

Abdominal Compartment Syndrome

Paul Wisniewski, D.O.

Medical Education

Cutting Edge Surgical Medical
Group

Apple Valley, Ca



Disclosures

None

Learning objectives

1. **Define** abdominal compartment syndrome and identify its primary, secondary, and recurrent causes.
2. **Recognize** the clinical signs, symptoms, and diagnostic criteria, including intra-abdominal pressure (IAP) and intra-abdominal perfusion pressure (IAPP).
3. **Explain** the steps for measuring IAP and calculating IAPP, and understand their significance in guiding treatment.
4. **Describe** the evidence-based medical and surgical management strategies to prevent morbidity and reduce mortality in patients with ACS.



Abdominal Compartment Syndrome (ACS)

- **Definition**
- Abdominal Compartment Syndrome (ACS) is a life-threatening condition characterized by sustained intra-abdominal hypertension (IAH) that leads to impaired organ perfusion and dysfunction. It commonly occurs in critically ill or injured patients and can be classified as primary (abdominal pathology), secondary (extra-abdominal pathology), or recurrent (after previous ACS) (Kirkpatrick et al., 2013).



Pathophysiology

- IAH occurs when the intra-abdominal pressure (IAP) rises above 12 mmHg. If not treated, elevated IAP (>20 mmHg) with new-onset organ dysfunction constitutes ACS (WSACS, 2013). IAH affects perfusion to abdominal organs, reduces venous return, impairs cardiac output, increases thoracic pressures, and can cause multiorgan failure (Malbrain et al., 2006).



Etiologies of ACS



Etiologies of ACS



Primary ACS: Trauma, intra-abdominal bleeding, pancreatitis, bowel obstruction, peritonitis, ruptured AAA



Secondary ACS: Sepsis, capillary leak, fluid resuscitation, burns, major surgery



Iatrogenic causes: Tight abdominal closure, over-resuscitation



Signs and Symptoms

ACS is often difficult to recognize due to nonspecific findings. Common clinical manifestations include:

- **Cardiovascular:** Hypotension, tachycardia, increased CVP or pulmonary artery pressures
- **Respiratory:** Increased peak airway pressures, decreased tidal volume, hypoxia, hypercapnia
- **Renal:** Oliguria (<0.5 mL/kg/hr), rising creatinine
- **Gastrointestinal:** Abdominal distension, increased girth, feeding intolerance
- **Neurological:** Elevated intracranial pressure (in brain-injured patients)

(Kirkpatrick et al., 2013; Malbrain et al., 2006)



Signs and Symptoms

System	Signs/Symptoms
Cardiovascular	Hypotension, tachycardia, elevated CVP
Respiratory	Decreased compliance, ↑ peak airway pressures, hypoxia
Renal	Oliguria, rising creatinine
GI	Abdominal distention, feeding intolerance
Neurological	Elevated ICP in head injury

(Kirkpatrick et al., 2013; Cheatham & Malbrain, 2009)



Diagnostic Criteria

Diagnostic Criteria

Intra-abdominal Hypertension Grades	IAP (mmHg)
Grade I	12–15
Grade II	16–20
Grade III	21–25
Grade IV	>25

ACS = Sustained IAP >20 mmHg with new organ dysfunction

Measurement: Via bladder pressure (25 mL saline, supine, transducer at mid-axillary line at iliac crest)



Diagnostic Criteria

- **Sustained IAP >20 mmHg AND**
- **New organ dysfunction/failure** (e.g., oliguria, hypoxia, increased ventilator demand)
- **Measurement**
- **Gold standard:** Bladder pressure measurement via Foley catheter.
- Measured with 25 mL saline instilled; transducer zeroed at mid-axillary line at iliac crest level (Cheatham & Malbrain, 2009).



Intra-Abdominal Perfusion Pressure (IAPP)

Definition

Intra-abdominal perfusion pressure (IAPP) is a calculated parameter used to assess abdominal organ perfusion. It reflects the pressure gradient driving blood flow to abdominal organs.

