Intra-abdominal hypertension and abdominal compartment syndrome in critical illness

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CHAPTER 2

Intra-abdominal hypertension and Abdominal Compartment Syndrome in critically ill patients: a narrative review of past, present and future steps.

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Abstract

Background and aims
Intra-abdominal hypertension is frequently present in critically ill patients and is an independent predictor for mortality. In this narrative review, we aim to provide a comprehensive overview of current insights into intra-abdominal pressure (IAP) monitoring, intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS). The focus of this review is on the pathophysiology, risk factors and outcome of IAH and ACS as well as on therapeutic strategies such as non-operative management, surgical decompression and management of the open abdomen. Finally, future steps are discussed including propositions of what a future guideline should focus on.

Conclusion
Pathological IAP is a continuum ranging from mild IAP elevation without clinically significant adverse effects to substantial increase in IAP with serious consequences to all organ systems. IAP monitoring should be performed in all patients at risk of IAH. Although continuous IAP monitoring is feasible, this is currently not standard practice. There are a number of effective non-operative medical interventions that may be performed early in the patient’s course to reduce IAP and decrease the need for surgical decompression. Abdominal decompression can be life-saving when ACS is refractory to non-operative treatment and should be performed expeditiously. The objectives of open abdomen management are fistula prevention and to achieve delayed fascial closure at the earliest possible time. There is still a lot to learn and change. The 2013 World Society of Abdominal Compartment Syndrome guidelines should be updated and multicentre studies should evaluate the effect of IAH treatment on patient outcome.
Background and Aims

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are conditions that result from increased intra-abdominal pressure (IAP). IAH is frequently present in critically ill patients and is an independent predictor for mortality.[1,2] The World Society of the Abdominal Compartment Syndrome (WSACS) was founded in 2004 (www.wsacs.org) and its early accomplishments were the development of uniform definitions for IAH and ACS and standardized techniques for IAP monitoring to facilitate research and improve patient care.[3] The definitions of IAP and ACS are based on consensus and the absolute IAP levels and thresholds used facilitate comparisons. However, in clinical practice pathological IAP is a continuum ranging from mild IAP elevations without clinically significant adverse effects to substantial increases in IAP with multi-organ failure.[3]

In this review, we aim to provide a comprehensive overview of current insights into IAP measurement, IAH and ACS. Its focus is on pathophysiology, risk factors and outcome of IAH and ACS as well as on therapeutic strategies such as non-operative management, surgical decompression and management of the open abdomen. Finally, future steps are discussed including propositions of what a future guideline should focus on.

How to measure intra-abdominal pressure

IAP is the steady-state pressure within the abdominal cavity.[4] Since the abdominal compartment generally transduces pressure evenly throughout the cavity, IAP can be measured in nearly every part of the abdomen.[5,6] Clinical assessment of IAP based on palpation of the abdomen is unreliable and clinically significant IAH may be present in the absence of abdominal distension.[7] IAP can be measured directly from catheters placed in the peritoneal cavity, for example during laparoscopy when the abdomen is insufflated with CO2 gas. Indirect IAP may be measured in hollow organs in the abdominal cavity with easy contact with the atmosphere: bladder, stomach, rectum or vagina.[6] In critical care IAP measurements through either bladder or stomach are most practical and intravesicular IAP measurement is considered the gold standard.[5] However, measurements may be inaccurate, for example when artefacts in the IAP waveform lead to signal over- or underdamping or when the pressure transducer is malpositioned relative to the reference point.[6]

Furthermore, regional inter-compartmental pressure differences between the upper and lower abdomen have been described in patients following liver transplantation.[8] A standardized approach to IAP measurement is important to ensure reproducibility[5], and the WSACS consensus definitions state that 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 midaxillary line (Table 1). The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml sterile saline.[3]

Table 1. World Society of Abdominal Compartment Syndrome (WSACS) Consensus Definitions

<table>
<thead>
<tr>
<th>Definition</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal intra-abdominal pressure (IAP) is approximately 5-7 mmHg in critically ill adults</td>
<td></td>
</tr>
<tr>
<td>Intra-abdominal hypertension (IAH) is defined as a sustained IAP ≥ 12 mmHg</td>
<td></td>
</tr>
<tr>
<td>Abdominal Compartment Syndrome (ACS) is defined as a sustained IAP &gt; 20 mmHg associated with new organ dysfunction or failure.</td>
<td></td>
</tr>
<tr>
<td>IAP is the steady-state pressure within the abdominal cavity.</td>
<td></td>
</tr>
<tr>
<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 transduced zeroed at the level of the midaxillary line.</td>
<td></td>
</tr>
<tr>
<td>The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml sterile saline.</td>
<td></td>
</tr>
</tbody>
</table>

Based on MLNG Malbrain et al., Intensive Care Medicine 2006 [3] and AW Kirkpatrick et al., Intensive Care Medicine 2013 [4] and available via the WSACS website (http://www.wsacs.org)

From a theoretical viewpoint, the only correct reference level is the mid-bladder level at the tip of the Foley catheter, but this level cannot be identified at the bedside. The WSACS recommended zero reference level is the midaxillary line at the level of the iliac crest as this bony structure is easy to palpate, even in obese patients.

