Abdominal Compartment Syndrome

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The term abdominal compartment syndrome (ACS) describes the clinical manifestations of the pathologic elevation of the intra-abdominal pressure (IAP). When the IAP exceeds 12 mm Hg it is referred to as intra-abdominal hypertension (IAH) while ACS generally sets in at an IAP in excess of 20 mm Hg. This syndrome is most commonly observed in the setting of severe abdominal trauma and in the aftermath of major abdominal operations. ACS affects mainly the respiratory, cardiovascular, renal, gastrointestinal and the central nervous systems. Fundamental to the development of ACS are the obstruction of venous return to the heart via the inferior vena cava and the splitting of the diaphragm due to elevated IAP. Preventing ACS by the identification of patients at risk and early diagnosis is paramount to its successful management. To this end a high index of suspicion is sine qua non. The management of established ACS requires clinical astuteness and decisiveness with a readily available and generous team support. The purpose of this review is to enhance awareness among clinicians about a subtle condition with a devastating impact on morbidity and mortality if undiagnosed.

Historical Milestones

Credit for the first recorded account of an abnormal increase in intra-abdominal pressure (IAP) goes to the French physician Etienne-Jules Marey in 1863. He published a paper titled "Physiologie Medicale de la Circulation du Sang" and noted that the effects that respiration produces on the thorax are inversely related to those in the abdomen. Wendlt, in 1873, measured the IAP through the rectum and noted that the higher the IAP the less the secretion of urine. That was the first description of organ dysfunction directly attributable to elevated IAH. In rapid succession others championed our understanding of ACS. Oderbrecht in 1875 cannulated the urinary bladder and reported that the IAP is normally positive. In 1890 Heinricius reported that IAP between 27 to 46 cmH$_2$O (19.8 to 33.8 mm Hg) was uniformly fatal to experimental animals and he attributed his observations to obstruction of ventilation, decreased end-diastolic volume and a low blood pressure.

In the United States, in a luminous paper in 1911, Haven Emerson reported convincing experimental observations. He noted that the contraction of the diaphragm is the main contributor of IAP during inspiration; that hypnosis and muscle paralysis induced by anesthesia with the concomitant loss of muscle tone results in a decrease in IAP; that elevated IAP leads to an increase in peripheral vascular resistance; and that high IAP results in the death of the animal from cardiac...
Abdominal Compartment Syndrome (ACS) is a condition in which the abdominal compartment pressure (ICP) rises to a level that compromises the functioning of vital organs, particularly the lungs and kidneys. This condition can develop acutely or subacutely due to various underlying conditions, such as trauma, burns, sepsis, or surgery. The rise in ICP can lead to decreased perfusion to the organs, which can result in organ dysfunction and failure. The presence of ACS can be identified by monitoring the IAP, which should be maintained below 12 mmHg to ensure adequate organ perfusion. The diagnosis of ACS is confirmed by measuring the IAP and assessing the clinical signs and symptoms associated with the condition. The management of ACS involves early recognition, monitoring, and intervention to control the IAP and prevent organ damage. The literature suggests that ACS is associated with a high mortality rate, and the recognition and management of this condition are crucial in improving patient outcomes.

### Etiology and Classification

The World Society for the Abdominal Compartment Syndrome (WSACS) has classified ACS into three categories: primary, secondary, and tertiary (recurrent) types. Primary ACS is defined as a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or radiological intervention. Secondary ACS is defined as due to conditions that do not originate from the abdominopelvic region. Tertiary (recurrent) ACS refers to ACS developing following resuscitation. Examples of etiologic factors include blunt or penetrating abdominal trauma, burns, severe acute pancreatitis (SAP), major thermal burns, and other conditions requiring massive fluid resuscitation. In Australia, patients who undergo an initial trial of non-operative management for solid organ injuries and who subsequently develop ACS are included in the primary category. Secondary ACS is defined as due to “conditions that do not originate from the abdominopelvic region.” Examples of causes of secondary ACS include sepsis and capillary leak. Patients who undergo an initial trial of non-operative management for solid organ injuries and who subsequently develop ACS are included in the primary category. Secondary ACS is defined as due to “conditions that do not originate from the abdominopelvic region.” Examples of causes of secondary ACS include sepsis and capillary leak. Patients who undergo an initial trial of non-operative management for solid organ injuries and who subsequently develop ACS are included in the primary category. Secondary ACS is defined as due to “conditions that do not originate from the abdominopelvic region.” Examples of causes of secondary ACS include sepsis and capillary leak.

