Abdominal Compartment Syndrome: A Decade of Progress

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Unlike many disease processes that surgeons treat in which progress is slow, the knowledge, definition, diagnosis, research, and treatment of abdominal compartment syndrome (ACS) have undergone dramatic improvements during the past decade. Considering that ACS was only well characterized and physiologically defined in the laboratory in 1985, and better clinically defined in 1989, rapid progress has occurred in both the diagnosis and treatment of this relatively young entity.[1,2] In fact, the mortality rate has dropped from 60% to between 34% and 37% in just the last 10 years.[3-5]

Abdominal compartment syndrome is defined as a sustained intra-abdominal pressure (IAP) >20 mmHg that is associated with new onset of organ dysfunction or failure.[6] Although often mistakenly used interchangeably, ACS is a separate and distinct entity from intra-abdominal hypertension (IAH), which is defined as a sustained or repeated pathologic elevation of the IAP ≥12 mmHg.[6] Intra-abdominal hypertension does not cause organ dysfunction, and that is the key difference between the 2 disease processes. Normal or mean IAP within the nondiseased abdominal cavity is between 2 and 5 mmHg, depending on overall body mass index (BMI), but can run as high as 12 mmHg in the obese adult[7] (Table 1). This chronic IAH does not produce organ injury and is purely a result of visceral obesity. It is also important to realize that normal IAP in the critically ill, who are almost always fluid overloaded and edematous, runs slightly higher, at 5 to 7 mmHg.[6]

Determining intra-abdominal pressure

The standardized method of determining IAP is by measuring the bladder pressure transmitted through a Foley catheter. This is correctly performed by clamping the catheter tubing beyond the rubber or plastic diaphragm that allows needle puncture access into the lumen of the catheter, and then instilling a maximum volume of 25 mL saline into the bladder.[6] Overdistending the bladder with excessive volumes of fluid will increase the bladder pressure and not reflect the true IAP.[8] The IAP should be measured at end expiration with the patient supine and relaxed or sedated, with the transducer zeroed at the mid-axillary line.[6] Having the patient slightly upright at >30 degrees has been clearly shown to produce erroneous results because IAP will increase as the head of the bed is elevated from 10 to 30 to 45 degrees.[9]

Once the IAP is measured, the abdominal perfusion pressure (APP) can be calculated. The APP is simply the mean arterial pressure minus the IAP. To maintain adequate perfusion to the viscera, an APP of at least 60 mmHg is desired.[6] For example, a critically ill patient with a mean arterial blood pressure of 80 mmHg and an IAP of 22 mmHg, has an APP of 58 mmHg, which is just below the critical level. This is when end-organ dysfunction can begin to occur.

Primary, secondary, and tertiary abdominal compartment syndrome

There are 3 different types of ACS: primary, secondary, and tertiary, which is also known as recurrent ACS. Primary ACS refers to ACS that occurs due to a primary intra-abdominal (or intrapelvic) cause, such as a ruptured abdominal aortic aneurysm, abdominal trauma, or retroperitoneal hemorrhage[10]; these are the most common causes. However, other rare conditions, such as malignant ascites, a giant ovarian tumor, a rectus sheath hematoma, and ACS after Roux-en-Y gastric bypass, have also been reported.[11-13] Almost any pathology that creates a space-occupying or expanding lesion within the abdominal or pelvic cavity can cause ACS.

Secondary ACS (also known as extra-abdominal compartment syndrome) refers to ACS that occurs as a result of massive bowel edema secondary to sepsis, capillary leak, conditions requiring massive fluid resuscitation, or burns.[14-16] Secondary ACS occurs most commonly after hemorrhagic shock requiring massive fluid resuscitation or severe burn injuries requiring massive fluid infusion. Although these situations result in total body anasarca, it is the swelling of the bowel that limits renal

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Abbreviations and Acronyms

ACS = abdominal compartment syndrome
APP = abdominal perfusion pressure
BMI = body mass index
IAH = intra-abdominal hypertension
IAP = intra-abdominal pressure
ICP = intracranial pressure
RVEDVI = right ventricular end diastolic volume index

perfusion and ventilation due to increased abdominal pressure. There is also an entity known as recurrent ACS (or tertiary ACS) in which ACS recurs after resolution of an earlier episode of either primary or secondary ACS. Recurrent ACS is relatively uncommon and tends to be the result of an overly aggressive attempt at abdominal closure in an edematous patient with an open abdomen.

