Understanding Intra-Abdominal Pressures

1 Contact Hour

Course Expires: May 31, 2018
Course Updated: October 14, 2014
First Published: October 14, 2011

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Acknowledgments
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Purpose and Learning Objectives
The purpose of Understanding Intra-Abdominal Pressures is to understand the signs and symptoms of the intra-abdominal hypertension that can lead to abdominal compartment syndrome and the rationale for trending intra-abdominal pressures. Malbrain, et al (2006) conducted a prevalence study in 13 intensive care units and assessed 97 patients. The overall prevalence of intra-abdominal hypertension was 58.8% (65% in surgical patients and 54.4% in medical patients).

After successful completion of this course, you will be able to:

1. State the etiology of abdominal compartment syndrome.
2. Understand the pathophysiology of intra-abdominal hypertension.
3. Identify the signs and symptoms of abdominal compartment syndrome.
4. Know how to measure intra-abdominal pressures.
5. State the grading of abdominal compartment syndrome.
6. Describe the management of elevated intra-abdominal pressures.
7. Describe nursing considerations.
8. State how/what the nurse should document.

Introduction
Recently, there has been a trend in medicine to more closely monitor bladder pressures. A decline in organ function, once mistakenly thought to be progression of illness, is now understood to be related to abdominal compartment syndrome. Undetected intra-abdominal hypertension can lead to a lethal condition known as abdominal compartment syndrome, caused by ischemia of the peritoneal organs.
Understanding the etiology and the signs and symptoms of abdominal compartment syndrome will assist the nurse in the early detection of this disease state. Knowing how to properly measure and trend intra-abdominal pressures will help in this imperative detection. Early identification is key to preventing and treating abdominal compartment syndrome through diligent nursing assessment and monitoring by trending intra-abdominal pressures (Walker, 2003). Since treatment can improve organ dysfunction, it is important that the diagnosis of intra-abdominal hypertension or abdominal compartment syndrome be considered in the appropriate clinical situation. The definition, risk factors, clinical presentation, diagnosis, and management, are reviewed here (Gestring, 2014).

Overview
Abdominal compartment syndrome is a term that describes the collective physiological response to elevations in intra-abdominal pressure.

Abdominal compartment syndrome is defined as new organ dysfunction or failure in the setting of sustained intra-abdominal pressures > 20 mmHg (Lee, 2012).

Any increase in pressure in the anatomically closed space of the abdominal cavity jeopardizes the functioning of peritoneal and retroperitoneal organs, and indirectly affects the cardiovascular, pulmonary, neurological and renal systems secondary to restrictions or obstructions in arterial flow.

This increase in pressure refers to the term intra-abdominal hypertension, and when undetected, can lead to abdominal compartment syndrome.

Test Yourself
Abdominal compartment syndrome is defined as:

A. New organ dysfunction or failure in the setting of sustained intra-abdominal pressures > 10 mmHg
B. New organ dysfunction or failure in the setting of sustained intra-abdominal pressures > 20 mmHg
C. Three or more organ dysfunctions or failure in the setting of sustained intra-abdominal pressures > 30 mmHg

The correct answer is: B. New organ dysfunction or failure in the setting of sustained intra-abdominal pressure > 20 mmHg.

Rationale: Abdominal compartment syndrome is defined as new organ dysfunction or failure in the setting of sustained intra-abdominal pressures > 20 mmHg.

Terminology
Intra-Abdominal Pressure (IAP)
A baseline level of pressure within the abdominal cavity is 0-5 mmHg in a healthy individual, and varies inversely with intrathoracic pressure during normal breathing. In critically ill patients, a consistent pressure of 5-7 mmHg can be expected (Lee, 2012). Obese, chronic ascites, and pregnant
patients will have higher baseline readings, but in these conditions the changes happen slowly and the body adjusts.

**Intra-Abdominal Hypertension (IAH)**
Consistent intra-abdominal pressures are 12 mmHg. This is an early indication of abdominal compartment syndrome and pressures of this level will begin to affect perfusion to the organs within the abdominal cavity (Gestring, 2014; Lee, 2012).

**Abdominal Perfusion Pressure (APP)**
APP is a measure of the adequacy of abdominal blood flow. APP is calculated by subtracting the IAP from the mean arterial pressure (MAP). APP in patients with IAH or ACS should be maintained at 60 mmHg or higher (Lee, 2012).

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

**Abdominal Compartment Syndrome**
Defined as sustained pressures of >20 mmHg with or without an APP <60 mmHg associated with new organ dysfunction or failure. Intra-abdominal pressures in this range can cause rapid decline in organ function and lead to multiple organ failure if not treated (Lee, 2012).