### Pathophysiology

**Gastrointestinal System**

Fundamental to the understanding of the pathophysiology of ACS is the understanding of the concept of abdominal perfusion pressure (APP) in much the same way as cerebral perfusion pressure (CPP) is in relation to raised intracranial pressure. APP is the difference between the mean arterial blood pressure and the IAP. A decrease in APP can lead to decreased perfusion to the gut, which can result in ischemia and necrosis of the intestinal mucosa. This can lead to bacterial translocation and sepsis, which are major contributors to the high mortality rate associated with ACS. The management of ACS involves the control of IAP and the restoration of APP to maintain adequate organ perfusion. This can be achieved through the use of abdominal dressings, the application of the Military Antishock Trousers (MAST), and the use of intra-abdominal drains. The use of these interventions can help to control the IAP and improve organ perfusion, which can lead to improved patient outcomes.
between the mean arterial pressure (MAP) and intra-abdominal pressure (IAP) which is represented mathematically as:

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

An APP of 50 mm Hg or higher is the optimum resuscitation goal in all critically ill patients.\(^5^1\)

Splanchnic ischemia (the "first hit") caused by hemorrhage is the initiating event\(^5^2,5^3\) leading to neutrophil priming, which promotes local and remote organ injury by several immune-mediated mechanisms\(^5^4,5^5\) that include free-radicals like superoxide, enzymes for membrane degradation like elastase, increased CD11b/CD18 expression for endothelial adhesion, elaboration of the chemo-attractant IL-8 to recruit more neutrophils and delayed apoptosis to sustain cytotoxicity.\(^5^6\)

Decompressive laparotomy (DL) as the definitive treatment of ACS brings with it fresh problems (the second hit) to this pathophysiological conundrum. ACS relieved by DL allows a bolus of inflammatory agents to enter the systemic circulation and constitutes an ischemia-reperfusion injury.\(^5^7\) Mesenteric lymphatic circulation has been implicated as the conduit for the systemic distribution of proinflammatory cytokines, metabolites of arachidonic acid, complement\(^5^8\) and bacterial translocation.\(^5^9\) This has been proposed as the mechanism for remote organ injury, capillary leak and endotoxemia in ACS (Figure 1).\(^6^0\,6^1\,6^3\)

**Cardiovascular System**

IAH affects the cardiovascular system by disturbing preload, myocardial contractility and afterload.\(^6^4\) Low preload may be due to absolute loss of intravascular volume or relative loss through the compression of the inferior vena cava (IVC) as it traverses the diaphragmatic hiatus. Pooling of blood in the pelvis and the lower extremities constitutes a loss and may be a factor in the pathogenesis of deep venous thrombosis.\(^6^5\)

The increased intra-thoracic pressure increases pulmonary vascular resistance and decreases right ventricular preload compromising the ability of the thin-walled right ventricle (RV) to effect adequate right ventricular ejection fraction (RVEF) and a consequent decrease in left ventricular end-diastolic volume and diminished left ventricular ejection fraction (LVEF). The resultant low cardiac output (CO) triggers a compensatory tachycardia and a consequent shortened cardiac cycle (at the expense of diastolic time) leading to impaired coronary blood flow. Thus, at the time the heart has to work the hardest, its own blood supply is impaired (Figure 1). The compensatory peripheral vascular resistance due to the low cardiac output together with the compressive effect of IAH on the aorta and its branches contribute towards the low APP.

**Respiratory System**

Raised IAP is transmitted across the diaphragm producing elevated intra-thoracic pressure, compressive atelectasis, increased alveolar dead space, reduced functional residual capacity, ventilation-perfusion mismatch and hypoxic bronchial artery vasoconstriction. By the same token, the splitting of the diaphragm leads to the need to use the accessory muscles of respiration in the presence of diminished chest wall compliance and so increases the work of breathing. In patients under mechanical ventilation, the decreased dynamic lung and
chest wall compliances result in elevated peak inspiratory and plateau pressures in attempts by the ventilator to deliver preset tidal volumes (Figure 2).66

Central Nervous System
The central venous pressure (CVP) in shock induced by ACS is often elevated due to the high intra-pleural pressure producing a functional obstruction of the jugular veins, resulting in a raised intracranial pressure (ICP). Therefore, the cerebral perfusion pressure is decreased in obedience to the Monroe-Kellie doctrine (Figure 2). The resultant cerebral hypoxia results in encephalopathy and affects vital regulatory centers in the brain, thereby disturbing homeostatic mechanisms.

