.org/survey.htm.

Etiology

The ACS can be diagnosed when there is increased IAP with evidence of end-organ dysfunction. While multiple causes of acute cardiopulmonary, renal, hepatosplanchnic or neurologic deterioration exist in the intensive care unit, it is important that we recognize the IAP as being an independent risk factor for this organ function deterioration. Hence the timely recognition of the underlying risk factors and predisposing conditions that lead to IAH and ACS is extremely important. Indications for IAP monitoring should be based on the presence/absence of these risk factors. Many conditions are reported in association with IAH/ACS, and they are summarized in Table 2.
Diagnosis

Clinical and radiologic examination

The abdominal perimeter or girth or clinical examination cannot be used as a surrogate for IAP since this is far from accurate with a sensitivity and positive predictive value of around 40-60% [6,7]. Radiologic investigation with plain radiography of the chest or abdomen, abdominal ultrasound or CT-scan are also insensitive to the presence of increased IAP.

Measurement of IAP

Since the abdomen and its contents can be considered as relatively non-compressive and primarily fluid in character, behaving in accordance to Pascal’s law, the IAP measured at one point may be assumed to represent the IAP throughout the abdomen [8,9].

In the strictest sense, normal IAP ranges from 0 to 5 mmHg [10]. Certain physiologic conditions, however, such as morbid obesity [11,12], ovarian tumors, cirrhosis or pregnancy, may be associated with chronic IAP elevations of 10-15 mmHg to which the patient has adapted with an absence of significant patho-physiology. In contrast, children commonly demonstrate low IAP values [13].

Different indirect methods for estimating IAP are used clinically because direct measurements are considered to be inaccurate with a sensitivity and positive predictive value of around 40-60% [6,7]. Radiologic investigation with plain radiography of the chest or abdomen, abdominal ultrasound or CT-scan are also insensitive to the presence of increased IAP.

### Table 1 Consensus definitions [3]

<table>
<thead>
<tr>
<th>Definition</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>IAP is the steady-state pressure concealed within the abdominal cavity</td>
</tr>
<tr>
<td>2</td>
<td>( \text{APP} = \text{MAP} - \text{IAP} )</td>
</tr>
<tr>
<td>3</td>
<td>( \text{FG} = \text{GFP} - \text{PTP} = \text{MAP} - 2 \times \text{IAP} )</td>
</tr>
<tr>
<td>4</td>
<td>IAP should be expressed in mmHg 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 mid-axillary line</td>
</tr>
<tr>
<td>5</td>
<td>The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml of sterile saline</td>
</tr>
<tr>
<td>6</td>
<td>Normal IAP is approximately 5-7 mmHg in critically ill adults</td>
</tr>
<tr>
<td>7</td>
<td>IAH is defined by a sustained or repeated pathologic elevation of IAP ≥ 12 mmHg</td>
</tr>
<tr>
<td>8</td>
<td>IAH is graded as follows:</td>
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<tr>
<td></td>
<td>Grade I: IAP 12-15 mmHg</td>
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<td></td>
<td>Grade II: IAP 16-20 mmHg</td>
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<tr>
<td></td>
<td>Grade III: IAP 21-25 mmHg</td>
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<tr>
<td></td>
<td>Grade IV: IAP &gt; 25 mmHg</td>
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<tr>
<td>9</td>
<td>ACS is defined as a sustained IAP &gt; 20 mmHg (with or without an APP &lt; 60 mmHg) that is associated with new organ dysfunction/failure</td>
</tr>
<tr>
<td>10</td>
<td>Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention</td>
</tr>
<tr>
<td>11</td>
<td>Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region</td>
</tr>
<tr>
<td>12</td>
<td>Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS</td>
</tr>
</tbody>
</table>

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.