**Test Yourself**
BP = 100/40 (60)
IAP = 15
APP =

A. 25
B. 45
C. 85

The correct answer is: B. 45.

*Rationale: MAP - IAP = APP*

**Causes of Abdominal Compartment Syndrome**
The WSACS (2011) categorizes the cause of IAH or ACS into two sections, primary and secondary conditions. Primary conditions are the ones that need surgical or interventional radiological treatment. Secondary conditions are due to medical causes that do not require surgery or radiological intervention as initial therapy (Lee, 2012).

**Primary:**
- Trauma- blunt and penetrating abdominal trauma, pelvic fractures, bowel perforation and hemorrhage
- Liver transplants
- Ruptured abdominal aortic aneurysm
- Post-operative bleeding
• Retroperitoneal hemorrhage
• Mechanical intestinal obstruction
• Abdominal surgery-decreased abdominal wall compliance secondary to post-surgical abdominal packing or, tight surgical closures
• Bleeding pelvic fractures

(Gestring, 2014; WSACS, 2011)

**Secondary:**

• Massive volume replacement status-post surgery or trauma
• Rapid fluid resuscitation in the setting of SIRS (Systemic Inflammatory Response Syndrome), peritonitis, pancreatitis, bowel obstruction, ileus, sepsis, ruptures, abdominal aneurysm, abdominal tumors, cirrhosis, ascites and full thickness burns
• Obstetrical conditions: preeclampsia, and pregnancy-related DIC
• Ascites
• Pancreatitis
• Ileus
• Sepsis
• Major burns
• Continuous ambulatory peritoneal dialysis
• Morbid obesity
• Sever intra-abdominal infection

(Gestring, 2014; WSACS, 2011)
**Pathophysiology: Intravascular Response**

- Increased intra-abdominal pressure
  
  - Decreased venous return (hypovolemia)
    
    - Compensatory vasoconstriction
      
      - Blood flow is shunted away from intra-abdominal organs to preserve cardiac and cerebral perfusion
        
        - Cellular hypoxia of the kidneys, liver, intestines (mesentery)

(Urden, Stacey, & Lough, 2010)

**Pathophysiology: Cellular Response**

- Intra-abdominal hypoperfusion and hypoxia
  
  - Cytokine release and vasodilation
    
    - Increased capillary wall permeability
      
      - Edema

  - Decreased production of Adenosine Triphosphate (ATP)

  - Sodium-potassium pump failure floods cells with excess sodium
    
    - Cellular edema and rupture
      
      - Further increase in ischemia

  - Decreased vascular volume
    
    - Anaerobic metabolism
      
      - Increased lactic acid

    - Metabolic acidosis

(Urden, Stacey, & Lough, 2012)
Sodium-Potassium Pump

This image is provided courtesy of Wikimedia (Public Domain).

Pathophysiology: Metabolic Acidosis

Continued lack of bowel perfusion affects gastrointestinal mucosa barrier function promoting bacterial translocation

Increased risk for sepsis

Metabolic acidosis worsens

(Urden, Stacy, & Lough, 2012)
Pathophysiology: Anatomical Response
As the cellular effects of abdominal compartment syndrome occur, the increase in intra-abdominal pressure limits the rise and fall of the diaphragm which:

- Splints the thoracic cavity compressing the inferior vena cava decreasing venous return to the heart
- Decreases preload and cardiac output compromising ejection fraction
- Decreases lung capacity
- Impaired gas exchange compromises cerebral perfusion and neurological function
- Decreases blood flow to the renal and mesenteric arteries

(Gestring, 2014)

Please note!
Gastrointestinal changes can be seen with IAP as low as 12 mmHg.

Signs and Symptoms: Cardiovascular
The increase in intra-thoracic pressure compresses the heart and major vessels, causing a tamponade-like picture, especially as abdominal pressures rise. Clinical findings show the following cardiovascular signs and symptoms:

- Hypotension
- Reflex tachycardia
- Decreased peripheral pulses
- Cold, clammy skin

(Lee, 2012)

From compression of the inferior vena cava, there is a decreased blood flow back to the heart. This decreases preload and then causes a decrease in cardiac output. Changes in Hemodynamic Monitoring Include:

- Increased central venous pressures from intra-thoracic pressure
- Increased pulmonary artery pressures
- Increased pulmonary capillary wedge pressure
- Increased systemic vascular resistance
- Decreased ejection fraction
- Decreased Cardiac Output

(Lee, 2012)
Test Yourself
Which of the following is cardiovascular sign of increased abdominal pressures:

A. Increased ejection fraction
B. Increased pulmonary artery pressures
C. Decreased systemic vascular resistance

The correct answer is: B. Increased pulmonary artery pressures.

