Professional Gaps

Most anesthesiologists are aware of the syndrome of IAH and its associated high morbidity and mortality rates. How perioperative anesthetic management may decrease or inappropriately increase this complication may be less well known. Current knowledge on how timely management and awareness may prevent IAH from progressing to ACS is presented.

Learning Objectives

At the completion of the activity, the reader will be able to:

1. Cite the incidence of IAH and ACS in critically ill patients
2. Understand the role of the World Society of Abdominal Compartment Syndrome in providing consensus and defining specific/unique language terms and guidelines for diagnosis, communication, and future research
3. Appreciate the diverse population of both medical and surgical patients at risk
4. List risk factors for the development of ACS
5. Describe the etiology
6. Present a brief history of the understanding of IAH and ACS
7. Indicate situations when delayed abdominal closure is indicated
8. Differentiate between the 3 types of ACS
9. List the grades of IAH according to intraabdominal pressure (IAP)
10. Recognize the part that obesity plays in IA
**Case**

A 60-year-old woman was admitted to the ICU with a presumed diagnosis of severe acute pancreatitis. She was 65 inches tall and weighed 101 kg. Heart rate was 117 beats per minute and blood pressure was labile, ranging from 70/50 to 90/75 mm Hg. She was given approximately 3.5 L of normal saline over 3 hours. Intraabdominal pressure was measured at 20 mm Hg. Antibiotics, vasopressors, and inotropes were administered and titrated according to weight and changing vital signs after placement of invasive monitors and endotracheal intubation. Despite these supportive measures, serum creatinine increased to over 4 mg/dL, as did inspired ventilation pressures, which exceeded 40 cm H₂O. Respiratory rate was increased to 30 breaths per minute and respiratory volumes decreased to 300 mL. Inspired oxygen was still administered at 70% in an attempt to maintain the patient’s blood oxygen level above 92%. Abdominal imaging reported a dilated bowel, and a nasogastric tube was placed. The intensivist requested surgical and anesthetic consults.

This 2-part series is a review of intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS). In Part 1, recent definitions are offered along with a history of the development and understanding of IAH and ACS. The incidence is noted and risk factors are outlined. The most important etiologies are listed. In Part 2, the pathophysiology is explained as well as means to a diagnosis. Management and treatment are considered, including damage control resuscitation and the reperfusion syndrome. Fluid management and anesthetic considerations, as well as care of the case presented, conclude the lesson.

**Introduction**

A physiologic compartment is an enclosed space where body organs are located and function. A syndrome is not a primary disease, but may occur from many pathologic conditions. The compliance or distensibility of any compartment is a major factor in predicting the effects of increasing compartment content—from causes such as swelling, hemorrhage, and infection—on intracompartmental pressure.

Compartment syndromes of the extremities and increased intracranial pressure have long been recognized as life-threatening conditions that require immediate treatment and decompression. Increased pressure within the centrally located abdominal cavity may carry equal or even greater urgency for attention. Hypoperfusion and ischemia of the gut are untoward complications of increased compartmental pressure.

The human body may be described as having 4 major compartments: the head, chest, abdomen, and extremities. These compartments are not isolated but interconnected and interactive. Within these major compartments, individual organs such as the kidneys, liver, heart, and eyes carry their own potential for compartment syndrome within enclosed physiologic spaces.

A compartment syndrome develops within a physiologic space or cavity when tissue and organ viability is threatened due to compromised circulatory perfusion pressure and decreased oxygenation. It occurs when the pressure within a fixed compartment or its potential space that may be circumscribed by bone and/or myofascial tissue becomes exceeded due to increasing compartmental content.

The term *multiple compartment syndrome* was used initially when more than one compartment was affected.¹ Later, the term *polycompartment syndrome* was introduced to define the interactive effects of
multiple or adjacent compartments, such as those between the abdominal and thoracic compartments (about which more below). This terminology was adopted to avoid confusion with the term multicompartment syndrome that had become more universally understood to describe multiple limb trauma.