### Table 2 Etiology, risk factors and predisposing conditions for IAH

| Related to decreased abdominal wall compliance |
| Mechanical ventilation, especially fighting with the ventilator and the use of accessory muscles |
| Use of PEEP or the presence of auto-PEEP |
| Basal pleuroneumonia |
| 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, gastrochisis or omphalocele |
| Burns with abdominal eschars |

| Related to increased intra-abdominal contents |
| Gastroparesis/gastric distention/ileus/Colon pseudo-obstruction |
| Abdominal tumor |
| Retroperitoneal/ abdominal wall hematoma |
| Enteral feeding |

| Related to abdominal collections of fluid, air or blood |
| Liver dysfunction with ascites |
| Abdominal infection (pancreatitis, peritonitis, abscess, …) |
| Hemoperitoneum |
| Pneumoperitoneum |

| 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 mm³ OR an activated partial thromboplastin time (APTT) more than two times normal OR a prothrombin time (PTT) below 50% OR an international standardized ratio (INR) more than 1.5) |
| Polytransfusion/trauma (> 10 units of packed red cells/24 hours) |
| Severe sepsis or bacteremia |
| Septic shock |
| Massive fluid resuscitation (> 5 l of colloid or > 10 l of crystalloid/24 hours with capillary leak and positive fluid balance) |
| Major burns |

*The combination of acidosis, hypothermia and coagulopathy has been forwarded in the literature as the deadly triad [82,83].
be too invasive [8,14]. These techniques include rectal, uterine, gastric, inferior vena caval and urinary bladder pressure measurement. Only gastric and bladder pressures are used clinically. Over the years bladder pressure has been forwarded as the gold-standard indirect method (Fig. 1). The bladder technique has achieved the most widespread adoption worldwide due to its simplicity and minimal cost [8,9]. However considerable variation is noted between the different techniques used, and recent data suggest to instill minimal volumes (10-25 ml) into the bladder for priming [15–17].

Recently, new measurement kits, either via a FoleyManometer (Holtech Medical, Kopenhagen, Denmark, at www.holtech-medical.com), an AbViser-valve (Wolfe Tory Medical, Salt Lake City, UT, USA, at www.wolfetory.com) or a balloon-tipped stomach catheter (Spiegelberg, Hamburg, Germany, at www.spiegelberg.de and Pulsion Medical Systems, Munich, Germany, at www.pulsion.com) [9] have become commercially available.

Abdominal perfusion pressure (APP) measurement

Analogous to the widely accepted and clinically utilized concept of cerebral perfusion pressure, calculated as mean arterial pressure (MAP) minus intracranial pressure, APP, calculated as MAP minus IAP, has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation [18–21].

\[ \text{APP} = \text{MAP} - \text{IAP} \]

APP, by considering both arterial inflow (i.e. MAP) and restrictions to venous outflow (i.e. IAP), has been demonstrated to be statistically superior to either parameter alone in predicting patient survival from IAH and ACS [21]. A target APP of at least 60 mmHg has been demonstrated to correlate with improved survival from IAH and ACS.

Pathophysiologic implications

IAH affects multiple organ systems in a graded fashion. In order to better understand the clinical presentation and management of disorders of IAH, one must understand the physiologic derangements within each organ system separately [22]. It is beyond the scope of this review to give a concise and complete summary of the pathophysiologic implications of raised IAP on end-organ function within and outside the abdominal cavity [23,24]. We will only discuss some key-messages related to each organ that will affect daily clinical practice. Afterwards we will more extensively discuss the impact on the heart and the lungs since this is the topic of this review.

Neurologic function

Acute IAH causes an increase in intracranial pressure due to augmentation in pleural pressure. Cerebral perfusion pressure will decrease due to a functional obstruction of cerebral venous outflow caused by the increased intrathoracic pressure due to the cephalad displacement of the diaphragm in combination with a reduced systemic blood pressure as a result of decreased preload and cardiac output.

