Lesson 45: Management of a Patient With Intraabdominal Hypertension And Abdominal Compartment Syndrome: Part 2

Written by: Jonathan R. Ashton, MD, Staff Anesthesiologist, Memorial Hospital of Carbondale, Carbondale, Illinois

Reviewed by: Adrian Martin, MD, Attending, General Surgeon, Memorial Hospital of Carbondale Carbondale, Illinois; Elizabeth A.M. Frost, MD, Professor of Anesthesiology, Icahn Medical Center at Mount Sinai, New York, New York

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Read this article, reflect on the information presented, then go online and complete the lesson post-test and course evaluation before the termination date below. (CME credit is not valid past this date.) You must achieve a score of 80% or better to earn CME credit.

TIME TO COMPLETE ACTIVITY: 2 hours
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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. Discuss the presentation and pathophysiologic effects
2. Emphasize the importance of integrating IAP measurements with clinical signs and symptoms in the diagnosis
3. List laboratory and imaging studies necessary to understand/appreciate severity
4. Suggest conservative medical and pharmacologic approaches
5. Describe interventional radiologic percutaneous interventions to prevent open decompression
6. List the indications and complications associated with surgical decompression and temporary/delayed abdominal closure
7. Recognize complications and alternative methods for limiting large fluid resuscitation for shock and sepsis
8. Understand the consequences of reperfusion syndrome
9. Draw up a plan for anesthetic management
10. Describe damage control resuscitation
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.

Pathophysiology

Increased intraabdominal pressure (IAP) and abdominal compartment syndrome (ACS) affect all bodily systems to a greater or lesser extent. Some of the more important complications are described.

Cardiovascular

IAH causes upward movement of the diaphragm increasing intra-thoracic pressure, cardiac compression and reduction of ventricular compliance and cardiac output. Pulsus paradoxus is an exaggerated decrease in systolic blood pressure (SBP) with inspiration that occurs in severe cases. These mechanisms are analogous to cardiac tamponade and, as has been named by some authors, a cardiac compartment syndrome. An IAP of as little as 10 mmHg can increase intra-thoracic pressure and reduce venous return and cardiac output. Compensatory cardiac responses include tachycardia from an intact sympathetic response. In severe cases, bradycardia may occur due to decreased coronary perfusion and increased systemic vascular resistance (SVR). These effects are more evident in patients who are hypovolemic and hypotensive or require high ventilation pressures.

Aortal compression results from IAH and increased afterload. The indices of filling pressures, central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP), may be deceptively elevated due to increased intra-thoracic pressure, making these monitors of limited use in estimation of volume replacement. Pulmonary vascular resistance (PVR) is increased from pulmonary parenchymal compression. Right ventricular end-diastolic volume (RVEDV), global end-diastolic volume (GEDV), esophageal Doppler monitoring (EDM) and stroke volume variation (SVV) are better indicators of intravascular volume status.
Abdominal distention obstructs venous return from both lower extremities and the inferior vena cava thus reducing blood return. IAH may also produce a mechanical narrowing of the inferior vena cava (IVC) at the diaphragmatic crura. Increased femoral venous pressures can promote formation of peripheral edema within the lower extremities and increase the risk of deep venous thrombosis (DVT). Relieving IAH and elevated femoral venous pressure have been reported to be accompanied by an increased incidence of pulmonary embolism as clots are dislodged from the legs and pelvis.

**Pulmonary**

Pulmonary effects of IAH include reduced spontaneous tidal volumes, decreased functional reserve capacity (FRC) and increased hypoxia and hypercarbia. Mechanically ventilated patients with IAH most often require increased peak inspiratory pressures and mean airway pressures due to reduced lung compliance and restrictive lung disease. High ventilation pressures carry an increased risk for alveolar barotrauma, volutrauma and pneumothorax. Extrinsic lung compression from an expanding abdominal cavity, compounded by the effects of large fluid resuscitation with redistribution, increases extravascular lung water, alveolar atelectasis, decreased oxygen diffusion and transport and increases the intrapulmonary shunt fraction.