Prevalence and pathophysiology

The prevalence of ACS is difficult to quantify and varies with the patient population that is studied and the acuity of those patients. In the general medical or surgical population, the incidence has been reported to be around 0.5% to 8%, but increases to 6% to 14% in trauma patients, depending on the classification of primary, secondary, or both.[14,17,18] The incidence is even higher in burn patients, occurring in 1% to 20%, depending on severity and percentage burn.[18]

The various cascades of molecular and cellular events that ultimately lead to ACS are multifactorial, with extensive interplay between the capillary membrane and interstitial interface, combined with neutrophil activation and hemodilution. I will now examine some of these models at the microvascular level. Regardless of which model is used as the example, the inciting event, whether it be trauma with hemorrhagic shock, sepsis, or burns, all lead to a capillary leak syndrome resulting in the extravasation of fluid into the interstitium and massive bowel wall edema. The bowel wall has an enormous capacity to hold fluid, and a patient can easily sequester several liters of fluid within the bowel wall and mesentery. This edema leads to increased pressure within the abdominal cavity, which ultimately leads to decreased renal perfusion, decreased bowel perfusion, and loss of ventilatory capacity.

Table 1. Grading System for Intra-Abdominal Hypertension

Grade	Internal pressure, mmHg	Description
Normal	<12	This accounts for the obese
1	12–15	
2	16–20	Unusual to have organ dysfunction in this range
3	21–25	Not equivalent to ACS unless organ dysfunction occurs
4	>25	With organ dysfunction the terminology changes to ACS

ACS, abdominal compartment syndrome.

A common cause of IAH and ACS is sepsis requiring massive fluid resuscitation. Clinical and basic science research has shown that interleukin-6, interleukin-8, and tumor necrosis factor–α all act directly on the endothelial cell of the capillary beds within the bowel wall to contribute to the capillary leak syndrome and bowel wall edema.[19,20] In addition, when the septic source is located within the abdomen, such as with appendicitis, the peritoneal fluid itself has been shown to contain elevated levels of interleukin-6 and tumor necrosis factor–α, which in turn prime neutrophils and fuel the inflammatory cascade and capillary leak.[21,22] *Neutrophil priming* refers to an enhanced and overly exaggerated secondary response to injury that occurs after an initial injury or stimulus.[23] Priming results in the neutrophil expressing certain adhesion molecules on its surface, specifically CD11b and CD18, which allows the neutrophil to adhere to the endothelial cell of the capillary and extravasate into the interstitium.[24,25]

Central vasodilation and shunting of blood from the periphery during sepsis result in hypotension, which is usually managed with a combination of fluid resuscitation and vasopressors. Vigorous fluid resuscitation during a phase of capillary leak alters the normal hydrostatic and oncotic pressures, resulting in the formation of intestinal edema because of a net efflux of fluid into the interstitium.[26] As the bowel edema worsens and IAP increases, upward pressure on the diaphragm increases central venous pressure, creating a pressure gradient against venous outflow and causing abdominal venous hypertension. The increased IAP also hinders lymphatic efflux of fluid from the abdomen, which only worsens the process.[26] All of these factors combining at once contribute to the growing edema within the bowel wall and mesentery, and IAP continues to increase. The septic patient who has been aggressively fluid resuscitated is at obvious risk for ACS.

Another common cause of IAH and ACS is abdominal trauma, leading to hemorrhagic shock requiring massive fluid resuscitation. In this model, the initial traumatic event leads to direct blood loss from vascular damage within the abdominal cavity. Whether the patient requires surgery to control the blood loss or is managed nonoperatively, the patient has lost a considerable amount of blood and requires either blood transfusion

or crystalloid resuscitation to restore their circulating blood volume. Similar to the septic model, a microvascular capillary leak syndrome is created in which fluid extravasates from the bloodstream and into the interstitium of the bowel wall and mesentery creating edema, but the inciting events are different. After trauma, blood loss followed by resuscitation or transfusion creates an ischemia-reperfusion injury to the bowel, which results in increased capillary permeability.[26] The main mediators of these events are interleukin-1, interleukin-6, interleukin-18, monocyte chemoattractant protein 1, and tumor necrosis factor—α.[27,28] With ongoing fluid resuscitation, decreased oncotic pressure from crystalloid use, and increased capillary permeability due to the mediators mentioned previously, all combine to cause a rapid infusion of fluid into the bowel interstitium and create edema leading to increased IAP.

