ABDOMINAL COMPARTMENT SYNDROME
AND INTRA-ABDOMINAL MONITORING

A LEARNING RESOURCE FOR INTENSIVE CARE NURSING STAFF

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Abdominal Compartment Syndrome:

Introduction:

Abdominal compartment syndrome (ACS) refers to organ dysfunction caused by intra-abdominal hypertension. It may be under-recognised because it primarily affects patients who are already quite ill and whose organ dysfunction may be incorrectly ascribed to progression of the primary illness. Since treatment can improve organ dysfunction, it is important that the diagnosis be considered in the appropriate clinical situation. ACS occurs when intra-abdominal pressure increases to the point that it exceeds pressure in the inferior vena cava and prevents venous return to the heart.

Definitions:

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome are distinct clinical entities and should not be used interchangeably.

Abdominal compartment syndrome:

For research purposes, ACS is defined as a sustained intra-abdominal pressure >20mmHg that is associated with new organ dysfunction. For clinical purposes, ACS is better defined as new organ-dysfunction without a strict intra-abdominal pressure threshold, since no intra-abdominal pressure can predictably diagnose ACS in all patients.

Aetiology and risk factors:

ACS can be classified as primary or secondary. Primary ACS is due to injury or disease in the abdomino-pelvic region e.g. abdominal trauma; haemoperitoneum; pancreatitis. Intervention (surgical or radiological) of the primary condition is often needed. Secondary ACS refers to conditions that do not originate in the abdomen or pelvis e.g. fluid resuscitation; sepsis; burns. Recurrent ACS defines a condition in which ACS develops again following previous surgical or medical treatment of primary or secondary ACS.

ACS generally occurs in patients who are critically ill due to any of a wide variety of medical and surgical conditions. Some of these include:

- Trauma – injured patients in shock who require aggressive fluid resuscitation are at risk for ACS.
- Burns – patients with severe burns (>30% total body surface area) with or without concomitant trauma are also at risk for ACS. Importantly, ACS must be distinguished from other intra-abdominal problems that occur in these critically ill patients e.g. necrotising enterocolitis, ischaemic bowel.
- Liver transplantation – a prospective cohort study found intra-abdominal hypertension (intra-abdominal pressure >25mmHg) following liver transplantation in 30% of patients.
- Abdominal conditions – massive ascites, abdominal surgery or intraperitoneal bleeding can increase intra-abdominal pressure.
- Retroperitoneal conditions – retroperitoneal pathologies, such as ruptured abdominal aortic aneurysm, pelvic fracture with bleeding, and pancreatitis, can lead to compartment syndrome.
- Medical illness – conditions that require fluid resuscitation e.g. sepsis and are associated with third spacing of fluids and tissue oedema can increase intra-abdominal pressure.
- Post-surgical patients – patients undergoing operations in which they are given large volume resuscitation, particularly with crystalloid in the face of haemorrhagic or septic shock, are at risk for ACS.

The development of secondary ACS is often related to the need for and extent of volume resuscitation. Careful attention needs to be paid to the amount of fluid being administered and alterations in fluid management may be needed in patients who are exhibiting early signs/symptoms of ACS.
Physiologic consequences: Intra-abdominal hypertension (IAH) can impair the function of nearly every organ system, thereby causing ACS.

- **Cardiovascular:** IAH decreases cardiac output by impairing cardiac function and reduced venous return.

- **Pulmonary:** Mechanically ventilated patients with IAH have increased peak inspiratory and mean airway pressures which can cause alveolar barotrauma. They also have reduced chest wall compliance and spontaneous tidal volumes, which combine to cause arterial hypoxaemia and hypercarbia. Pulmonary infection is more common among patients with IAH.

- **Renal:** Several mechanisms contribute to renal impairment in patients with IAH. Renal vein compression increases venous resistance, which impairs venous drainage. This appears to be the major cause of renal impairment. Renal artery vasoconstriction is induced by the sympathetic nervous and rennin-angiotensin systems, which are stimulated by the fall in cardiac output. The end result is progressive reduction in glomerular perfusion and urine output. Oliguria generally develops at an intra-abdominal pressure of approximately 15mmHg, while anuria usually develops at an intra-abdominal pressure of approx 30mmHg.

