Abdominal compartment syndrome

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Abstract

Abdominal compartment syndrome (ACS) is a devastating condition for the critically unwell patient. Initially described as solely affecting surgical patients, ACS is now also recognized in the medical intensive care setting. Without prompt and definitive treatment mortality rates approach 70% as multi-organ failure develops. Over the past decade our understanding, recognition and management of ACS has evolved. The World Society of Abdominal Compartment Syndrome published updated guidelines in 2013 to draw consensus and improve patient outcomes. ACS is the end sequela of raised intra-abdominal pressure (IAP), defined as a sustained IAP >20 mmHg with or without an abdominal perfusion pressure <60 mmHg and associated with new organ dysfunction. Intravesical measuring of IAP is the gold standard diagnostic technique. Surgical decompressive laparotomy and open abdomen with temporary abdominal closure measures is the definitive treatment. This article summarizes the updated consensus definitions, pathophysiology, diagnostic investigation and management to help the junior surgical trainee faced with ACS.

Keywords Abdominal compartment syndrome (ACS); abdominal perfusion pressure (APP); decompressive laparotomy; intra-abdominal hypertension (IAH); intra-abdominal pressure (IAP); open abdomen

Compartment syndrome is a surgical emergency which is often incorrectly attributed to solely affecting the limbs. It is important to recognize that the abdomen too is a confined cavity within which compartment syndrome can manifest.

It is only since 1989 that abdominal compartment syndrome (ACS) has sparked interest amongst the medical community.[1] Historically, it was considered an epiphenomenon of major abdominal trauma. Our knowledge of ACS has since evolved. ACS is now recognized to manifest in a host of surgical and non-surgical conditions including pancreatitis, burns, sepsis and following aggressive fluid resuscitation.[2] Early recognition and definitive management are crucial in improving outcomes. The mortality rate from untreated ACS is alarming, approaching 70%.[3,4] It contributes to multi-organ failure in those already critically unwell; with ACS being an independent predictor of poor patient outcome.[1,3] Consensus around its management has been somewhat elusive. In 2004 the World Society of Abdominal Compartment Syndrome (WSACS) standardized the terminology and clinical management of ACS, with their most recent update being published in 2013.[5] The aim of this article is to provide an overview of the pathophysiology, clinical presentation and management of ACS.

Definitions

The concept of intra-abdominal pressure (IAP) and its clinical relevance was first reported by Marey and Burt who described the respiratory effects caused by elevated IAP.[3] It was a decade later in 1989 that the term ACS emerged. This followed Fietman's et al. observation of four patients who had undergone open repair of ruptured abdominal aortic aneurysms and postoperatively developed increased ventilatory pressures, decreased urinary output and significant abdominal distension in the absence of intra-abdominal bleeding.[1,3] Since then, explicit definitions have been published by the WSACS to facilitate consensus development with the aim of improving patient outcomes[5] (Box 1).

The IAP is the steady-state pressure within the abdominal cavity. It is not static, varying with respiration (increasing with inspiration, and decreasing with expiration) and the intra-abdominal volume.[1–7] IAP and intra-abdominal volume have a synergistic relationship. As the intra-abdominal volume increases, whether it is occupied by solid organs, pathological masses or fluid, the compliance of the abdominal cavity reduces and causes the IAP to increase. The normal IAP is 0–5 mmHg.[1–3,5,7] This increases to 5–7 mmHg in the critically unwell.[3,6,7]

The abdominal perfusion pressure (APP) is analogous to the cerebral perfusion pressure.[2] It quantifies the blood flow pressure within the abdomen. It is defined as the difference between the mean arterial pressure and the IAP.[1–7] APP has been identified as a surrogate marker of patient mortality,[7] with a pressure of less than 60 mmHg thought to represent an important threshold.[1,2,6] Current guidelines, however, refute the use of APP as an endpoint for resuscitation.[5]