The same holds true for massive fluid resuscitation after burns, but again, the molecular mediators are slightly different. After a large burn, basic science research has shown that there are increased systemic levels of vascular endothelial growth factor, interleukin-6, interleukin-8, and tumor necrosis factor—α.[29-31] After a burn, it is these systemic up-regulators of the inflammatory response that create endothelial cell leakage from the capillary bed and result in a net fluid flux into the interstitium of the bowel and all the tissues of the body. For this reason, plasma exchange, which decreases the systemic levels of inflammatory cytokines in the bloodstream, has beneficial effects after a major burn.[32] But just as in the other models, the result is a capillary leak syndrome and bowel edema that increases IAP and leads to IAH.

In summary, regardless of the inciting event, an inflammatory cascade of events and resuscitation leads to increased hydrostatic pressure, decreased oncotic pressure, ischemia and reperfusion injury, and increased capillary permeability. This leads to interstitial edema, bowel wall edema, and increased IAP, which then leads to increased central venous pressure, both of which cause mesenteric venous hypertension and decreased lymphatic flux out of the abdomen.[23] The result is progressively swollen and more edematous bowel and IAH or ACS.

Risk factors

Independent and widely substantiated risk factors for development of ACS include massive fluid resuscitation, multiple transfusions (>10 U packed RBC 24 hours), hypothermia (core temperature <33°C), base deficit/acidosis (pH <7.2), and BMI >30[6,16-18,33,34] (Table 2). High-volume fluid resuscitation sufficient to cause ACS has been defined as >3,500 mL given in 24 hours.[35]

Table 2. Independent Risk Factors for Abdominal Compartment Syndrome

Massive crystalloid fluid resuscitation (>5,000 mL in 24 h)
Multiple transfusions (>10 U packed RBC in 24 h)
Hypothermia (core temperature <33°C)
Base deficit/acidosis (pH <7.2)
Body mass index (>30)

However, other authors believe the risk of ACS does not increase substantially unless >5,000 mL are given within 24 hours.[6] Abdominal trauma and abdominal surgery also increase the risk for ACS, but the association is weaker.[33-35] The literature also cites many other risk factors, such as peritonitis, sepsis, burns, and pancreatitis, but these are risk factors for ACS only in that they all require fluid resuscitation, which is the actual underlying risk factor itself.[6]

Abdominal compartment syndrome and multiple organ failure

Once the bowel has become massively swollen and IAP is greatly increased, organ dysfunction occurs. Comparing trauma patients with similar Injury Severity Scores, the incidence of multi-organ failure in those with and without ACS is 32% to 55% vs 8 to 12%, respectively.[17,36] In addition, in the same cohort of trauma patients, the mortality rate of those with ACS is 43% to 64% vs 12% to 17% in those without ACS.[17,36]

Renal

A well-described presentation in the trauma or septic patient who has received massive fluid resuscitation is a progressively more swollen and tense abdominal wall, followed by decreasing urine output, despite ongoing fluid infusion. A bladder pressure measurement showing an IAP >20 mmHg combined with evidence of decreased renal perfusion is diagnostic of ACS. The underlying pathological process that induces renal failure from increased IAP is increased renal venous resistance and increased renal arteriolar resistance, which leads to reduced renal arterial flow and decreased glomerular filtration rate. Rabbit, canine, and rodent studies have all shown that renal blood flow is reduced when IAP is increased to >20 mmHg.[37-39] Glomerular filtration rate decreased to <25% at a pressure of 20 mmHg and dropped to 7% when the pressure was increased to 40 mmHg.[38] Renal vascular resistance increased >500%, which was 15 times higher than the systemic vascular resistance.[38] Renal dysfunction from increased IAP is due to direct renal vein compression and cortical arteriolar compression, as confirmed by these animal studies.[37-39] This was later confirmed in a human study

showing a significantly increased resistive index in the renal arteries as well.[40]

In addition, the increased renal vascular resistance leads to up-regulation of antidiuretic hormone, aldosterone, renin, and angiotensin.[41-43] This worsens the entire process because increased levels of antidiuretic hormone and aldosterone result in sodium and water retention, which can increase bowel edema, and increased levels of renin and angiotensin increase renal vascular resistance even more, and worsen renal perfusion.[41-43]

Respiratory

Increased IAP pushes up directly against the diaphragm, limits total lung capacity, and decreases functional residual capacity. It also decreases pulmonary and respiratory system static compliances and increases airway resistance, alveolar-arterial oxygen gradient, and respiratory dead space.[44] As the diaphragm is pushed up, compressive atelectasis of the lower lobes occurs and this impairs oxygenation, increases intrapulmonary shunting, and increases dead-space perfusion.[45] There is also a global increase in intrathoracic pressure, which is transmitted to the smaller airways, creating increased airway pressures.[46-48] This reduces chest wall compliance and degree of recoil after inspiration, creating hypercarbia and ventilatory failure.[46,47] Just like in the abdomen, increased intrathoracic pressure increases pulmonary vascular resistance, which worsens oxygenation.[45-48]

Some very interesting research in porcine models has shown that IAH in the face of acute lung injury or acute respiratory distress syndrome induces and worsens pulmonary edema.[49,50] With intra-abdominal pressures between 20 and 30 mmHg for several hours, the extravascular lung water content increased considerably, by up to 30%.[49,50] This is believed to be due to increased intrathoracic pressures impeding pulmonary venous return and increasing pulmonary vascular resistance.

