

Review

Abdominal compartment syndrome

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Abstract

Intra-abdominal hypertension (IAH) associated with organ dysfunction defines the abdominal compartment syndrome (ACS). Elevated intra-abdominal pressure (IAP) adversely impacts pulmonary, cardiovascular, renal, splanchnic, musculoskeletal/integumentary, and central nervous system physiology. The combination of IAH and disordered physiology results in a clinical syndrome with significant morbidity and mortality. The onset of the ACS requires prompt recognition and appropriately timed and staged intervention in order to optimize outcome. The history, pathophysiology, clinical presentation, and management of this disorder is outlined.

Keywords: compartment, abdomen, syndrome, hypertension

Introduction

Intra-abdominal pressure (IAP) and its effects on respiration and the abdominal contents has been the subject of scientific study since the 19th century. Marey hypothesized a reciprocal relationship between intra-thoracic pressure and IAP [1]. Bert obtained pressure measurements from anesthetized animals and concluded that diaphragmatic descent caused a rise in IAP, supporting Marey's hypothesis [1]. The potentially profound effect of IAP on organ function was also of interest to early investigators. Wendt inferred IAP from rectal measurements and noted a progressive decline in urine output with increasing IAP [1]. Bradley and Bradley [2] measured renal plasma flow and glomerular filtration rate, and monitored pressures in the inferior vena cava and renal veins while manipulating IAP, and concluded that the decreased renal plasma flow and glomerular filtration rate seen with increased IAP was a

function of elevated renal venous pressure. Heinrichs noted a steady decline in inspired air with respiratory failure and death occurring with IAP above 27–46 cmH₂O in anesthetized cats and guinea pigs [1]. Emerson, following a series of elaborate experiments, concluded that excessive IAP diminished venous return to the heart, resulting in cardiovascular failure [1]. Coombs [3] demonstrated the additive effect of hemorrhage and diminished circulating blood volume on cardiovascular compromise from elevated IAP.

Baggot [4], in 1951, described the clinical effects of abdominal wound closure under tension after a dehiscence or 'abdominal blow-out'. He cited the example of infant death after particularly forcible reductions of abdominal viscera during repair of congenital abdominal wall defects. He also noted the similarly high mortality associ-

ated with analogous procedures in adults with high-tension repairs of acquired abdominal wall defects. Referencing earlier investigations, he concluded that death was a result of respiratory dysfunction. Baggot coined the phrase 'acute tension pneumoperitoneum', believing that trapping a large volume of air within the abdomen during wound closure caused the elevation in IAP. He recommended that tight abdominal closures and dressings be abandoned in favor of loose dressings placed on the open abdomen, primarily to prevent entry of microbes. Interestingly Ogilvie [5], more than a decade earlier, described a 'dodge that has twice helped me out' in order to avoid closing a 'burst abdomen' under tension. He describes the use of Vaseline impregnated canvas or cotton cloth sutured to the wound edges in order to cover abdominal contents. After this he enhanced epithelialization with 'pinch grafts ... liberally sprinkled' on the granulating wound surface. He recommended a waiting period of several months to allow for wound contracture before any attempt at repair of the resultant ventral hernia.

Despite these early contributions, the clinical and pathophysiologic significance of elevated IAP went largely unrecognized. Given the significant mortality associated with repair of congenital abdominal wall defects, pediatric surgeons developed the prosthetic silo technique for gradual reduction of abdominal viscera. This methodology resulted in a marked reduction in mortality in these patients and revisited the topic of the adverse consequences of compressed abdominal viscera and elevation in IAP [6]. Also, the advent of laparoscopy renewed interest in the physiologic consequences of elevated IAP associated with pneumoperitoneum. Several investigators demonstrated altered hemodynamics associated with elevation in IAPs above 20 cmH₂O. Although these investigations demonstrated alteration in various cardiovascular indices, no adverse clinical effects occurred. In keeping with the findings of Coombs [3], the authors of one such study [7] recommended caution with the use of laparoscopy in patients with impaired cardiovascular function, anemia, or hypovolemia.

