Intra-abdominal hypertension and abdominal compartment syndrome in critical illness
Smit, Marije

DOI:
10.33612/diss.192064066

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2021

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):

Copyright
Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment.

Take-down policy
If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): http://www.rug.nl/research/portal. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.
CHAPTER 1

General introduction
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]

Definitions according to the World Society of Abdominal Compartment Syndrome (WSACS) are shown in Table 1.

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

<table>
<thead>
<tr>
<th>Condition</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal intra-abdominal pressure (IAP)</td>
<td>Is approximately 5-7 mmHg in critically ill adults</td>
</tr>
<tr>
<td>Intra-abdominal hypertension (IAH)</td>
<td>Is defined as a sustained IAP ≥ 12 mmHg</td>
</tr>
<tr>
<td>Abdominal Compartment Syndrome (ACS)</td>
<td>Is defined as a sustained IAP &gt; 20 mmHg associated with new organ dysfunction or failure</td>
</tr>
</tbody>
</table>

IAP is the steady-state pressure within the abdominal cavity.

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)

Historical perspective

IAP was studied long before the first intensive care unit (ICU) was established in 1953.[3] In a review of the literature published in 1911, Emerson noted that there are many points of view from which the matter of IAP has been approached by clinicians. Marey pointed out that the effects of respiration on the thorax are inverse of those present in the abdomen in 1863, but he did not describe any tests. Braune tested abdominal pressure by the use of rectal bougies in 1865 and concluded that it varied according to the amount of voluntary abdominal contraction. Furthermore, he noted that abdominal pressure was lowest in the horizontal, and highest in the vertical position.[4] Wendt performed a further study of rectal pressures in 1876 and concluded that an increase in abdominal pressure was associated with a decrease in urine output.[5] Moritz (in 1895) tested pressures by a bougie balloon in the stomach connected with a recording water manometer. He noted that accurate IAP can be determined only by a cannula in the peritoneal cavity.[4]

Almost 100 years later Kron, who is often credited for “rediscovering” the study of IAP, described a series of 11 postoperative patients who developed oliguria due to elevation of IAP above 30 mmHg in 1984. Operative re-exploration and decompression in seven patients resulted in immediate diuresis. Four patients who were not re-explored developed acute kidney injury (AKI) and died. Kron concluded that an IAP above 25 mmHg in a postoperative patient with oliguria and an adequate blood volume is an indication for re-exploration and decompression of the abdomen. The IAP level of 25 mmHg seems arbitrarily chosen and is unsupported by his data which illustrate that decompression was performed when IAP increased above 30 mmHg. Kron also described his technique for measuring IAP through a transurethral catheter.[6] However, Richards actually already described a series of 4 patients with postoperative
anuric renal failure associated with tense abdominal distention in 1983. The etiology was unknown, but all 4 patients had bleeding complications and AKI improved after reoperation. No measurements of IAP were obtained in these patients, but a parallel was drawn to a study in dogs, where the level of IAP was the cause of renal failure. When IAP increased above 20, anuria occurred. Reduction of IAP immediately increased urine output.[7] The term (intra-)abdominal compartment syndrome was first used in 1989 by Fietsam et al.[8] Four patients with ruptured abdominal aneurysms developed increased IAP after repair. Clinical signs included increased ventilatory pressure, increased central venous pressure, and decreased urinary output associated with massive abdominal distension not due to bleeding. Decompressive laparotomies were performed in the ICU with placement of Marlex mesh. In two additional patients, at the completion of the aneurysmectomy the abdominal wall was left open with interposition of Marlex mesh. Opening the abdomen was associated with improvements in central venous pressure, urinary output, ventilator pressure, arterial carbon dioxide tension and oxygenation. The authors concluded that recognition and treatment of this condition by opening the abdominal wound or delayed closure may affect outcome in some cases.

The accuracy and reproducibility of IAP measurements worldwide during the 1990’s and early 2000’s were variable and threshold values to define IAH and ACS lacked consensus.[9-11] Some used the terms IAH and ACS interchangeably. Hence it was difficult to compare different studies.

IAH was usually defined between 12 and 25 mmHg, depending on the effects on organ function. The pressure at which organ dysfunction becomes apparent in the majority of patients is most appropriate in defining IAH.[12] However, the exact IAP that defines IAH has long been a subject of debate. For ACS, a definition incorporating one numerical value was needed in order to improve comparisons between studies and to design future clinical trials. Although the critical IAP that defines ACS was subject to debate, of greater importance than one exact numerical value was the development of organ dysfunction and failure. Consensus definitions were discussed at the 2004 International ACS Consensus Definitions Conference and the subsequent article “Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions” listed 12 definitions. This included definitions for IAH and ACS. Since this article reflects a consensus meeting of experts in the field, some of the statements are based on expert opinion only.[12]

The World Society of the Abdominal Compartment Syndrome (WSACS) was founded in 2004 and its mission has been to promote research, foster education, and improve the survival of patients with IAH and/or ACS. The development of uniform definitions for IAH and ACS as well as standardized techniques for IAP monitoring to facilitate research
and improve patient care were important early accomplishments of the WSACS.[12] Since 2004, WSACS developed recommendation statements, patient care algorithms and guidelines. In 2013 “Intra-abdominal Hypertension and Abdominal Compartment Syndrome: updated consensus definitions and clinical practice guidelines from WSACS” was published.[13] The definitions of IAH and ACS remained unchanged. (Table 1) To better reflect the full scope of the Society’s interests and activities, its name was changed to WSACS – the Abdominal Compartment Society in 2017.[14]

Aims of this thesis

1. To investigate the prevalence and outcome of increased IAP in critically ill patients.
2. To investigate the relationship between IAP, biomarkers of acute kidney injury (AKI) and intestinal injury.
3. To explore the relationship between IAP, body mass index (BMI) and central obesity.
Outline of this thesis

Chapter 2 is a narrative review in which we aim to provide a comprehensive overview of current insights into IAP monitoring, IAH and 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. Future steps are discussed including propositions of what a future guideline should focus on.

In chapter 3 the results of a retrospective study of ACS and intra-abdominal ischemia performed in 59 patients with severe acute pancreatitis are outlined.

In chapter 4 the results of a human model of IAH are reported. Markers of acute systemic inflammation and AKI were analyzed in a study of 50 living kidney donors randomly assigned between hand-assisted laparoscopic nephrectomy and open nephrectomy.

Chapter 5 outlines a prospective observational study in 186 cardiothoracic surgical patients which aimed to investigate the relationship between IAP, central obesity and BMI and the relationship between IAP, inflammation and renal function.

Chapter 6 and chapter 7 are based on the results of a prospective, observational, single-centre cohort study where 503 patients with high risk of IAH admitted to the ICU were included. Chapter 6 describes the prevalence and outcome of IAH and ACS. In chapter 7 biomarkers of AKI and intestinal injury in relation to IAH and ACS are analyzed.

In chapter 8 the studies included in this thesis are summarized and reviewed. Future perspectives are discussed.
References