**Renal**

IAH decreases glomerular filtration rate (GFR) secondary to decreased cardiac output, decreases renal arterial blood flow and renal vein compression, impairing renal venous blood outflow. Decreased GFR increases serum BUN and creatinine (renal insufficiency, azotemia and uremia). On average, oliguria begins at an IAP of 15 mmHg and anuria at IAP of 30 mmHg.

Decreased cardiac output and hypotension decrease renal artery blood flow and activate the renin-angiotensin system. Plasma renin activity on angiotensinogen produces angiotensin I which is converted to angiotensin II by angiotensin converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor and stimulates the release of both aldosterone and antidiuretic hormone (ADH). Thus, IAH may be considered a pre-renal cause of acute renal failure and acute tubular necrosis.

The kidney is enclosed in a fibrous capsule. A hematoma within the capsule from trauma or coagulopathy may compound the effects of rising kidney interstitial edema adversely effecting tissue perfusion and organ function. Increasing interstitial renal edema from IAH is thought to create an intrarenal compartment syndrome.

**Gastrointestinal**

Twenty-five percent of the circulatory volume capacity may be in the splanchnic circulation. Initially, increasing IAP and sympathetic vasoconstriction compress mesenteric veins and increase venous return. However, as IAP further increases, venous return decreases as mesenteric veins and the inferior vena cava (IVC) become more obstructed. Also mesenteric vein compression impairs venous return with subsequent development of intestinal edema further increasing IAP. Thus a vicious cycle evolves. Mesenteric blood flow may be reduced when IAP is only elevated to 10 mmHg. In addition, hepatic artery and portal venous blood flow may be reduced with only modest increases in IAP of 10-20 mmHg.

All intra-abdominal and retroperitoneal organs demonstrated decreased blood flow with increasing IAP during in vitro studies. However, this same study found that blood flow to the adrenal glands
increased, considered to be a sympathetic response enabling catecholamine release in the presence of shock.

The end result of reduced venous return and increased abdominal interstitial edema is hypo-perfusion, bowel ischemia, decreased intra-mucosal pH, feeding intolerance and systemic metabolic lactic acidosis. Gut hypo-perfusion with loss of the mucosal barrier integrity and immunity protection allow subsequent bacterial translocation, (cytokine transfer and other excitatory immunomodulators) sepsis and multiple system organ failure (MSOF).36

Liver

Flow in the hepatic artery and vein as well as the portal venous system are diminished from decreased cardiac output as well as extrinsic compression of the liver and a theoretical anatomic narrowing of hepatic veins passing through the diaphragm. Hepatic congestion reduces portal vein blood flow increasing azygos blood flow and gastro-esophageal collateral blood volume. Esophageal varices are at greater risk of rupture.37 Hepatic metabolism of glucose, albumin synthesis, coagulation factor production as well as detoxification mechanisms are impaired.

Abdominal Wall

Visceral edema, abdominal packs, and free intraperitoneal fluid all distend the abdomen and reduce abdominal wall compliance. Excess fluid resuscitation in response to shock leads to sequestration and increases intra-abdominal edema. Rectus abdominis muscle blood flow reduction may occur at an IAP of only 10 mmHg. Impaired wound healing, a high rate of fascial and skin dehiscence and predilection to development of necrotizing fasciitis have been found in cases where the abdomen was closed under tension.

Central nervous system

The cranium has four primary components including the skull, brain parenchyma, cerebral spinal fluid (CSF) and blood volume. The Monro-Kellie hypothesis states that a change in volume in one of these components requires a reciprocal change in volume in the other non-osseous components.38 Blood volume and blood flow as well as CSF are the homeostatic buffer mechanisms for maintaining normal intracranial pressure (ICP) of 5-12 mmHg. When these buffer mechanisms are depleted, ICP rises steeply. ICP greater than 15 mmHg is referred to as intracranial hypertension and ICP greater than 25 mmHg may cause cerebral herniation.

