INTENSIVE CARE

Intra abdominal hypertension and the abdominal compartment syndrome

Paul Hayden

Department of Critical Care, Guys and St. Thomas’ NHS Foundation Trust, London SE1 7EH, UK

KEYWORDS
Abdominal compartment syndrome; Perfusion pressure; Laparostomy

Summary
The patho-physiological significance of raised intra-abdominal pressure, known as Intra Abdominal Hypertension, with subsequent organ dysfunction and failure, known as Abdominal Compartment Syndrome, has recently been demonstrated to occur relatively frequently in mixed populations of critically ill patients. Clinical diagnosis is unreliable, so routine measurement of intra abdominal pressure should be undertaken, particularly in specific groups of patients known to be at high risk. Whilst definitive therapy requires surgical abdominal decompression, less invasive therapies have been investigated and, if initiated early, may help to minimise progression of the condition. Clearly defined indications for surgical intervention remain elusive however and require prospective investigation. This review summarises the patho-physiology of the syndrome, its diagnosis and surveillance, and current management strategies, both medical and surgical.

© 2007 Elsevier Ltd. All rights reserved.

Introduction
Over the past 20 years, the patho-physiological significance of raised intra abdominal pressure, known as Intra Abdominal Hypertension (IAH), with subsequent organ dysfunction and failure, known as Abdominal Compartment Syndrome (ACS), has become increasingly acknowledged. Although many high-risk groups have been identified, consensus evidence-based management remains elusive.

This review summarises the patho-physiology of the syndrome, its diagnosis and surveillance, and current management strategies, both medical and surgical.

E-mail address: paulhayden@lineone.net.
Epidemiology

Once thought of as an epi-phenomenon of major trauma, it is now acknowledged that IAH and ACS are far more common than previously thought, being prevalent in the medical and general surgical populations as well as in those with major trauma. Two recent prospective observational multi-centre studies have suggested that in mixed ICU populations, IAH may occur in approximately 30–50% of patients with ACS occurring in 4–8% of these.3,4 The development of ACS whilst on the ICU has also been shown to be a significant independent risk factor for increased mortality.

Patho-physiology

The abdominal compartment syndrome is unique as a compartment syndrome in that it affects virtually all organ systems within the body. Organ compromise begins insidiously and at variable intra-abdominal pressures depending on the organ system investigated and the individual’s abdominal compliance. Chronically increased intra-abdominal volume, such as that caused by obesity, pregnancy or cirrhotic ascites does not usually cause significant IAH or ACS because the abdominal wall can increase its compliance over time. Strategies to increase abdominal compliance have also shown benefit in reducing the effects of ACS.

Cardiovascular system

As the pressure rises within the abdomen, it begins to compress the inferior vena cava, leading to a reduction in preload with consequent reduction in cardiac output and blood pressure. Additionally, pressure on the abdominal aorta increases vascular resistance leading to a further reduction in cardiac output. The rise in abdominal pressure also increases intra-thoracic pressure via transmission across the diaphragm. As intra-thoracic pressure (ITP) increases, it further impedes cardiac output via reduction in venous return and a rise in pulmonary vascular resistance. This rise in intra-thoracic pressure however artifically increases central venous pressure, making this parameter unhelpful as a guide to fluid resuscitation and volumetric techniques such as the PiCCO™ system have been validated as an alternative.5

Respiratory system

Raised intra-abdominal pressure acts directly across the diaphragm, compressing the basal lung segments resulting in alveolar collapse, impairing ventilation/perfusion relationships with subsequent hypoxaemia and hypercarbia. The rise in intra-thoracic pressure that occurs makes ventilation increasingly difficult. In order to prevent hypventilation and hypercarbia, ventilatory pressures are often raised above accepted safety thresholds increasing the risk of ventilator induced lung injury. Measurement of airway pressures is confounded by the increased intra-thoracic pressure caused by the
trans-diaphragmatic effect of raised IAP. One useful calculation of ‘effective’ airway pressure is by subtracting IAP/2 from the measured airway pressures on the ventilator.  

Gastrointestinal and hepato-biliary systems

Rising intra-abdominal pressure impedes splanchnic blood flow leading to a vicious cycle of worsening gut ischaemia and bowel oedema, further elevating IAP. Splanchnic ischaemia may promote bacterial translocation, exacerbating the sepsis response.

