Intra-abdominal Hypertension and Abdominal Compartment Syndrome

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Interest in intra-abdominal hypertension and abdominal compartment syndrome has intensified in recent years as the recognition of this condition has increased, thereby providing a potential therapeutic target to improve outcomes for critically ill patients [17]. According to a recent systematic review, the incidence of abdominal compartment syndrome varies between 0.5% and 36.4% of trauma patients, mortality is nearly universal if untreated, and for treated patients, overall survival is improved across all study cohorts, though survival rates range widely, from 25% to 75% [23].

Definition and Grading

Increased intra-abdominal pressure can result from multiple causes, such as volume occupying lesions, hemorrhage, ascites, overwhelming infection, and surgical packing. Definitions and a grading system have been developed by the World Society of the Abdominal Compartment Syndrome in an attempt to standardize the approach to intra-abdominal hypertension both clinically and in research. A normal intra-abdominal pressure is 2–7 mm Hg [19]. Intra-abdominal hypertension is the condition in which the patient has an elevated intra-abdominal compartment pressure, typically defined at a level of 12–15 mm Hg [19]. If untreated, the condition may progress to abdominal compartment syndrome, which occurs at an intra-abdominal pressure of greater than 20 mm Hg and is associated with end-organ dysfunction [19]. The grades of intra-abdominal hypertension and the associated intra-abdominal pressures are as follows:

Grade I: 12–15 mm Hg
Grade II: 16–20 mm Hg
Grade III: 21–25 mm Hg
Grade IV: >25 mm Hg

An additional parameter that has been suggested as an endpoint of resuscitation is the abdominal perfusion pressure (APP). Abdominal perfusion pressure is calculated by subtracting the intra-abdominal pressure (IAP) from the mean arterial pressure (MAP): APP = MAP – IAP. An APP equal to or greater than 50 mm Hg has been demonstrated to be a good indicator of survival in patients with intra-abdominal hypertension or abdominal compartment syndrome [6].

Several risk factors for the development of abdominal compartment syndrome have been identified, including high-volume resuscitation, hypothermia, acidosis, ileus, and multisystem organ dysfunction [12, 13].

Measurement of Intra-abdominal Pressure

Intra-abdominal compartment pressure is typically measured in the intensive care setting, but because it is fairly straightforward to do, it can easily be measured anywhere in the inpatient setting. The technique involves measuring the pressure within the bladder in a supine patient at end expiration with a relaxed abdominal wall [21]. Fifty milliliters of saline are introduced into the bladder via the urinary drainage catheter, which is then clamped. The patient is placed in a level supine position, and the resulting pressure within the bladder is transduced at a height level to the patient's bladder [25]. Elevation of the head can result in artificially elevated values and thus should be avoided. Intra-vesicular pressure can then be transduced using a number of techniques. These include the use of an interposition T-piece, direct cannulation of the urinary catheter using a transducer-based needle, or the insertion of a continuous transduction method using a three-way Foley catheter. Perhaps the simplest technique was

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described by Harrahill [11]. In this technique, after the 50 milliliters of saline is infused in the bladder, the column of urine generated in the Foley catheter that is raised straight up is measured in centimeters. This measurement correlates with intra-abdominal pressure. It should be remembered that this measurement is in cm H_2O, and the units defined in the grading system are in mm Hg. To convert from cm H_2O to mm Hg, multiply the value in cm by 0.74 [11]. A few additional key conversions to remember are that 16 cm H_2O is equal to 12 mm Hg and 27 cm H_2O is equal to 20 mm Hg.

Screening

Intra-abdominal hypertension and/or abdominal compartment syndrome is difficult to identify through physical examination alone. Recommendations from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome have identified patients who should be screened and/or surveilled for IAH or ACS.

These criteria include the following:

1. New intensive care unit admission
2. Evidence of clinical deterioration
3. Any two risk factors (listed in Table 23.1)

In patients who meet these criteria, bladder pressures should be monitored. However, the optimal frequency of pressure measurements is still unknown. Our usual practice is to check serial intra-abdominal pressures using the bladder pressure measurement technique every 4 h as long as the patient has one or more risk factors for the development of abdominal compartment syndrome.

Pathophysiology

Several mechanisms can lead to intra-abdominal hypertension and abdominal compartment syndrome. Primary intra-abdominal hypertension may develop as a result of retroperitoneal hemorrhage and infection. Secondary intra-abdominal hypertension, which is more common, refers to intra-abdominal hypertension developing in the setting of extra-abdominal or extrapelvic pathology. Secondary intra-abdominal hypertension develops when a large volume of resuscitation fluid (either crystalloid and/or blood products) is required to treat septic shock or hemorrhagic shock [24]. No matter the cause of shock, the pathophysiologic response is similar. Shock leads to the development of capillary leak. This capillary leak, combined with volume resuscitation, leads to extravasation of fluid from the intravascular space into the interstitial space. The release of inflammatory mediators leads to worsening of the condition, which develops into "intestinal distress syndrome" [20]. The increase in volume in the interstitial compartment leads to generalized edema throughout the body. Although commonly seen in the subcutaneous tissue, this process also occurs with the intra-abdominal viscera and the contents of the retroperitoneal space. This then leads to increased intra-abdominal pressure as the abdominal contents take on more fluid and increase volume, although the fascia of the abdominal wall does not increase in compliance. Under the constraints of a relatively non-compliant abdominal wall fascia, the volume of the intra-abdominal contents reaches a maximum, after which the intra-abdominal pressure begins to increase.