- **Gastro-intestinal:** The gut appears to be one of the organs most sensitive to increases in intra-abdominal pressure. Mesenteric blood flow was reduced at an intra-abdominal pressure as low as 10mmHg in one animal study. Intestinal mucosal perfusion is decreased at an intra-abdominal pressure of approx 20mmHg according to both animal and human studies. Celiac artery and superior mesenteric artery blood flow are decreased at an intra-abdominal pressure of approx 40mmHg, according to one animal study.

The impact of IAP on mesenteric perfusion seems to be greater among patients who had haemorrhage or are hypovolaemic. IAH also compresses thin-walled mesenteric veins, which impairs venous flow from the intestine and causes intestinal oedema. The intestinal swelling further increases IAP, initiating a vicious cycle. The end result is worsened hypoperfusion, bowel ischaemia, decreased intramucosal pH, and lactic acidosis.

Hypoperfusion of the gut may incite loss of the mucosal barrier, with subsequent bacterial translocation, sepsis, and multiple system organ failure. Supporting this notion, bacterial translocation has shown to occur at an IAP of only 10mmHg in the presence of haemorrhage.

- **Hepatic:** The liver’s ability to remove lactic acid is impaired by increases of IAP as small as 10mmHg. This occurs even in the presence of a normal cardiac output and mean arterial blood pressure. Thus, lactic acidosis may clear more slowly than expected despite adequate resuscitation.

- **Central nervous system:** Intracranial pressure (ICP) transiently increases during the short-lived elevation of IAP that occurs with coughing, defecating or emesis. ICP similarly appears to be elevated in the presence of persistent IAH. The elevated ICP is sustained as long as IAH exists, which can lead to a critical decrease in cerebral perfusion and progressive cerebral ischaemia.
Clinical presentation:
It is desirable to recognise intra-abdominal hypertension (IAH) early, so it can be treated before progressing to ACS.

Symptoms:
Most patients who develop ACS are critically ill and unable to communicate. The rare patient who is able to convey symptoms may complain of malaise, weakness, lightheadedness, dyspnoea, abdominal bloating or abdominal pain.

Physical signs:
Nearly all patients with ACS have a tensely distended abdomen. Despite this, physical examination of the abdomen is a poor predictor of ACS. Progressive oliguria and increased ventilatory requirements are also common in patients with ACS. Other findings may include hypotension, tachycardia, an elevated JVP, jugular distension, peripheral oedema, abdominal tenderness or acute pulmonary compensation. There may also be evidence of hypoperfusion, including cool skin, restlessness, or lactic acidosis.

Measurement of intra-abdominal pressure:
Intra-abdominal pressure can be measured indirectly via the bladder. Measurement of bladder pressure is the standard method to screen for intra-abdominal hypertension and ACS.

Equipment required for monitoring intra-abdominal pressure:
- Dressing trolley
- Dressing pack
- A single transducer set
- 500ml 0.9% Sodium Chloride
- Pressure infusor bag
- 25ml 0.9% Sodium Chloride for instillation (from 50ml saline bag)
- 30ml leur-lock syringe
- Sterile gloves
- Green needle
- Chlorhexidine wipe
- Clamps
**Procedure for Monitoring Intra-abdominal Pressure:**

In the measurement of intra-abdominal pressure (IAP), the bladder is partially infused with 25ml of fluid which is generally taken to be the compliant range of the bladder in adult patients. The current procedure involves using a Foley catheter with the patient positioned supine.