Rationale: Increased pulmonary artery pressures is a cardiovascular sign of increased abdominal pressures.

Signs and Symptoms: Pulmonary
From the increase in abdominal pressure, intra-thoracic pressures increase as the abdomen presses upward towards the lungs. Clinical findings show the following pulmonary signs and symptoms:

- Hypoventilation
- Tachypnea
- Dyspnea
- Hypoxia
- Atelectasis
- Edema
- Increased alveolar dead space

(Lee, 2012; Urden, Stacy, & Lough, 2012)

Mechanical ventilation changes include:

- Increased peak inspiratory pressure
- Decreased tidal volume

(Gestring, 2014; Lee, 2012)

Signs and Symptoms: Renal
From compression of the inferior vena cava, there is a decreased blood flow back to the heart. This decreases preload and then causes a decrease in cardiac output. When there is a decrease in cardiac output, there is a decrease in renal perfusion. Renal signs and symptoms include:

- Initial decrease in urine output leading to oliguria when IAP>12 mmHg
- Increased BUN and Creatinine
Anuria is associated with complete Abdominal Compartment Syndrome, or IAP >20 mmHg

Acute Tubular Necrosis (ATN) and Acute Renal Failure (ARF) in advanced stages

(WSACS, 2012)

**Signs and Symptoms: Neurological**
From the increase in IAP leading to increased pressures to the thoracic compartment, this puts back pressure on the jugular veins and decreases drainage of cerebrospinal fluid and blood, leading to increased intracranial pressure (Lee, 2012). Neurological signs and symptoms include:

- Headache
- Nausea and vomiting
- Lethargy
- Confusion
- Visual changes
- Palsies or stroke-like symptoms
- Intubated and sedated patients may exhibit subtle neurological changes

(Gestring, 2014; Lee, 2012)

**Signs and Symptoms: Gastrointestinal and Hepatic**
Lee (2012) states that IAH on the organs leads to diminished gut perfusion which leads to ischemia, acidosis of the mucosal bed, capillary leak, intestinal edema, and translocation of gut bacterial. Gastrointestinal and Hepatic signs and symptoms include:

- Hypoactive or absent bowel sounds
- Abdominal distention
- Increased liver function tests (LFTs)
- Increased lactic acid
- Nausea and vomiting

(Gestring, 2014; Lee, 2012)
Grading of Abdominal Compartment Syndrome

There are multiple scales to grade abdominal compartment syndrome based on variable intra-abdominal pressures. The lower the IAP, the lower the risk of abdominal compartment syndrome. The higher the IAP, the higher the risk of abdominal compartment syndrome.

It is important to note that the signs and symptoms of abdominal compartment syndrome worsen with increasing intra-abdominal pressure. Worsening abdominal compartment syndrome is often seen with an increasing trend of the intra-abdominal pressure.

The WSACS (2012), states the following grading system for intra-abdominal hypertension:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Intra-abdominal pressure, mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>12-15</td>
</tr>
<tr>
<td>II</td>
<td>16-20</td>
</tr>
<tr>
<td>III</td>
<td>21-25</td>
</tr>
<tr>
<td>IV</td>
<td>&gt;=25 (<strong>greater than/equal to)</strong></td>
</tr>
</tbody>
</table>

Measuring Intra-Abdominal Pressures

The importance of IAP monitoring is to trend readings in order to detect the onset of intra-abdominal hypertension which could lead to abdominal compartment syndrome (Lee, 2012).

- In Grade I, the goal is to maintain normovolemia (normal fluid volume).
- In Grade II, volume is often administered.
- In Grade III, decompression of the abdominal cavity is considered.
- In Grade IV, re-exploration of the abdomen is contemplated.

(Forrant, 2009)
Risk Factors of IAH/ACS
According to the World Society of Abdominal Compartment Syndrome, there are four categories that places a patient at an increased risk 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**
   - Gastroparesis
   - Ileus
   - Colonic pseudo-obstruction

3. **Increased abdominal contents**
   - Hemoperitoneum
   - Pneumoperitoneum
   - Ascites
   - Liver dysfunction

4. **Capillary leak/fluid resuscitation**
   - Acidosis
   - Hypotension
   - Hypothermia (Core temperature <33 degrees C)
   - Polytransfuction (>10 units of blood/24 hours)
   - Coagulopathy (platelets <55000/mm2) or prothrombin time (PT) >15 seconds or partial thromboplastin time (PTT) >2 times normal or international standardized ratio (INR) >1.5
   - Massive fluid resuscitation (>5L/24 hours)
   - Pancreatitis
   - Oliguria
   - Sepsis
   - Major trauma
   - Burns
   - Damage control laparotomy
Measuring Intra-Abdominal Pressures
The most accurate and direct measurement of intra-peritoneal pressure is via the intra-peritoneal catheter which is placed into the abdomen, but this is an extremely invasive procedures with high risks (Lee, 2012).