Clinically relevant changes in IAP occur at head-of-bed increases above 20 degrees[9] and when measured at 30 and 45 degrees head-of-bed elevation, the IAP is respectively 4 and 9 mmHg higher on average than in the supine position.[10]

Normal IAP is approximately 5-7 mmHg in critically ill adults[4], but there is a positive correlation between IAP and body mass index (BMI).[11] Baseline IAP levels are between 9 and 14 mmHg in morbidly obese patients.[10]

IAP monitoring

The WSACS recommends IAP is measured every 4-6 hours in critically ill patients with one or more risk factors for the development of IAH or ACS and protocols for IAP measurement should be developed for each ICU.[5] Continuous IAP monitoring is feasible, but is currently not standard practice.[12] The development of new technologies in IAP monitoring are promising. For example, the Accuryn urinary catheter (Potrero Medical, San Francisco, CA) measures IAP from a small balloon seamlessly integrated directly below the catheter tip. IAP will be measured and displayed at the pressing of a button. It is technically possible to continuously monitor IAP using this system. Furthermore, the Accuryn urinary catheter continuously measures urine output.[13]

In 2004 the cost of intermittent IAP measurements were estimated to be between 20-80
euros per patient. Set-up costs for continuous IAP monitoring are higher by at least a factor 3. Set-up cost of IAP monitoring using a microchip were estimated to be a factor 40 higher than intermittent intravesicular measurement.[6] In the Netherlands in 2020, the cost of the Accuryn urinary catheter is approximately 200 euro per patient once the set-up cost of the re-usable monitor (approximately 9800 euro) has been covered.

**Abdominal compliance**

Abdominal compliance ($C_{ab}$) is defined as a measure of the ease of abdominal expansion. This is determined by the elasticity of the abdominal wall and diaphragm. It should be expressed as the change in intra-abdominal volume (IAV) per change in IAP (in ml/mmHg). Since the abdominal pressure-volume relationship is curvilinear, a small decrease in IAV, for example by drainage of abdominal free fluid, may result in a substantial decrease in IAP in patients with ACS. Measurement of $C_{ab}$ at the bedside is difficult, which may explain why it has been called a neglected parameter in critically ill patients. Measurement can only be done in case of a change in IAV, for example when drainage of intra-abdominal free fluid is performed. Normal $C_{ab}$ is around 250-450 ml/mmHg. Decreased $C_{ab}$ means that the same increase in IAV will result in a greater increase in IAP compared to normal. Factors associated with decreased $C_{ab}$ include male gender, android body composition, fluid overload and prone positioning. Treatment of patients with decreased $C_{ab}$ and patients with IAH are largely based on the same principles.[14,15]

**Pathophysiology**

Increased abdominal pressure has adverse effects on many different organ systems. Most often this involves the heart, lungs and kidneys, but the intestines, liver and brain may also be affected.

The abdominal compartment syndrome is comparable to a compartment syndrome in general, where increased pressure within an anatomic compartment leads to decreased blood flow and ultimately to tissue hypoxia.[16]

The pathophysiology of IAH and ACS is not completely understood, but a cascade is set in motion leading to a vicious cycle of deterioration and multi-organ failure.[17] Elevated IAP leads to compression of the vena cava causing reduced venous return to the heart. This decrease in pre-load leads to reduced cardiac output resulting in reduced blood flow to the organs (including kidneys and intestines). Furthermore, high intrathoracic pressures may result from elevation of the diaphragm due to high IAP. Thoracic compliance is reduced leading to increased airway pressures or increased work of breathing. Fluid resuscitation will further increase edema of the abdominal and thoracic wall and decrease abdominal and thoracic compliance. Edema of the bowel will increase bowel volume adding to this vicious cycle of increasing IAP.[18] Decreased splanchnic perfusion leads to decreased
mucosal blood flow and bacterial translocation, as Diebel described in a rodent model of ACS.[19] Bowel ischemia and multi-organ failure may follow.[20] Hepatic venous, arterial and microcirculatory blood flow decreases significantly with even slight increases in IAP. Hepatic dysfunction may lead to decreased clearance of plasma lactate, further adding to metabolic acidosis.[21]

The increase in intra-thoracic pressure impairs the venous return of the brain, thereby increasing intracranial pressure (ICP) and consequently decreasing cerebral blood flow.[22]

