Part One: Pathophysiology and Pressure Measurement of Pelvic and Abdominal Compartment Syndromes

Introduction

A Compartment Syndrome (CS) exists when the increased pressure in a closed anatomic space threatens the viability of surrounding tissue. Within the body there are 4 compartments, the head, the chest, the abdomen and the extremities. Within each compartment; an individual organ or a region with multiple organs can develop a CS. A CS is not a disease, as such it can have many causes and it can develop within many disease processes.

Scalea et al. was the first to allude to the term Multiple Compartment Syndrome (MCS) in a study of 102 patients with increased intra-abdominal (IAP), intrathoracic, and intracranial pressure (ICP) after severe brain injury. Since the term multi or multiple CS is mostly often used in relation to multiple limb trauma with CS requiring fasciotomy, the term Polycompartment Syndrome was finally coined in order to avoid confusion. Part one of this article will focus on Pelvic Compartment Syndrome, Abdominal Compartment Syndrome (ACS) and intra-abdominal pressure measurement as well as briefly outlining the recently published consensus definitions for intra-abdominal hypertension (IAH) and ACS.

Pathophysiology
The increased compartment pressure (CP) will exert a direct force on the original compartment and its contents by increasing venous resistance and decreasing perfusion pressure, as well as on distant compartments (Figure 1). The impact on end-organ function and viability within and outside the original cavity can be devastating.

ACS: abdominal compartment syndrome
CCS: cardiac compartment syndrome
ECS: extremity compartment syndrome
HCS: hepatic compartment syndrome
ICS: intracranial compartment syndrome
RCS: renal compartment syndrome
OCS: orbital compartment syndrome
PCS: pelvic compartment syndrome
TCS: thoracic compartment syndrome

Table 1. Consensus definitions

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<tr>
<th>Definition</th>
<th>Description</th>
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<tr>
<td>Definition 1</td>
<td>IAP is the steady-state pressure concealed within the abdominal cavity.</td>
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<td>Definition 2</td>
<td>APP = MAP – IAP</td>
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<td>Definition 3</td>
<td>FG = GFP – PTP = MAP – 2 * IAP</td>
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<td>Definition 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>
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<td>Definition 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>
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<td>Definition 6</td>
<td>Normal IAP is approximately 5-7 mmHg in critically ill adults.</td>
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<td>Definition 7</td>
<td>IAH is defined by a sustained or repeated pathologic elevation of IAP</td>
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**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 abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention.

**Definition 11**  
Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region.

**Definition 12**  
Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

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The World Society on 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 with an interest in intra-abdominal hypertension (IAH) and ACS. Recently the first consensus definitions have been published. Table 1 summarises these consensus definitions: a sustained increase in IAP equal to or above 12 mmHg defines IAH where ACS is defined by a sustained IAP above 20 mmHg with new onset organ failure. While Table 2 lists some possible risk factors for the development of IAH.

**Table 2. Risk factors for the development of IAH and ACS**

**A. Related to diminished abdominal wall compliance**  
- Mechanical ventilation, especially fighting with the ventilator and the use of accessory muscles  
- Use of positive end expiratory pressure (PEEP) or the presence of auto-PEEP  
- Basal 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, gastroschisis or omphalocoele
• Burns with abdominal eschars

B. Related to increased intra-abdominal contents

• Gastroparesis
• Gastric distention
• Ileus
• Volvulus
• Colonic pseudo-obstruction
• Abdominal tumour
• Retroperitoneal/abdominal wall hematoma
• Enteral feeding
• Intra-abdominal or retroperitoneal tumour
• Damage control laparotomy

C. Related to abdominal collections of fluid, air or blood

• Liver dysfunction with ascites
• Abdominal infection (pancreatitis, peritonitis, abscess,…)
• Haemoperitoneum
• Pneumoperitoneum
• Laparoscopy with excessive inflation pressures
• Major trauma
• Peritoneal dialysis

D. Related to capillary leak and fluid resuscitation

• Acidosis* (pH below 7.2)
• Hypothermia* (core temperature below 33°C)
• Coagulopathy* (platelet count below 50000/mm3 OR an activated partial thromboplastin time (APTT) more than 2 times normal OR a prothrombin time (PTT) below 50% OR an international standardised ratio (INR) more than 1.5)
• Polytransfusion / trauma (> 10 units of packed red cells / 24 hours)
• Sepsis (as defined by the American – European Consensus Conference definitions)
• Severe sepsis or bacteraemia
• Septic shock
• Massive fluid resuscitation (> 5 liters of colloid or > 10L 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 (129, 130).