Intra-abdominal hypertension (IAH) and ACS are the sequelae of sustained pathologically elevated IAP. IAH is defined as an IAP equal to or greater than 12 mmHg. IAH is further subcategorized into four groups based on increasing pressures (Grade 1: IAP 12–15 mmHg, Grade 2: IAP 16–20 mmHg, Grade 3: IAP 21–25 mmHg, Grade 4: IAP >25 mmHg).[3,5,7]

ACS is a constellation of signs and symptoms of end organ dysfunction or failure when the IAP is greater than 20 mmHg, with or without an APP less than 60 mmHg.[1–6] ACS can be further categorized into primary, secondary and recurrent (Box 1 and Table 1). Primary ACS arises from injury or disease within the abdominopelvic cavity. Secondary ACS manifests from conditions that originate from outside of the abdominopelvic cavity. Recurrent ACS is the persistence of ACS despite radiological or surgical management.[3,5,7] Irrespective of the subtype, the pathophysiology and clinical approach to ACS remains the same.

Measuring intra-abdominal pressure

Clinical examination of the abdomen has a poor sensitivity and positive predictive value (40–60%) for diagnosing raised IAP.[6,8]

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The World Society of Abdominal Compartment Syndrome consensus definitions[5]

Intra-abdominal pressure (IAP)
- Steady-state pressure concealed within the abdominal cavity
- Normal IAP <5 mmHg
- IAP in the critically ill: 5–7 mmHg

Intra-abdominal hypertension (IAH)
- Persistently elevated IAP ≥ 12 mmHg
- Subcategorized into four grades of increasing severity:
  - Grade 1: IAP = 12–15 mmHg
  - Grade 2: IAP = 16–20 mmHg
  - Grade 3: IAP = 21–25 mmHg
  - Grade 4: IAP >25 mmHg

Abdominal perfusion pressure (APP)
- APP = MAP[a] − IAP
- Analogous to cerebral perfusion pressure

Abdominal compartment syndrome (ACS)
- Constellation of signs and symptoms due to elevated IAP >20 mmHg (with or without APP <60 mmHg) within the abdominopelvic cavity with new organ dysfunction or failure

Primary ACS
- Results from an injury/disease in the abdominopelvic region requiring surgical or interventional radiological intervention

Secondary ACS
- Results from conditions that do not originate in the abdominopelvic region, e.g. hypothermia

Recurrent ACS
- Recurring ACS despite medical/radiological/surgical treatment

[a]MAP, mean arterial pressure.

Box 1

Accurately assessing the IAP is dependent upon using a reliable and reproducible method. Various techniques for directly and indirectly measuring IAP, both intermittently and continuously, have been described in the literature.[2,3,5] Direct measurement involves the introduction of an intra-peritoneal catheter into the abdomen. Indirect measurements of IAP are obtained via the insertion of catheters into the bladder, rectum, uterus or inferior vena cava. Comparatively, indirect methods yield a similar diagnostic accuracy as direct measures.[7] Thus, due to its cost-effectiveness, feasibility and non-invasiveness, the gold standard for measuring IAP is intravesically.[5] Disadvantages of this technique include an inability to continuously measure IAP and its contraindication in bladder trauma, neuropathic bladders and when pelvic packs are in situ.[8]

Techniques for measuring the IAP via the intravesical route also vary.[2,3] Figure 1 illustrates a simple and commonly utilized method. Irrespective of the technique, it is essential that the patient is fully supine with maximal abdominal relaxation, and that measurements are taken at the end of expiration with the transducer placed at the mid-axillary level and expressed in mmHg.[5]

Epidemiology and risk factors

Quantifying the incidence and prevalence of ACS using historic data has been hindered by the inconsistencies in defining IAH and ACS, the varied patient populations studied and the trend for aggressive fluid resuscitation in critically unwell patients.[3,7] As a result, observational studies vastly vary with regards to their reported incidences and prevalences of ACS.[3] In any given mixed surgical-medical intensive care unit (ICU) patient population, the incidence of IAH is thought to range from 25% to 56%.[2,9,10] Of these, up to 10% have ACS.[2,10]

ACS is widely recognized as a detrimental complication of major trauma. The paradigm shift in managing trauma over the last few decades, with damage control surgery, leaving the abdomen open, and moderating fluid resuscitation, has coincided with a decrease in the incidence of primary ACS.[7,9] Severe trauma, burns and pancreatitis patients are also particularly susceptible to the development of ACS.[7] In relative terms, however, ACS is rare.