The 1980s ushered in a renewed interest in the pathophysiologic effects of elevated IAP. Several authors published reports of impaired organ function (particularly renal) associated with presumed elevated IAP, with clinical improvement after abdominal decompression. Kron *et al* [8], in 1984, reported the first series in which IAP was measured and used as a criterion for abdominal decompression, followed by improvement in organ function. Kron *et al* were the first to use the phrase 'abdominal compartment syndrome' (ACS).

Pathophysiology

The 'normal' barometric environment of the abdominal compartment and its regulation has long been a subject of

interest. Hammermilk is credited with providing the first definitive statement on normal IAP. In 1858 he concluded that the normal intra-abdominal environment was a vacuum and believed that the visceral surfaces of its contents were opposed by a 'horror vacui'. Measurement of IAP was described by Braune in 1865; he attempted to measure positive IAP by the use of rectal bougies. He found the pressures within the abdomen varied with position (lowest horizontal and highest vertical) and contraction of abdominal musculature. His studies were criticized because the measurements were based on barometric conditions within hollow viscera. Odebrecht in 1875 tested pressures within the urinary bladder and confirmed the findings of Braune [1]. Multiple investigators have since confirmed the normal pressure environment of the abdomen to be atmospheric or subatmospheric, and to vary inversely with intra-thoracic pressure during normal spontaneous ventilation [1,3,9].

Measurement of intra-abdominal pressure

Contemporary measurement of IAP outside of the laboratory is accomplished by a variety of means. These include direct measurement of IAP by means of an intra-peritoneal catheter, as is done during laparoscopy. Bedside measurement of IAP has been accomplished by transduction of pressures from indwelling femoral vein, rectal, gastric, and urinary bladder catheters. Of these methods, measurement of urinary bladder and gastric pressures are the most common clinical applications [8–12]. In 1984 Kron *et al* [8] reported a method by which to measure IAP at the bedside with the use of an indwelling Foley catheter. Sterile saline (50–100 cm³) is injected into the empty bladder through the indwelling Foley catheter. The sterile tubing of the urinary drainage bag is cross-clamped just distal to the culture aspiration port. The end of the drainage bag tubing is connected to the Foley catheter. The clamp is released just enough to allow the tubing proximal to the clamp to flow fluid from the bladder, then reapplied. A 16-gauge needle is then used to Y-connect a manometer or pressure transducer through the culture aspiration port of the tubing of the drainage bag. Finally, the top of the symphysis pubic bone is used as the zero point with the patient supine (Fig. 1).

An alternative bedside technique has been described in which intragastric pressure measurements are taken from an indwelling nasogastric tube. This method has been validated and found to vary within 2.5 cmH₂O of urinary bladder pressures [12]. Of these techniques, measurement of urinary bladder pressure appears to have gained widest clinical acceptance and application [9,13,14].

The terms intra-abdominal hypertension (IAH) and ACS have sometimes been used interchangeably. It is important to recognize the distinction between these entities. IAH exists when IAP exceeds a measured numeric para-

Figure 1



Measuring the abdominal compartment pressure having injected fluid into the bladder, clamping distal to the aspiration port, and hooking up the pressure transducer apparatus to the aspiration port.

meter. This parameter has generally been set at between 20 and 25 mmHg [10,13]. ACS exists when IAH is accompanied by manifestations of organ dysfunction, with reversal of these pathophysiologic changes upon abdominal decompression [9,10,13–15].

The adverse physiologic effects of IAH impact multiple organ systems. These include pulmonary, cardiovascular, renal, splanchnic, musculoskeletal/integumentary (abdominal wall), and central nervous system [9,13–15].

Pulmonary dysfunction

Elevated IAP has a direct effect on pulmonary function. Pulmonary compliance suffers with resultant progressive reduction in total lung capacity, functional residual capacity and residual volume [9]. This is manifested clinically by elevated hemidiaphragms on chest radiography. These changes have been demonstrated with IAP above 15 mmHg [16]. Respiratory failure secondary to hypoventilation results from progressive elevation in IAP. Pulmonary vascular resistance increases as a result of reduced alveolar oxygen tension and increased intrathoracic pressures. Ultimately, pulmonary organ dysfunction is manifest by hypoxia, hypercapnia and increasing ventilatory pressure. Decompression of the abdominal cavity results in nearly immediate reversal of respiratory failure [9].

