ARTICLE

Intra-abdominal hypertension and the abdominal compartment syndrome

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The phenomenon of intra-abdominal hypertension (IAH) and its progression to abdominal compartment syndrome (ACS) is a common and frequently under-recognised condition in critically ill patients. IAH is defined as an intra-abdominal pressure (IAP) greater than 12 mmHg. The ACS occurs once IAH is associated with organ dysfunction.

Early detection is the cornerstone of management in the critically ill patient and the regular measurement of IAP in patients at risk for developing raised IAP is encouraged. The intravesical technique of measurement is relatively non-invasive and provides excellent correlation with direct measurement of IAP. The consequences of ACS are multi-systemic, resulting in organ dysfunction. The respiratory, cardiovascular, renal and gastrointestinal systems are particularly affected. The vicious cycle of organ dysfunction may be perpetuated by excessive fluid resuscitation. Early intervention is essential to prevent complications, which carry significant morbidity and, if untreated, possible mortality. Definitive management of this condition is the prompt surgical decompression of the abdomen followed by temporary abdominal closure.

For the past decade it has been recognised that intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) cause significant morbidity and mortality in critically ill patients.[1] Recognition of the prevalence of this syndrome combined with early detection has led to a significant reduction in patient morbidity and mortality.[2] ACS is a condition that has for a long time gone unnoticed and contributed to poor outcomes in critically ill patients.[3] Understanding the pathophysiology, risk factors and complications associated with this condition will lead to early intervention and improved outcome. In this article, emphasis is placed on the recent consensus guidelines published by the World Society of the Abdominal Compartment Syndrome, outlining standards for measurement as well as diagnostic criteria for IAH and ACS.[4]

Pathophysiology

The abdominal cavity can be regarded as a closed space and the pressure within this space is a reflection of its compliance. Since the abdominal contents consist primarily of fluid and are non-compressible, the pressure at any point is representative of the IAP throughout the abdomen.[5] IAP fluctuates with ventilation, increasing with inspiration and decreasing with exhalation, reflecting movement of the diaphragm.[6] IAP is also directly affected by the volume of the solid organs and hollow viscera. The latter may be filled with fluid, air or faeces. Blood, ascites or other space-occupying lesions (tumours, gravid uterus, etc.) also influence IAP. Finally, factors that limit the expansion of the abdominal wall such as burn eschars and oedema can raise the IAP.[7]

The boundaries of the abdominal cavity are the spine posteriorly, the costal margin and abdominal muscles anteriorly, and the diaphragm and pelvis representing the superior and inferior borders respectively. The pressure within this space is determined by the compliance of its walls and the character of the intra-abdominal contents. The pressure volume relationship of the abdominal compartment can be represented by an elastance curve (Fig. 1).

From Fig. 1 it is clear that once a critical volume is reached, any further increase in intra-abdominal volume results in a dramatic increase in pressure. The consequences of raised IAP are multi-systemic, resulting in severe organ dysfunction.

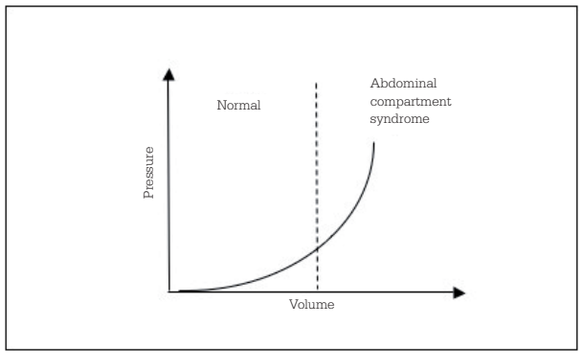

Fig. 1. Abdominal pressure-volume relationship.

Respiratory function is impaired because of increased IAP being transmitted into the thoracic cavity. In the unventilated patient this rapidly leads to respiratory failure. In the mechanically ventilated patient, IAH results in increased airway pressure, a decrease in functional residual capacity (FRC) and ventilation-perfusion mismatching. All of these are potential causes of hypoxaemia. Respiratory compliance is an important parameter to monitor when primary fascial closure is attempted in patients at risk for ACS.