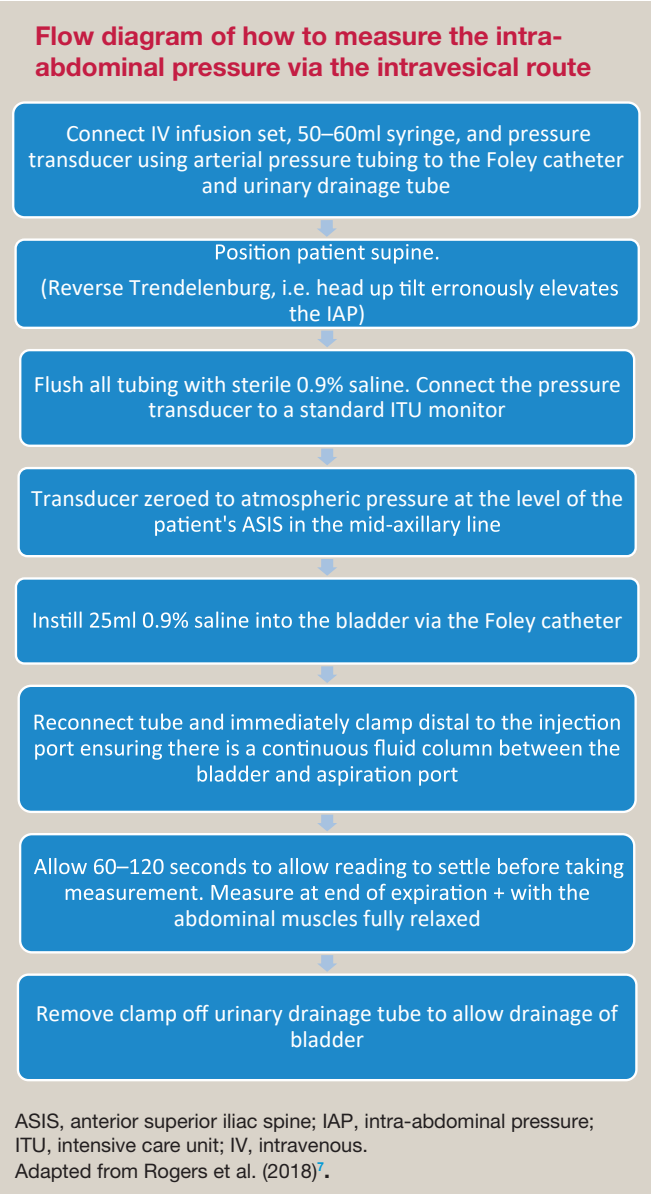
Despite the true epidemiology of ACS being difficult to ascertain, there is convincing evidence that ACS is an independent predictor of patient outcomes. IAH and ACS are both risk factors for increased morbidity and mortality in ICU patients.[2,3,10]

The aetiology of abdominal compartment syndrome

Primary ACS	Secondary ACS	Recurrent ACS
Abdominal trauma	Massive transfusion	Any primary or secondary cause
Damage-control laparotomy	Sepsis	
Pancreatitis	Hypothermia	
Intra/retroperitoneal haemorrhage	Coagulation	
Pneumoperitoneum	Major burns	
GI obstruction	Dialysis	
Ruptured AAA	Aggressive fluid resuscitation	
Peritonitis		
Liver dysfunction with ascites		
Abdominal packing		
Ileus		

ACS, abdominal compartment syndrome; AAA, abdominal aortic aneurysm; GI, gastrointestinal.