Hepatic function

The liver appears to be particularly susceptible to injury in the presence of elevated IAP. Animal and human studies have shown impairment of hepatic cell function and liver perfusion even with only moderately elevated IAP of...
10 mmHg [25,26]. Furthermore acute liver failure, decompensated chronic liver disease and liver transplantation are frequently complicated by IAH and the ACS [27,28]. In the management of these patients it might be useful to measure the plasma disappearance rate for indocyaninegreen as this correlates not only with liver function and perfusion but also with IAP [29,30]. With increasing IAP there is, decreased hepatic arterial flow, decreased venous portal flow and increase in the portacollateral circulation, this all exerts physiological effects with decreased lactate clearance, altered glucose metabolism and mitochondrial function.

Renal function

IAH has been associated with renal impairment for over 150 years [1]. It is only recently however that a clinically recognized relationship has been found [31-34]. The etiology of renal impairment in IAH is not entirely well established, however it may be multifactorial: reduced renal perfusion, reduced cardiac output and increased systemic vascular resistance and alterations in humoral and neurogenic factors.

Gastrointestinal function

IAH has profound effects on splanchnic organs, causing diminished perfusion, mucosal acidosis and setting the stage for multiple organ failure [35]. The pathologic changes are more pronounced after sequential insults of ischemia-reperfusion and IAH. It appears that IAH and ACS may serve as the second insult in the two-hit phenomenon of the causation of multiple organ dysfunction syndrome [36,37]. Recent clinical studies have demonstrated a temporal relationship between ACS and subsequent multiple organ failure [35,38,39]. 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 multiple organ failure where the lymph flow conducts gut-derived pro-inflammatory cytokines to remote organs. IAP inversely correlates with pH [40-42]. IAP inversely correlates with plasma disappearance rate for indocyaninegreen [29]. IAH triggers a vicious cycle leading to intestinal edema, ischemia, bacterial translocation and finally multiple organ dysfunction syndrome [43-45].

Abdominal wall and endocrine function

Increased IAP has been shown to reduce abdominal wall blood flow by the direct, compressive effects leading to local ischemia and edema [46]. This can decrease abdominal wall compliance and exacerbate IAH [47]. Abdominal wall muscle and fascial ischemia may contribute to infectious and non-infectious wound complications (e.g. dehiscence, herniation, necrotizing fasciitis) often seen in this patient population.

Cardiac and respiratory effects of increased abdominal pressure

Cardiovascular function

Due to the cephalad movement of the diaphragm pleural pressure and intrathoracic pressure will increase. This will result in a difficult preload assessment because traditional filling pressures will be erroneously increased. When IAP rises above 10 mmHg cardiac output drops due to an increase in afterload and a decrease in preload and left ventricular compliance. Systemic vascular resistance increases due to mechanical compression of vasculary beds and a reduction in preload due to drop in stroke volume and a reduction of venous return [48-51]. Mean arterial blood pressure may initially rise due to shunting of blood away from the abdominal cavity but thereafter normalizes or decreases [21,52]. Table 3 schematically shows the cardiovascular effects of IAH:

- cardiovascular dysfunction and failure are common in IAH or ACS;

<table>
<thead>
<tr>
<th>Table 3 Cardiovascular effects related to IAP$^a$</th>
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<tbody>
<tr>
<td>Diaphragm elevation ↑</td>
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<tr>
<td>Pleural and intrathoracic pressure ↑</td>
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<tr>
<td>Difficult preload assessment</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure ↑</td>
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<tr>
<td>Central venous pressure ↑</td>
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</tbody>
</table>
| Transmural filling pressure =
| Intrathoracic blood volume index =
| Global enddiastolic blood volume index =
| Right ventricular end-diastolic volume =
| Right, global and left ventricular ejection fraction =
| Extra vascular lung water =
| Stroke volume variation ↘ |
| Pulse pressure variation ↘ |
| Systolic pressure variation ↘ |
| Inferior vena caval flow ↓ |
| Venous return ↓ |
| Left ventricular compliance and contractility ↓ |
| Downward and right shift of the Frank Starling curve |
| Cardiac output ↓ |
| Systemic vascular resistance ↑ |
| MAP ↘ = |
| Pulmonary artery pressure ↑ |
| Pulmonary vascular resistance ↑ |
| Heart rate ↘ |
| Lower extremity hydrostatic venous pressure ↑ |
| Venous stasis, edema, ulcers ↑ |
| Venous thrombosis ↑ |
| Pulmonary embolism$^b$ ↑ |
| Mixed venous oxygen saturation ↓ |
| Central venous oxygen saturation ↓ |

$^a$ Cardiovascular effects are exacerbated in case of hypovolemia, hemorrhage, ischemia, auto-PEEP or high PEEP ventilation.