The familiar term limb compartment syndrome, which is associated with trauma and swelling, results from an increase in pressure in an enclosed muscle compartment. Measurement of normal osseofascial compartment pressure is below 10 to 12 mm Hg. Pressures above 30 mm Hg are believed to be critical and threaten tissue viability due to decreased blood perfusion. Limb fasciotomy is often required to restore blood perfusion. Increased pressure within the confined space results in excessive muscle compression with cellular injury. Without decompression of the affected area, irreversible tissue injury and necrosis can result in acute kidney injury, coagulopathies, acute lung injury, rhabdomyolysis, shock, and ultimately death.

Recently, another type of limb compartment syndrome, referred to as well-leg compartment syndrome, has been described as a complication of lengthy laparoscopic surgery performed in the Trendelenburg position. Impaired arterial perfusion and reduced femoral venous drainage due to pneumoperitoneum are presumed causative. Corrective recommendations include minimal elevation of the legs above the heart, placing the former in a horizontal position every 2 hours, and avoiding pneumatic compression devices and thromboembolism-deterrent stockings in procedures lasting over 4 hours. Great toe oximetry has also been suggested.

The abdomen is an enclosed compartment with both rigid walls consisting of the costal arch, spine, and pelvis, and flexible walls including the abdominal wall and diaphragm. These boundaries outline the abdominopelvic cavity, including the pelvic/bone girdle. The urinary bladder is in the pelvis, and pressure within it is used to measure intraabdominal pressure (IAP). Should the parenchymal contents of this cavity become distended from disease, infection, edema, or hemorrhage, the normal IAP may become pathologically elevated to create a condition referred to as abdominal compartment syndrome (ACS), which jeopardizes normal organ perfusion, oxygenation, and function. Due to clinical unfamiliarity, ACS is probably underrecognized and unreported, especially in critically ill medical and/or surgical or trauma patients. It is observed more often in surgical and trauma patients than in medical patients.

Intraperitoneal and to a lesser extent retroperitoneal compartments are more compliant than extremity fascial compartments. However, there is an end point of compliance and distention, at which the intracompartmental pressure will dramatically rise and impair oxygenation and nutritional blood flow.

Definitions

The World Society of the Abdominal Compartment Syndrome (WSACS) (www.wsacs.org), founded in 2004, drafted a consensus of definitions in 2006 intended to improve communication and research for various representative terms regarding ACS. These definitions have recently been reviewed and updated, as follows.

Intraabdominal pressure is defined as the steady-state pressure concealed within the abdominal cavity, and may be measured directly by transducing a needle inserted into the abdomen or indirectly. Due to simplicity and low cost, urinary bladder volume has become the standard for indirect measurement. Guidelines ensure reproducibility. Under normal conditions, IAP ranges from 0 to 5 mm Hg, is approximately 5 to 7 mm Hg in patients in the ICU, and increases to around 10 mm Hg in critically ill
adults. In chronic conditions such as morbid obesity, cirrhosis with ascites, and pregnancy, IAP may be as high as 9 to 14 mm Hg. Intraabdominal pressure in patients after recent abdominal surgery, sepsis, organ failure, or those who have received large-volume resuscitation may exceed 20 mm Hg.

Intraabdominal hypertension (IAH) is defined as a sustained IAP of at least 12 mm Hg. A grading system to describe severity includes:

- grade 1: IAP of 12 to 15 mm Hg
- grade 2: IAP of 16 to 20 mm Hg
- grade 3: IAP of 21 to 25 mm Hg
- grade 4: IAP greater than 25 mm Hg

Several subtypes of IAH are described. Hyperacute IAH is an elevated IAP lasting a few seconds, which occurs during laughing, coughing, straining, and sneezing. Acute IAH develops over hours from trauma and/or intraabdominal hemorrhage. Subacute IAH builds over days, usually in critically ill medical patients. Chronic IAH progresses over months in patients with morbid obesity and cirrhosis with ascites, or those who are pregnant.

Cerebral perfusion pressure is a net pressure gradient related to adequate cerebral blood flow. Cerebral perfusion pressure is expressed by the equation: cerebral perfusion pressure = mean arterial pressure – intracranial pressure or central venous pressure. An equivalent abdominal term, referred to as abdominal perfusion pressure, is calculated as: abdominal perfusion pressure = mean arterial pressure – IAP.