ICP transiently increases during short-lived periods of elevated intra-abdominal pressure such as coughing, defecation or emesis. Intracranial pressure (ICP) appears to be elevated with persistent/chronic IAH. A gradual increase in ICP over time allows for buffer mechanisms to compensate; but acute changes from either traumatic injury and/or acute illness may result in rapidly escalating intracranial pressures. Increased abdominal pressure and/or intra-thoracic pressure may also affect pressures in the cranium by impairing cerebral venous outflow. Obstructed lumbar venous plexus blood outflow may create increased ICP from displacement of lumbar CSF cephalad. Decreased venous return from either the head or back impeding removal of CO2 may increase cerebral vasodilation increasing ICP.39 Intracranial hypertension lowers CPP leading to cerebral ischemia.
Poly-compartment Syndrome

A poly-compartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures. Primary poly-compartment syndrome is defined as a pathological rise of pressure from injury or disease directly related to the affected compartment such as intracranial hematoma or limb fracture. Secondary poly-compartment syndrome symptoms are based on pressure transmission from one compartment to another such as elevated thoracic or chest pressures secondary to elevated abdominal pressures. Large fluid resuscitation for shock and sepsis is a major mechanism predisposing to secondary poly-compartment syndrome. Poly-compartment syndromes are uncommon but have significant consequences. For example, the abdominal compartment may play a key role in the cause and/or effect of pathophysiology in what has previously been referred to as cardiorenal and hepatorenal syndromes, more descriptively referred to as cardio-abdominal-renal syndrome (CARS) and hepato-abdominal-renal syndrome (HARS). When medical management of these critically ill patients has been maximized, surgical procedures such as decompressive craniotomy, sternotomy, pericardiectomy, fasciotomy and laparotomy may be the only treatment options remaining to offer physiologic improvement.

Diagnosis of IAH/ACS

IAH/ACS results from a broad number of primary disease processes and major injuries. The presentation and diagnosis are diverse and varied. Clinical signs and symptoms combined with elevated IAP measurements help confirm the diagnosis. Clinical parameters for assessing early compromised organ function are recognized. Pulmonary failure may be assessed by worsening arterial blood gases; renal failure is evaluated by serum creatinine level and urine output; hepatic failure is measured by liver function tests, coagulation profile and albumin and cardiac failure measured by filling pressures, systemic vascular resistance (SVR) and cardiac output. Lung ultrasound evaluates lung water; and transesophageal echo and esophageal Doppler monitoring provide information pertinent to the need for vasopressors.

Most patients are critically ill and unable to communicate. Associated symptoms include general malaise, weakness, lightheadedness, dyspnea, abdominal bloating and/or abdominal pain. Physical signs are an ashen, pale and cyanotic appearance. Patients may present with a tense distended abdomen as well as dyspnea, fever, tachycardia and hypotension. Progressive oliguria and increased supportive ventilation requirements may occur as well as increased edema/anasarca and elevated jugular venous distention. Other indicators of hypo-perfusion include cool skin, obtundation and altered level of consciousness and restlessness.

Laboratory findings show multiple abnormalities in complete blood counts, chemistry and blood gases depending on primary pathology. Decreased hematocrit may be present in trauma, hemorrhage and large fluid resuscitation. White blood cell count may be elevated in sepsis. Metabolic and respiratory acidosis, increased creatinine and abnormal liver function tests (LFTs) occur. Image findings on chest x-ray are decreased lung volumes, atelectasis and elevated hemi-diaphragms. The CT abdomen may show abdominal distention, hemoperitoneum, bowel wall thickening, infiltration of the retro-peritoneum, extrinsic compression of the IVC, renal compression and bilateral inguinal herniation.

