

## Evaluation & Management of Abdominal Compartment Syndrome

### Goal

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- Define abdominal compartment syndrome and diagnostic dilemmas
- Discuss initial management strategies
- Effectively identify patients with a high probability of requiring surgical decompression

### Definitions

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**IAH** – Intraabdominal hypertension

**IAP** – Intraabdominal pressure

**ACS** – Abdominal compartment syndrome

**APP** – Abdominal perfusion pressure

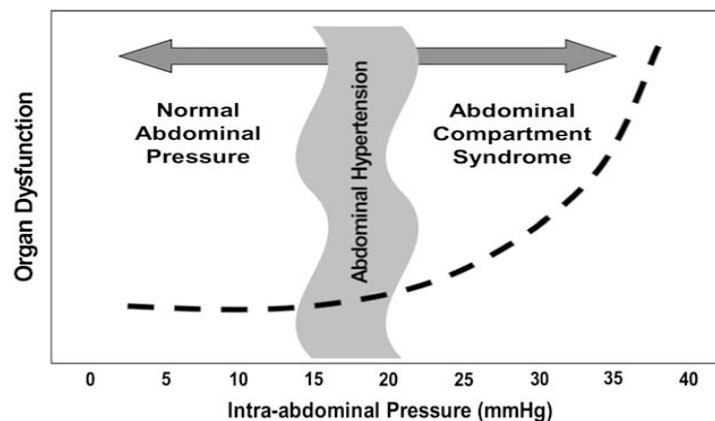
**MAP** – Mean arterial pressure

**APP = MAP – IAP**, APP > 60 mmHg associated with improved survival from IAH and ACS

**ACS** – IAH induced new organ dysfunction without a strict IAP threshold, since no IAP can accurately diagnose ACS in all patients. In practical terms, ACS is often defined as IAP >20 mmHg (with or without APP < 60 mmHg) and evidence of new organ dysfunction.

**Primary ACS** – Due to disease or injury in the abdomino-pelvic region, such as abdominal trauma, hemoperitoneum, and pancreatitis, frequently requires early surgical or interventional radiological intervention.

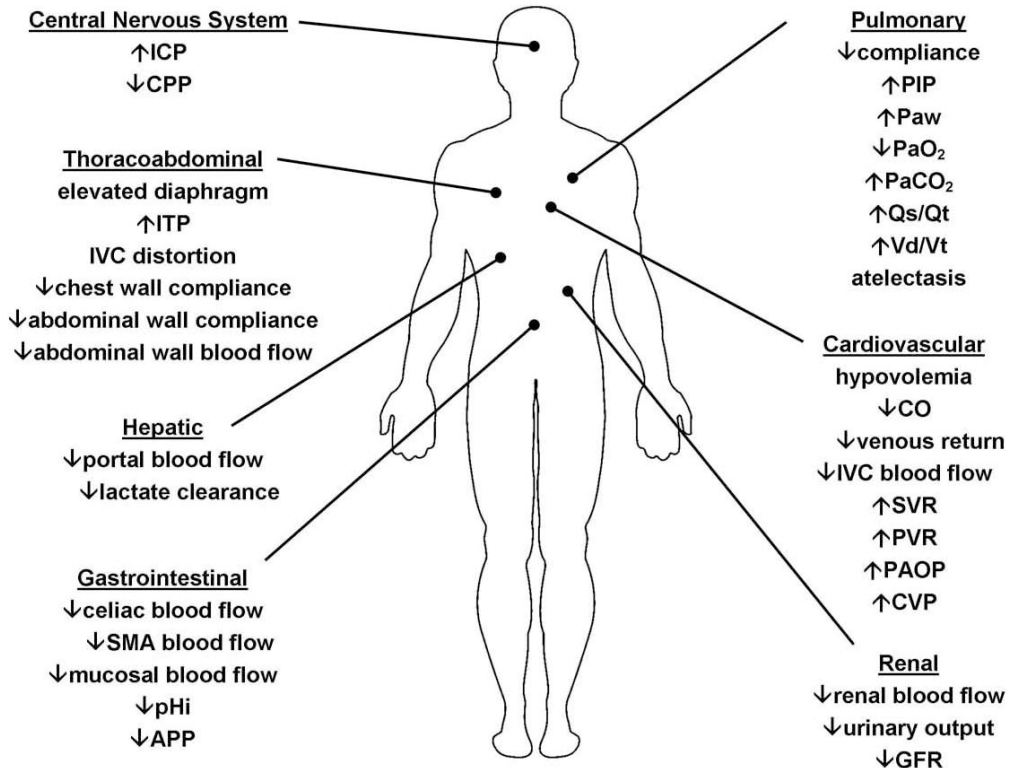
**Secondary ACS** – Due to conditions that do not originate within the abdomen such as fluid resuscitation, burns, and sepsis.



**Reference:** Scheppach et al. **Abdominal Compartment Syndrome**. Best Practice and Research Clinical Gastroenterology 2009; 25-33

This serves as a guideline only. Individual circumstances can vary and clinical judgment should always be used. When in doubt, consult with the Trauma Attending On-call.

## Physiological Consequences of IAH



**Reference:** Cheatham et al. **Abdominal Compartment Syndrome: Pathophysiology and Definitions.** Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine 2009; 10-17

**Cardiovascular:** Impaired venous return to the right heart and increased left ventricular afterload both lead to decreased cardiac output, lower organ perfusion pressure and blood pressure. This leads to the need for more fluids, further worsening any existing intra abdominal hypertension.