Hepatic artery blood flow is impaired at a relatively low IAP, at least in animal models, although this remains insidious until late on in the clinical course of untreated ACS. Portal vein blood flow is also compromised both by direct compression and reduced cardiac output leading to hepatic dysfunction. Collateral flow is increased as is variceal wall stress raising the likelihood of variceal haemorrhage.

Renal system

The effect of raised IAP on the kidney is multifactorial. Anti-Diuretic Hormone secretion increases as part of the stress response. Reduced cardiac output impairs anterograde flow to the kidney with a reduction in efferent flow leading to compromised renal perfusion gradient. Direct ureteric compression may also occur. IAH and ACS are now regarded as significant independent risk factors for the development of Acute Kidney Injury (AKI).  

Neurological system

The rise in intra thoracic pressure induced by an increase in IAP impedes venous flow from the cranial circulation. This causes a rise in intracerebral pressure and impairs cerebral perfusion pressure. Several studies have shown improvement in ICP by strategies to reduce IAP in traumatic brain injury (TBI) patients. Some authors have called for routine measurement of IAP in all patients admitted with TBI, particularly those with polytrauma.

Methods of diagnosis and surveillance

Clinical assessment of abdominal distension, both by examination and abdominal perimetry has been shown to be inaccurate. Whilst the gold standard in terms of accuracy is directly measured intra-abdominal pressure using a transducer placed within the abdominal cavity such as that used in laparoscopic surgery, it is a high risk and impractical procedure for routine use in the ICU with the potential for infection, haemorrhage and cannula displacement. Therefore, indirect measures of IAP have been developed. Perhaps the easiest to perform and most investigated of these is the trans-vesical technique in which a pressure transducer is attached to a urinary catheter. This technique has been utilised since the early 1980s and has undergone several modifications to reduce the potential for complications. It has the benefit of being simple to perform, safe, and reproducible. However, in those with bladder pathology or in patients without urinary catheters (for example due to anuric renal failure), it may be desirable to use an alternative technique. Rectal, vaginal and femoral vascular techniques have all been investigated although intra-gastric and oesophageal measurement has been the most validated. Simultaneous measurement of intra-oesophageal pressure and intra-gastric pressure also allows a calculation of the trans-diaphragmatic pressure difference to be made which is useful to assess the effect of raised intra-abdominal pressure on the intra-thoracic pressure.

High risk groups

IAP measurement should be considered in all patients known or suspected to be at high risk of developing IAH and ACS (see Fig. 2). Clearly, those patients requiring major abdominal surgery, particularly for traumatic injuries, are at particularly high risk. Patients with severe capillary leak phenomena and those receiving high volume fluid resuscitation, such as severe pancreatitis and burns, should also be considered for monitoring with one recent study quoting the incidence of IAH in pancreatitis at 78%.

![Figure 2 High-risk groups.](image-url)
The optimal frequency of measurement is however unknown. One suggested strategy is to measure all high-risk patients intermittently (for example, 4–6 hourly) and then if IAP rises above 12 mmHg, to increase the frequency of measurement to either hourly or continuously whilst strategies to reduce IAP are commenced. Following treatment for IAH/ACS, measurement of IAP should be continued to observe for recurrent ACS.

Management of intra-abdominal hypertension

Once ACS supervenes, even with aggressive management, mortality remains high. Preventative measures to prevent the progression from IAH to ACS should therefore be considered (see Fig. 3).

Because invasive techniques are associated with increased complications, medical and minimally invasive options may be considered unless ACS has already developed, at least in patients with secondary ACS. For those with primary ACS the available evidence, albeit primarily based on retrospective analysis of large trauma cohorts, is to perform abdominal decompression as early as possible.

Medical/minimally invasive options

Decompression of the bowel

Ileus and bowel obstruction lead to bowel dilatation and mucosal oedema which subsequently raise intra-abdominal pressure. This results in a vicious circle of worsening organ perfusion and haemodynamic compromise requiring further volume resuscitation, exacerbating the rise in IAP. Measures to promote bowel motility such as correction of electrolyte abnormalities and avoidance of drugs known to impair motility such as opiates should be routine with the use of gastro-prokinetics (metoclopramide, erythromycin) or colo-prokinetics (neostigmine) when necessary. Endoscopically-guided bowel decompression may also be beneficial.