Direct methods of IAP measurement include insertion of a needle into the peritoneum directly or during laparoscopy. Less invasive and indirect methods use intragastric, intracolonic, urinary bladder and
central venous catheters. Transducing the pressure within these organs is believed to correlate with surrounding pressures. The urinary bladder has evolved as the standard screening measurement (recommended by the WSACS in 2013). With a standard vascular transducer improvised to connect to a Foley catheter, reproducible pressure measurements are best taken in the supine position at end-expiration. The transducer is zeroed at the mid-axillary line in adults or pubic symphysis in children. Correlation between bladder and direct intra-abdominal measurement is satisfactory. Relative contraindications include bladder hematomas, uro-genital anomalies, recent bladder surgery and presence of neurogenic bladder.

Many clinicians initially assess signs such as abdominal distension and increasing girth as well as oliguria and hemodynamics. But clinical assessment alone is believed to be less than optimal having a sensitivity of around 40%. The addition of IAP measurement is advocated as the standard of care. There should be a low threshold for measuring IAP in patients at risk of IAH/ACS who present with severe trauma, AAA repair, bowel obstruction, pancreatitis, peritonitis or organ transplant.

**Management and treatment**

Conservative medical care for IAH/ACS includes adequate analgesia and sedation. Tasdogan et al. studied the effects of propofol versus dexmedetomidine infusion in 40 patients with severe sepsis post abdominal surgery. The IAP dropped significantly more in the dexmedetomidine group versus the propofol group. At 24 hours, IAP was 12.35 mmHg and 18.1 mmHg in the dexmedetomidine and propofol group respectively; and at 48 hours, 13.9 mmHg vs 18.7 mmHg.

Ileus is not uncommon following colectomy and patients maintained on narcotics and sedatives. Evacuation of intestinal contents is performed using nasogastric and rectal tube drainage. Endoscopic gastrointestinal decompression may be considered if nasogastric tube and rectal tube decompression are inadequate. Pro-kinetic agents including metoclopramide which works through dopamine antagonism and erythromycin (EEM) which acts via a direct contractile effect from the motilin receptor may also be used. Aperients, laxatives, cathartics and enemas may be considered for constipation/obstipation. Alvimopan has peripheral μ-opioid antagonist properties and is approved to treat post-operative ileus in the hospital setting.

A severe form of a-dynamic ileus also known as Ogilvie’s syndrome may develop in both medical and surgical patients. It is a non-mechanical pseudo-obstruction that consists of massive dilation of the colon in the absence of mechanical obstruction and may be caused by ineffectual colonic motility from excessive sympathetic stimulation (catecholamine infusion) and/or parasympathetic dysfunction. A spontaneous bowel perforation occurs in 3% with 50% mortality. Reduction colonoscopy is technically difficult and complicated by perforation or recurrence. The obstruction may respond to a slow IV infusion of neostigmine, an anticholinesterase. Positive response to neostigmine with flatus and or stool occurs in 30 minutes with a median response time of 4 minutes. Bowel perforation may be a complication with neostigmine usage. Neostigmine is relatively contraindicated in patients with bradycardia and reactive airway disease (RAD). Patients on beta-blockers are more sensitive to the cholinergic effects of neostigmine. The procedure requires EKG monitoring and bedside atropine.

Several other therapies have been studied. Octreotide is a synthetic somatostatin analogue that reduces portal venous pressure in esophageal varices. It has also been studied for use in treatment of bowel ischemia and reperfusion injury by limiting oxidative damage from free radicals and increasing glutathione antioxidant levels. Melatonin, a secretory product of the pineal gland with free radical
scavenging and anti-oxidative properties is said to have anti-inflammatory effects and inhibits activation of neutrophils by free-radicals thus limiting reperfusion injury.\textsuperscript{48} Extra-corporeal techniques such as hemodialysis and/or ultrafiltration may be effective in removing excess fluids and edema.

Ventilatory support may require high airway pressures for satisfactory oxygenation and carbon dioxide elimination, which increases the risk of volutrauma/barotrauma as well as further increasing IAP. Decreased tidal volume, use of pressure-limited mode and permissive hypercapnia have been suggested in patients requiring high inspiratory and mean airway pressures for extended periods of time.