Abdominal perfusion pressure has been proposed as a reliable hemodynamic indicator of blood flow to the intraabdominal organs. It measures arterial inflow against restriction to venous outflow. Maintaining an abdominal perfusion pressure of at least 60 mm Hg is recommended to improve survival in disease states. However, in chronically hypertensive patients this number may be modified upward. A continuous measurement of IAP versus an intermittent measurement increases the accuracy of interpreting trends. Obtaining these measurements should be accompanied by an antiseptic protocol to avoid nosocomial infections. Abdominal perfusion pressure is said to be diagnostic and a guide to manage disease severity, and should be included with other indices such as pH, base deficit, arterial lactate levels, and hourly urinary output in predicting outcome.
Abdominal compartment syndrome is defined as a sustained IAP of at least 20 mm Hg that is associated with additional organ dysfunction/failure, such as acute renal or respiratory insufficiency or failure. There are 3 subcategories of ACS. Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiology procedures. Secondary ACS refers to conditions that do not originate from the abdominopelvic region, such as extraabdominal trauma, shock, sepsis, and burns. Tertiary (or recurrent) ACS refers to relapse of previously treated ACS.

The normal IAP in a well child is 0 mm Hg. Both IAH and ACS are defined by sustained IAP elevations greater than 10 mm Hg with new or worsening organ dysfunction due to lower blood pressures in children.

A few additional terms were included in the 2013 consensus. A polycompartment syndrome is a condition in which compartmental pressures in 2 or more anatomic compartments are elevated (eg, the abdomen and chest). Abdominal compliance quantifies the ease of abdominal expansion, is determined by the elasticity of the abdominal wall and diaphragm, and is expressed as the change in intraabdominal volume per change in IAP. An open abdomen is any abdomen requiring a delayed or modified temporary abdominal closure when the skin and fascia are not closed until IAP normalizes.

Although ACS is arbitrarily stated to be a sustained IAP of at least 20 mm Hg, organ dysfunction has been reported to occur earlier, at an IAP of 10 mm Hg, when gut-wall edema may impair oxygen delivery with bacterial translocation and release of cytokines and free radicals (this is addressed in part 2).

History

Many contributors over the last century and a half have added to our understanding of IAH and ACS. Knowledge of the effects of increased IAP on individual organ systems has furthered medical and surgical management. For example, surgery involving repair of ruptured abdominal aortic aneurysms (AAA) and resuturing of dehisced abdominal wounds with retention sutures may create a “tight abdomen”; this practice has been reevaluated. Delayed closure techniques have been developed.

A comprehensive review of IAPs written over 100 years ago included observations and patient histories from the second half of the 19th century. The author described how Marey in 1863 and Burt in 1870 documented the effects of inspiration and expiration of the diaphragm on increasing and decreasing IAP. In 1865, Braune first measured IAP through the rectum. In 1873, Wendt noted “less secretion of urine” with increasing IAP when measured from the rectum. Oderbrecht measured abdominal pressure from the urinary bladder in 1875, and Henricius identified impaired diaphragmatic excursion and decreased cardiac diastolic distention. Henricius also appreciated that rapid abdominal distention was much less tolerated than abdominal distention that developed gradually. Elevated IAP impaired movement of the diaphragm with subsequent adverse cardiovascular effects of increased vascular resistance and decreased cardiac contractility, causing death in animal models. In 1923, Thorington and Schmidt, while studying experimental ascites, described the relationship of increased IAP on renal dysfunction and the restoration of renal function and urinary output after abdominal decompression. An anesthesiologist appreciated high mortality following abdominal wound dehiscence in 1951. He offered the terms “abdominal blow-out” and “acute tension pneumoperitoneum” from excessive free air trapped during abdominal closure, which increased IAP, and recommended delaying closure of the wound.
Measurement of IAP via intravesicular, intragastric, and intracolonic pressure in animal models was commonplace in the 1920s and 1930s, but it was Sönderberg and Westin who, in 1970, described a strong correlation between IAP and urinary bladder pressure during laparoscopy in humans.\textsuperscript{12}

Studies by Kron and colleagues in the early 1980s rediscovered IAP as a cause of unexplained oliguria and renal failure in postoperative patients with abdominal distention.\textsuperscript{13} Using IAP measurements taken from the urinary bladder to assess urgency of abdominal re-exploration and decompression could restore urinary output, decreased by high IAP/IAH. The term ACS was coined from observing complications following repair of ruptured AAA.