Joseph et al68 studied the effects of raising the IAP using pneumoperitoneum on ICP and CPP. They demonstrated that IAH increases ICP and concluded that laparoscopy would be harmful in patients with head injury. Subsequently, reports of the detrimental effects of pneumoperitoneum on ICP69 and the beneficial effects of DL on ICP70 appeared confirming the earlier assertions. It has, therefore, been proposed that the monitoring of IAP in patients with head trauma and abdominal trauma be routine.71,72

Renal System
The effect of IAH on the renal system is multi-factorial and includes decreases in CO, renal perfusion pressure (RPP), glomerular filtration gradient (GFG), microcirculatory flow, direct compression of the renal cortex, and increased renal venous pressure (RVP).73 Of these, it appears that increased RVP plays the most significant role in the pathogenesis of IAH-induced renal failure (Figure 1).74 The reduction of RPP and the reduction in the GFG (the net force acting across the glomerulus) as well as the effects of the hormones vasopressin, renin-angiotensin and aldosterone probably work in concert to produce renal failure in IAH.

The Abdominal Wall
IAH reduces the abdominal wall blood flow by the direct compressive effect. Blood flow to the rectus abdominis muscle is reduced by 60% at an IAP above 10 mm Hg. As collagen deposit and resistance to infection are directly proportional to tissue perfusion and oxygenation, elevated IAP adversely affects wound healing and leads to wound dehiscence.75

Diagnosis
ACS has been described as the presence of a tensely distended abdomen, elevated intra-abdominal and peaked airway pressure, inadequate ventilation with hypoxia and hypercarbia, impaired renal function, and a documented improvement of these features after DL.76 While the virtues of clinical assessment cannot be debated, the detection of IAH by palpation of the abdomen has proved to be insensitive in differentiating IAPs of 10 from 20 mm Hg77 and serial measurement of the abdominal circumference did not show any correlation with the IAP.78 Therefore, the identification of patients at risk of developing IAH carries with it the responsibility for a deliberate and diligent monitoring of IAP, especially in patients that are comatose or intubated.

The abdominal CT scan has a role in identifying ACS in selected patients by the following subtle findings:
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- Round-belly sign: Abdominal distention with an increased ratio of anteroposterior-to-transverse abdominal diameter (ratio >0.80; P <0.001);
- Collapse of the vena cava;
- Bowel wall thickening with enhancement;
- Bilateral inguinal herniation.79

In the 144 years since the publication of the findings of Etienne-Jules Marey,4 the techniques of IAP have gone through several modifications. A detailed description of each technique is beyond the scope of this article but those interested can find them in the WSACS textbook.80 The first method described to find clinical applicability was by Kron et al.12,81 This method entails instillation of 60 mL of saline through the bladder using an indwelling Foley’s catheter. The outlet is then clamped and through a wide-bore needle connected to a manometer or pressure transducer and the pressure is measured. This method has several drawbacks, which include the disruption of the otherwise closed drainage system of the bladder, thereby not safeguarding sterility. The other drawbacks are that the use of needles predisposed healthcare workers to needle-stick injuries and was time-consuming.

Iberti et al15,82 introduced a modification that had some of the shortcomings of the previous method with the advantage that it is simpler, less time-consuming and needed less manipulation. About a decade later, Cheatham and Safcsak83 introduced a modification that was revised by Malbrain84 which serves as the current standard for the intra-vesical technique. Using a three-way Foley’s catheter, investigators in Australia85 have shown that a continuous measurement of IAP is possible and of practical advantage in critically ill patients.