The cardiovascular manifestations are mainly caused by inadequate filling of the heart due to the high intrathoracic pressure. High intrathoracic pressure increases central venous pressure and pulmonary artery wedge pressure without increasing the right or left ventricular end-diastolic volume. Simultaneously, left ventricular afterload is increased due to a rise in systemic vascular resistance. Consequently cardiac output is affected by a reduction in stroke volume, secondary to the reduction in preload and increase in afterload. The effect is similar to cardiac tamponade and a marked pulsus paradoxus may be noted. Assessment of a patient's volume status may therefore be misleading in the setting of raised IAP. In mechanically ventilated patients with IAH the arterial pressure trace may show a systolic pressure variation despite adequate intravascular volume. Intravenous fluid administration may increase cardiac filling and output acutely, but is detrimental due to aggravation of bowel oedema (Fig. 2).[8]

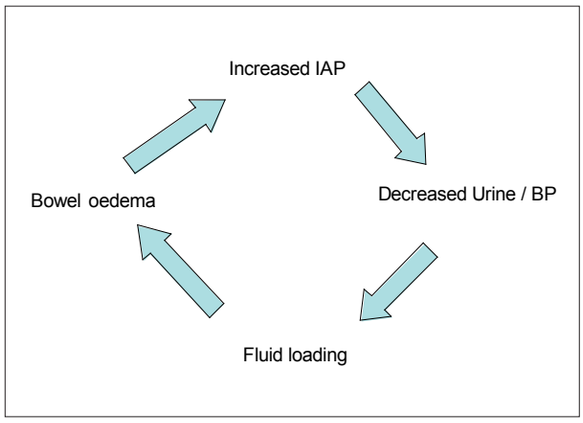

Fig. 2. The vicious cycle of abdominal compartment syndrome.

Cerebral perfusion is compromised due to increases in jugular venous pressure caused by high intrathoracic pressure impeding venous return from the brain. This leads to raised intracranial pressure and decreased cerebral blood flow. Later a fall in systemic blood pressure will aggravate cerebral perfusion. Patients who already have a brain injury are particularly at risk.

Perfusion of the intra-abdominal organs is decreased due to reduced cardiac output and increased splanchnic vascular resistance. Like cerebral perfusion pressure, which is the mean arterial pressure (MAP) minus the intracranial pressure, abdominal perfusion pressure (APP) is the MAP – IAP and has been proposed as a more accurate predictor of visceral perfusion and a possible end-point for resuscitation.[9] APP has been shown to be statistically superior to other common resuscitation end-points such as arterial pH, base deficit, lactate, and hourly urine output. A target APP of 60 mmHg has been correlated with improved survival in patients with IAH and ACS.[9]

A decrease in urine output is often the first clinical indication of ACS in the patient whose IAP is not being monitored. Renal function is impaired primarily by a reduction in renal blood flow and not by direct compression of the renal outflow tract. Decreased renal perfusion pressure (RPP) and associated decreased glomerular filtration have been shown to be the key factors in the development of IAH-induced renal failure.[9-11] Filtration gradient (FG) is the mechanical force across the glomerulus and equals the difference between the glomerular filtration pressure (GFP) and the proximal tubular pressure (PTP). In the presence of IAH, the PTP can be assumed to be equal to the IAP and the GFP is estimated as being equal to the MAP – IAP. It is therefore evident that changes in IAP will have a greater impact on renal function than MAP.[12] Recently, experiments in pigs exposed to IAH where measurement of the renal resistive index in the segmental and arcuate arteries of both kidneys using Doppler techniques was used, showed a linear relationship with intravesical measurements of IAP.[13]

Effects on the micro-circulation in animals have shown that decompression of ACS is associated with the release of cytokines into the portal circulation, resulting in a systemic inflammatory syndrome similar to that seen after resuscitation of subjects with haemorrhagic shock.[14] In fact, decompression of ACS can be seen as a 'second hit' when appropriately sequenced with haemorrhagic shock, mimicking an ischaemia-reperfusion injury.[15,16] This would explain the continued deterioration of patients into multisystem failure and death after delayed surgical decompression.