The experience of surgeons treating infants with gastroschisis or omphalocele further contributed to understanding the concept of “loss of abdominal domain” or inability of the abdominal cavity to accommodate normal visceral organs, as well as the life-threatening cardiac, pulmonary, and GI complications that occur when the abdomen is primarily closed in the presence of elevated IAP (as discussed in part 2).\textsuperscript{14}

Introduction of laparoscopy in the 1980s emphasized the injurious effects of elevated IAP on cardiac, pulmonary, renal, GI, hepatic, and cerebral function.\textsuperscript{15} Pneumoperitoneum decreased preload, increased afterload, and decreased cardiac output; pneumoperitoneum also decreased lung compliance and functional residual capacity and impaired gas exchange. Hepatic, renal, mesenteric, and GI blood flow are all impaired. Increased intracranial pressure occurs from raised intrathoracic pressure, the Trendelenburg position, and increased carbon dioxide. Intraabdominal pressures greater than 12 to 13 mm Hg over 1 hour during laparoscopy have been shown to decrease perfusion and cause anaerobic metabolism within the rectus abdominis muscle wall.\textsuperscript{16} Appreciation of these changes by anesthesiologists and surgeons has led to improved recognition and management of both IAH and ACS in critically ill patients.

**Incidence and Epidemiology**

The incidence of IAH and ACS is difficult to determine due to the insidious character in severely traumatized and critically ill patients who often have many comorbidities. Most studies evaluating the incidence of ACS have been performed in trauma patients, with estimates of incidence varying considerably. A review of 97 ICU admissions, of which 59% were medical and 41% surgical, indicated an overall incidence of IAH with an IAP of at least 12 mm Hg of 50.5% and an overall incidence of ACS with an IAP of at least 20 mm Hg of 8.2%.\textsuperscript{17}

In another prospective study, IAP measurements were obtained in critically ill patients admitted to the ICU requiring bladder catheterization. Of 83 patients, 33% developed IAH defined by an IAP of at least 12 mm Hg, and 53% died. Abdominal compartment syndrome defined by an IAP of at least 20 mm Hg developed in 12% with 80% mortality.\textsuperscript{18} Regueira and colleagues analyzed 81 consecutive patients with septic shock admitted to an ICU; 93% of the surgical patients and 73% of the medical patients developed IAH with an IAP of at least 12 mm Hg.\textsuperscript{19} Other researchers monitored 128 major trauma victims without abdominal injuries but receiving aggressive fluid resuscitation. Secondary ACS developed in 11 (9%). Mortality was 54%.\textsuperscript{20} Finally, in a review by Sugrue, approximately 35% of ICU patients developed IAH, and 5% progressed to ACS.\textsuperscript{21}

Thus, IAH and ACS are not uncommon, and due to the potentially prolonged course and grave prognosis of these disorders, awareness of these diagnoses should be a prominent and early consideration in critically ill patients.
Etiology and Patients at Risk

Patients with injury or disease originating in the abdominopelvic region, such as abdominal trauma, hemoperitoneum from vascular injury or rupture, and severe acute pancreatitis, are at risk for developing primary ACS. Patients with conditions that have not originated in the abdomen or pelvis, such as shock, sepsis, and burns, but who have received large crystalloid resuscitation, and patients sustaining extraabdominal trauma, are all at risk for developing secondary ACS. Abdominal compartment syndrome resulting from severe pancreatitis has been referred to as both primary ACS due to abdominal collections and inflammation and secondary ACS due to fluid resuscitation.\textsuperscript{22,23}

Large-volume fluid resuscitation, mainly with crystalloids, may be partially responsible for either primary or secondary ACS. Little has been written concerning onset time in regard to primary or secondary ACS. Some overlap between primary and secondary ACS classifications may be due to subtle distinctions between intraabdominal, intraperitoneal, and retroperitoneal nomenclature.