Tertiary abdominal compartment syndrome occurs when the syndrome develops after initial treatment for abdominal compartment syndrome. In other words, it develops after the abdominal cavity has already been decompressed. This is occasionally referred to as chronic abdominal compartment syndrome. Repeated massive fluid resuscitation may result in an increased inflammatory response, bowel edema, and ascites. This can result in an abdominal compartment syndrome requiring further opening of the abdominal cavity and placement of a possibly less restrictive abdominal dressing [2].

Quaternary abdominal compartment syndrome occurs when the syndrome develops in the setting of hernia repair or abdominal wall closure. The greater use of abdominal wall reconstruction techniques, rather than "hole patch" incisional hernia repairs, understandably leads to an increased volume of viscera contained within the abdominal cavity. This has resulted in the recognition of a quaternary abdominal compartment syndrome which develops following the abdominal wall reconstruction [18].

Table 23.1 Risk factors for intra-abdominal hypertension and/or abdominal compartment syndrome

Acute respiratory failure with increased intrathoracic pressures	Major trauma and/or burns
Gastroparesis	Ileus
Colonic pseudo-obstruction (Ogilvie's syndrome)	Ascites
Hemoperitoneum	Acidosis
Hypotension	Hypothermia
Massive transfusion [12]	Large volume resuscitation
Oliguria	Sepsis

Effects on Organ Systems

Cardiac

Intra-abdominal hypertension results in decreased cardiac output and decreased overall cardiac function. This is mani-

fested clinically as hypotension, one of the hallmarks of abdominal compartment syndrome. Cardiac function is dependent upon preload, contractility, and afterload, and intra-abdominal hypertension affects all of these components of cardiac function in different ways [5].

Preload: Intra-abdominal hypertension leads to cephalad displacement of the diaphragm into the thoracic cavity. This increase in intra-abdominal pressure is transmitted to the thoracic cavity and pleural spaces. Besides the direct compression of the inferior vena cava (IVC) by the increased intra-abdominal pressure, the transmitted increase in intrathoracic pressure also reduces IVC blood flow. With marked flow reduction through the IVC, venous return is reduced and subsequently, so is preload. Additionally, measurements of preload via vascular catheters become inaccurate.

Contractility: Increased intrathoracic pressure and displacement of the diaphragm affect cardiac contractility. Direct cardiac compression from the increase in intrathoracic pressure decreases cardiac contractility. Additionally, the increase in intrathoracic pressure leads to in an increase in pulmonary vascular resistance that detrimentally affects right and left ventricular function. Together, both of these mechanisms decrease cardiac contractility.

Afterload: As cardiac output decreases due to decreased preload, peripheral vascular resistance will increase in compensation in order to maintain mean arterial pressure. Additionally, the relative under perfusion of the kidney leads to derangements in the renin angiotensin system, which also increases peripheral vascular resistance. The increase in peripheral vascular resistance increases afterload and thereby diminishes cardiac output.

Pulmonary

As the diaphragm becomes displaced into the thoracic cavity, several changes occur that affect the pulmonary system. The decreased compliance of the abdominal wall increases intra-abdominal pressure, which is distributed throughout the abdominal cavity, including against the diaphragm. This direct pressure on the diaphragm not only decreases the size of the thoracic cavity but also inhibits the excursion ability of the diaphragm, impacting pulmonary function [22].

Ventilation: As the diaphragm is pushed into the thoracic cavity, the size of the thoracic cavity decreases, and chest wall compliance decreases. The changes in chest wall compliance are often compounded by the effects of interstitial edema. Tidal volume decreases, impacting ventilation, which leads to a subsequent rise in airway pressures [15]. Clinically, one hallmark of abdominal compartment syndrome is elevated peak airway pressures.

Oxygenation: A decrease in size of the thoracic cavity due to increased pressure against the diaphragm also leads to a reduction in functional residual capacity. Atelectasis develops in response to increased intrathoracic pressure and can lead to pulmonary vascular shunting and ventilation perfusion mismatch. All of these mechanisms lead to the inability to oxygenate. Another hallmark of abdominal compartment syndrome is decreasing oxygen saturation.