$^b$ Upon decompression.
accurate assessment and optimization of preload, contractility, and afterload is essential to restore end-organ perfusion and function;

our understanding of traditional hemodynamic monitoring techniques and parameters, however, must be reevaluated in IAH/ACS since pressure-based estimates of intravascular volume as pulmonary artery occlusion pressure and central venous pressure (CVP) are erroneously increased:

- the clinician must be aware of the interactions between intrathoracic pressure (ITP), IAP, positive end expiratory pressure (PEEP), and intracardiac filling pressures;
- misinterpretation of the patient’s minute-to-minute cardiac status may result in the institution of inappropriate and potentially detrimental therapy;
- Transmural™ filling pressures, calculated as the endexpiration value (ee) minus the ITP better reflect preload [49]:

\[ \text{CVP}^{\text{TM}} = \text{CVP}_{ee} - \text{ITP} \]
\[ \text{PAOP}^{\text{TM}} = \text{PAOP}_{ee} - \text{ITP} \]

- a quick estimate of transmural filling pressures can also be obtained by subtracting half of the IAP from the end expiratory filling pressure:

\[ \text{CVP}^{\text{TM}} = \text{CVP}_{ee} - \text{IAP}/2 \]
\[ \text{PAOP}^{\text{TM}} = \text{PAOP}_{ee} - \text{IAP}/2 \]

- the surviving sepsis campaign guidelines targeting initial and ongoing resuscitation towards a CVP of 8-12 mmHg [53] and other studies targeting a MAP of 65 mmHg [54] should be interpreted with caution in case of IAH/ACS to avoid unnecessary over- and under-resuscitation!

- volumetric estimates of preload status such as right ventricular end diastolic volume index or global end diastolic volume index, are especially useful because of the changing ventricular compliance and elevated intrathoracic pressure [51,55-58];
- functional dynamic hemodynamic parameters should be used to assess volume responsiveness [59];
- the cardiovascular effects are aggravated by hypovolemia and the application of PEEP [60-64], whereas hypervolemia has a temporary protective effect [65].

**Pulmonary function**

The interactions between the abdominal and the thoracic compartment pose a specific challenge to the intensive care unit physicians [66]. Both compartments are linked via the diaphragm and on average a 50% (range 25-80%) transmission of IAP to the intrathoracic pressure has been noted in previous animal and human studies [51]. Patients with primary ACS will often develop a secondary acute respiratory distress syndrome (ARDS) and will require a different ventilatory strategy and more specific treatment than a patient with primary ARDS [67,68]. The major problem lays in the reduction of the functional residual capacity (FRC). Together with the alterations caused by secondary ARDS this will lead to the so-called “baby-lungs” Fig. 2 schematically illustrates the respiratory effects of IAH. Figs. 3 and 4 show the effect of increased IAP during venti-

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**Figure 2** Respiratory effects of IAH. CVP: central venous pressure; DVT: deep vein thrombosis; EDV: end diastolic volume; IAP: intra-abdominal pressure; PAOP: pulmonary artery occlusion pressure; PE: pulmonary embolism; P™: transmural pressure.
lator dyssynchrony on endtidal CO\textsubscript{2} production and the effect of curarization in an individual patient. Table 4 schematically shows the pulmonary effects of IAH. Some key-issues to remember are:

- IAH decreases total respiratory system compliance by a decrease in chest wall compliance, while lung compliance remains unchanged [47,69].
- Best PEEP should be set to counteract IAP whilst in the same time avoiding over-inflation of already well-aerated lung regions:

  \[
  \text{Best PEEP} = \text{IAP}
  \]

- The ARDS consensus definitions should take into account PEEP and IAP values.