Patients with various disease processes have been identified as being at increased risk for developing IAH and ACS by the WSACS. These broad, possibly overlapping categories include conditions that cause 1) diminished abdominal wall compliance, 2) increased GI contents, 3) increased intraabdominal contents, and 4) fluid resuscitation resulting in fluid sequestration.\textsuperscript{5} Examples of risk factors with the potential for compromising abdominal wall compliance include complicated abdominal surgery as well as damage control laparotomy that provides hemostasis, injury repair, and decontamination, and often requires temporary abdominal closure. Obesity and burn eschars as well as severe osteoporosis may also reduce abdominal wall compliance. Increased GI contents may result from ileus, volvulus, colonic pseudo-obstruction, obstructing colon tumors, and gastroparesis.

Increased abdominal contents may include intraabdominal infections, abscesses, hematomas, tumors, ascites, pancreatitis, peritonitis, liver and spleen trauma, and ruptured AAA.

Large-volume fluid resuscitation with a positive fluid balance exceeding 1.5 to 2 L in 24 hours results in subsequent redistribution/sequestration in the gut wall.\textsuperscript{24} Other predisposing conditions include high ventilation pressure requirements, a prone or upright position, peritoneal dialysis, laparoscopy with excessive inflation pressures, and liver or renal transplants. A study by Holodinsky and colleagues included 2,500 surgical and medical ICU patients. Risk factors observed were clustered into 3 themes, identified by 1) the volume of fluids administered, 2) the degree of respiratory support needed, and 3) the presence of shock or hypotension.\textsuperscript{25} Other risk factors included metabolic derangements such as metabolic and respiratory acidosis and hyperchloremic acidosis from excess normal saline fluid administration/resuscitation and obesity.

Obesity may be understated as a risk factor. A large percentage of the population is overweight or obese (35%). Central obesity is associated with increased intraperitoneal fat. Intraabdominal pressure measurements performed during bariatric surgery were found to be increased in relation to the number of obesity-related comorbidities such as hypoventilation syndrome, pseudotumor cerebri, and metabolic syndrome.\textsuperscript{26} Acute increases in IAP/IAH may be less well tolerated in these patients with chronically elevated IAPs.\textsuperscript{27}
REFERENCES

Post-test

1. Diseases in which patients may be at risk for IAH and ACS include all of the following except:
   a. pancreatitis
   b. cirrhosis
   c. coronary artery disease
   d. sepsis, shock, and burns

2. The WSACS consensus definition of IAP measurement at which ACS occurs is ______.
   a. 4 mm Hg in children
   b. 15 mm Hg in geriatric patients
   c. 20 mm Hg in adults
   d. updated annually

3. Categories of patient risk factors include all of the following except:
   a. increased GI contents
   b. increased intraabdominal contents
   c. increased abdominal compliance
   d. excessive fluid resuscitation and sequestration

4. Primary diseases or injuries that may lead to IAH and ACS include ______.
   a. ruptured AAA
   b. sepsis, burns, and shock
   c. major torso trauma
   d. all of the above

5. The abdomen is defined by all of the following rigid borders except:
   a. the costal margin
   b. the diaphragm
   c. the spine
   d. the pelvis
6. **Compartment syndrome develops in a physiologic space when______.**
   a. tissue viability is threatened  
   b. hypotension develops  
   c. tissue oxygenation is compromised  
   d. all of the above

7. **Risk factors for IAH include______.**
   a. hypertension  
   b. positive fluid balance of 3 L  
   c. spontaneous respiration  
   d. a blood oxygen level of 120 mm Hg of the inspired oxygen fraction of 0.3

8. **Abdominal compartment syndrome was initially used in describing complications following:**
   a. abdominal cancer surgery  
   b. abdominal aortic aneurysm surgery  
   c. abdominal trauma surgery  
   d. abdominal exploratory surgery

9. **Effects of pneumoperitoneum during laparoscopic surgery include______.**
   a. increased intracranial pressure, decreased cerebral perfusion, and decreased blood oxygen level  
   b. increased afterload, increased diastolic filling, and decreased cardiac output  
   c. decreased hepatic artery blood flow, increased portal vein blood flow, and increased mesenteric blood flow  
   d. decreased pulmonary gas exchange, decreased functional residual capacity, and decreased lung compliance

10. **Central obesity components include______.**
    a. hypoventilation syndrome  
    b. metabolic syndrome  
    c. pseudotumor cerebri  
    d. all of the above