Much of the recent research has focused on the use of increased PEEP to match and overcome the abdominal pressure. In other words, have the PEEP set to match the abdominal pressure pushing up from below to avoid lung compression. Interestingly, high levels of PEEP ($\geq$12 mmHg) will increase IAP considerably, in both patients with and without IAH.[51] A summary of this research shows that with low levels of IAH (<13 mmHg), adding PEEP to match at the same pressure will counterbalance the effects of IAH and maintain normal oxygenation and hemodynamics, and prevent atelectasis and loss of lung volume.[52] However, with rising levels in IAP and ACS, a sufficient amount of PEEP necessary to match this pressure in the 18 to 22 mmHg range results in a 26% loss of cardiac output, decreased total respiratory compliance, decreased chest wall compliance, and did not improve arterial oxygenation.[53] Attempts at matching the IAP with the PEEP is not recommended because the excessive pressure created in the chest has detrimental effects on cardiac output and increases the risk of barotrauma, without any improvement in oxygenation.[53]

Cardiac

To understand the effects of IAH and ACS on the heart, only 2 main factors need to be kept in mind: a decreased venous return and a greatly increased afterload. This is important to understand because increasing venous return (increasing the central venous pressure) and reducing afterload will restore the cardiac output in these patients.

Impairment of venous return from the abdominal cavity to the heart, and specifically from the inferior vena cava, begins to occur with IAPs $\geq$15 mmHg.[54] Once the pressure has reached $\geq$20 mmHg, there is substantial collapse of the mesenteric and renal veins, as well as the vena cava, which results in a considerable drop in venous return.[55] Ironically, because of the increased abdominal pressure pushing on the vena cava and pushing upward against the diaphragm and increasing the intrathoracic pressure as well, the central venous pressure will be falsely elevated. Research in a porcine model showed that an IAP of 30 mmHg will artificially increase the central venous pressure by a factor of 4.[56] The novice clinician might think the patient is euvolemic when in fact, because of continued volume loss into the bowel interstitium, the patient is intravascularly volume depleted. This is evidenced by the marked improvement in blood pressure and overall hemodynamics with intravascular volume expansion.[50,57-59]

The second factor that decreases cardiac output is the elevated afterload. The afterload is elevated due to increased systemic vascular resistance, mostly from the abdominal cavity, and increased intrathoracic pressure.[54-58] Increased abdominal pressure has similar physiological effects as to increased end expiratory pressure in the chest cavity. Just as increased PEEP expands the lungs, which then constantly apply direct pressure to the mediastinum, so too does the constant direct pressure against the diaphragm from the swollen viscera. The direct cardiac compression and increased intrathoracic pressure transmitted upward from the abdominal cavity limit ventricular compliance and right ventricular end diastolic volume.[51-53] In addition, there is direct compression of the arterioles within the mesentery of the bowel and other viscera that account for a substantial increase in the afterload.

A situation is created in which there is decreased venous return to the heart, direct cardiac compression, and greatly increased afterload. The cardiac output will markedly diminish. The central venous pressure will be falsely elevated, although the patient might be volume depleted and the right ventricle inadequately filled. Therefore, appropriate resuscitation is not guided by central venous pressure or urine output, but rather by the right ventricular end diastolic volume index, which has proven itself superior to the pulmonary artery occlusion pressure and other indices of resuscitation.[60-62]

Cerebral

There is also a direct, although less obvious, relationship between increased IAP and increased intracranial pressure (ICP). For some time, clinical observations in trauma patients with concomitant head and abdominal injuries have shown that decompressive laparotomy for ACS would also unintentionally improve intracranial hypertension in these patients. A substantial fall in ICP would occur as soon as the abdomen was opened in the operating room. This has been shown in both case reports and retrospective studies.[63,64] Interestingly, in a single study in trauma patients without ACS (but mean IAP of 27 mmHg, range 21 to 35 mmHg), decompressive laparotomy resulted in a rapid drop in the ICP by at least 10 mmHg in all patients. In 6 patients, the improvement was transient and all 6 died; in the remaining 11 patients with sustained improvement in ICP, all survived and recovered.[64]