Cardiovascular dysfunction

Elevated IAP is consistently correlated with reduction in cardiac output. This has been demonstrated with IAP above 20 mmHg [17]. Reduction in cardiac output is a result of decreased cardiac venous return from direct compression of the inferior vena cava and portal vein. Increased intrathoracic pressure also results in reduced

inferior and superior vena cava flow. Maximal resistance to vena cava blood flow occurs at the diaphragmatic caval hiatus. This is related to the abrupt pressure gradient between the abdominal and chest cavities. Elevated intrathoracic pressure causes cardiac compression and reduction in end-diastolic volume. Elevations in systemic vascular resistance result from the combined effect of arteriolar vasoconstriction and elevated IAP. These derangements result in reduced stroke volume that is only partly compensated for by increases in heart rate and contractility. The Starling curve is thus shifted down and to the right, and cardiac output progressively falls with increasing IAP [9,16,17]. These derangements are exacerbated by concomitant hypovolemia [3].

Increased intrapleural pressures resulting from transmitted intra-abdominal forces produce elevations in measured hemodynamic parameters, including central venous pressure and pulmonary artery wedge pressure (PAWP). Significant hemodynamic changes have been demonstrated with IAP above 20 mmHg [9,16]. Animal models have shown that approximately 20% of IAP is transmitted to the chest cavity from upward bulging of the hemidiaphragms [17]. Accurate prediction of end-diastolic filling pressures by means of equations that subtract a component of pleural pressure from PAWP have not been demonstrated to be consistently reliable, however [16,18]. Recent technological advances have allowed measurement of right ventricular end-diastolic volumes by means of a rapid thermistor flow-directed pulmonary artery catheter. This technology has been shown to be a more accurate predictor of left ventricular end-diastolic volume and cardiac index than PAWP measurements [18,19]. The cardiovascular environment produced by elevated IAP may be more reliably discerned by reliance on this methodology for hemodynamic measurements.

Renal dysfunction

Graded elevations in IAP are associated with incremental reductions in measured renal plasma flow and glomerular filtration rate. This results in a decline in urine output, beginning with oliguria at IAP of 15–20 mmHg and progressing to anuria at IAP above 30 mmHg [2,9,20]. The mechanism by which renal function is compromised by elevated IAP is multifactorial. Early investigations [2] pointed to elevated renal venous pressure as a means that is sufficient to account for renal insufficiency associated with IAH. Later investigators criticized these studies for failure to establish the effect of direct ureteral compression on renal dysfunction. Subsequent investigations showed no significant difference in renal dysfunction when ureteral stents were used in a subgroup of patients [20].

The adverse renal physiology associated with IAH is pre-renal and renal. Prerenal derangements result from altered cardiovascular function and reduction in cardiac output

with decreased renal perfusion. Reduced cardiac output is not solely responsible for renal insufficiency associated with elevated IAP because correction of cardiac indices does not completely reverse impairment in renal function. Renal parenchymal compression produces alterations in renal blood flow secondary to elevated renal vascular resistance. This occurs by compression of renal arterioles and veins. Resistance changes have been measured with graded elevation in IAP. Renal vascular resistance ranges from 500% or greater at 20 mmHg to 1500% or greater at 40 mmHg, and is many times greater than simultaneously measured systemic vascular resistance [20].

The combined effect of prerenal and renal derangements produces progressive reduction in renal plasma flow and glomerular filtration. This results in elevated levels of circulating renin, antidiuretic hormone, and aldosterone, which further elevate renal and systemic vascular resistance. The result is azotemia with renal insufficiency and renal failure that is only partly correctable by improvement in cardiac output [2,9,20].

Portosystemic visceral dysfunction

Splanchnic blood flow abnormalities that result from IAH are not limited to the kidneys. Impaired liver and gut perfusion have also been demonstrated with elevation in IAP. Severe progressive reduction in mesenteric blood flow has been shown with graded elevation in IAP from approximately 70% of baseline at 20 mmHg, to 30% at 40 mmHg. Intestinal mucosal perfusion as measured by laser flow probe has been shown to be impaired at IAP above 10 mmHg, with progressive reductions in flow corresponding to increased measured abnormalities in mesenteric perfusion. Metabolic changes that result from impaired intestinal mucosal perfusion have been shown by tonometry measurements that demonstrate worsening acidosis in mucosal cells with increasing IAH [21]. Similarly, measured abnormalities in intestinal oxygenation have been shown with elevations of IAP above 15 mmHg. Impairment in bowel tissue oxygenation occurs without corresponding reductions in subcutaneous tissue oxygenation, indicating the selective effect of IAP on organ perfusion [22]. Not surprisingly, reductions in mesenteric flow have been shown to be greatly exacerbated in the setting of resuscitation after hemorrhagic shock [23].

