Abdominal Compartment Syndrome (ACS)

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The Operative Review of Surgery. 2023; 1:142-149.

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Definitions

Definition [1]

- *Intraabdominal Pressure (IAP)*: Steady-State Pressure Concealed within the Abdominal Cavity
  - Abdominal Perfusion Pressure (APP) = MAP – IAP
  - Normal IAP: 5-7 mmHg in Critically Ill Adults
- *Intraabdominal Hypertension (IAH)*: Sustained IAP ≥ 12 mmHg
- *Abdominal Compartment Syndrome (ACS)*: Sustained IAP > 20 mmHg that is Associated with New Organ Dysfunction/Failure
  - *Primary ACS*: ACS that Originates from Injury or Disease in the Abdominopelvic Region
  - *Secondary ACS*: ACS that Originates from Injury or Disease Outside of the Abdominopelvic Region
  - *Recurrent ACS*: ACS that Recurs After a Previously Treated ACS
- *Polycompartment Syndrome*: Two or More Anatomical Compartments Have Elevated Compartmental Pressures
  - Compartments Include Abdomen, Thorax, Head, and Extremities [2]
  - The Compliance of Each Compartment is Key to Determining the Transmission of Pressure Between Compartments [2]

Chronic Elevation

- IAP May Be Chronically Higher in Those with Ascites, Pregnant, or Obese (7-14 mmHg) [3,4]
- Acute Increases in IAP May Be Less Well Tolerated if Superimposed on Chronic IAH [5]

Intraabdominal Hypertension Grading [1]

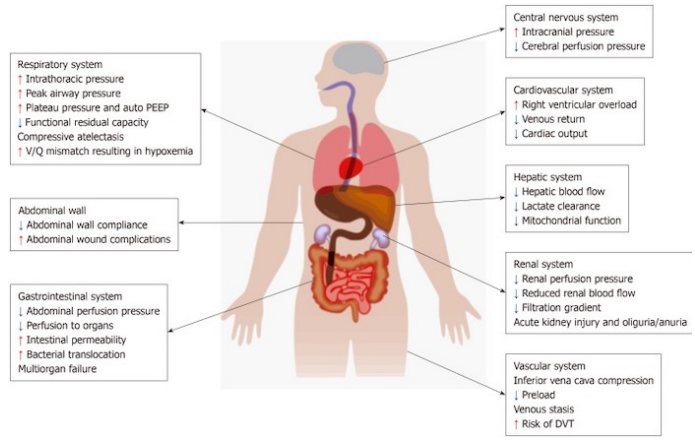
- Grade I: IAP 12-15 mmHg
- Grade II: IAP 16-20 mmHg
- Grade III: IAP 21-25 mmHg
- Grade IV: IAP > 25 mmHg

Pediatric Changes [1]

- *Intraabdominal Hypertension*: Sustained IAP > 10 mmHg
- *Abdominal Compartment Syndrome*: Sustained **IAP > 10 mmHg** that is Associated with New Organ Dysfunction/Failure

Pathophysiology

Pulmonary Effects [6,7]



Effects of Abdominal Compartment Syndrome [7]

- Intraabdominal Hypertension Causes Upward Displacement of the Diaphragm
- Upward Displacement of the Diaphragm Causes:
  - Increased Intrathoracic Pressure
  - **Decreased Chest Wall Compliance**
  - Decreased Functional Residual Capacity (FRC)
  - Increased Pulmonary Vascular Resistance
  - Compression Atelectasis
- Increased Intrathoracic Pressure Causes:
  - Increased Pleural Pressure
  - **Increased Airway Pressures (Peak, Mean, and Plateau)**
- Effects on Gas Exchange:
  - Hypercarbia and Hypoxia
  - Increased Ventilation-Perfusion Mismatch with Dead-Space Ventilation and Intrapulmonary Shunting

Cardiovascular Effects [6-8]

- Intraabdominal Hypertension Causes Increased Intrathoracic Pressure
- Increased Intrathoracic Pressure Causes:
  - Decreased Preload Due to Increased Central Venous Pressure (CVP) and Pulmonary Artery Pressure (PAP)
  - Increased Left Ventricle Afterload Due to Vascular Resistance

    - Increased Right Ventricle Afterload Can Cause Septal Deviation to the Left, Further Decreasing Preload
- Results in Decreased Cardiac Output and High Peripheral Vascular Resistance

Renal Effects [6,9]

- Intraabdominal Hypertension Causes:
    - Poor Renal Perfusion Due to Decreased Cardiac Output
    - Renal Venous Resistance
    - Direct Renal Compression with Shunting of Blood from the Cortex to the Medulla
    - Activation of the RAAS System Leading to Water and Sodium Retention
- Results in **Renal Impairment and Oliguria/Anuria**
    - *First Clinical Sign of Intraabdominal Hypertension

Gastrointestinal Effects [6-8]

- Intraabdominal Hypertension Causes:
    - Decreased Cardiac Output
    - Increased Splanchnic Vascular Resistance
- Results in Poor Mesenteric Blood Flow
- Risk for Mucosal Ischemia and Perforation

Hepatic Effects [7,8]