Table 1



ASIS, anterior superior iliac spine; IAP, intra-abdominal pressure; ITU, intensive care unit; IV, intravenous.
Adapted from Rogers et al. (2018)[7].

Figure 1

Pathophysiology

The pathophysiology of ACS is multi-factorial affecting most organs (Figure 2). Impaired capillary blood flow is the orchestrator of a sequelae of events resulting in cardiorespiratory, renal, neurological and gastrointestinal dysfunction. Extrinsic compression on abdominal vessels and viscera restricts arterial inflow and venous return. Anaerobic respiration and lactic acidosis follow, in response to cellular hypoperfusion. Tissue ischaemia ensues and if left untreated multi-organ failure and death follow. It has been proposed that an IAP of 15 mmHg is the threshold for the development of circulatory disturbances.[11] The renal and pulmonary systems are particularly vulnerable to the effects of ACS with signs of their dysfunction often manifesting first.

Cardiovascular

ACS has profound effects on the cardiovascular system. It reduces cardiac output and venous return, whilst also increasing the systemic vascular resistance. This is a consequence of direct compression of the central vessels and heart. As the IAP rises, there is progressive cephalic displacement of the diaphragm, which in turn increases the intra-thoracic pressure (ITP). This abdomino-thoracic transmission means that approximately 50% of the IAP is transferred to the thorax. The external pressure exerted onto the heart reduces ventricular compliance and contractibility, thereby reducing end diastolic volume.[3,6,9]

Simultaneously, the elevated IAP and ITP compress the splanchnic vessels, portal vein and inferior vena cava, reducing venous return to the heart. To maintain homeostasis, systemic vascular resistance increases and the renin–angiotensin–aldosterone system is activated.[6,8] Cardiac output is reduced due to a reduction in stroke volume caused by the reduced preload and increased afterload.[3,6,9] Venous stasis resulting from the reduced venous return also increases the risk of venous thromboembolic events. Prophylactic anticoagulation is therefore particularly important in ACS to reduce the occurrence of deep vein thromboses and/or pulmonary embolisms.[8]

Respiratory

The pulmonary insult caused by ACS presents clinically as hypoxia, hypercapnia and elevated ventilatory pressures. This arises secondary to the mechanical effects of an elevated IAP. Diaphragmatic splinting from the raised IAP and ITP (abdomino-thoracic transmission causes ITP to rise in response to elevated IAP) has a restrictive effect on the lungs, reducing their functional capacity. Basal atelectasis, reduced lung compliance, increased pulmonary dead space and increased ventilatory pressures occur. This produces a ventilation–perfusion mismatch with the resulting hypoxia and hypercapnia stimulating vasoconstriction, further exacerbating the mismatch. Increased ventilatory pressures are thus required to compensate for the pulmonary dysfunction and to maintain adequate oxygenation and ventilation. This can result in barotrauma, and so the cycle of pulmonary insults continues.[2,3,6–9]

Neurological

The neurological effects seen in ACS, as with other organ systems, occur in a graded fashion. The overarching effect is that intracranial pressure increases, whilst cerebral perfusion pressure (cerebral perfusion pressure = mean arterial pressure − intracranial pressure) reduces. Central venous outflow obstruction, from the external pressure exerted on the intra-thoracic vasculature as the ITP increases in response to the increased IAP, impedes cerebral venous return. The venous stasis within the internal jugular vein obstructs the venous drainage of the brain, increasing the intracranial pressure. This functional obstruction to cerebral venous outflow therefore contributes to a reduction in the cerebral perfusion pressure. In addition, the reduction in systemic blood pressure caused by reduced cardiac output also contributes to the reduction in cerebral perfusion pressure.[2,3,6–9]

An alternative mechanism proposed to explain the neurological effects seen is the diminished blood flow to the lumbar venous plexus as a result of the reduced blood flow through the inferior vena cava. Cerebral spinal fluid (CSF) absorption at the lumbar cistern is subsequently reduced, causing increased pressure within the CSF to be propagated up the spinal canal to the brain, resulting in further impediment to cerebral venous outflow.[6]