Impaired bowel perfusion has been linked to abnormalities in normal physiologic gut mucosal barrier function, resulting in a permissive effect on bacterial translocation. This may contribute to later septic complications associated with organ dysfunction and failure [24].

Adverse effects of IAP on hepatic arterial, portal, and microcirculatory blood flow have also been shown with pressures above 20 mmHg. A progressive decline in perfusion through these vessels occurs as IAP increases,

despite cardiac output and systemic blood pressure being maintained at normal levels. Splanchnic vascular resistance is a major determinant in the regulation of hepatic arterial and portal venous blood flow. Elevated IAP can become the main factor in establishing mesenteric vascular resistance and ultimately abdominal organ perfusion [25]. These abnormalities are amplified in the setting of hypovolemia and hemorrhage, and are only partly correctable by physiologic and resuscitative improvements in cardiac output [21–25].

Although technically not a component of the abdominal cavity itself, the abdominal wall is also adversely impacted by elevations in IAP. Significant abnormalities in rectus muscle blood flow have been documented with progressive elevations in IAP. These perfusion abnormalities are roughly on par with changes in abdominal visceral perfusion with graded increases in IAP. Clinically, this derangement is manifest by complications in abdominal wound healing, including fascial dehiscence, and surgical site infection [26].

Central nervous system dysfunction

Elevations in intracranial pressure (ICP) have been shown in both animal and human models with elevated IAP. These pressure derangements have been shown to be independent of cardiopulmonary function and appear to be primarily related to elevations in central venous and pleural pressures. The exact mechanism of elevated ICP associated with IAH remains to be definitively elucidated, but appears to be a function of impaired cranial venous outflow. Elevated IAP has been demonstrated to coexist with obesity and increased abdominal girth. This is proposed as a chronic form of IAH and has been hypothesized as a mechanism for benign ICP, which is also referred to as pseudotumor cerebri. Abdominal decompression and weight loss via bariatric surgery have been shown to reverse benign ICP associated with IAH [9,27].

Clinical presentation

ACS exists when elevated IAP or IAH is associated with organ dysfunction. Mechanistically this occurs when there is a pressure–volume disparity between the abdominal cavity and its contents. The result is elevated IAP, causing the adverse physiologic consequences described above.

Incidence and risk factors

The exact incidence of ACS is yet to be established, but it is clearly increased in certain population groups. These include patients with severe blunt and penetrating abdominal trauma, ruptured abdominal aortic aneurysms, retroperitoneal hemorrhage, pneumoperitoneum, neoplasm, pancreatitis, massive ascites, and liver transplantation [14]. Massive fluid resuscitation, accumulation of blood and clot, bowel edema, and forced closure of a non-compliant abdominal wall are common factors among

Table 1

Risk factors for abdominal compartment syndrome

Severe penetrating and blunt abdominal trauma
Ruptured abdominal aortic aneurysm
Retroperitoneal hemorrhage
Pneumoperitoneum
Neoplasm
Pancreatitis
Massive ascites
Liver transplantation
Abdominal wall burn eschar

these patients [28]. Additionally, circumferential abdominal burn eschars cause extrinsic compression of the abdominal wall, leading to increases in IAP [9]. Among the trauma population, the group that is especially at risk includes those patients undergoing abbreviated or 'damage control' laparotomy, especially with intra-abdominal packing [9,28]. In one prospective series of 145 patients who were identified as being at risk for development of the ACS [10] the incidence was reported as 14%. The incidence following primary closure after repair of ruptured abdominal aortic aneurysm is reported in one series as 4% [9].

Risk factors for ACS are summarized in Table 1.

Diagnosis

The ACS exists when IAH is associated with organ dysfunction that is reversible upon abdominal decompression. The patients at risk have been previously described. Organ dysfunction occurs in multiple systems, as previously mentioned.