- Intraabdominal Hypertension Causes:
    - Decreased Cardiac Output
    - Increased Splanchnic Vascular Resistance
- Results in Decreased Hepatic Artery and Portal Vein Blood Flow
- Results in Decreased Liver Function and Lactate Clearance

Central Nervous System Effects [6,7]

- Intraabdominal Hypertension Causes Increased Intrathoracic Pressure
- Increased Intrathoracic Pressure Causes Increased Jugular Venous Pressure and Impairs Cerebral Venous Return
- Results in Increased Intracranial Pressure (ICP) and Decreased Brain Perfusion

Extremity Effects [6]

- Intraabdominal Hypertension Causes:
    - Increased Central Venous Pressure
    - Increased Peripheral Vascular Resistance
    - Decreased Cardiac Output
- Results in Decreased Peripheral Perfusion of the Extremities

Risk Factors

Reduced Abdominal Wall Compliance [7,10]

- Obesity
- Abdominal Surgery
- Prone Positioning
- Rectus Sheath Hematoma
- Burns with Abdominal Eschar
- Mechanical Ventilation with High PEEP
- Ventilator Dyssynchrony

Increased Intraluminal Contents [7,10]

- Gastric Distention
- Gastroparesis
- Ileus
- Small Bowel Obstruction
- Colonic Pseudo-Obstruction
- Volvulus
- Intraabdominal Tumor
- Retroperitoneal Tumor
- Damage Control Laparotomy
- Enteral Feeding
- Pregnancy

Abdominal Cavity Collections [7,10]

- Ascites
- Hemoperitoneum
- Pneumoperitoneum
- Major Trauma
- Laparoscopy with Excessive Inflation Pressures
- Peritoneal Dialysis
- Abdominal Inflammation-Peritonitis (Pancreatitis)
- Abdominal Abscess

Capillary Leak and Fluid Resuscitation [7,10]

- Acidosis
- Hypothermia
- Coagulopathy
- Massive Transfusion
- Trauma
- Sepsis

- Large Volume Fluid Resuscitation
- Major Burns
- Liver Transplant

Presentation

Symptoms [11]

- Most Patients are Critically Ill and Unable to Communicate
- Fatigue and Malaise
- Dyspnea
- Lightheadedness
- Abdominal Pain
- Abdominal Distention

Physical Exam

- Tense and Distended Abdomen
- Abdominal Exam is a Poor Predictor of ACS [12-14]

Additional Findings [6-9]

- Pulmonary:
    - Increased Airway Pressures
    - Hypercarbia and Hypoxia
    - Difficulty Weaning from the Ventilator
- Cardiovascular:
    - Decreased Cardiac Output
    - Increased Peripheral Vascular Resistance
- Renal Injury and Oliguria/Anuria
- Intestinal Ischemia and Perforation
- Elevated Intracranial Pressure (ICP)

Diagnosis

Diagnosis

- Definitive Diagnosis is Made by the Measurement of Intraabdominal Pressure (IAP) in the Setting of **New Organ Dysfunction/Failure** [1]
    - Requires IAP > 20 mmHg

- Perform with a Low Threshed of Suspicion [15]

Measurement of IAP [16]

- Intravesical (Bladder Pressure) – Standard Method
- Intragastric
- Intracolonic
- IVC Catheters

Requirements for Accurate Measurement [1]

- Measured at End-Expiration
- Measured in the Supine Position
- Ensure that Abdominal Muscle Contractions are Absent (Sedation)
- Transducer Should be Zeroed at the Midaxillary Line

How to Measure Bladder Pressure (Procedure) [17]

- Place Foley Catheter and Drain the Bladder
- Clamp Foley Catheter
- Zero Transducer at the Midaxillary Line
- Instill Sterile Saline into the Bladder (Maximum 25 cc) [1]
- Insert an 18 Gauge Needle Attached to a Pressure Transducer into the Aspiration Port
    - Some Commercially Available Catheters Permit Measurement by a Needle-Less Connection System
- Measure the Pressure at End-Expiration
    - Wait for 60 Seconds After Instillation of Fluid to Allow Detrusor Muscle Relaxation

Treatment

Definitive Treatment

- Definitive Treatment is by **Decompressive Laparotomy** with Temporary Abdominal Closure [1]
- Immediate Complications of Decompression:
    - Bolus of Lactic Acid, Potassium and Other Anaerobic Byproducts (Induced Arrhythmia)
    - Decrease in Preload (Induced Hypotension)
    - Respiratory Alkalosis
- May Be Able to Avoid Laparotomy in Select Cases:
    - Massive Ascites – Percutaneous Catheter Decompression/Paracentesis [18]
    - Burn Eschars Causing Mechanical Limitations – Escharotomy [19]

Options to Temporize or Prevent Progression of IAH to ACS [20]

- Place in the Supine Position and Avoid Elevating the Head of Bed
- Improve Abdominal Wall Compliance:
    - Sedation and Analgesia
    - Paralysis
- Reduce Intraabdominal Volume:
    - Orogastric/Nasogastric Tube Decompression
    - Foley Catheter
    - Percutaneous Catheter Decompression/Paracentesis
- Low Tidal Volume & High PEEP
- Limit Fluid Administration

Morbidity and Mortality

- Failure to Recognize Can Cause Multisystem Organ Failure and Death
- High Mortality Once ACS Develops (40-100%) [21-25]

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