- Explain the procedure to the patient.
- Ensure the patient is **lying supine and flat**.
- Prime the transducer set with 0.9% sodium chloride & insert the diaphragm into the transducer.
- Place bag of 500ml saline inside pressure infuser bag and inflate to 300mmHg.
- Ensure the **transducer position is maintained at the level of the mid-axillary line**.
- Zero the transducer system on the monitor.
- Apply clamp on drainage tubing: clamp immediately below the urinary catheter sampling port.
- Open dressing pack and place the following on to the sterile field: sterile gloves; 50ml bag 0.9% sodium chloride; 30ml leur-lock syringe, chlorhexidine wipe and green needle for drawing up saline.
- Wash hands and put on sterile gloves.
- Using the 30ml syringe, draw up **25ml** saline.
- Clean the urinary catheter sampling port with the chlorhexidine wipe. With the needle still attached to the 30ml syringe, insert the needle into the catheter sampling port and **slowly** instill the saline.
- Using an alcohol swab, wipe the sample port and insert the needle from the transducer set.
- Abdominal pressure should now appear on the patient’s monitor.
- IAP is measured at 30 to 60 seconds to allow for bladder muscle relaxation. Measure pressure at **end** of expiration.
- Once IAP pressure has been obtained, remove the green needle from the sample port and safely dispose of all sharps as per hospital policy.
- Remove the clamp from the tubing and allow urine to run freely.
- Remember to discount 25ml from the next urine volume to account for the saline instilled.
- Document the abdominal pressure on the patient’s monitoring chart and report findings to medical staff. Intra-abdominal hypertension is defined as sustained intra-abdominal pressure > 12mmHg.
- Check with medical staff if the procedure needs to be repeated later.

**Important:**

IAP should be expressed in mmHg and measured at **end-expiration** in the **complete supine flat** position after ensuring that abdominal muscle contractions are absent and with the **transducer zeroed at the level of the midaxillary line**.
Intra-abdominal hypertension (IAH) is defined as a sustained intra-abdominal pressure >12 mmHg.

Abdominal compartment syndrome (ACS) is defined as a sustained intra-abdominal pressure >20 mmHg that is associated with new organ dysfunction.

Increasing Physiologic Compromise
IAP 12 – 15 mmHg

- Increased Systemic Vascular Resistance (SVR)
- IAP against diaphragm makes breathing difficult
- Decreased gut perfusion
- Increased Ischemia
- Decreased wound perfusion (poor healing)
- Decreased Cardiac Output
- Decreased preload
- Vena caval compression
- Decreased Urinary Output (UOP)
- Increased SIRS/Cytokine release
- Lower extremity Venous Pooling (DVT risk)

Onset of Multiple Organ Dysfunction Syndrome (MODS)
IAP > 20 mmHg

- Increased peak pressure, difficult ventilation and oxygenation, VIL/ARDS
- Increased gut ischemia, Impending necrosis
- Brain swelling and ischemia
- Cardiovascular instability
- Vena caval flattening
- Anuria/Acute Renal Failure (ARF)
- Further worsening of acidosis
Intra-abdominal hypertension (IAH) is defined as a sustained intra-abdominal pressure >12 mmHg.

Document IAP on ICU chart.

Inform medical staff if IAP > 12mmHg.

<table>
<thead>
<tr>
<th>Bladder Pressure</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-15mmHg</td>
<td>Monitor</td>
</tr>
<tr>
<td>16-25mmHg</td>
<td>Monitor</td>
</tr>
<tr>
<td>26-35mmHg</td>
<td>Decompression</td>
</tr>
<tr>
<td>&gt;35mmHg</td>
<td>Decompression &amp; re-exploration</td>
</tr>
</tbody>
</table>

Reference values obtained from: Meldrum et al., Am J Surg 1997
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

Measure IAP/APP at least every 4-6 hours or continuously.
Titrate therapy to maintain IAP ≤ 15 mmHg and APP 60 mmHg