Clinical manifestations of organ dysfunction include respiratory failure that is characterized by impaired pulmonary compliance, resulting in elevated airway pressures with progressive hypoxia and hypercapnia. Extremely high driving pressures may be required to maintain minimally sufficient tidal volumes, often with loss of delivered tidal volume by distension of ventilatory tubing. Some authors report pulmonary dysfunction as the earliest manifestation of ACS [14]. Chest radiography may show elevated hemidiaphragms with loss of lung volume [29].

Hemodynamic indicators include elevated heart rate, hypotension, normal or elevated PAWP and central venous pressure, reduced cardiac output and elevated systemic and pulmonary vascular resistance [9,29]. Measurement of right ventricular end-diastolic volume may be a more accurate predictor of a patient's position on the Starling curve [18,19].

Table 2

Abdominal compartment grading system

Grade	Bladder pressure (mmHg)	Recommendation
I	10–15	Maintain normovolemia
II	16–25	Hypervolemic resuscitation
III	26–35	Decompression
IV	>35	Decompression and re-exploration

Impairment in renal function is manifest by oliguria progressing to anuria with resultant azotemia. Renal insufficiency as a result of IAH is only partly reversible by fluid resuscitation. Renal failure in the absence of pulmonary dysfunction is not likely to be the result of IAH [14,29].

Elevated ICP is an additional clinical manifestation of ACS [29]. Clinical confirmation of IAH requires bedside measurements indicative of IAP. These techniques include transduction of gastric, rectal, and bladder pressures [8,11,12]. A technique for measurement of bladder pressure has been described by Kron *et al* [8] (discussed above). Experimental and clinical data indicate that IAH is present above an IAP of 20 mmHg [10,13].

Management

Definitive management of ACS is based on optimal timing and staging of abdominal decompression and is predicated on early identification of at-risk patients.

Surveillance for IAH and ACS requires close monitoring of relevant physiologic parameters, including indicators of IAP. The decision to intervene surgically is based on the clinical decision that improvement in organ dysfunction can best be accomplished by abdominal decompression, which is the treatment required [9,14].

Prevention

The earliest and potentially most effective means of addressing this disorder is by recognition of patients who are at risk and pre-emptive interventions designed to minimize the chances for development of IAH. These decisions are primarily made during laparotomy and involve choices regarding the decision to terminate an operation because of overwhelming nonoperative disorders in patient physiology (hypothermia, acidosis, coagulopathy) and the method of abdominal wound closure [30]. Various types of mesh closures of the abdominal wall and other alternative means of abdominal content coverage have been described [5,9,13,14,31,32]. There is evidence [31] that ACS may be preventable by use of absorbable mesh in high-risk injured patients undergoing laparotomy. Achieving optimal resuscitation rather than over-resuscita-

tion is a potentially preventable complication in intensive care management. Multiple indicators of effective resuscitation have been evaluated. Lactate, base deficit, and gastric mucosal pH appear to be reliable indicators to guide resuscitative interventions [33].

Surgical intensive care unit management

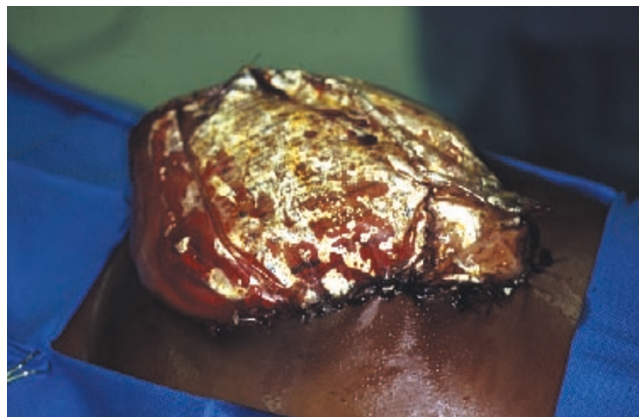
Identifying patients in the intensive care unit (ICU) at risk for developing ACS with constant surveillance can help lead to prevention. A further strategy is based on recognition of IAH and resultant organ dysfunction. A four-stage grading scheme based on IAP has been developed, tested, and proposed as a useful ACS management tool (Table 2) [10]. These stages are based on measured bladder pressures. This methodology correlates worsening organ dysfunction with increasing bladder pressures, with 100% of patients showing pulmonary, cardiovascular, and renal dysfunction with pressures greater than 35 mmHg. Meldrum *et al* [10] perform simple bedside decompression for bladder pressures from 26 to 35 mmHg, but recommend formal abdominal exploration with pressures greater than 35 mmHg in anticipation of significant intra-abdominal ischemia. This is based on impaired bowel capillary perfusion at IAP greater than 35 mmHg.