Pathophysiology of AKI
The pathophysiology of AKI is complex and has not been completely elucidated. Decreased cardiac output leading to decreased renal perfusion contributes to AKI, but multiple factors play a role.[17] Oliguria may be one of the earliest signs of ACS. [23] IAH reduces renal blood flow and glomerular filtration rate through compression of the renal veins and arterial vasoconstriction. This leads to activation of the renin-angiotensin-aldosterone hormone system (RAAS) signalling cascade. RAAS regulates blood pressure, fluid and electrolyte balance, as well as systemic vascular resistance. When renal blood flow is reduced, renin is secreted into the circulation by juxtaglomerular cells. Angiotensinogen is converted to angiotensin I by renin, and angiotensin I is subsequently converted to angiotensin II by the angiotensin-converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor leading to an increase in systemic vascular resistance. Angiotensin II also stimulates the secretion of aldosterone from the adrenal cortex. Aldosterone causes the renal tubules to increase the reabsorption of sodium and water in the blood, which increases the volume of extracellular fluid in the body. The intended effect is to increase renal perfusion.[24] Vasoconstriction may also be mediated by the sympathetic nervous system. Although mean arterial blood pressure (MAP) may increase at first, it soon stabilizes or decreases.[18] The inflammatory response triggered by IAH may also add to AKI.[16,17] In IAP > 20 mmHg, an increase in circulating levels of a variety of inflammatory mediators has been described in rats.[25] Renal tissue and plasma tumour necrosis factor-alpha (TNF-alpha) and IL-6 levels were significantly higher in rats undergoing increases in IAP compared to sham rats. There was an association with histopathological changes in renal tissue and renal tubular changes were most prominent.[26] In a study of living kidney donors, an increase in inflammatory mediators was found in patients whose IAP was only 12 mmHg.[27] In this study, living kidney donors were randomly assigned to hand-assisted laparoscopy (with induction of a pneumo-peritoneum of 12 mmHg) and open nephrectomy. Postoperative levels of CRP and interleukin-6 (IL-6) were higher in the laparoscopy group even though procedure time was shorter.
Epidemiology

Prevalence of IAH and ACS

There are substantial differences in incidence and prevalence within subgroups of patients, for example medical, surgical and trauma patients. Reported incidences of IAH and ACS by case mix were summarized by De Waele in 2011 (Table 2).[18] These data were based on the limited number of publications available at the time and varying definitions of IAH and ACS were used. In more recent studies, IAH and ACS have been defined according to the WSACS. These studies are compared to the incidences reported by De Waele in Table 2.

Data from a mixed medical-surgical Intensive Care Unit where all consecutive ICU patients were included showed that ACS occurred in 3% of patients.[28] In a mixed multi-center ICU population of 491 consecutive ICU patients ACS occurred in 6% of patients. The difference between the studies was attributed to the difference in case mix.[29]

A study in 81 trauma patients showed the incidence of ACS was 0% and IAH was 75%.[30] In a study of 503 high risk ICU patients prevalences of IAH and ACS were 33% and 3.6% respectively.[31] Highest prevalence of ACS occurred in subgroups of patients with pancreatitis (57%), after orthotopic liver transplant (OLT) (7%) and after abdominal aorta surgery (5%).

IAH persists and may increase as critically ill patients increasingly survive initial insults. [32] Overall ACS is decreasing (Table 2), possibly through early recognition and targeted management of IAH.[32] Other factors probably include improved peri-operative and intensive care management including restrictive peri-operative fluid management and meticulous haemostasis. However, ACS continues to be a major problem in specific subgroups, for example in the obese and in pancreatitis.[31]

Outcome

IAH is an independent predictor for mortality.[1,2,28] ICU mortality is significantly higher compared to those who never developed IAH (6-30% vs 1-11%)[28] and the grade of IAH is inversely related to outcome.[29] The 90-day mortality of ACS remains high at 39-76%, but shows a large variation.[29,31] Besides patient selection, variation may well reflect differences in patient management. In addition to increased mortality, IAH and ACS are associated with increased length of ICU stay, and increased severity of organ failure, particularly renal and respiratory failure.[31,33] This is illustrated by an increased requirement for renal replacement therapy and an increased duration of mechanical ventilation.[31] Timing of treatment is important in preventing progression of AKI and development of chronic kidney disease.[18]
Table 2. Comparison of IAH and ACS prevalences in 2011 and 2021