**Respiratory:** Displacement of the diaphragm cranially, impairing thoracic compliance and causing basal atelectasis, functionally similar to a restrictive ventilation disorder. Oxygenation is further impaired by ventilation perfusion mismatch. Part of this can be overcome with high levels of PEEP.

**Cerebral:** Elevated intra-thoracic pressure causes increased jugular venous pressure, which may result in increased intra-cranial pressure and cerebral edema. This may warrant earlier decompression in patients with traumatic brain injuries.

**Renal:** Oliguria ensues secondary to decreased venous outflow, reduced arterial perfusion, and direct parenchymal compression.

## I. **Clinical Presentation**

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**Physical Findings** – Nearly all patients with ACS have a tensely distended abdomen, however physical examination of the abdomen is a poor predictor of ACS. Findings such as progressive oliguria and increased ventilator requirement are common. Other findings such as hypotension, tachycardia, elevated jugular venous pressure, peripheral edema, abdominal tenderness, or acute pulmonary decompensation may be present.

**Imaging** – Is not helpful in the diagnosis of ACS.

## II. **Risk Factors**

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### 1. **Diminished abdominal wall compliance**

- Acute respiratory failure, especially with elevated intrathoracic pressure
- Abdominal surgery with primary fascial closure
- Major trauma/burns
- High body mass index, central obesity

### 2. **Increased intra-luminal contents**

- Gastroparesis, Ileus
- Colonic pseudo-obstruction

### 3. **Increased abdominal contents**

- Hemo/pneumoperitoneum
- Ascites/liver dysfunction

### 4. **Capillary leak/fluid resuscitation**

- Hypotension, Hypothermia
- Polytransfusion, Coagulopathy, Massive fluid resuscitation
- Pancreatitis
- Oliguria
- Sepsis
- Major trauma/burns

## III. **Diagnostic Evaluation**

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Normal IAP in adults is **5-7 mmHg**, IAH defined as **IAP > 20 mmHg**.

Measurement using **bladder pressure** is the standard method to screen for IAH/ACS .

Measurements require free movement of the bladder wall and may not be accurate in patients with:

- Intra-peritoneal adhesions
- Pelvic fracture/hematoma
- Pelvic packs
- Neurogenic bladder

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Bladder pressure measurement should be:

1. Expressed in mmHg
2. Measured at end-expiration
3. Performed in the supine position
4. Zeroed at the level of the mid-axillary line
5. Performed with an instillation volume of 25 ml saline
6. Measured 30-60 seconds after instillation to allow bladder detrusor muscle relaxation

#### **IV. Medical Treatment Options to Reduce IAP**

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Medical management may be used when IAH presents with early end organ dysfunction and can be trialed for 24-48 hours if improvement is noted.

##### **1. Improve abdominal wall compliance**

- Sedation and analgesia
- Neuromuscular blockade
- Avoid head of bed > 30 degrees

##### **2. Evacuate intraluminal contents**

- Nasogastric decompression
- Rectal decompression
- Gastro/colic prokinetic agents

##### **3. Evacuate abdominal fluid collections**

- Paracentesis
- Percutaneous drainage

##### **4. Correct positive fluid balance**

- Avoid excessive fluid resuscitation
- Diuretics
- Colloids/Hypertonic fluids
- Hemodialysis/Ultrafiltration

##### **5. Organ Support**

- Maintain APP > 60 mmHg with vasopressors
- Optimize ventilation, alveolar recruitment
- Tidal volume reduction, a pressure-limited mode, and/or permissive hypercapnia may be required
- PEEP may reduce ventilation-perfusion mismatch and improve hypoxemia
- Consider using volumetric preload indices

## V. Surgical Decompression

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Indicated for **primary ACS**, occasionally for secondary ACS.

General agreement that surgical decompression is indicated for ACS that is refractory to other treatment options, however a specific threshold for surgical decompression has not been reached.

Decompressing the abdomen prior to the development of ACS is becoming increasingly common and improves survival.

Failure to recognize IAH prior to development of ACS may lead to multiorgan failure and death.

Mortality for patients who have progressed to ACS ranges from 40-100 %.

### **Important Considerations:**

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1. Standard decompression is via midline incision, and can be performed in the operating room if the patient is medically stable or at the bedside.
2. During operative decompression make incision large enough so as to prevent mesenteric ischemia, by ensuring that the mesentery is not compressed by the fascial edge
3. Maintain an open abdomen using temporary abdominal wall closure to prevent recurrent ACS
4. Temporary abdominal closure can be performed in many different ways and no one method has been shown to be superior. Site specific methods or attending preference can be used to determine which closure is best.
5. Most patients, if decompressed early prior to the development of organ failure, will usually tolerate primary fascial closure in 5-7 days, however this can take as long as 14 days. Earlier closure requires careful diuresis and fluid restriction as tolerated by the patient.
6. Those who remain critically ill past this time frame may require either split thickness skin grafting of the exposed viscera with planned fascial closure 9-12 months later or cutaneous advancement flap.

### **Algorithm for Management of IAH/ACS**

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1. If IAP measurement  $\geq 12$  mmHg, initiate treatment to reduce IAP and avoid excessive fluid resuscitation and optimize organ perfusion.
2. Serial IAP measurements every 4 hours while patient is critically ill. If  $\leq 12$  mmHg consistently, IAH resolved.
3. If IAP  $\geq 20$  mmHg with new associated organ failure as a direct consequence, the patient has ACS, initiate treatment for underlying etiology.
4. If patient continues to have progressive organ dysfunction and IAP  $\geq 25$ , perform surgical decompression.