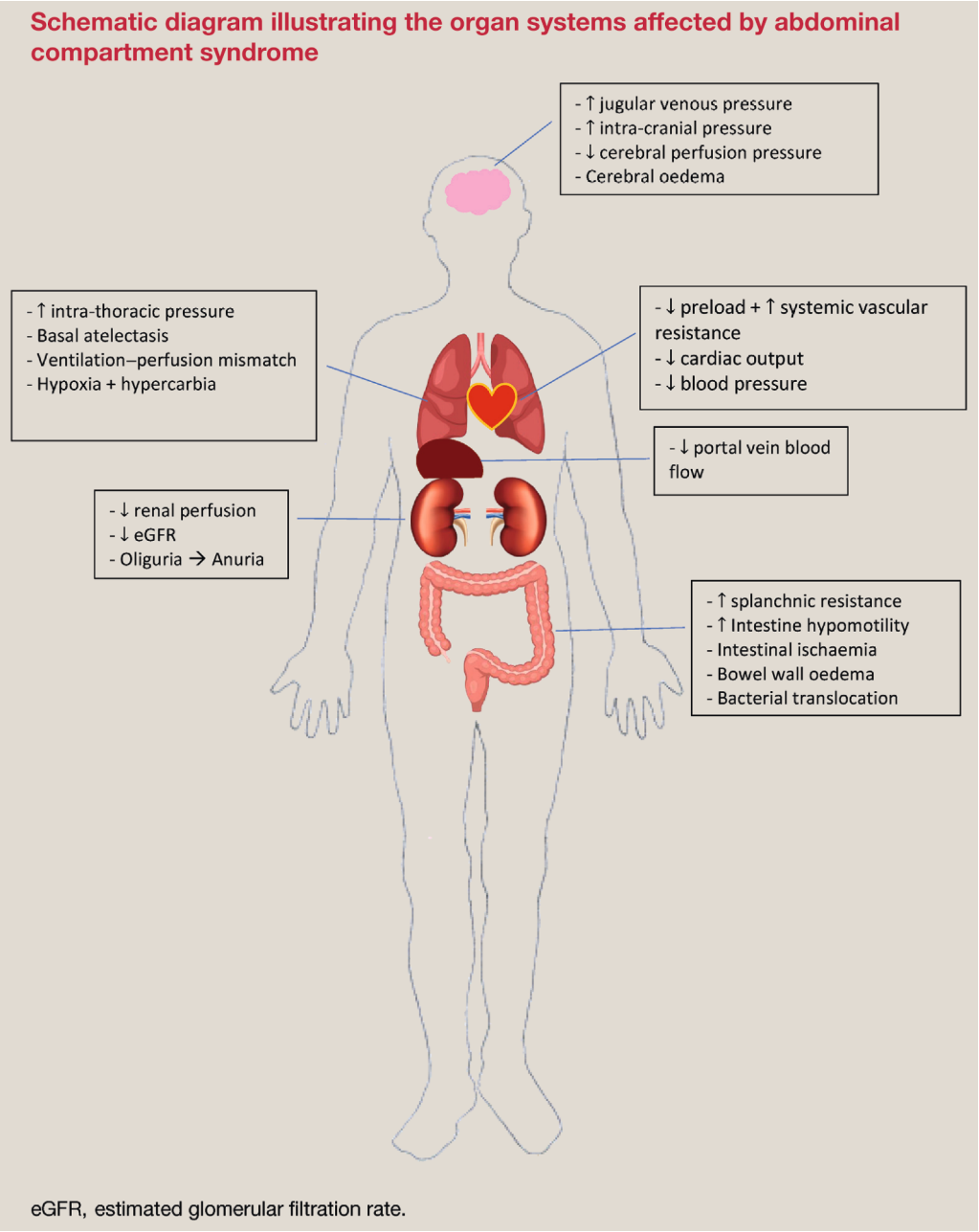


Figure 2

Renal

Oliguria progressing to anuria is often the first sign of ACS.[2,3,6,7] Reduction in the glomerular filtration rate (GFR) and urinary output arises from a combination of: decreased arterial inflow secondary to reduced cardiac output; venous outflow obstruction; compressed renal parenchyma, and activation of a cascade of hormonal and enzymic reactions.[3] In response to reduced renal blood flow, the juxtaglomerular cells are stimulated to secrete renin, activating the renin–angiotensin–aldosterone system, with the net result being water and sodium retention in an attempt to increase the blood pressure so as to increase renal arterial blood flow. Catecholamines are also released in response to reduced renal blood flow. Vasoconstriction results, which further reduces the glomerular filtration rate and renal blood flow, thus exacerbating an already compromised renal function.[2,3,6–8]

Gastrointestinal

Extrinsic pressure on the splanchnic and mesenteric vessels and on the intra- and retro-peritoneal organs reduces venous return and precipitates venous congestion. The resulting intestinal hypomotility causes intra- and extra-luminal fluid accumulation. Intestinal wall oedema exerts further pressure to an already elevated IAP. Tissue ischaemia and metabolic acidosis results. Bacterial translocation precipitates intra-abdominal sepsis, which in addition raises IAP and a vicious cycle is created.[2,3,6,7,9]

Blood flow within the hepatic artery, vein and portal venous system is also reduced. To compensate, flow through portosystemic collaterals increases. This results in hypoperfusion of the liver, causing reduced metabolite clearance, altered glucose metabolism and reduced energy substrate production.[3,6,11]

Other

Abdominal wall distensibility is limited, and its perfusion diminishes as the IAP increases. Wound healing therefore becomes compromised and fascial integrity progressively reduces. As IAP continues to increase, wound infections and dehiscence may occur.[6]

Management