Neuromuscular blockade may reduce intrinsic abdominal tone, improve efficiency of mechanical ventilation and reduce carbon dioxide production and retention. Administration of PEEP may reduce ventilation-perfusion mismatch and improve hypoxemia/hypoxia.

Vasopressors and inotropes dobutamine, dopamine, noradrenaline have been advocated to add hemodynamic support. The primary objective of treatment should always focus on the primary pathology. Supportive medical management of the underlying pathology may be of limited efficacy but should be initially attempted as it may provide for optimization of refractory IAH/ACS and allow further consideration of advantages and disadvantages of major surgery.

Prior to performing open decompression of IAH/ACS, a less invasive percutaneous catheter drainage (PCD) method is recommended for temporarily relieving IAH due to intra-peritoneal and/or retroperitoneal fluid, ascites and/or blood. Ultrasound and CT scan guidance locate fluid collections. Hemorrhage, visceral perforation, infection, fluid shifts and electrolyte imbalance are possible complications of PCD. In one report of 31 patients, 25 patients (81%) were treated successfully with PCD and avoided surgery. Drainage of greater than 1000 cc and lowering of IAP by 9 mmHg within the first four hours were associated with successful decompression.\textsuperscript{49} Parameters said to improve in children following PCD include IAP, abdominal perimeter, urine output, creatinine clearance, PaO2, PaCO2 and gastric retention.\textsuperscript{50} When conservative means have been inadequate, surgical abdominal decompression may become imperative.

Guidelines for open surgical decompression include an IAP pressure above 25 mmHg and an abdominal perfusion pressure below 50 mmHg.\textsuperscript{5} Evidence-based management studies indicate that surgical decompression performed early when IAP is 25-30 mmHg versus 30-40 mmHg reduce ICU, ventilator and hospital days and improve survival.\textsuperscript{51} Due to complications and high patient acuity, surgical decompression survival rates for ACS remain around 50%.\textsuperscript{52} The acute physiology and chronic health evaluation (APACHE II) and injury severity score (ISS) are generally high.

Open abdominal surgical decompression may have origins in techniques learned in the battlefield. The concept of “damage control” was drawn from US Navy ship salvage techniques to maintain mission integrity after the ship had been struck and damaged. This concept was extended to the management of acutely injured patients. The goal of “damage control laparotomy” is primarily to inspect and repair injuries, providing hemostasis and decontamination. Patients often received massive crystalloid and blood fluid resuscitation which often predisposes to sequestration producing a distended abdomen at the time of closure.

Damage control resuscitation is medical management to complement damage control laparotomy and open decompressive exploratory laparotomy. Following surgery, patients are taken to the ICU for stabilization and, if necessary, correction of coagulopathy (INR>1.5), hypothermia (<33 C) and acidosis
(pH < 7.2) referred to as the “lethal triad”.  

A strategy implemented to prevent coagulopathy includes

1. administering higher ratios of plasma and platelets to red blood cells,
2. limiting crystalloids, and
3. permissive hypotension. 

Although controversial in modern current civilian practice, the concepts of permissive hypotension evolved historically when increased patient survival among young soldiers was observed when endpoints of systolic blood pressures were believed acceptable at 70-80 mmHg and 85 mmHg respectively in WWI and WWII. 

Reperfusion syndrome may occur at the time of abdominal decompression and produce hemodynamic instability with arterial hypotension and dysrhythmias. Vigilant and prepared management require fluids, blood products, vasopressors/inotropes, as well as all other resuscitation supportive medications and methods. Upon reperfusion of ischemic organs, toxic oxygen metabolites and inflammatory mediators are released and make for an unstable course. Metabolic acidosis, hyperkalemia and a variety of pro-inflammatory mediators from both peripheral and mesenteric vasculature occur after decompression and reperfusion. These mediators compromise capillary integrity and capillary leak occurs leading to multiple system organ failure (MSOF). Systemic sequelae related to reperfusion of ischemic organs, shock and sepsis include systemic inflammatory response syndrome (SIRS), acute lung injury (ALI), and adult respiratory distress syndrome (ARDS).