In the last few years, it became more popular to measure the intra-abdominal pressure via the bladder. Bladder pressure monitoring is the least invasive method, it is reliable, and it is easier to perform than direct intra-peritoneal measuring. Due to the need for a cardiac monitor with invasive pressure monitoring capabilities to measure this pressure, it is often mainly done in intensive care units.

Image of Intra-Abdominal Pressure Monitoring

Image provided with permission from ConvaTec (Unomedical), 2011.

Pre-Procedure
Before beginning any procedure, it is important to educate the patient and/or the family about intra-abdominal pressure monitoring. The patient/family should know what the nurse is doing, why they are doing it, how they are performing the measurement, and the importance of monitoring for abdominal compartment syndrome.

Equipment
The following equipment is recommended to perform an intra-abdominal bladder pressure:

- Foley catheter (the patient must be catheterized to obtain an intra-abdominal pressure reading)
- Transducer set-up (flushed and primed with saline)
• Pressure cable for the cardiac monitor
• 250 or 500 ml bag of normal saline
• Blunt cannula
• 60 mL syringe
• Chlorhexadine
• Clamps
• Sterile male adapters
• Gloves

The Set-Up
Policy and procedures may differ between hospitals, please check with your facility before beginning intra-abdominal pressure monitoring.

The biggest variance noted in literature review was the amount of normal saline flushed into the bladder immediately before receiving your measurement.

The following steps were formulated from the American Association of Critical Care Procedural Manual (AACN, 2011).

Beginning Steps per AACN: Priming the tubing (2011)
1. Wash hands and don gloves
2. Prime the transducer set-up with the normal saline and inflate the pressure bag to 150 mmHg
3. Attach the pressure cable from the transducer set to the monitor
4. Level the fluid interface (zeroing stopcock) to the symphysis pubis
5. Attach the blunt cannula to the stopcock at the end of the transducer tubing
6. Attach the 60 mL syringe to stopcock
7. Fill with 20 mL of NS from the flush bag

The Procedure
1. Before beginning the measurement:
   • Place the patient in a complete supine position (HOB 0 degrees)
Level the transducer to the patient’s symphysis pubis

2. Clamp the foley drainage tubing just below the aspiration port

3. Cleanse the aspiration port with Chloraprep (or other agent per your hospital protocol)

4. Place the blunt cannula through the aspiration port on the foley catheter

5. Turn the stopcock off to the transducer, and open the stopcock to the 60 ml syringe

6. Inject the 20 mL of normal saline via the aspiration port on the foley catheter over 10 seconds

7. Turn the stopcock open to the transducer

Reading the Waveform

1. After 20 seconds have elapsed, read the mean pressure on the cardiac monitor

2. Measure the IAP at end-expiration:
   - On a non-vented patient, end-expiration is the higher point on the waveform
   - On a vented patient, end-expiration is the lower point on the waveform

Post-Procedure

1. Remove the blunt cannula & transducer set-up from aspiration port and remove the blunt cannula

2. Maintain sterility on the transducer set by applying the male adapter to where the blunt cannula was located on the stopcock

3. Remove the clamps from the foley drainage tubing

4. Save the transducer set-up for the next measurement

5. Don’t forget to remove your gloves and clean your hands

(AACN, 2011)

Important!
There are devices that have been designed to streamline this process. When using a device, follow the manufacture’s instructions.

Key Points to Remember

It’s important to remember:

- Subtract 20 mLs from the total urine output for that hour
• Record the bladder pressure on the patient’s flowsheet

• Monitor intra-abdominal pressures every 2 to 4 hours or more frequently based on clinical need

(AACN, 2011)

**Test Yourself**

It is not important to subtract the mls that were instilled for the bladder pressure reading from the hourly output.

A. True  
B. False

The correct answer is: B. False.

*Rationale: Subtract 20 mLs from the total urine output for that hour.*

**Documentation**

Documentation includes the following:

- Patient & family education
- Assessment findings before obtaining intra-abdominal pressures
- Intra-abdominal pressure value
- Post-procedure assessment
- Changes in the patient’s assessment indicating onset of intra-abdominal hypertension or abdominal compartment syndrome
- Unexpected outcomes
- Additional interventions
- Reportable conditions

(AACN, 2011)

**Treatment & Nursing Considerations**

Along with the IAP reading, the nurse will need to correlate this pressure with other assessment findings associated with abdominal compartment syndrome as previously mentioned.