- During lung protective ventilation, the plateau pressures should be limited to transmural plateau pressures \(P_{\text{pl}}\) below 35 cmH\textsubscript{2}O:

  \[
  P_{\text{pl}}^{\text{TM}} = P_{\text{plat}} - \text{IAP}/2
  \]

- The PAOP criterion in ARDS consensus definitions is futile in case of IAH and should be adapted (most patients with IAH and secondary ARDS will have a PAOP above 18 mmHg).
- IAH increases lung edema, within this concept monitoring of extravascular lung water index seems warranted [70].
- The combination of capillary leak, positive fluid balance and raised IAP poses the patient at an exponential danger for lung edema.

Figure 3  Upper panel: Trend tracing of endtidal CO\textsubscript{2} (ETCO\textsubscript{2}) on the left axis and APP on the right axis in a mechanically ventilated patient. Lower panel: Trend tracing of IAP. Line A (16:50) marks the beginning of ventilator dyssynchrony due to fighting and abdominal muscle contractions with increased IAP up to 30 mmHg, increased ETCO\textsubscript{2} and decreased APP. Line B (20:40) marks the end of dyssynchrony with normalization of all parameters after the start of a continuous infusion with cisatracurium. This case nicely demonstrates the interactions between the abdominal and thoracic compartments.

Figure 4  Another case demonstrating the interactions between the abdominal and thoracic compartments. Trend tracings of endtidal CO\textsubscript{2} (ETCO\textsubscript{2}) on upper panel and IAP on lower panel. The arrows indicate administration of cisatracurium bolus during ventilator dyssynchrony due to fighting and abdominal muscle contractions with increased IAP up to 22 mmHg and increased ETCO\textsubscript{2}.
Body position affects IAP:
- Putting an obese patient in the upright position can cause ACS [71].
- The abdomen should hang freely during prone positioning [30].
- The anti-Trendelenburg position may improve respiratory mechanics, however it can decrease splanchnic perfusion [72].

The use of curarization should be balanced against the beneficial effect on abdominal muscle tone resulting in decrease in IAP and improvement of APP, and the detrimental effect on lung mechanics resulting in atelectasis and sur-infection [73].

The presence of IAH will lead to pulmonary hypertension via increased intrathoracic pressure with direct compression on lung parenchyma and vessels and via the diminished left and right ventricular compliance. In this case the administration of inhaled NO or ilomedine (prostacyclin) may be justified.

The effect of IAP on parenchymal compression is exacerbated in case of hemorrhagic shock or hypotension.

Clinical management

The management of patients with IAH is based on three principles [74,75]:
- specific procedures to reduce IAP and the consequences of ACS;
- general support (intensive care) of the critically ill patient;
- optimization after surgical decompression to perhaps counteract some of the specific adverse effects associated with decompression.

Medical treatment

Before surgical decompression is considered less invasive medical treatment options should be optimized. Different medical treatment procedures have been suggested to decrease IAP [19]. These are based on five different mechanisms:

- improvement of abdominal wall compliance;
- evacuation of intraluminal contents;
- evacuation of abdominal fluid collections;
- correction of capillary leak and positive fluid balance;
- specific treatments.

Table 5 gives an overview of the different medical treatment options.

Surgical decompression

Although decompression remains the only definite management for ACS, the timing of this procedure still remains controversial. During the intervention specific anesthetic challenges need to be solved and after decompression the patient is at risk for ischemia reperfusion injury, venous stasis and fatal pulmonary embolism [76]. Maintaining adequate preload and APP are the key to success [18,21,60].