Although clinically this has been shown to be true, basic science research has not yet provided a clear explanation as to why this occurs. A prospective study performed in ICU patients showed that increased IAP will increase ICP and hypothesized that this occurs due to increased intrathoracic pressures that create a functional obstruction to cerebral venous outflow.[65] Additional research has better defined the actual pathophysiological connection. Animal models in pigs and mice that artificially created pneumoperitoneum and increased the IAP to 20 mmHg, resulted in increased levels of interleukin-6 in the cerebrospinal fluid and disrupted the blood–brain barrier.[66,67] Another porcine model showed that increased IAP will narrow the inferior vena cava at the level of the diaphragm and limit the drainage from the lumbar venous plexus and central nervous system.[68] The combination of these events is likely the underlying reason that a sudden decrease in IAP, such as seen after a decompressive laparotomy, results in a decrease in ICP.

Splanchnic

All of the structures within the abdominal cavity are also compressed, and this will cause regional hypoperfusion to all of the organs in the splanchnic bed. This effect might be most pronounced in the liver. Animal studies have shown that even with IAP of only 6 to 10 mmHg, portal venous blood flow is reduced considerably, and that at 20 mmHg, the portal venous flow and hepatic arterial flow were reduced by 35% and 55%, respectively.[69,70] Decreased blood flow can also be detected in the stomach during times of IAH using measurements of intramucosal gastric pH.[34,71,72] And the intestine itself will also show signs of decreased blood flow, especially in the regions of the least collateral flow, as shown by elevated levels of D-lactate and ischemia of the cecal mucosa.[73,74]

Diagnosis

Despite IAH and ACS occurring in a variety of patients and intensive care settings, 2 surveys among burn and pediatric physicians showed that only 47% could correctly define ACS, 25% had never measured bladder pressure, and bladder pressure was only measured routinely by 31%.[75,76] Knowledge of the pathophysiology and a high index of suspicion are necessary to diagnose IAH before it progresses to ACS.

Most commonly, the patient would be intubated and on mechanical ventilation in the ICU setting. The rare occasions that IAH occurs in the awake, nonventilated patient are usually due to large intra-abdominal tumors, large space-occupying lesions, or ruptured abdominal aortic aneurysms.[11,12,77] In addition to the tense abdomen found on physical examination in these awake patients, continuous reports of difficulty breathing due to abdominal pressure and lack of diaphragmatic excursion will be a prominent symptom. These patients will also report obvious abdominal pain and feeling of fullness. They display profound orthopnea, or an inability to lay supine without tremendous difficulty breathing, and will remain upright at all times to allow gravity to help relieve the pressure from the diaphragm.

Much more often, IAH is diagnosed in the ICU setting when the patient is already critically ill, ventilated, and sedated. The patient will have received substantial amounts of crystalloid infusion for their condition. The patient would be total body fluid overloaded and edematous. Keep in mind that their underlying condition does not necessarily have to be of abdominal origin because sepsis, burns, and hemorrhagic shock can also cause IAH and ACS due to large volume resuscitation.

On physical examination, the abdomen is very tense and distended. The extremities often show considerable edema. The face and neck are also likely to be swollen, as are the penis and genitalia. Often the physician is alerted to the possibility of IAH after receiving multiple calls about the ventilator sounding because of high airway

pressures. Additionally, a trend of decreasing urine output or rising creatinine can signal the impending renal failure. Unfortunately, the sensitivity of the physical examination to detect high IAP was shown to only be around 40% to 60% in 2 separate prospective studies.[78,79] Therefore, it is not the most reliable diagnostic method.

As discussed previously, measuring bladder pressure is considered the gold standard and should be done with a maximum volume of 25 mL saline. Overdistending the bladder with excessive volumes of fluid (eg, 50 to 100 mL) will artificially increase the bladder pressure.[8] The IAP should be measured at end expiration with the patient supine, and relaxed or sedated, with the transducer zeroed at the mid-axillary line.[6] Having the patient upright at >30 degrees has been clearly shown to produce erroneous results.[9] In addition, a single measurement is not sufficient. Recommendations from an international conference of experts are that if ≥2 risk factors for IAH or ACS are present, a baseline measurement should be obtained and then serial measurements performed during the patient's critical illness.[18] Other experts have recommended that continuous IAP monitoring is the best solution because it allows a more accurate trend to be determined.[80]

How have we improved?