The important thing to remember is to maintain airway and provide adequate ventilation.
The nurse should closely monitor blood pressure, heart rate, pulse oximetry, and respiratory rate.

If a pulmonary catheter is in place and more advanced hemodynamic monitoring can be achieved, trend and monitor the central venous pressure, pulmonary artery pressure, and cardiac output. Strict intake and output should be measured and frequent electrolyte checks should be done. Some hospitals have a policy to measure abdominal girths, but the research since the 1980’s state that abdominal growth is not a reliable tool to estimate abdominal pressure. It is important to assess the patient's bowel sounds (Forrant, 2009).

If there is an upward trend to a mildly elevated IAP (IAP 12-15 mmHg), the following interventions can be done:

- Sedation
- Pain control
- Avoid prone position. Instead reposition bed to a reverse Trendelenburg without flexion at the hips
- Carefully assess fluid administration: do not over resuscitate, use goal directed volumes, avoided unneeded fluid boluses, concentrate all drops, and aim for a neutral to negative fluid balance by day 3
- Nasogastric tube
- Enemas
- Bowel prokinetic agents such as erythromucin, metoclopramide

**Treatment & Nursing Considerations**

If there is an upward trend to a Grade II or Grade III (IAP 16-20 mHg), the patient should receive even closer monitoring. The following interventions can be done:

- All previous listed
- Enteral nutrition at trophic levels only
- Colloids plus diuretics
- Hemofiltration/dialysis to remove excess fluid
- Ultrasound or CT the abdomen to identify free fluid, space occupying lesions amenable to drainage
- Paracentesis catheter to drain any free fluid
- CT or US guided drainage or abscesses or hematomas

If the patient advances to a Grade IV (IAP >20 mmHg), the following interventions can be done:

- All previous listed
- Neuromuscular blockade and infusion
- Colonoscopy to decompress distended colon
- Stop enteral nutrition

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• Surgical evacuation of any tumors, masses
• Surgical consultation to plan decompressive laparotomy if the above interventions fail, IAP exceeds 25 mmHg or organ failure ensues

**Important!**
Like any hemodynamic number, it is important to look at the trend of the number and correlate it with the patient's signs and symptoms.

### How to Manage Abdominal Compartment Syndrome

• Closely monitor hemodynamics, pulmonary, and renal status.

• Watch for signs of impaired gas exchange, decreased urinary output, and changes in acid-base balance.

• Keep in mind that the patient is at high risk of ventilator-associated pneumonia since elevating the head of bed for a patient with IAH increases intra-abdominal pressure. Instead, use reverse Trendelenburg's position to elevate HOB 30 degrees (Measure IAP when patient is flat).

• If an urgent decompressive laparotomy in the operating room or bedside is needed, the peritoneal cavity is usually left open post-op with a covered exposed dressing. This urgent laparotomy provides rapid relief of intra-abdominal pressure (Brush, 2007).

Surgical decompression is the only definitive treatment once IAH progresses to abdominal compartment syndrome (Brush, 2007). Continuation of intra-abdominal pressures will be important during the post-operative period. Thorough assessment and prevention is essential. At early stages of increased abdominal pressures; optimal fluid resuscitation is imperative. Over-resuscitation can exacerbate edema, while under-resuscitation perpetuates ischemia.

**Did You Know?**
If an urgent decompressive laparotomy in the operating room or bedside is needed, the peritoneal cavity is usually left open post-op with a covered exposed dressing. This urgent laparotomy provides rapid relief of intra-abdominal pressure (Brush, 2007).

### Post Emergency Surgery
Post-laparotomy, there are many risks. Serious adverse side effects include:

• Risk of a massive washout of anaerobic products

• Profound hypotension

• Asystole

Nursing care should focus on risk assessment and early identification of clinical signs and symptoms of abdominal compartment syndrome.
Continuation of intra-abdominal pressures will be important during the post-operative period (Lee, 2012). Often the patient will be left with an open abdomen until the abdominal pressure resides (Lee, 2012).

**Conclusion**

In this course, you learned:

- Understanding the signs of syndromes of intra-abdominal hypertension is important for a nurse.
- A critical care nurse will need to closely monitor the intra-abdominal bladder pressures via an indwelling urinary catheter in order to trend intra-abdominal pressures.
- An increase in these pressures is a red flag for a severe condition of abdominal compartment syndrome.

**References**


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