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.

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

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. 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.

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. 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. Target APP values may be achieved by judicious fluid management and the use of vasoactive agents. Vasoconstrictors, however, may further decrease splanchnic perfusion. The current recommendations suggest targeting an APP of 50 - 60 mmHg.

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.

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. 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. McNelis et al. showed that excessive fluid resuscitation in non-trauma surgical patients was an independent risk factor for ACS. Balogh et al. retrospectively looked at trauma patients resuscitated to ‘supranormal’ end-points (i.e. oxygen delivery index of 500 - 600 ml/min/m²) 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 H2O) in this group of patients.23 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.14 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.39 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.14 Diuretic therapy in combination with colloid can be considered to mobilize 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.8 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.46-48 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 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.66

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 unrecognized 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.

3. Malbrain ML, De laet I, Cheatham M. Consensus conference definitions and recommendations on intrathoracic hypertension (IHH) and the abdominal compartment syndrome (ACS) – the long road to the final publications, how did we get there? Acta Clin Belg Suppl. 2007; (1): 44-58.