Patient responses to successful decompression of IAP and corrections in homeostasis are generally achieved within three days. Mobilization of extravascular fluid and diuresis results in negative fluid balance. Improving IAP should be accompanied by improved lung compliance, cardiac output, oxygenation and ventilation, renal function and urine output. Weaning of supportive inotropic infusions and high-pressure ventilation requirements may then be possible.

Decompressive laparotomy in patients can be achieved by either a midline laparotomy or transverse subcostal full-thickness incision. Subcutaneous linea alba fasciotomy (SLAF) is a less invasive surgical option that involves three short horizontal skin incisions leaving the peritoneum and skin intact for visceral protection. In a collective analysis of 161 patients undergoing full-thickness surgical decompression via midline laparotomy, the mean reported IAP before surgical decompression was 34.6 mmHg and fell to 15.5 mmHg after decompression. Indices of multiple organ systems functions improved. In analyzing the first 10 patients with severe acute pancreatitis (SAP) undergoing SLAF at the Meilahti Hospital in Helsinki, the mean preoperative IAP was 31 mmHg and fell to 20 mmHg immediately postoperatively with a mean decrease of 10 mmHg. SLAF is effective in about 50-70% of patients and carries fewer complications than an open abdominal approach.

The OR is preferred for open decompression as it provides superior aseptic condition, lighting, equipment and personnel. However, in urgent situations, ICU bedside decompression negates transfer of critically patient with multiple monitors and infusions. Upon completing open decompression, a temporary abdominal closure (TAC) technique may be the best option to maintain a lower IAP at levels for satisfactory organ function. The potential for evisceration/dehiscence is also lessened. Open decompression may be accompanied by an increased risk of adhesions, fluid shifts/losses from exposed bowel and peritoneum, bowel injury, sepsis, graft infections, herniation and entero-cutaneous fistulae with significant morbidity and mortality. Other complications include ileus, malnutrition and skin and
fascia lateralization.

Lateralization of the abdominal wall is the phenomenon where the musculature and fascia of the abdominal wall, most exemplified by the rectus abdominal muscles and the enveloping fascia contract laterally away from the midline. Vacuum-assisted fascial closure for TAC is believed to reduce the incidence of fascia lateralization, adhesions and reduces the occurrence of hernias. Final closure is possible within 5-7 days following decompressive surgery and TAC. Normalization of IAP and improving organ function and fluid balance are indicators for proceeding with definitive closure.

**Fluid Considerations**

Whether crystalloids or colloids are best for immediate resuscitation in shock, sepsis, etc. has been debated. Multiple fluids have been studied including isotonic sodium chloride solution, lactated Ringers solution, Plasmalyte®, hypertonic saline, albumin, purified protein fraction, fresh frozen plasma, hetastarch, pentastarch and dextran 70.

Advocates of colloid resuscitation argue for the greater oncotic pressure provided which decreases sequestration. However, severely ill patients with renal and liver failure may be more susceptible to greater capillary leakage of colloid material from the pulmonary vasculature. The presence of inflammatory mediators damages pulmonary capillary membranes and the lymphatic system can be overwhelmed. Synthetic colloid solutions including hetastarch, pentastarch, and dextran 70 have high molecular weights and remain in the intravascular space longer than the natural colloids including purified protein fraction (88% albumin and 12% globulin), fresh frozen plasma and albumin.

The European Society of Intensive Care Medicine (ESICM) advises against the use of colloids such as hydroxyethyl starches (HES) in patients with severe sepsis, burns or at risk of acute kidney injury.59 In addition, colloids are not recommended for patients with head injury and gelatins and HES should not be administered to organ donors.