Open abdomen treatment (or laparostomy) was initially intended for patients with diffuse intra-abdominal infections, and often used in combination with a planned relaparotomy approach. Due to the increased awareness of the deleterious effects of IAH, open abdomen treatment, either prophylactic or therapeutic, is more common in the intensive care unit [38,77]. Several methods for temporary abdominal closure are have been described (we refer the reader to more specific textbooks for further information [77]):

- Moist gauze used to be the preferred method of covering the abdomen, but this is no longer used.
- "Bogota bag": a plastic sheet is cut from a sterile 3L irrigation bag, and sewn to the skin or fascia.
Respiratory effects of increased intra-abdominal pressure

Increased resource utilization, decreased economic productivity, and increased mortality among a wide variety of patient populations [23, 78]. Despite its obvious clinical implications, attention is not paid to IAP, IAH and ACS. Only a few medical and surgical intensivists believe in the concept and actively attempt its prevention and treatment [5].

We must study and learn from the past and, at the same time, proactively “invent” the future. As aptly described by Dr. Ivatury and Sugerman [79], IAH/ACS is “…a clinical entity that had been ignored for far too long...the mystery of IAH and ACS continues to unfold, transgressing the boundaries of acute and chronic illness and medical and surgical specialties.” The future of IAH and ACS is in our hands and the results of recent multicenter studies confirm the importance of IAH and ACS on patient outcome [78, 80, 81]. It is time to pay attention, this was the title of a recent review [24] and the slogan of the Third World Congress on Abdominal Compartment Syndrome (WCACS2007) held in Antwerp, Belgium in 2007, March 22-24 (www.wcacs.org).

Conclusions

First suggested in 1863 by Marey, ACS is a constellation of the physiologic sequelae of increased IAP, termed IAH. Recent observations suggest an increasing frequency of this complication in all types of patients. The presence of IAH and ACS are significant causes of organ failure, increased resource utilization, decreased economic productivity, and increased mortality among a wide variety of patient populations [23, 78].

Medical treatment options for IAH and ACS

Table 5 Medical treatment options for IAH and ACS

<table>
<thead>
<tr>
<th>Improvement of abdominal wall compliance</th>
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<tr>
<td>Sedation</td>
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<tr>
<td>Pain relief (not fentanyl!) [84]</td>
</tr>
<tr>
<td>Neuromuscular blockade [20, 73, 85-87]</td>
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<tr>
<td>Body positioning [29, 30, 72]</td>
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<tr>
<td>Negative fluid balance</td>
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<tr>
<td>Skin pressure decreasing interfaces [29]</td>
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<tr>
<td>Weight loss [88]</td>
</tr>
<tr>
<td>Percutaneous abdominal wall component separation [89, 90]</td>
</tr>
</tbody>
</table>

Evacuation of intraluminal contents

- Gastric tube and suctioning [91-94]
- Gastroprokinetics (erythromycin, cisapride, metoclopramide) [95-97]
- Rectal tube and enemas [91-94]
- Colonoprokinitics (neostigmine, prostigmine bolus or infusion) [98-100]
- Endoscopic decompression of large bowel
- Ileostomy

Evacuation of peri-intestinal and abdominal fluids

- Ascites evacuation in cirrhosis [101-106]
- CT- or US-guided aspiration of abscess
- CT- or US-guided aspiration of hematoma
- Percutaneous drainage of (blood) collections
- Ascites evacuation in cirrhosis [90, 107, 108]

Correction of capillary leak and positive fluid balance

- Albumin in combination with diuretics (furosemide) [60, 109, 110]
- Correction of capillary leak (antibiotics, source control, ...)
- Colloids instead of cristalloids [111, 112]
- Dobutamine (not dopamine!) [113]
- Dialysis or CVH with ultrafiltration [114-116]
- Ascorbic acid in burn patients [117, 118]

Specific therapeutic interventions

- Continuous negative abdominal pressure (CNAP) [119, 120]
- Negative external abdominal pressure (NEXAP) [121-123]
- Targeted APP [18, 19, 51]
- (Experimental: Octreotide and melatonin in secondary ACS) [124, 125]

- Towel clip closure is often used as an initial method of temporary abdominal closure after damage control surgery.
- Removable prosthetic material. Examples are the zippers, Wittman patch (which uses a Velcro closure system, etc).
- Vacuum assisted fascial closure systems.

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Respiratory effects of increased intra-abdominal pressure