Recognition of ACS and IAP measurement

Regular monitoring of the IAP of 'at risk' patients is essential for the early detection of this condition.

Clinical assessment by palpating the abdomen is notoriously unreliable. Normal IAP varies from about 0 to 5 mmHg. The reading should be expressed in mmHg (1 mmHg = 1.36 cm H_20) and measured at the end of expiration with the patient in the supine position. The zero point should be at the level of the mid-axillary line.[4] Changes in body position (e.g. supine, prone, head up) and the presence of muscle contractions (detrusor and abdominal wall) directly impact on pressure measurement.[5]

The gold standard for IAP measurement is direct needle puncture and transduction of the pressure within the abdominal cavity (e.g. during peritoneal dialysis or laparoscopy).[17] However, this is not always practical in the critically ill patient and carries significant risk. Indirect measurement has become the routine practice in intensive care units.

Various techniques of indirect measurement have been described and validated, e.g. transduction of balloon-tip catheters in the bladder, colon, uterus and stomach.[5,18-20] The 'bladder' technique has achieved widespread acceptance due to its simplicity and cost effectiveness.[21] Several methods have been described for continuous IAP measurement and although they appear promising, further clinical validation is needed before they can be recommended for routine use.[19]

Regardless of the technique used, certain key principles must be followed to ensure accurate and reproducible measurement. There is disagreement in the literature as to the units in which IAP should be expressed. Earlier studies using water manometer techniques reported their results in cm H_2O, whereas newer electronic transducers report the pressure in mmHg.[22] Another source of confusion has been the zero reference point for the abdomen. Some authors have suggested the symphysis pubis, mid-axillary line or phlebostatic axis, each of which produces different results within the same patient.[23] The most contentious issue in the measurement of IAP has been the priming volume to be instilled in the bladder to ensure a continuous fluid column between the transducer and bladder wall. Several studies have shown that high volumes may increase bladder pressure, especially at higher IAP, leading to inaccurate measurement.[24,25] According to the new consensus guidelines, the standard volume for intravesical IAP measurement is 25 ml sterile saline and the zero point should be taken as the mid-axillarly line.[23]

A practical approach to the intravesical measurement of IAP:

- The patient is positioned supine.
- A T-piece with a three-way stopcock is placed between the urinary catheter and the drainage tubing to reduce the risk of needle-stick injury and the need for multiple needle insertions into the sample port.
- The T-piece is connected to a water manometer traditionally used to measure central venous pressure (CVP) or an electronic transducer.
- The zero point is taken at the level of the mid-axillary line.
- The urinary tubing is emptied and clamped distal to the T-piece.
- 25 ml of sterile saline is instilled into the bladder via the 3-way stopcock.
- If the pressure is recorded in cm H_2O this should be converted to mmHg.
- The clamp is opened and the volume instilled in the bladder subtracted from the patient's urinary output.

Intra-abdominal hypertension

IAH refers to a pressure greater than 12 mmHg and is based on the pressure at which organ dysfunction occurs.[2] Pathological IAH represents a continuum ranging from mild elevations to substantial increases in IAP resulting in severe organ failure. Therefore the following grading system has been introduced and recently modified to stratify patients with IAH:26

- grade 1: IAP 12 - 15 mmHg
- grade 2: IAP 16 - 20 mmHg
- grade 3: IAP 21 - 25 mmHg
- grade 4: IAP > 25 mmHg.

IAH is also sub-classified into four groups based on the duration of symptoms:[27]

- *Hyperacute* IAH refers to transient rises in IAP such as coughing, straining, sneezing, defaecating or physical activity.
- *Acute* IAH develops over a period of hours as a result of trauma or intra-abdominal haemorrhage. This form of IAH commonly progresses to the development of ACS.
- *Subacute* IAH occurs over a period of days and is most commonly seen in medical patients
- *Chronic* IAH occurs over a period of months or years such as pregnancy, morbid obesity, tumours or ascites and may put the patient at risk of developing acute or subacute IAH.