During the last 10 years, there have been 3 major changes in the management of the critically ill, traumatized, and burn patients that have led to a lower incidence and improvements in the overall mortality rate of ACS. They are adoption of massive transfusion protocols and 1:1 blood to plasma transfusion strategies; widespread use of damage control and open abdomen approaches to the polytraumatized abdominal cavity; and increased use of plasma and colloids in the resuscitation of burn patients.

It has been clearly proven in both the laboratory and in clinical practice that massive resuscitation with IV crystalloid solutions causes IAH and ACS.[81] The game-changing and now widely adopted use of blood and plasma in a 1:1 ratio for resuscitation, with much less reliance on crystalloid, has dramatically reduced the prevalence of ACS in trauma centers.[82-84] In addition, massive transfusion protocols have allowed resuscitation of the exsanguinating patient to continue for hours with a dramatic reduction in the incidence of anasarca and ACS.[83,84] In fact, resuscitation with a crystalloid to packed RBC ratio of >1.5:1 had a 2-fold higher risk of ACS than for those patients receiving less crystalloid compared with blood.[82]

The same results have been found in resuscitating the burn patient. Several studies have shown that transfusion of colloids or plasma will decrease the overall amount of crystalloid required for resuscitation.[85-87] One prospective study showed that transfusion of plasma to burn patients (>40% total body surface are) helped to maintain the IAP at <25 mmHg, and only 1 patient in the group resuscitated with crystalloid maintained an IAP <25 mmHg.[87]

And third, by simply not closing the abdominal cavity after laparotomy and using damage control and temporary abdominal closure methods, the incidence of ACS has been additionally reduced.[88]

Management

The presentation and clinical severity of the critically ill patient with IAH or ACS can vary. It is important to determine if and how the presence of increased IAP might or might not be affecting each patient on an individual basis. Therefore, all guidelines that exist can be modified according to the presenting clinical scenario. For example, a patient with 30% total body surface area burn and inhalation injury who is making good clinical progress on his fourth hospital day, but is found to have a distended abdomen and a bladder pressure of 18 mmHg, can be managed nonoperatively in ways that will be discussed here later. On the contrary, a patient who is recovering in the ICU just 8 hours after repair of a ruptured abdominal aortic aneurysm and has peak airway pressures of 50 mmHg, a very tight abdomen, bladder pressure of 35 mmHg, and has had minimal urine output since surgery, needs urgent decompression. Ultimately, it is important to realize that although there are recommendations for nonoperative management of these patients, the definitive management is to perform a laparotomy to release the pressure and provide a temporary abdominal closure until the disease process is reversed and the swelling abates. Hesitation to perform surgery can lead to death, and the morbidity from a laparotomy in experienced hands is minimal.

A more scientific way to determine if a patient with elevated abdominal pressure but minimal signs of ACS requires decompression is to measure the APP. As mentioned previously, the APP is the mean arterial pressure minus the IAP. Although never subjected to a prospective trial, an APP of ≥50 mmHg was shown to improve the chances of survival in a retrospective study of surgical patients.[89] Although not validated, most authorities now recommend attempting to maintain the APP at a level between 50 and 60 mmHg or higher, if possible, to ensure adequate blood flow to the viscera.[18]

Before performing a decompressive laparotomy, the nonoperative management of IAH can be divided into the following steps: sedation and paralysis to relax the abdominal wall, evacuation of intraluminal contents, evacuation of large abdominal fluid collections, optimization of APP, and correct a positive fluid balance[90] (Table 3).

Table 3. Nonoperative Management of Abdominal Compartment Syndrome

Sedation and paralysis to relax the abdominal wall
Evacuation of intraluminal contents
Evacuation of large abdominal fluid collections
Optimize abdominal perfusion pressure
Correct a positive fluid balance

Sedation/paralytics

The first step in the management of elevated IAP is to ensure adequate sedation and, if necessary, paralysis. Anxiety and restless behavior when fighting to breath against mechanical ventilation will increase abdominal wall muscular activity and increase IAP.[33,35] Complete paralysis will relax the abdominal wall musculature and allow additional expansion of the abdominal domain to decrease the overall IAP. A single prospective study that examined the effects of neuromuscular blockade on IAP found that bolus administration of cisatracurium significantly decreased IAP from a mean of 18 mmHg to 14 mmHg ($p = 0.01$) within 15 minutes; however, no change in urinary output or APP occurred.[91] Intra-abdominal pressure returned to the baseline level within 2 hours in all patients.