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>IAH 2011* (%)</th>
<th>IAH (includes ACS) 2021 (%)</th>
<th>ACS 2011** (%)</th>
<th>ACS 2021 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major abdominal surgery</td>
<td>NA</td>
<td>33-41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aorta surgery total</td>
<td>39.7*</td>
<td>5.2*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aorta surgery- elective</td>
<td>33.3*</td>
<td>4.4*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aorta surgery- emergency</td>
<td>61.5*</td>
<td>7.7*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver transplant</td>
<td>NA</td>
<td>59.5*</td>
<td>31</td>
<td>7.1*</td>
</tr>
<tr>
<td>Major trauma</td>
<td>50</td>
<td>58.8-72.8*</td>
<td>13-36</td>
<td>0*</td>
</tr>
<tr>
<td>Mixed ICU</td>
<td>30-54</td>
<td>39-48.9*</td>
<td>5-12</td>
<td>2.6*</td>
</tr>
<tr>
<td>Septic shock</td>
<td>51-76</td>
<td>18.9-68***</td>
<td>33</td>
<td>28****</td>
</tr>
<tr>
<td>Severe acute pancreatitis</td>
<td>59-84</td>
<td>85.7*</td>
<td>25-56</td>
<td>57.1*</td>
</tr>
</tbody>
</table>

NA not available
ICU intensive care unit
IAH intra-abdominal hypertension,
ACS abdominal compartment syndrome
* [31]
# [30]
** [28,29] [58]
## 2011 numbers are those published by de Waele et al.[18]
***[59,60] [61]
****[59,60]

Risk factors
A number of studies of IAH and ACS risk factors have been published, but interpretation is difficult due to significant heterogeneity between the studies. A systematic review and meta-analysis including 14 studies and 2500 critically ill adults was conducted in 2013 (Tables 3A and 3B).[34] A presenting diagnosis of sepsis, abdominal infection, abdominal surgery, hepatic failure/cirrhosis, gastro-intestinal (GI) bleeding and ileus were risk factors for development of IAH among ICU patients (Table 3A). Risk factors for ACS were studied in trauma and severe acute pancreatitis patients (Table 3B). Although several risk factors transcended across presenting patient diagnoses (for example large-volume crystalloid resuscitation and the presence of shock/hypotension), many were specific to the type of patient population under study. The authors stated that their findings were partially limited by clinical heterogeneity and the quality of statistical analyses conducted in the included studies and therefore these risk factors should be considered candidate evidence-based risk factors until formally evaluated in a prospective multi-centre observational study. However, thus far this is the best available evidence.
Table 3A. Significant risk factors for intra-abdominal hypertension among intensive care unit patients adapted from Holodinsky et al. [34]

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Patient population</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>Mixed ICU patients</td>
<td>5.10 (1.92-13.58)</td>
</tr>
<tr>
<td>Age (per year increase)</td>
<td>Mixed ICU patients</td>
<td>2.75 (1.01-3.09)</td>
</tr>
<tr>
<td><strong>Presenting diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sepsis</td>
<td>Mixed ICU patients</td>
<td>2.38 (1.34-4.23)</td>
</tr>
<tr>
<td>Abdominal infection</td>
<td>Mixed ICU patients</td>
<td>2.49 (0.48-13.0)</td>
</tr>
<tr>
<td>Abdominal surgery</td>
<td>Mixed ICU patients</td>
<td>1.93 (1.30-2.85)</td>
</tr>
<tr>
<td>Post-laparotomy</td>
<td>Trauma</td>
<td>5.72 (1.50-21.43)</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Mechanically ventilated mixed ICU patients</td>
<td>4.73 (1.96-11.41)</td>
</tr>
<tr>
<td>Hepatic failure/cirrhosis</td>
<td>Mechanically ventilated mixed ICU patients</td>
<td>2.07 (2.07-28.81)</td>
</tr>
<tr>
<td>GI bleeding</td>
<td>Mechanically ventilated mixed ICU patients</td>
<td>3.37 (1.43-7.94)</td>
</tr>
<tr>
<td>Ileus</td>
<td>Mixed ICU patients</td>
<td>2.05 (1.40-2.98)</td>
</tr>
<tr>
<td>Liver dysfunction*</td>
<td>Mixed ICU patients</td>
<td>2.25 (1.1-4.58)</td>
</tr>
<tr>
<td><strong>Disease severity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APACHE II score (per point increase)</td>
<td>Pancreatitis</td>
<td>1.652 (1.131-2.414)</td>
</tr>
<tr>
<td><strong>Metabolic derangement/organ failure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base deficit</td>
<td>Trauma</td>
<td>1.15 (1.01-1.33)</td>
</tr>
<tr>
<td>Acidosis*</td>
<td>Mixed ICU patients</td>
<td>1.93 (1.12-3.45)</td>
</tr>
<tr>
<td><strong>Shock/hypotension</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vasopressor use</td>
<td>Mechanically ventilated mixed ICU patients</td>
<td>2.33 (1.02-5.35)</td>
</tr>
<tr>
<td>Shock</td>
<td>Mixed ICU patients</td>
<td>4.68 (1.93-6.44)</td>
</tr>
<tr>
<td>Hypotension*</td>
<td>Mixed ICU patients</td>
<td>2.12 (1.05-4.50)</td>
</tr>
<tr>
<td>CVP (per mmHg)</td>
<td>Mixed ICU patients</td>
<td>1.3 (1.1-1.6)</td>
</tr>
<tr>
<td><strong>Respiratory status/failure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peep &gt; 10 cm H₂O</td>
<td>Mechanically ventilated mixed ICU patients</td>
<td>2.41 (1.57-3.70)</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td>Mechanically ventilated mixed ICU patients</td>
<td>1.87 (1.22-2.87)</td>
</tr>
<tr>
<td>ARDS</td>
<td>Mixed ICU patients</td>
<td>3.61 (1.60-9.06)</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>Mixed ICU patients</td>
<td>6.78 (1.94-59.03)</td>
</tr>
<tr>
<td><strong>Fluid resuscitation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-ICU crystalloid* ##</td>
<td>Trauma</td>
<td>1.40 (1.00-1.96)</td>
</tr>
<tr>
<td>Fluid balance* ##</td>
<td>Mixed ICU patients</td>
<td>5.22 (2.03-7.45)</td>
</tr>
<tr>
<td>24 hour fluid balance* ##</td>
<td>Pancreatitis</td>
<td>1.004 (1.001-1.006)</td>
</tr>
<tr>
<td>Fluid collections*</td>
<td>Pancreatitis</td>
<td>2.015 (1.298-3.129)</td>
</tr>
<tr>
<td>Fluid resuscitation (&gt;3.5 L crystalloid or colloid)</td>
<td>Mixed ICU patients</td>
<td>2.17 (1.30-3.63)</td>
</tr>
</tbody>
</table>