Reducing intra-abdominal fluid and oedema

Increased bowel oedema and intra-abdominal fluid collections are recognised as both a cause of IAH and a sequel. Strategies to facilitate removal of intra-abdominal fluid have therefore been investigated and advocated. Techniques for renal replacement therapy (RRT) are particularly useful to tightly control body fluid balance and can be employed to achieve negative fluid balance following the initial resuscitative period. Frusemide ± human albumin solution has also been shown to assist in achieving negative fluid balance and redistribution of fluid accumulating in the extra vascular space as a result of capillary leak.

An alternative strategy is to limit overall fluid administration in the first place. Several well conducted trials in critically ill patients have shown outcome benefit when restrictive fluid balance is achieved. Early aggressive fluid resuscitation guided by haemodynamic endpoints such as serum lactate, central venous pressure or central venous oxygen saturation has been shown to reduce the

**Figure 3** Proposed management guideline for IAH/ACS.
overall volume of fluid administered and improve outcome. Minimally invasive options include percutaneous drainage of intra-abdominal collections, under CT or Ultrasound guidance. Several studies have shown outcome benefits when these minimally invasive techniques are employed compared with medical therapy alone or versus surgical intervention. The rate at which the intra-abdominal collections are drained can also be varied, which may limit the severity of reperfusion injury caused by sudden decompression of the abdomen, a significant problem found when performing surgical abdominal decompression.

Increasing abdominal compliance

Measures to increase the compliance of the abdominal cavity, whilst not able to reverse the pathological cause of IAH/ACS, may be used to improve symptoms and may ‘buy time’ for definitive therapy. The main strategy involved is the use of muscle relaxants. Negative pressure cuirasses have also been investigated to effectively increase intra-abdominal volume and reduce pressure. These techniques remain at a research stage although they have been validated in both animal models and human subjects.

Surgical techniques—abdominal decompression

The gold standard for treatment of established ACS is surgical decompression of the abdomen. Although this is now a relatively common-place procedure, recommendations for the intra-abdominal pressure at which it should take place and in whom remain unknown. Pre-emptive surgical decompression has been advocated in high-risk patients to prevent the development of ACS although there are no convincing prospective studies so far to support this. A systematic review of the literature concerning abdominal decompression was recently reported by De Waele and colleagues. They identified eighteen trials including a total of 250 patients. Mortality following decompression approached 50% but in many of the trials, mortality without decompression approached 100%.

Following successful decompression, the abdomen may be left ‘open’ to heal by secondary intention, forming a ventral hernia with poor cosmetic and functional results, or closed via a variety of strategies. These may be divided into primary or delayed closure (temporary abdominal closure or TAC).

Primary closure following ACS frequently leads to the development of tertiary ACS which in turn is associated with poor outcome. Early signs of failure are an increased ventilatory pressure or haemodynamic instability during approximation of the wound edges. This should influence the clinician to consider alternative TAC techniques.

There are several types of TAC. The ‘Bogota Bag’ is a sterile 3 l fluid administration bag cut into a flat sheet that is sewn across the abdominal wall defect. Whilst this allows visual inspection of the abdominal contents, it is non-expansive and is strongly associated with the development of recurrent ACS.

Vacuum Assisted Closure (VAC) devices help to draw the wound margins together, removing slough from the granulating tissue forming across the cavity and facilitating reduction of the defect size. Low pressures should be applied to prevent the development of fistulae. However, at present this is the preferred technique as it improves the cosmetic and functional result with reduced operative intervention.

Minimising reperfusion sequelae

The sudden reduction in intra-abdominal pressure during abdominal decompression causes a marked reperfusion syndrome with release of products of anaerobic metabolism, potentially inducing cardiac arrest. Preventative strategies include aggressive volume resuscitation prior to decompression, infusion of sodium bicarbonate and mannitol although there is no evidence base for this in the context of ACS. Free radical scavengers and anti-oxidant therapies have also been investigated as therapies. At present, the rationale for these techniques is limited to anecdotal evidence and small mammal models however.

Conclusions

Intra-Abdominal Hypertension is a relatively common problem in critically ill patients which if untreated may lead to Abdominal Compartment Syndrome and poor outcome. Clinical assessment is unreliable. Therefore monitoring is required which is easy and safe to perform via the vesical or oesophago–gastric route. A treatment strategy aimed at restrictive fluid balance following initial resuscitation combined with good practice to promote bowel motility may minimise worsening of IAH and degradation to ACS. If surgical decompression is to be performed, it should
be considered prophylactically in high-risk candidates and undertaken early in those with clinical deterioration. Prospective trials are desperately needed to improve evidence-based management of the syndrome. It is hoped that a multi-centre interventional trial will soon commence to facilitate this.

References