**Evacuate intraluminal contents**

Insert nasogastric and/or rectal tube

Initiate gastro-/colonic prokinetic agents

Minimize enteral nutrition

Administer enemas

Consider colonoscopic decompression

Discontinue enteral nutrition

**Evaluate intraperitoneal space occupying lesions**

Abdominal ultrasound to identify lesions

Abdominal computed tomography to identify lesions

Percutaneous catheter drainage

Consider surgical evacuation of lesions

**Improve abdominal wall compliance**

Ensure adequate sedation & analgesia

Avoid prone position, head of bed > 20 degrees

Consider reverse Trendelenberg position

Consider neuromuscular blockade

**Optimize fluid administration**

Avoid excessive fluid resuscitation

Resuscitate using hypertonic fluids, colloids

Fluid removal through judicious diuresis once stable

**Optimize systemic/regional perfusion**

Aim for zero to negative fluid balance by day 3

Resusciate using hypertonic fluids, colloids

Fluid removal through judicious diuresis once stable

**Goal-directed fluid resuscitation**

Maintain abdominal perfusion pressure (APP) ≥ 60 mmHg

Hemodynamic monitoring to guide resuscitation

Vasopressive medications to keep APP ≥ 60 mmHg

If IAP > 25 mmHg (and/or APP < 50 mmHg) and new organ dysfunction/failure is present, patient’s IAH/ACS is refractory to medical management. Strongly consider surgical abdominal decompression.

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INTRA-ABDOMINAL HYPERTENSION (IAH) ASSESSMENT ALGORITHM

- Patients should be screened for IAH/ACS risk factors upon admission and with new or progressive organ failure
- If two or more risk factors are present, a baseline IAP measurement should be obtained.
- If IAH is present, serial IAP measurements should be performed throughout the patient’s critical illness.

**Patient has TWO or more risk factors for IAH/ACS upon either ICU admission or in the presence of new or progressive organ failure**

**Measure patient’s IAP to establish baseline pressure**

IAP measurements should be:
1. Expressed in mmHg (1 mmHg = 1.36 cm H₂O)
2. Measured at end-expiration
3. Performed in the supine position
4. Zeroed at the iliac crest in the mid-axillary line
5. Performed with an installation volume of no greater than 25 mL of saline [1 mL/kg for children up to 20 kg] (for bladder technique)
6. Measured 30-60 seconds after installation to allow for bladder detrusor muscle relaxation (for bladder technique)
7. Measured in the absence of active abdominal muscle contractions

**Sustained IAP ≥ 12 mmHg?**

- **YES**
  - Patient has IAH
  - Notify patient’s doctor of elevated IAP. Proceed to IAH/ACS management algorithm.

- **NO**
  - Patient does not have IAH
  - Observe patient. Recheck IAP if patient deteriorates clinically.

**Risk Factors for IAH / ACS**

1. Diminished abdominal wall compliance
   - Acute respiratory failure, especially with elevated intrathoracic pressure
   - Abdominal surgery with primary fascial or tight closure
   - Major trauma/burns
   - Prone positioning, head of bed > 30 degrees
   - High body mass index (BMI), central obesity

2. Increased intra-luminal contents
   - Gastroperistasis
   - Ileus
   - Colonic pseudo-obstruction

3. Increased abdominal contents
   - Hemoperitoneum/pneumoperitoneum
   - Ascites/liver dysfunction

4. Capillary leak/fluid resuscitation
   - Acidosis (pH < 7.2)
   - Hypotension
   - Hypothermia (core temperature < 33°C)
   - Polytransfusion (>10 units of blood/24 hrs)
   - Coagulopathy (platelets < 55,000/mm² OR prothrombin time (PT) > 15 seconds OR partial thromboplastin time (PTT) > 2 times normal OR international standardised ratio (INR) > 1.5)
   - Massive fluid resuscitation (> 5 L/24 hours)
   - Pancreatitis
   - Oliguria
   - Sepsis
   - Major trauma / burns
   - Damage control laparotomy

**IAH Grading**

- Grade I: IAP 12-15 mmHg
- Grade II: IAP 16-20 mmHg
- Grade III: IAP 21-25 mmHg
- Grade IV: IAP ≥ 25 mmHg

*Grade IV is IAP ≥ 25 mmHg*

**Abbreviations**

IAH - intra-abdominal hypertension
ACS - abdominal compartment syndrome
IAH - intra-abdominal pressure

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