Risk factors for IAH/ACS

Patient factors

- Acidosis (pH < 7.2)
- Hypothermia (core temperature < 33°C)
- Polytransfusion (>10 U packed red blood/24 h)
- Coagulopathy (platelets < 55 000/mm^3 or activated partial thromboplastin time two times normal or higher or prothrombin time < 50% or international standardised ratio > 1.5)
- Sepsis (American-European Consensus Conference definitions)
- Bacteraemia

Abdominal factors

- Intra-abdominal haemorrhage
- Intra-abdominal infection/abscess
- Peritonitis
- Liver dysfunction/cirrhosis with ascites
- Mechanical ventilation
- Use of positive end-expiratory pressure (PEEP) or the presence of auto-PEEP
- Pneumonia
- Abdominal surgery, especially with tight fascial closures
- Massive fluid resuscitation (>5 l colloid or crystalloid/24 h)
- Gastroparesis/gastric distention/ileus
- Volvulus
- Pneumoperitoneum
- Major burns
- Major trauma
- High body mass index (>30)
- Intra-abdominal or retroperitoneal tumors
- Prone positioning
- Massive incisional hernia repair
- Acute pancreatitis
- Distended abdomen
- Damage control laparotomy
- Laparoscopy with excessive inflation pressures
- Peritoneal dialysis.

Abdominal compartment syndrome

As the term implies, the ACS is not related to an absolute pressure but rather refers to a critical pressure at which microcirculatory flow is impaired and organ dysfunction and failure occur. Therefore, ACS is the natural progression of pressure-induced end-organ changes if IAH is not treated timeously. Many definitions exist to describe ACS but generally the 'triad' of ACS is used, which includes:

- a pathological state caused by an acute increase in IAP above 20 - 25 mmHg which
- adversely affects end-organ function or causes serious wound complications, and in which
- abdominal decompression has beneficial effects.[28,29]

Failure to recognise and treat ACS is uniformly fatal whereas appropriate treatment results in improved organ function and overall patient survival.[30] In contrast to IAH, ACS should not be graded, but rather be seen as an 'all or nothing' phenomenon.[3]

Management of IAH and ACS

The only effective measure to treat critically raised IAP is surgical decompression. Certain other strategies have been recommended to prevent and treat organ dysfunction due to elevated IAP.[31,32] In patients at risk for developing ACS, it may be prudent to leave the abdomen open at the time of the first laparotomy. Measuring the intravesical pressure intraoperatively immediately after closure of the abdominal wound is also recommended.

Appropriate management of IAH and/or ACS is based upon four general principles:

- serial monitoring of IAP
- optimisation of systemic perfusion and organ function
- institution of certain medical procedures to reduce IAP
- prompt surgical decompression for refractory IAH.[4]

Abdominal perfusion pressure

As mentioned previously, APP is a useful end-point in resuscitation of patients with IAH. Although not yet subjected to prospective randomised controlled trials, Cheatham *et al.*[9] showed that maintaining an APP ≥ 60 mmHg discriminated between survivors and non-survivors by day 3 in patients with IAH. Indiscriminant fluid administration places the patient at risk for secondary ACS and should be avoided.[31,32] Target APP values may be achieved by judicious fluid management and the use of vasoactive agents. Vasocontrictors, however, may further decrease splanchnic perfusion. The current recommendations suggest targeting an APP of 50 - 60 mmHg.[4]

Increased muscle tone due to various causes including pain, agitation and patient ventilator asynchrony can increase IAP. It is therefore intuitive to provide adequate analgesia and anxiolysis in patients with IAH.[1] Diminished abdominal wall compliance may increase IAP, especially in patients with tight abdominal wall closures. Neuromuscular blockade (NMB) has been effectively used to reduce IAP temporarily in patients with IAH.[33] Although not without adverse effects, including prolonged paralysis, NMB has been used to reduce IAP while other interventions are being performed to reduce IAP.