Table 3. Treatment options for compartment syndrome

1. Improvement of compartment wall compliance

• Sedation
• Pain relief (not fentanyl!)
• Neuromuscular blockade
• Body positioning
• Negative fluid balance
• Skin pressure decreasing interfaces
• Weight loss
• Percutaneous abdominal wall component separation
• Escharotomies

2. Evacuation of intra-compartmental contents

• Gastric tube and suctioning
• CSF, ascites, pleural or pericardial drainage
• Rectal tube and enemas
• Chest tube and suctioning
• Endoscopic decompression of large bowel
• Colostomy or ileostomy
• CT- or US-guided aspiration of abscess
• CT- or US-guided aspiration of hematoma
• Pericardectomy

3. Correction of capillary leak and positive fluid balance

• Albumin in combination with diuretics (furosemide)
• Correction of capillary leak (antibiotics, source control,…)
• Colloids (Hypertonic-Voluven® instead of cristalloids)
• Dobutamine (not dopamine!)
• Dialysis or CVVH with ultrafiltration
• Ascorbinic acid in burn patients

4. Specific therapeutic interventions

• Continuous negative external pressure (VAC®)
• Targeted compartment perfusion pressure

5. Rescue therapy

• ICS: decompressive craniectomy
• ACS: decompressive laparotomy
• TCS: decompressive sternotomy
• ECS: decompressive fasciotomy
• PCS: decompressive gluteal fasciotomy
• RCS: renal decapsulation
• HCS: hepatic decapsulation
• CCS: decompressive pericardiotomy
• OCS: orbital decompression

Pelvic Compartment Syndrome – PCS
In the pelvic region three major compartments (gluteus medius/minimus compartment, gluteus maximus compartment, and iliopsoas compartment) can be distinguished from the smaller compartment of the tensor fasciae latae muscle. Pelvic compartment syndromes are rare and a clear history of trauma is often lacking. The PCS is often associated with drug and alcohol abuse, infections (necrotising fasciitis) and the use of anticoagulant therapy. Increased pelvic CP may eventually increase IAP and affect kidney function due to bilateral ureteral obstruction and renal failure caused by a massive intrapelvic haematoma with increased retroperitoneal pressure. Decompressive fasciotomy of the gluteal compartment is the treatment of choice.

Abdominal Compartment Syndrome
In many ways the abdomen could be compared to the head with its’ partially rigid sides (spine and pelvis), not unlike the skull, an anchorage above (costal arch) and partially flexible sides (abdominal wall and diaphragm). Both are filled with organs: small and large intestine, liver, kidneys, spleen in the abdomen and like the head contains the brain which is surrounded by a third space filled with peritoneal fluid like the cerebrospinal fluid (CSF) and perfused by the mesenteric arteries with a mesenteric and venous capacitance blood volume. However, the abdomen is further complicated by the movable diaphragm, the shifting costal arch, the contractions of the abdominal wall, and the intestines that may be empty or filled with air, liquid or fecal mass.
The term ACS was first used by Fietsam et al. in the late 1980’s to describe the pathophysiologic alterations resulting from IAH secondary to aortic aneurysm surgery: “In four patients that received more than 25 litres of fluid resuscitation increased IAP developed after aneurysm repair. It was manifested by increased ventilatory pressure, increased central venous pressure, and decreased urinary output. This set of findings constitutes an abdominal compartment syndrome caused by massive interstitial and retroperitoneal swelling... Opening the abdominal incision was associated with dramatic improvements.”

**Monitoring of Intra-abdominal Pressure**

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. IAP increases with inspiration (diaphragmatic contraction) and decreases with expiration (diaphragmatic relaxation). In the strictest sense, normal IAP ranges from zero to 5 mmHg. Certain physiologic conditions, however, such as morbid obesity, ovarian tumours, 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 pathophysiology. In contrast, children commonly demonstrate low IAP values. The clinical importance of any IAP must be assessed in view of the baseline steady-state IAP for the individual patient. The gold standard IAP measurement method is via the bladder with a FoleyManometer (Holtech Medical, Copenhagen, Denmark) or an AbViser valve (Wolfe-Tory, Utah, USA), while continuous IAP measurement can be performed via a balloon-tipped catheter in the stomach (Spiegelberg, Hamburg, Germany or CiMON, Pulsion Medical Systems, Munich, Germany).

**Abdominal Perfusion Pressure (APP) Measurement**

Analogous to the widely accepted and clinically utilised 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 by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP). - 

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

**Clinical Management**

The management of patients with polycompartment syndrome is based on 3 principles:
- Specific medical and surgical procedures to reduce the compartment pressure (Table 3)
  - Improvement of compartment wall compliance
  - Evacuation of intra-compartment contents
  - Correction of capillary leak and positive fluid balance
  - Specific treatments
  - Rescue treatments
- General and organ support (intensive care) of the critically ill patient
- Optimisation and prevention of specific adverse events after surgical decompression (ischaemia/reperfusion)

**Conclusion**

First suggested in 1863 by Marey, ACS is the end-stage of the physiologic sequellae of increased IAP, termed IAH. Recent observations suggest an increasing frequency of this complication in all types of patients. Even chronic elevations of IAP seem to affect the various organ systems in the body. The presence of IAH and ACS are significant causes of organ failure, increased resource utilisation, decreased economic productivity, and increased mortality among a wide variety of patient populations. Despite its obvious clinical implications, too little attention is paid to IAP, IAH and ACS. Although there is much research interest in the subject, there are still too many unanswered questions, which cloud our understanding of the pathophysiology of this syndrome.

In the second part of this article, in the Autumn Issue of ICU Management, we will discuss the remaining compartment syndromes – Orbital (OCS), Intracranial (ICS), Thoracic (TCS), Cardiac (CCS), Limb or extremity (ECS), Hepatic (HCS), and Renal RCS as well as their interactions.

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