* Risk factor was not clearly defined

## Although the odds ratio in these studies appeared to increase per liter of fluid, this was unclear in the original manuscript. Abdominal infection was defined as infection of the peritoneal cavity confirmed by radiology or microbiology, or pancreatitis, abscess, or other. Sepsis was defined according to consensus definitions. Respiratory failure was defined as PaO₂/FiO₂ < 300 mmHg, APACHE II acute physiology and chronic health evaluation II, CVP central venous pressure, GI gastro-intestinal, PEEP positive end-expiratory pressure, ARDS acute respiratory distress syndrome.
Table 3B. Significant risk factors for abdominal compartment syndrome among intensive care unit patients adapted from Holodinsky et al.[34]

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Patient population</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient to OR within 75 mins of ED admission</td>
<td>Trauma</td>
<td>102.7 (9.65-999.9)</td>
</tr>
<tr>
<td>Disease severity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>APACHE II score &gt; sample mean of 20.3</td>
<td>Severe acute pancreatitis</td>
<td>1.143 (1.012-1.292)</td>
</tr>
<tr>
<td>Glasgow-Lrmie score &gt; sample mean of 9.1</td>
<td>Severe acute pancreatitis</td>
<td>1.221 (1.000-1.493)</td>
</tr>
<tr>
<td>Metabolic derangement / organ failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature ≤ 34°C</td>
<td>Trauma</td>
<td>22.9 (1.39-378.25)</td>
</tr>
<tr>
<td>Hemoglobin ≤ 80 g/L</td>
<td>Trauma</td>
<td>252.2 (9.89-999.9)</td>
</tr>
<tr>
<td>Base deficit ≥ 12</td>
<td>Trauma</td>
<td>3.5 (1.37-839.50)</td>
</tr>
<tr>
<td>Urine output ≤ 150 ml in 24 hours</td>
<td>Trauma</td>
<td>64.1 (5.48-749.68)</td>
</tr>
<tr>
<td>Serum creatinine &gt; sample mean of 217.7 umol/L</td>
<td>Severe acute pancreatitis</td>
<td>1.115 (1.02-1.219)</td>
</tr>
<tr>
<td>Shock/hypotension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure &lt; 86 in ED</td>
<td>Trauma</td>
<td>4.9 (1.78-13.99)</td>
</tr>
<tr>
<td>GAP CO2 ≥ 16</td>
<td>Trauma</td>
<td>&gt;999.9 (22.1-999.9)</td>
</tr>
<tr>
<td>Cardiac index &lt; 2.6 L/min/m²</td>
<td>Trauma</td>
<td>12.5 (1.02-153.64)</td>
</tr>
<tr>
<td>Respiratory status/failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory rate &gt; sample mean of 19.7 breaths/min</td>
<td>Severe acute pancreatitis</td>
<td>1.004 (1-1.008)</td>
</tr>
<tr>
<td>Resuscitation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crystalloid ≥ 3 L in ED</td>
<td>Trauma</td>
<td>23 (6.38-83.10)</td>
</tr>
<tr>
<td>Pre-hospital crystalloid</td>
<td>Extremity injury</td>
<td>1.99 (1.07-3.73)</td>
</tr>
<tr>
<td>PRBC ≥ 3 units in ED</td>
<td>Trauma</td>
<td>5.6 (1.03-30.83)</td>
</tr>
<tr>
<td>Crystalloid (L): PRBCs (units) ratio &gt; 1.5:1</td>
<td>Blunt trauma</td>
<td>3.6 (1.3-9.7)</td>
</tr>
</tbody>
</table>