Renal

Intra-abdominal hypertension has direct effects on renal vasculature, specifically compression of the renal veins and renal arterioles. These compressive effects lead to increased vascular resistance in the kidney, thereby compromising renal blood flow. This is especially true of the renal veins where the intravascular pressure is much less than in the renal arteries. The resultant decrease in relative perfusion can lead to renal failure from a prerenal cause [26]. If acute tubular necrosis develops, a patient could potentially develop anuric renal failure, which is linked to worse outcomes in critically ill patients. Relative hypoperfusion of the nephron leads to upregulation of the renin angiotensin system and aldosterone. The activation of this hormonal axis leads to further increases in vascular vasoconstriction together with systemic effects of increased afterload. Increased levels of angiotensin II will lead to increased renal vascular resistance and contribute to a decreased glomerular filtration rate, manifested as impaired renal function, further worsening renal circulation.

Gastrointestinal

It has been postulated that elevated intra-abdominal pressure could affect perfusion of organs [14]. Specifically, an IAP of 20 mm Hg leads to a reduction of flow of up to 25% in the portal vein. Reduction of blood flow may also affect capillary circulation to the mucosa of the gastrointestinal tract. Subsequent reperfusion injury may increase systemic inflammation and worsen intra-abdominal hypertension [14].

Central Nervous System

A relationship between intra-abdominal pressure and intracranial pressure has been described [4]. Specifically, intracranial pressure drops by 10 mm Hg after decompressive laparotomy in patients with abdominal compartment syndrome. This may be a result of relieving direct impedance to cerebral venous drainage or by modifying inflammatory mediators which contribute to intracranial pressure. These findings have opened up the possibility for decompressive laparotomy as an adjunct to treatment of refractory elevated

intracranial pressure. The specific indications and role for this procedure in patients with elevated intracranial pressure are controversial and have not been clearly determined currently [16].

Management of Intra-abdominal Hypertension and the Abdominal Compartment Syndrome

There is a continuum of therapy for intra-abdominal hypertension and abdominal compartment syndrome, which culminates in decompressive laparotomy. The first steps involve decreasing the volume of the abdominal compartment (Table 23.2). This can be accomplished in several ways. The use of a nasogastric tube to decompress the stomach can decrease intra-abdominal pressure. If there are space-occupying lesions such as ascites, cysts, or abscesses, these can often be addressed with percutaneous drainage to decrease the intra-abdominal volume they occupy. If these measures do not accomplish a decrease in intra-abdominal pressure and/or result in an abdominal perfusion pressure of more than 50 mm Hg, the compliance of the abdominal wall can be increased pharmacologically to reduce intra-abdominal pressure. This approach is typically used in the intubated and mechanically ventilated patient and can be accomplished via neuromuscular blockade or by increasing sedation. These measures should be considered carefully because in critically ill patients, neuromuscular blockade can have adverse effects, including secondary infections and critical illness neuromyopathy, resulting in increased costs and length of stay [1] (Table 23.3).

Table 23.2 Nonoperative management of intra-abdominal hypertension and abdominal compartment syndrome

1. Evacuate intraluminal contents
2. Evacuate intra-abdominal space-occupying lesions
3. Optimize abdominal wall compliance
4. Optimize fluid administration
5. Optimize systemic and& regional perfusion

Table 23.3 Goals of managing the open abdomen

Cover and protect abdominal contents
Prevent evisceration
Prevent or treat ACS
Protect the fascia
Minimize loss of domain
Facilitate reoperation
Keep the patient warm and dry
Prevent hypothermia
Prevent adhesion formation
Remove infectious material

Decompressive Laparotomy

If medical therapy does not result in physiologic normalization in patients with suspected abdominal compartment syndrome, surgery is the sole remaining treatment. As such, the extreme end of the treatment spectrum is decompression of the abdominal cavity via laparotomy. Decompressive laparotomy entails opening the abdominal wall from the xiphoid to the pubic symphysis. This allows maximal decompression and leaves the patient with an open abdomen [3]. Particularly important is to ensure that adequate volume resuscitation has occurred before the abdominal cavity is opened. Endpoints of resuscitation, including central venous pressure and urinary output, may be artificially suppressed in a patient with intra-abdominal hypertension and abdominal compartment syndrome [14].

Decompressive laparotomy is effective at decreasing intra-abdominal pressure, but intra-abdominal hypertension persists in most patients [8, 9]. Mortality remains high—between 37% and 49% [8, 9]. Survival is highest when organ dysfunction is reversed, which suggests that timing of decompressive laparotomy may be important, thus reinforcing the importance of monitoring intra-abdominal pressures in critically ill patients. However, the effect of decompressive laparotomy on organ function is not uniform, and some studies found that decompressive laparotomy did not affect organ function [9]. Improvement in oxygenation and urinary output was the most pronounced effects of decompressive laparotomy [8].

Published case reports describe the use of decompressive laparotomy as a salvage maneuver in the intensive care unit [10]. However, in this scenario, which involves a patient who is severely critically ill, rapidly decompensating, and unable to tolerate transport to the operating room, anesthesia and perioperative care are understandably challenging. Decompressive laparotomy under these circumstances can be extremely challenging and fraught with complications.

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