Alternative means for surgical decision making are based on clinical indicators of adverse physiology, rather than on a single measured parameter. In the setting of IAH, abdominal decompression has been recommended with any coexisting deterioration in pulmonary, cardiovascular, or renal function. Additionally, with IAH that is unresponsive to standard intervention and with indicators of bowel ischemia (acidosis by tonometry or dusky bowel seen through transparent coverage material), decompression is recommended [9,34]. Worsening hypercapnia and pulmonary compliance have been identified as critical indicators of pulmonary failure that warrant emergent abdominal decompression in the setting of IAH [13].

Abdominal decompression and wound management

Once the decision is made to proceed to surgical decompression and the need for intervention is established, the location and possibly transportation requirements for performing this procedure must be decided. A decision to perform the decompression in the ICU is a function of the ventilatory requirements of the patient and the risk associated with transport to the operating room. Although optimal respiratory support may be available in the ICU, this location is generally suboptimal for controlling surgical bleeding. The potential for major intra-abdominal hemorrhage varies, but it can be significant in patients with ACS. Operative planning must include contingencies for management of surgical bleeding encountered when decompression is performed in the ICU, which may require repacking and immediate transport to the operating room. It is mandatory that an operating room be immediately

Figure 2



The abdomen was reopened due to abdominal compartment syndrome and approximated with an intravenous bag, sterile side down.

available and appropriately staffed before beginning an ICU abdominal decompression. Patients who require high airway pressures for adequate gas exchange require transport on a high-flow pressure ventilator powered by a battery source [14].

Abdominal decompression may itself precipitate adverse physiologic and metabolic events that should be anticipated. These include a large increase in pulmonary compliance with resultant elevation in minute ventilation and respiratory alkalosis unless appropriate ventilatory changes are instituted. 'Washout' of accumulated intra-abdominal products of anaerobic metabolism may result in a bolus of acid and potassium systemically delivered to the heart. This may result in an adverse cardiac event such as an arrhythmia or asystole. Anticipating, recognizing, and treating these effects is of critical importance [9,14].

Under most circumstances following abdominal decompression, immediate primary fascial closure is obviated. Alternative means for coverage of the abdominal contents include skin closure with towel clips or suture, abdominal wall advancement flaps, plastic or silicone coverage, and mesh interposition grafts (Fig. 2). Patients undergoing decompressive laparotomy are by definition at risk for future redevelopment of ACS, and strong consideration should be given to providing for re-exploration and a staged closure. This may include fascial closure after a period of 7–10 days versus placement of split thickness skin grafts on a granulating surface followed by delayed repair of the resulting abdominal wall hernia after several months [9,13,14,30–32]. Finally, early management of the open abdomen must include recognition for significant fluid losses and fluid replacement [14].

Outcomes

The ACS is a condition with a potentially high lethality that must be recognized early and effectively managed in order to optimize outcome. Most deaths associated with ACS are due to sepsis or multiple organ failure. Mortality associated with this condition has been reported in 10.6–68% of patients [9,10,14,28]. In one series [14], nonsurvivors tended toward a more fulminant course, with the majority of deaths occurring within the first 24 h of injury. There is some evidence that the syndrome may be prevented in high-risk patient groups by selective mesh closure of the abdominal wall after laparotomy [28,31].

Further study is needed to better establish the incidence, long-term and short-term morbidity, and mortality of this condition.

Conclusion

The abdominal compartment syndrome is defined as intra-abdominal hypertension associated with organ dysfunction. Adverse physiology has been demonstrated in pulmonary, cardiovascular, renal, musculoskeletal/integumentary, and central nervous system function. Identification of patients at risk, early recognition, and appropriately staged and timed intervention is key to effective management of this condition.

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