Evacuation of intraluminal contents

Many critically ill patients will at some point have a gastrointestinal ileus. Gastric, small bowel, and colonic distension can all increase IAP substantially, and simple endoluminal decompression is a rapid and effective way to decrease an elevated IAP. Therefore, all patients with elevated IAP should have a nasogastric tube placed. Patients with colonic ileus or acute colonic pseudo-obstruction (eg, Ogilvie syndrome) should undergo either colonoscopic decompression or be given neostigmine.[92,93]

Evacuation of large abdominal fluid collections

Patients with ascites (eg, burns, liver failure, or malignancy) or those recovering from abdominal trauma can have large collections of fluid or blood within the abdominal cavity contributing to elevated IAP. In these cases, it is often the presence of the fluid rather than swollen bowel from resuscitation that causes IAH. It would make sense that simply draining the fluid would alleviate the problem. Percutaneous drainage of the fluid in these situations has shown great success in burn and oncology patients, avoiding the need for a decompressive laparotomy.[94-96] However, immediate success must be tempered by the fact that recurrent ACS is always possible as the fluid re-accumulates, and so continued surveillance is necessary. One study using percutaneous drainage to treat elevated IAP in burn patients found that 44% of the patients eventually required a decompressive laparotomy.[94] Unfortunately, in the trauma patient with liver or spleen injuries, the blood will quickly loculate and aggregate into pockets, which can make percutaneous drainage difficult. In addition, ACS that develops in the trauma patient is much more commonly due to swollen bowel from massive fluid resuscitation rather than free fluid. One multi-institutional study of traumatic liver injuries found percutaneous drainage to be effective in a small minority of the total patients.[97]

Optimize abdominal perfusion pressure

Because IAP from swollen viscera can be relatively constant, to increase APP the mean blood pressure must be elevated. This can be done with fluid infusion. Because the central venous pressure will likely be artificially elevated, the right ventricular end diastolic volume index (RVEDVI) should be used to guide resuscitation to first ensure that the patient is euvolemic and not under-resuscitated.[60-62] Random boluses or fluid challenges should be discouraged because a transient increase in blood pressure will do nothing to ameliorate the underlying problem, and excessive fluid infusion will worsen the edema of the bowel. Multiple reports have documented that excessive crystalloid resuscitation worsens IAH and ACS and can cause secondary ACS.[16,73,98,99] Urine output will also be an unreliable marker of resuscitation because the venous and arteriolar compression within the renal cortex will limit urine production.[100,101]

The more goal-directed measures are necessary to guide resuscitation without committing over-resuscitation and worsening the bowel edema. The target for the RVEDI is debatable, but fluid infusion that allows continued increase up to the plateau point is the goal. In other words, continue fluid infusion until the right ventricle is full. Good research in this area has shown that although the optimal right ventricular volume for each individual person will vary and be dependent on ejection fraction and contractility, a volume <90 mL/m^2 is associated with a high response rate to fluid administration.[102] Most patients will continue to be responsive to fluid infusion up to 130 mL/m^2.[103] Other helpful measures that support the RVEDVI are the base deficit and lactate levels, which remain good end points to guide resuscitation.

With this in mind, fluid is then given to optimize the RVEDVI and base deficit and lactate are normalized. Ideally, the mean blood pressure would also rise and improve the APP. Once the RVEDVI is optimized, additional fluid infusion will not be helpful and will only worsen the bowel edema. At this point, it would be good to note that one prospective, randomized study did show that colloid-based resuscitative strategies were

associated with a decreased incidence of ACS in burn patients, mostly due to decreasing the total volume of fluid required.[86] It is a good strategy to switch to colloid infusion for ongoing fluid requirements after the RVEDVI is optimized. Another option is to use hypertonic crystalloid resuscitation, which, in one study, was associated with a substantially decreased fluid requirement and higher APP.[104]

Correct a positive fluid balance

In direct contradistinction to the previous discussion, the clinician will sometimes find him- or herself faced with a patient who has already been grossly over-resuscitated and not only has ACS, but also congestive heart failure and total body fluid overload. In these cases, it has usually been overly aggressive resuscitation in the operating room for exsanguinating extra-abdominal trauma that leads to a patient succumbing to iatrogenic ACS several hours later. In fact, several studies have shown that the underlying events that lead to ACS might more likely be the physician-ordered fluid resuscitation rather than the traumatic event itself.[98,99,105] In these cases, the extra fluid needs to be removed without creating a situation of hypotension, hypoperfusion, or acidosis. Although the idea of a gentle diuresis is appealing, the reality of removing the already third-spaced fluid from the interstitium without causing intravascular volume depletion and hypotension is almost impossible in the short term, especially if the patient shows any signs of hemodynamic instability. This difficulty is compounded by the ACS-induced renal compression and oliguria. Although diuretics can be attempted, success is unlikely. Therefore, the early institution of renal replacement therapy with fluid removal by continuous hemofiltration or ultrafiltration is the most appropriate therapy in these cases.[106,107]