Current trauma resuscitation of the severely injured patient focuses on restoration of clotting factors, as well as depleted and dysfunctional platelets through the concept of damage control resuscitation, with proactive transfusion of a higher ratio of plasma and platelets to red blood cells.60 Massive transfusion using protocols with higher ratios of plasma and platelets to packed red blood cells appear to be associated with improved survival.61 Many authors have demonstrated favorable outcomes associated with plasma and platelet ratios approximating whole blood including decreased costs, blood product utilization, organ failure rates, open abdomens, and six hour, 24 hour and 30 day mortality.53 Large volume crystalloid-based resuscitation is associated with a higher frequency of ACS, and mortality. Incidence of pulmonary complication, gastrointestinal motility dysfunction and coagulation disturbances are also higher.

Critically ill patients require individual management. Many crystalloid and hypertonic solutions as well as colloids and blood components are available and have been studied. Initially resuscitation may require significant fluid and blood component administration. Inotropic support, diuresis and renal dialysis are often needed.
Anesthetic considerations in ACS

Anesthetic considerations in any critically ill patient should include placement of invasive lines for both venous access and monitoring due to hemodynamic instability. Strict aseptic techniques must be maintained. The importance of preventing hypothermia, one of the components of the lethal triad cannot be overemphasized to prevent infection and adverse cardiac events. Use of forced-air warming blankets and intravenous fluid warmers throughout is imperative. Entropy monitoring and cerebral oximetry may help with assessment and management of cerebral oxygenation and cognitive function.

Etomidate maintains hemodynamic stability. Disadvantages include pain on injection, adrenal suppression and myoclonic activity. Effects of adrenal suppression have been observed after a single dose. Ketamine, a dissociative and analgesic drug is an alternative induction agent for use in labile patients due to its central sympathetic activity provided the autonomic nervous system is intact. However, ketamine may increase ICP which may already be elevated from a primary condition or from IAH/ACS. At low titrated induction doses most modern IV anesthetics are acceptable and unlikely to produce gross hypotension. Propofol may be a good choice of anesthetic when ischemia-reperfusion injury is anticipated. It inhibits lipid peroxidase production and enhances the antioxidant production of glutathione in vivo. Propofol, also increases the fluidity of the erythrocyte membrane preventing hemolysis as shown in a study of human erythrocytes. Sevoflurane has similar anti-oxidant properties. Desflurane and sevoflurane are rapidly titrated and eliminated from the body should abrupt hemodynamic depression be encountered. N2O is not advised due to the potential for further bowel or other air-filled space distension and reduction in FIO2 delivery. Moreover it negates protective effects of inhalational agents. Morphine releases histamine which produces peripheral vasodilation and hypotension. Fentanyl and sufentanil afford greater hemodynamic stability.

Muscle relaxation by decreasing muscle tone, and oxygen consumption, reduces abdominal pressure. Side effects of succinylcholine may be increased in patients with renal compromise and include dysrhythmias, hyperkalaemia and myoglobinuria. However, the rapid onset may make it the drug of choice in select patients. Pharmacodynamic and pharmacokinetic effects of muscle relaxants may be unpredictable due to compromised hepatic metabolism, decreased protein synthesis and hepatobiliary and renal clearance.

Management of the case

The patient was taken to the operating room as the IAP reached 30 mmHg. The airway had been secured. Anesthesia was administered with low dose fentanyl, rocuronium and desflurane. Oxygen was adjusted to maintain SpO2 >94%. Arterial blood gases showed a pH of 7.31, paCO2 of 45 mmHg, paO2 95 and a base deficit of -8. Minute ventilation was increased and 50 meq of bicarbonate were administered for combined respiratory and metabolic acidosis. The abdomen was opened and decompressed. Peritoneal irrigation was performed and the pancreas was inspected for necrosis as well as the bowel for ischemia, obstruction and perforation. Partial pancreatectomy of a necrotic segment was performed with multiple lysis of adhesions. Temporary abdominal closure was chosen using vacuum-assisted fascial closure (VAFC). The patient was returned to the ICU, sedated with dexmedetomidine. Intra-abdominal pressure decreased, abdominal swelling improved with supportive care. Primary abdominal wound closure was completed 3 days later. After a protracted course, the patient’s trachea was extubated. She required care in a rehabilitation facility for 3 months and was finally able to return home.
Summary

In critically ill patients, the centrally located abdomen plays an interactive role of cascading pathology involving all organ systems. Both surgical and medical critically ill patients have been found to have a high incidence of increased intra-abdominal pressure in the ICU when urinary bladder IAP measurements are added to the clinical assessment.