Aside from the intra-vesical route, the same principle has been applied in measuring the IAP from catheters inserted into the stomach. A novel method using a fully automated system, the Air-Pouch System (or Spiegelberg System), measures IAP and gastric pH via a nasogastric tube.86 This system has been tested in an experimental setup with an excellent correlation (r²=0.99) with direct insufflator pressure.87 Clinical validation in ICU patients and during laparoscopic surgery has shown excellent correlation (r²=0.96) with the standard intra-vesical method.88 There are on-going efforts to improve on the accuracy and reliability of the current techniques of IAP measurement using piezo-resistive techniques that are showing promising results.89

Other routes have been described for the measurement of IAP using the foregoing principle, but none of these have found widespread clinical use. These are the rectum,90 the uterus91 and the inferior vena cava (IVC).92

The problem with the rectal route is that residual faecal matter may block the catheter tip and give an erroneous reading and nursing staff have an aversion for this route. The uterine method has a distinct disadvantage in that it can only be used in females and may be resisted by virgins. The other disadvantage is that it carries the risk of introducing infection and cannot be used in patients with uterine bleeding. The IVC route employs the placement of a central venous catheter through either the SVC or through the femoral veins and is guided into place within the abdominal compartment. The disadvantage of the IVC route is the risk of catheter-related infections and the technical difficulty in the initial insertion.

Management

A subset of ACS can be prevented and to this end, there is no substitute for clinical astuteness, sound judgment and good operative technique. Early goal-directed therapy using the APP in critically ill or traumatized patients during the resuscitation phase may avert the dangers of over-enthusiastic fluid resuscitation. Confining DCS to the shortest time possible to give opportunity to correct acidosis, hyperthermia and coagulopathy—the so-called deadly triad of ACS—is appropriate. Gentle handling of tissue intra-operatively and the avoidance of primary closure of the abdomen under tension are the defining qualities and the hallmarks of good surgical handicap. It is generally advocated that when the abdominal contents can be viewed protruding above the wound margin when looking across the table at the level of the anterior abdominal wall, temporary abdominal closure (TAC) is the option of choice.93,94

Burch et al95 proposed a four-category grading system for IAH as follows:

| Grade | Pressure Range |
|-------|----------------|
| I     | 10-15 cm H₂O (7.5-11 mm Hg) |
| II    | 15-25 cm H₂O (11-18 mm Hg) |
| III   | 25-35 cm H₂O (18-25 mm Hg) |
| IV    | > 35 cm H₂O (>25 mm Hg) |

The authors advocated conservative management for grades I and II and DL for grades III and IV. At first glance, this is useful and practical but, in reality, many surgeons are skeptical about the whole concept of IAH and ACS and would not readily take a patient with, say severe pancreatitis with an IAP of 25 mm Hg, from the ICU to the OR for a DL. This is where the problem lies: the decision to subject a critically ill medical patient with hemodynamic instability (somebody else’s headache!) to the additional hazards of the trauma of surgery and general anaesthesia. On the contrary, many published papers show ample evidence that such intervention re-
sults in substantial benefit to the patient with improved survival and diminished morbidity.  

There are critically ill patients with IAH and ACS in whom a trial of conservative (medical) management would be appropriate. This may wholly, or in part, be due to four reasons: DL is associated with a less than perfect survival outcome (38-71%); the complications and the cost of management of patients with TAC may be prohibitively high; there are subsets of patients in whom DL may not be necessary; and consent for operation may have been withheld.

The evidence for conservative management is limited by the lack of randomized studies. Therefore, a lot of what is published is based on limited experience and anecdotal reports. Massive distension of the abdomen by postoperative ileus or intra-abdominal pathology may contribute significantly to IAH for which correction of underlying etiologic factors like electrolyte imbalance or the use of prokinetic drugs like erythromycin, metoclopramide, neostigmine, insertion of nasogastric and rectal tubes or colonoscopic decompression may be accompanied by a reduction of IAP. Faecal impaction and irremovable constipation may respond to suppositories, enemas or manual disimpaction. In patients under mechanical ventilation, the use of neuromuscular blockade may be a temporizing measure by paralyzing the muscles of the abdominal wall and considerably lowering the IAP. Diuretics and dialysis might help in removing excess interstitial fluid that has contributed to visceral edema in cases where massive fluid requirement for resuscitation led to IAH and ACS such as in burns and SAP. Tapping of a massive ascites may be rewarded with an immediate reduction in IAP, but it is also in these patients with liver failure that the coagulopathy may contraindicate DL.