ACS abdominal compartment syndrome
OR operating room
ED emergency department
APACHE II acute physiology and chronic health evaluation II
GAP CO2 gastric mucosal CO2 minus end tidal CO2
PRBC packed red blood cells

Management

IAH has consistently been associated with morbidity and mortality in observational studies. However, it remains uncertain whether treating or preventing this condition improves patient outcomes.[4]

To date, there have been no intervention studies to answer the important question which patient needs non-operative management or surgical management of ACS. Therefore the position and timing of decompression laparotomy in ACS are still unknown.[35]

Non-operative management of ACS

In a 2008 study of 83 patients admitted to the ICU, 10 patients developed ACS (12%). These patients all received nasogastric suctioning, rectal decompression, diuretics, deep sedation and neuromuscular blockade. None underwent surgical decompression. Two patients (20%) survived with non-operative ACS management.[33]

Non-operative management has advanced since 2008 and there are a number of effective non-operative interventions that may be performed to reduce IAP and thereby decrease the need for surgical decompression.[4][36]
In 2013 the WSACS published an IAH/ACS Medical management algorithm consisting of 5 therapeutic interventions[4] (Table 4). However, overall quality of evidence available to guide development of recommendations was low.[4] Small trials have investigated single measures and its effect on IAP, not on outcome.

**Table 4. IAH/ACS Medical management algorithm [4]**
Adapted with permission according to open access CC BY Licence 4.0 from Kirkpatrick et al. (https://link.springer.com/article/10.1007/s00134-013-2906-z)

- The choice (and success) of the medical management strategies listed below is strongly related to both the etiology of the patient’s IAH / ACS and the patient’s clinical situation. The appropriateness of each intervention should always be considered prior to implementing these interventions in any individual patient.
- The interventions should be applied in a stepwise fashion until the patient’s intra-abdominal pressure (IAP) decreases.
- If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm.

Patient has IAP ≥ 12 mmHg

**Begin medical management to reduce IAP (GRADE 1C)**

Measure IAP at least every 4-6 hours or continuously. **Tritrate therapy to maintain IAP ≤ 15 mmHg (GRADE 1C)**

**Evacuate intraluminal contents**

- Insert nasogastric and/or rectal tube
- Abdominal ultrasound to identify lesions
- Initiate gastro-/colo-prokinetic agents (GRADE 2D)
- Minimize enteral nutrition
- Administer enemas (GRADE 1D)
- Consider colonoscopic decompression (GRADE 1D)

**Evacuate intra-abdominal space occupying lesions**

- Ensure adequate sedation & analgesia (GRADE 1D)
- Remove constrictive dressings, abdominal eschars
- Consider reverse Trendelenberg position
- Consider surgical evacuation of lesions (GRADE 1D)
- Consider surgical evacuation of lesions (GRADE 1D)

**Improve abdominal wall compliance**

- Avoid excessive fluid resuscitation (GRADE 2C)
- Aim for zero to negative fluid balance by day 3 (GRADE 2C)
- Consider reverse Trendelenberg position
- Consider neuro-muscular blockade (GRADE 1D)

**Optimize fluid administration**

- Optimize fluid administration
- Goal-directed fluid resuscitation
- Resuscitate using hypertonic fluids, colloids
- Fluid removal through judicious diuresis once stable

**Optimize systematic/regional perfusion**

- Hemodynamic monitoring to guide resuscitation
- Consider hemodialysis/ultrafiltration

**STEP 1**

- Fluid removal through judicious diuresis once stable
- Consider hemodialysis/ultrafiltration

**STEP 2**

- Hemodynamic monitoring to guide resuscitation
- Consider hemodialysis/ultrafiltration
- Fluid removal through judicious diuresis once stable

**STEP 3**

- Consider hemodialysis/ultrafiltration
- Fluid removal through judicious diuresis once stable
- Consider hemodialysis/ultrafiltration