Fluid resuscitation

Fluid administration is still a controversial subject in resuscitation but remains vital in managing the haemodynamically unstable patient at risk for organ failure. Owing to the effects of mechanical ventilation, hypovolaemia may aggravate the pathophysiological effects of IAH.[6,34] McNelis *et al.*[35] showed that excessive fluid resuscitation in non-trauma surgical patients was an independent risk factor for ACS. Balogh *et al.*[36] retrospectively looked at trauma patients resuscitated to 'supranormal' end-points (i.e. oxygen delivery index of 500 - 600 ml/min/m^2) and found that this group of patients were twice as likely to develop IAH, ACS, organ failure, and to

die than the patients in the restrictive fluid strategy group. Hypertonic fluid resuscitation in burn patients (> 40% body surface area) has been associated with decreased fluid requirements, reduced peak inspiratory pressures, and significantly higher APP levels. Isotonic fluid resuscitation is associated with a 3.5-fold increased risk for developing IAH (IAH > 30 cm H_2O) in this group of patients.[37] Similarly, burn patients were randomised to receive either crystalloid or colloid fluid resuscitation. The crystalloid group required significantly more fluid to maintain adequate urine output and developed significantly higher peak IAH. Based on these studies, the consensus recommendations in terms of fluid strategies advocate judicious fluid resuscitation in patients with or at risk for IAH/ACS and that hypertonic fluids and colloid-based fluids be considered in patients with IAH to prevent the progression to secondary ACS.[4]

Many patients at risk for IAH/ACS have associated gastrointestinal ileus.[1,2] Fluid and air within the hollow viscera can increase IAP and increase the risk of developing IAH/ACS. Drainage of fluid either nasogastrically or rectally or endoscopic decompression can decrease IAP in patients with IAH.[17]

The use of prokinetic agents also appears to hold promise, particularly in patients with colonic pseudo-obstruction by decreasing the luminal size.[38] IAH represents an absolute contraindication to the institution of enteral feeding, as enteral nutrition increases intestinal oxygen consumption and undigested feed can lead to gas formation by bacterial fermentation, leading to a further increase in IAP.

Early institution of renal replacement therapy either by intermittent dialysis or continuous haemofiltration/ultrafiltration with fluid removal in patients with oliguria or anuria has been reported and prevents the progression to secondary ACS.[39] Diuretic therapy in combination with colloid can be considered to mobilise oedema fluid once the patient is haemodynamically stable.[4]

A relatively new and minimally invasive technique to decrease IAP has been recommended to treat IAH or secondary ACS due to free intra-abdominal fluid, air or blood.[40-42] Performed under ultrasound or computed tomography guidance, percutaneous decompression appears to be effective and avoids the need for surgical decompression in appropriate patients.

Abdominal decompression

In patients with IAH refractory to medical management or organ dysfunction/failure, surgical decompression remains the mainstay of treatment and may be life-saving.[43-45] It must be emphasised that delays in surgical decompression and ignoring persistently raised IAP is associated with increased mortality.[9] Even presumptive decompression or leaving the abdomen open in patients at risk for developing IAH/ACS appears to improve survival.

Temporary abdominal closure (TAC) is required to protect the abdominal contents after surgical decompression. Various techniques have been described, e.g. towel clips, 'vacuum pack closure', 'Bogota bag', Wittmann pouch and vacuum-assisted closure.[46-49] All of these techniques involve encasing exposed bowel to create an air-tight seal but allowing for intra-abdominal volume to increase. It is important to a have a system that allows for drainage of peritoneal fluid, and the modified sandwich-vacuum pack technique developed by the Groote Schuur Trauma Unit functions well in our ICU.[46]

If decompressed early, before the development of organ failure, most patients tolerate primary fascial closure within 5 - 7 days. Those who remain critically ill with loss of abdominal compliance, require split-thickness skin grafting or primary fascial closure 9 - 12 months later. Cutaneous advancement flap allows earlier fascial closure.[9]

Conclusion

ACS is a condition that has in the past gone unrecognised as a major factor contributing to poor outcomes in critical illness. In the past decade major advances have been made in understanding the pathophysiology of this condition and in the early detection of patients at risk of developing IAH/ACS. Frequent measurement and a heightened awareness of the clinical signs of raised IAP will ensure prompt recognition of IAH and early intervention to ensure improved patient outcome.

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