Decompressive laparotomy

Although there is a role for the nonoperative strategies just mentioned, mostly in the stable or minimally symptomatic patient, the definitive therapy to treat ACS is a decompressive laparotomy. This is unarguably the fastest and most effective way to reduce elevated IAP. One study that analyzed 250 patients with ACS found that the mean IAP changed from 35 mmHg to 16 mmHg ($p < 0.001$) after surgical decompression.[108] Delays in performing laparotomy are associated with excessive morbidity and mortality rates up to 88%.[92,108] The symptomatic patient with signs of organ distress or failure should not be treated with nonoperative techniques.

The goals of a decompressive laparotomy are 5-fold: decrease the elevated IAP to stop organ dysfunction; allow room for continued expansion of the abdominal viscera during ongoing resuscitation; provide temporary abdominal coverage/closure as the disease process resolves; prevent excessive fascial retraction, which makes subsequent definitive abdominal closure much more difficult; and allow a means for continued evacuation of fluid from the abdominal cavity (Table 4). No technique currently in use is ideal to accomplish all of these goals, but several different techniques have been developed. It is beyond the scope of this article to describe all of the current techniques for temporary abdominal closure, but I will describe the important steps briefly.

Table 4. Critical Goals and Steps in Performing a Decompressive Laparotomy

Goals
Decrease the elevated intra-abdominal pressure to stop the organ dysfunction
Allow room for continued expansion of the viscera during ongoing resuscitation
Provide temporary abdominal closure
Prevent excessive fascial retraction
Allow a means for continued evacuation of fluid from the abdominal cavity
Steps
Perform a complete fasciotomy of the linea alba to allow evisceration
Separate the underlying viscera from the abdominal wall with an impermeable drape/barrier
Suture a prosthetic mesh, vacuum sponge, or silicone elastomer to the fascia to prevent fascial retraction
Place drains or a vacuum sponge to allow ongoing removal of fluid from the abdominal cavity

Regardless of the exact technique used, the main points are to perform a complete fasciotomy of the linea alba to allow evisceration, separate the underlying viscera from the abdominal wall with an impermeable drape/barrier or bowel bag, prevent excessive fascial retraction by suturing a prosthetic material to the fascia (or using a vacuum sponge or elastic/silicone elastomer bands), and allow ongoing evacuation of fluid from the abdominal cavity using drains or a vacuum sponge. The first point, to perform a complete fasciotomy, is very important because the degree to which the IAP decreases is a function of the degree to which the fascia is released. Interestingly, in most patients after a decompressive laparotomy the IAP is reduced but not normalized, and can remain high for some time after surgery.[108] In addition, the recuperation of organ dysfunction is not immediate and persistent organ failure can occur despite decompression, which ultimately leads to death.[108,109] The second step is also crucial because the bowels will adhere and stick to the cut fascial edge within 72 hours. After this time, sharply dissecting the bowel from the fascial edge is technically very difficult and places the patient at extremely high risk for enterocutaneous fistula if an

inadvertent enterotomy occurs. Keeping the bowels off of the fascia is critical to allow a delayed abdominal wall closure. For this same reason, some type of preventative measure to keep the fascia from retracting too far laterally is necessary. Suturing mesh to the cut edge, using a vacuum sponge, silicone elastomer sheeting, or silicone elastomer bands, all are effective.[110] Finally, because the edematous bowel will continue to "weep" and the ongoing production of peritoneal fluid will continue to collect within the abdominal cavity, a means of evacuation with drains or vacuum sponges is necessary, otherwise, the fluid will become trapped under the impermeable bowel drape and recurrent ACS can occur.

CONCLUSIONS

Abdominal compartment syndrome is a life-threatening but treatable disease that requires good clinical skills to diagnose and manage effectively. A high index of suspicion is necessary and measurements of bladder pressure should be performed in all high-risk patients so that effective intervention can be performed rapidly. Although nonoperative interventions have a role in the stable and minimally symptomatic patient, these techniques should not be used as a surrogate for or delay a decompressive laparotomy, which is the treatment of choice. During the last 10 years, there have been substantial improvements in our understanding and ability to treat ACS. The adoption of massive transfusion protocols and 1:1 blood to plasma transfusion strategies in trauma, as well as increased use of plasma and colloids for resuscitation in burn patients, has reduced the prevalence of this disease. In addition, the widespread use of damage control and open abdomen techniques has resulted in a dramatic reduction in the mortality rate for these critically ill patients. May this progress continue.

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