**STEP 4**

If IAP > 20 mmHG and new organ dysfunction / failure is present, patient’s IAH / ACS is refractory to medical management. Strongly consider surgical abdominal decompression (GRADE 1D)
In 2007 the first prospective trial of neuromuscular blockade in IAH management, reporting temporary reductions in IAP in 9 out of 10 patients was published.[37] In a prospective, blinded trial of epidural versus intravenous postoperative pain therapy, the two therapies were demonstrated to have equivalent efficacy for reducing IAP.[38] Drainage of any intra-abdominal fluid collections identified is a safe and effective technique for reducing IAP.[39] Removal of even a few hundred millilitres of fluid can result in a marked decrease in IAP in the presence of significant IAH and loss of abdominal compliance.[36]

*Surgical decompression*

“Open the abdomen and keep it open” was the adage used in the early 2000’s when surgical management was considered the only therapeutic option available for the patient with ACS.[40] Currently there is still no consensus on indications for decompression, both in terms of IAP values and of timing [41], but abdominal decompression can be life-saving when ACS is refractory to non-operative treatment and should then be performed expeditiously.[36]

However, the evidence supporting decompressive laparotomy is limited, as only small non-randomized trials have been performed. In 2010 a study protocol of a multicentre, randomized, controlled study investigating decompressive laparotomy versus percutaneous puncture with placement of abdominal catheter in patients with abdominal compartment syndrome during acute pancreatitis was published.[42] However, the results of this study are still pending. In a recent systematic review and meta-analysis 15 articles and 286 patients (including children) were included.[43] Decompressive laparotomy resulted in a significantly lower IAP and improvement in hemodynamic, respiratory and renal parameters. Mortality after decompressive laparotomy remained high at 49.7% in adult patients. There was a correlation between timing of decompressive laparotomy and mortality. The results of this meta-analysis confirm the recommendation that decompressive laparotomy should be considered when medical options fail, even though it remains unclear which patients would benefit most from decompressive laparotomy.

Among the surgical techniques used to decompress the abdomen are median laparotomy and bilateral transverse subcostal laparotomy.[44] A third, less invasive option is the subcutaneous linea alba fasciotomy (SLAF) where 3 short, horizontal skin incisions are made.[45] This technique is effective in only 50-70% of patients.[44] Few data support the use of other techniques than median laparotomy.[46]

*Open abdomen*

After surgical decompression, the fascial edges of the abdomen are not approximated (laparostomy). The abdominal contents are exposed and protected with a temporary cover.[46] Since the open abdomen may require multiple re-operations and there is a risk
of significant complications such as entero-atmospheric fistulas, loss of abdominal wall domain and large hernias,[47], open abdomen management should immediately follow surgical decompression. The objectives of open abdomen management are to prevent complications and to achieve the earliest possible delayed fascial closure.[48]

Negative pressure wound therapy (NPWT) or vacuum therapy is the procedure currently recommended in treatment of the open abdomen.[49] Roberts et al. performed a systematic review to determine the comparative efficacy and safety of NPWT versus alternate temporary abdominal closure techniques in critically ill adults with open abdominal wounds.[50] In this study of 1018 adults, 2 randomized controlled trials and 9 cohort studies (3 prospective and 6 retrospective) were included. These were all controlled cohort studies. Methodological quality of the prospective studies was moderate and low for most of the retrospective studies. The limited data suggested that NPWT may be linked with improved outcomes versus alternate temporary closing techniques. Cirocchi et al. performed a review and meta-analysis on the effectiveness of NPWT in patients treated with open abdomen.[51] In this study, data of controlled and uncontrolled studies were pooled, 1225 patients were analyzed and 723 (59%) underwent NPWT. Comparing the NPWT group and the group without NPWT, there was no statistically significant difference in fascial closure, postoperative 30-day overall morbidity, postoperative entero-atmospheric fistulae rate (2.1 vs 5.8%, p = 0.57), postoperative bleeding rate and postoperative abdominal abscess rate. There were significant differences in the postoperative mortality rate (28.5% vs 41.4%, p=0.03) and in the length of stay in the ICU. The data should be interpreted with substantial caution given the non-randomized nature of many of the included studies, the small sample sizes and high variability between studies. Furthermore, the pooling of controlled and uncontrolled data is a confounding factor and limits the validity of the conclusions further. Both studies highlight the need for randomized controlled trials with homogeneous inclusion criteria to assess the use of NPWT for the management of open abdomen.

Kirkpatrick et al. performed a single-centre randomized controlled trial where 45 adults with abdominal injury or intra-abdominal sepsis were randomly allocated to ABThera (n=23) or Barker’s vacuum pack (n=22).[52] The cumulative incidence of primary fascia closure at 90 days was similar between groups. However, 90-day mortality was improved in the ABThera group (p=0.04). This survival difference did not seem to be mediated by an improvement in peritoneal fluid drainage, fascia closure rates, or markers of systemic inflammation.