The WSACS has assigned definitions to IAH and primary and secondary ACS to serve as guidelines for diagnosis, management and treatment. Most important are the pathophysiologic effects of ACS which must be recognized and treated urgently. Early and aggressive management includes medical supportive care and interventional radiology which may be able to prevent operative decompression, a last resort.

IAH/ACS is not uncommon. Evolving evidence-based science and experience will enhance management recommendations. Intervention strategies should continue to be studied and explored, especially a more rational approach to fluid replacement, guided by such monitors as stroke volume variation and esophageal Doppler monitoring. Awareness of patients at risk and early treatment decreases use of prolonged expensive medical and surgical resources and improves outcome.

Dr. Elizabeth A.M. Frost, who is the editor of this continuing medical education series, is clinical professor of anesthesiology at The Mount Sinai School of Medicine in New York City. She is the author of Clinical Anesthesia in Neurosurgery (Butterworth-Heinemann, Boston) and numerous articles. Dr. Frost is past president of the Anesthesia History Association and former editor of the journal of the New York State Society of Anesthesiologists, Sphere. She is also editor of the book series based on this CME program, Preanesthetic Assessment, Volumes 1 through 3 (Birkhäuser, Boston) and 4 through 6 (McMahon Publishing, New York City).
REFERENCES


Post-test

1. **Cardiac effects of ACS include all but:**
   a. Decreased venous return
   b. Decreased cardiac output
   c. Decreased ventricular compliance
   d. Decreased pulmonary vascular resistance (PVR)

2. **Advantages of using temporary abdominal closure (TAC) and vacuum-assisted fascial closure include:**
   a. Reduced occurrence of hernias
   b. Decreased incidence of dehiscence/evisceration
   c. Reduced occurrence of adhesions and fascia lateralization
   d. All the above

3. **Guidelines for performing the procedure of urinary bladder measurement of IAP include all except:**
   a. Semi-recumbent
   b. End expiration measurement
   c. Zero transducer at mid-axillary line in adults
   d. Vascular transducer connected to a Foley catheter

4. **Which anesthetic agent is best avoided in managing patients with IAH/ACS?**
   a. Propofol
   b. Nitrous Oxide
   c. Desflurane
   d. Fentanyl

5. **Diagnostic criteria of IAH/ACS might include:**
   a. Decrease in cardiac output
   b. Decreasing creatinine clearance
   c. Metabolic alkalosis
   d. Decreasing ICP
6. **Pulmonary effects of IAH include:**
   a. Reduced spontaneous tidal volumes
   b. Increased functional reserved capacity
   c. Hypocarbia
   d. Increased lung compliance

7. **Damage control laparotomy does NOT include:**
   a. Repair injuries
   b. Obtain hemostasis
   c. Decontamination
   d. Pericardiocentesis

8. **Best anesthesia management for critically ill patients includes:**
   a. Fluid warmer and forced air blanket warmer
   b. Entropy and cerebral oximetry
   c. Invasive lines for strict hemodynamic monitoring
   d. All the above

9. **Recommendations for preventing coagulopathy in critically ill and traumatized patients include all the following except:**
   a. Permissive hypotension
   b. Permissive hypercarbia
   c. Limiting crystalloid
   d. Administering high ratios of fresh frozen plasma and platelets to packed red blood cell

10. **Which finding is not included in the ‘lethal triad’ of critically ill surgical and medical patients?**
    a. Hypothermia
    b. Acidosis
    c. Coagulopathy
    d. Hyperthermia