Formula

$$\text{IAPP} = \text{MAP} - \text{IAP}$$

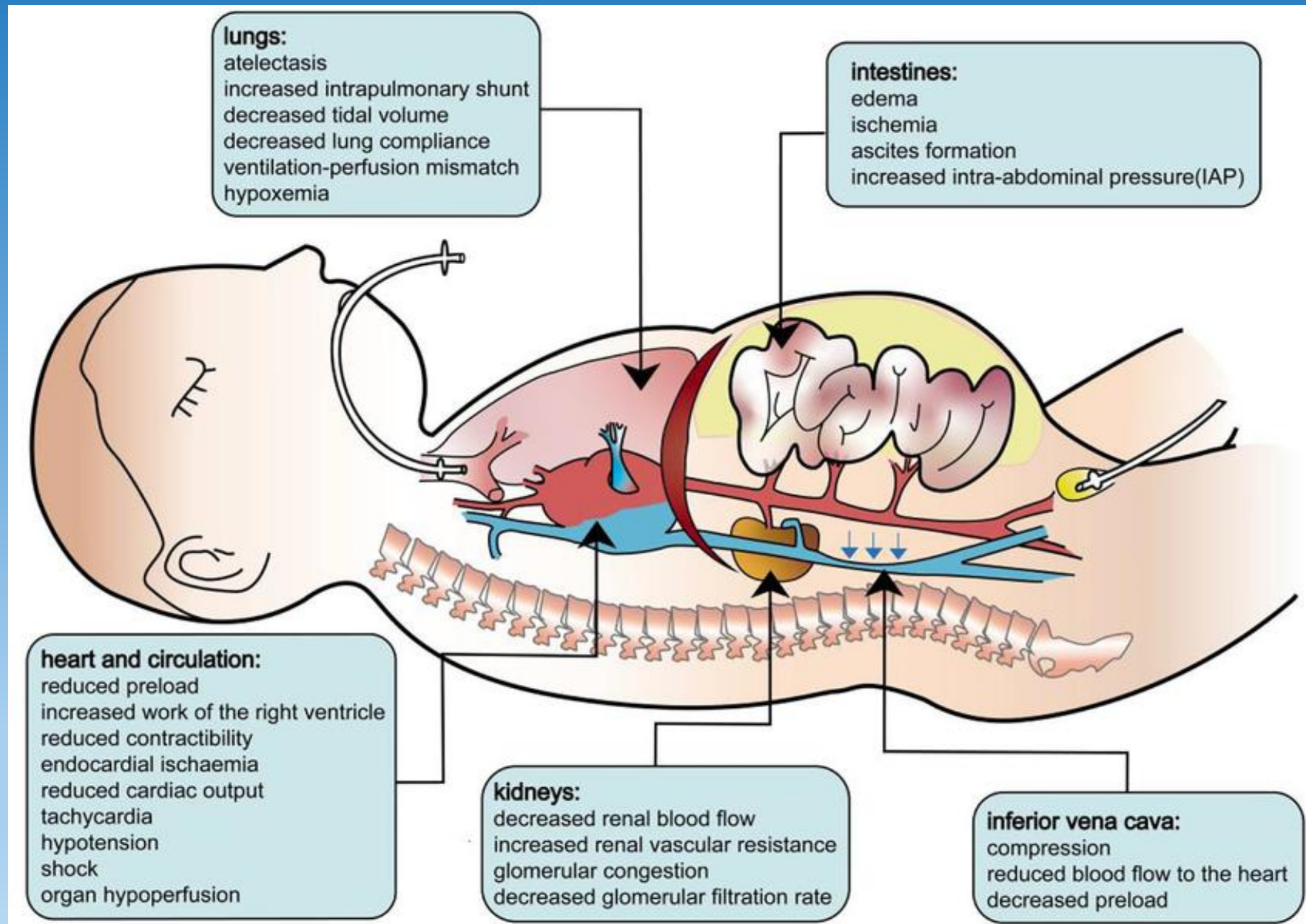
(MAP: Mean Arterial Pressure, IAP: Intra-Abdominal Pressure)

Clinical Significance

- **IAPP < 60 mmHg** is associated with **reduced organ perfusion and increased mortality** (WSACS, 2013)
- Stronger predictor of survival than IAP alone
- Goal-directed resuscitation should aim for **IAPP > 60 mmHg**

(Malbrain et al., 2006; Vidal et al., 2008)





Management

Medical Management (IAP 12–20 mmHg)

- Gastric/rectal decompression
- Sedation/neuromuscular blockade
- Diuresis or dialysis
- Abdominal wall relaxation
- Fluid restriction or reversal of over-resuscitation

Surgical Management (ACS diagnosis)

- Decompressive laparotomy
- Temporary abdominal closure (VAC, Bogota bag)
- Maintain IAPP > 60 mmHg post-decompression

(Malbrain et al., 2014)



Management

- **Medical Management (IAP 12–20 mmHg):**
- Evacuate intraluminal contents (NGT, rectal tube)
- Sedation and neuromuscular blockade
- Diuretics or dialysis for fluid overload
- Optimize abdominal wall compliance
- Minimize positive fluid balance
- Elevate HOB <30°



Management

- **Surgical Management (ACS / IAP >20 mmHg with organ failure):**
- **Decompressive laparotomy:** Immediate intervention of choice
- Temporary abdominal closure techniques (Bogota bag, negative pressure dressing)
- Mortality with surgical decompression ranges from 25–75% depending on etiology and timing (Sugrue et al., 2002; De Waele et al., 2006)



Prognosis and Mortality

Mortality in untreated ACS approaches **100%** (Balogh et al., 2013)



With surgical decompression, mortality remains high:

Trauma patients: 40–60%

Sepsis/burns: Up to 75%

Early detection and decompression
improves survival significantly (Cheatham et al., 2000; Malbrain et al., 2006)



Key Points

- Early recognition and bladder pressure monitoring are crucial.
- IAP >12 mmHg should prompt close monitoring.
- IAP >20 mmHg with organ dysfunction requires urgent decompression.
- Delays in diagnosis increase mortality exponentially (Kirkpatrick et al., 2013).



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