Where operative intervention is indicated in the treatment of ACS, the operative technique involves opening the abdomen, managing the open abdomen and closing it. The abdomen may be opened for damage-control surgery where the operation is abbreviated by a reduction of IAP. Faecal impaction and intracellular necrosis. This is then followed by a temporary large amount of acidic metabolites from the mesenteric aim to increase the circulatory volume, to neutralize a carbonate and 50 g of mannitol is administered. This is a cocktail made of one liter of normal saline, sodium bicarbonate and 50 g of mannitol is administered. This is aimed to increase the circulatory volume, to neutralize a large amount of acidic metabolites from the mesenteric vascular bed and to enhance diuresis to prevent acute tubular necrosis. This is then followed by a temporary abdominal closure which is aimed at ensuring the IAP is not raised by a primary fascial closure (PFC) and to permit easy access to the contents of the abdomen after resuscitation of the patient. Managing the open abdomen is fraught with many problems that include sepsis and negative nitrogen balance from loss of fluid from the wound.

Four methods TAC have been described: towel clip closure, Bogota bag closure, mesh closure, and vacuum-assisted wound closure. The towel clip closure method is the cheapest and easiest method and entails using a series of towel clips to hold the edge of the skin at distances 2 to 3 cm apart after hemorrhage has been controlled. The Bogota bag uses a split infusion bag sutured to the edge of the wound to cover the visera. Mesh closure employs absorbable meshes on the fascial edges with the omentum, if present, interposed between the mesh and the bowels to minimize the risk of fistula formation. The vacuum-assisted technique involves placing a fenestrated non-adherent plastic sheet inside the abdomen extending bilaterally under the anterior abdominal wall followed by a layer of polyurethane sponge that is cut to size and sutured to the edge of the wound incorporating fenestrated tubes connected to vacuum. These are covered with a final air-tight adhesive film.

Closure of the open abdomen is planned when edema has subsided and PFC is feasible. Gradual wound closure is attempted using interrupted transverse sutures on the superior and inferior edges of the incision. The technique of component separation is helpful to achieve PFC and involves the partial separation of the muscles of the anterior abdominal wall at the level of their aponeuroses on the abdominal recti in a vertical direction while preserving the neurovascular bundles. A new technique was reported from Sweden using vacuum-assisted closure and gradual shortening of non-absorbable meshes sutured to the wound edges with good results. The use of tissue expanders to aid in PFC is known as well as the use of pedicle or free latissimus dorsi flaps to reconstruct the abdominal wall.

Finally, we must not forget the courageous, lone surgeon working in austere parts of the world without devices for vacuum-assisted wound closure or who may not be familiar with using them. The use of a skin only closure may be a safe and wise option in the aftermath of DCS under such circumstances. The incisional hernia thus created can be repaired at a later date and would amount to an acceptable trade-off for the patient to end up with a temporary disability (hernia) than to risk death by ACS.
Abdominal compartment syndrome (ACS) is a serious condition characterized by increased intra-abdominal pressure (IAP), which can lead to organ dysfunction and failure. The high IAP applies pressure on vital abdominal organs, compromising blood flow and oxygen delivery, particularly to the kidneys and liver. ACS is often associated with traumatic abdominal injuries, sepsis, postoperative states, and severe burns.

### Key Points
1. **Etiology**: ACS can be primary or secondary to other conditions. Primary ACS is seen in individuals with severe injuries or medical conditions leading to large fluid shifts within the abdomen. Secondary ACS develops when abdominal injuries or medical conditions increase IAP.
2. **Pathogenesis**: Increased IAP reduces blood flow to abdominal organs, particularly the kidney and liver, leading to organ dysfunction and failure. The reduced perfusion can result in metabolic acidosis, renal failure, and multiple organ dysfunction syndrome (MODS).
3. **Clinical Manifestations**: ACS manifests with a variety of symptoms, including abdominal pain, altered mental status, oliguria, and hypotension. Early recognition and intervention are crucial to prevent complications.
4. **Diagnosis and Management**: Measurement of IAP is crucial for diagnosis and management. Therapeutic options include early tracheal intubation, ventilator management, fluid resuscitation, and prompt surgical therapy.
5. **Outcome**: The mortality associated with ACS is high, with approximately 50% of patients dying due to multi-organ failure.

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### Conclusion
Abdominal compartment syndrome is a significant medical emergency requiring prompt diagnosis and management to prevent multi-organ failure and mortality. Recognition and timely intervention are critical to improve outcomes in patients with ACS.