A laparostomy registry entitled Open Abdomen Route by European Registry of Abdominal Wall Hernias has been implemented in 2015 by CAMIN (surgical working group for military and emergency surgery) of DGAV (German Society for General and Visceral Surgery).[48]
ACS was the reason for open abdomen treatment in 18% of the 82 patients. Average duration of open abdomen treatment was 19 days (range 2-120 days). Small bowel fistula occurred in 8 of 82 patients (9.8%); in 2 of these patients after treatment for ACS.

In the International Register of Open Abdomen (IROA) 649 adult patients with open abdomen were registered, 2.9% after ACS. Entero-atmospheric fistulas developed in 8.9% of patients. There was a linear correlation between the days of open abdomen and time to nutrition with development of entero-atmospheric fistulas.[53]

Treatment of entero-atmospheric fistulas is very complex, therefore this complication should be prevented whenever possible after decompressive laparotomy: by performing fascial closure at the earliest possible opportunity.[47]

Future steps
First of all, recent reports indicate that a new disease like COVID-19 and the increasing application of extra-corporeal life support in the ICU, may impact the prevalence of IAH and its complications.[54,55] Further study will follow, but in the meantime IAP monitoring may be considered in these new clinical contexts.

Secondly, IAP monitoring should be easier, accessible and preferably continuous. Ball et al. noted in 2006 that IAP measurement was routine in the critically ill in their hospital and that IAP was considered the “fifth vital sign”. [56] However, this is not standard care in every ICU. When IAP measurements become easier and less time-consuming IAP trends may be easily followed and IAP may indeed become a vital sign. This will be an opportunity to collect an abundance of data, allowing us to learn more about the dynamics of IAP in different clinical settings or during interventions as well as individual differences. The development and distribution of a cost-effective and continuous IAP monitoring device to be used in every ICU should be followed closely.

Thirdly, an update in the 2013 WSACS guidelines should be considered. A future guideline should focus more on the clinical context accompanying IAP increase than on specific numbers, bearing in mind that pathological IAP is a continuum and the clinical context ultimately determines the need for therapy. Furthermore, the guideline should take into account that it is not always possible or practical to measure IAP in the supine position and allow for IAP measurement in different positions. It is standard of care in the ICU to elevate the head-of-bed to 30 degrees to prevent ventilator associated pneumonia.[57] Since IAP increases when the head of bed is elevated, inaccuracies may occur.[9,10] Therefore we propose a simple reference table correcting IAP for head of bed position (Table 5). This reference table should be validated in a prospective study where IAP measurements are repeated in different positions per patient.
Table 5. Proposed reference table for correction of intra-abdominal pressure in different head-of-bed positions

<table>
<thead>
<tr>
<th>Head of bed elevation (degrees)</th>
<th>IAP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-20</td>
<td>Pressure measured</td>
</tr>
<tr>
<td>20-40</td>
<td>Pressure measured - 4</td>
</tr>
<tr>
<td>&gt;40</td>
<td>Pressure measured - 9</td>
</tr>
</tbody>
</table>

IAP = intra-abdominal pressure

Finally, the consequence of the heterogeneous patient population at risk for ACS in combination with the low incidence of ACS is that intervention studies can only be performed in large multi-centre studies. A multi-centre study which randomises between early decompression surgery with open abdomen management and bundles of non-operative treatment in ACS patients might answer some of the many remaining important clinical questions. Furthermore, the effect of treatment of IAH on outcome needs further study. IAH has consistently been associated with morbidity and mortality in observational studies, but it is uncertain whether treating or preventing this condition improves patient outcomes. A large multi-centre trial randomizing patients at risk of IAH between bundles of non-operative management in IAH (but not ACS) versus no treatment of IAH should be performed as soon as possible. We recommend this study is performed in a homogeneous patient population with a relatively high prevalence of IAH, for example in patients with severe acute pancreatitis.

Conclusion

Pathological IAP is a continuum ranging from mild IAP elevations without clinically significant adverse effects to substantial increases in IAP with serious consequences to all organ systems in the body. In ACS increased pressure within the abdominal compartment leads to decreased blood flow and ultimately to multi-organ failure. IAP monitoring should be performed in all patients at risk of IAH. Although continuous IAP monitoring is feasible, this is currently not standard practice. There are a number of effective non-operative medical interventions that may be performed early in the patient’s course to reduce IAP and decrease the need for surgical decompression. Abdominal decompression can be life-saving when ACS is refractory to non-operative treatment and should be performed expeditiously. The objectives of open abdomen management are fistula prevention and to achieve delayed fascial closure at the earliest possible time. There is still a lot to learn and change. First and foremost, the 2013 WSACS guidelines should be updated and multicentre studies should evaluate the effect of IAH treatment on patient outcome.
References