Prevention where possible is crucial for the management of ACS. Equally, delays in its management increases the risk of mortality.[6] Despite definitive treatment, mortality rates approach 50%; nearing to 100% if left untreated.[9] In 2013, the WSACS published recommendations for the management of ACS. Whilst these recommendations provide a treatment standard, they are based on low-quality evidence.[5,9] As with the management of most surgical conditions, a medical or surgical treatment approach to managing ACS can be used. The aim is to reduce the IAP before irreversible organ damage develops.

Medical management of ACS

Medical management of ACS focusses on reducing the intra-abdominal volume, and/or increasing the abdominal wall compliance.[9] Broadly speaking there are five treatment arms which can be utilized simultaneously to achieve this: 1) improve abdominal wall compliance; 2) evacuate intra-luminal contents; 3) evacuate abdominal fluid collections and other space occupying lesions; 4) optimize fluid administration; and 5) optimize systemic and regional perfusion in order to optimize organ support.[1,2,5,9,11] Figure 3 illustrates this.

The contribution of pain and anxiety to an increased abdominal wall tone is often forgotten. Whilst pain and anxiety can be difficult to assess in intubated patients, it is important to recognize that muscle tension can be relieved by administrating adequate analgesia, anxiolytics and muscle relaxants.[1,5] Epidurals should frequently be used. Neuromuscular blockade with the of use of neostigmine has been found to improve abdominal wall compliance. Its unpleasant systemic side effects, however, limits its use. These interventions should be used as temporizing measures whilst definitive management is sought.[1,5,7]

Ileus is common in ACS. Gastric decompression with a nasogastric tube is advocated. Rectal cannula is reserved for pronounced colonic distension after conservative measures (e.g. phosphate enemas, correction of electrolyte abnormalities, avoiding drugs which impair gut motility such as opioids, and use of prokinetic agents such as metoclopramide or erythromycin) have been used to stimulate colonic motility. If the above non-invasive measures fail to relieve the ileus, neuromuscular blockade with neostigmine and/or endoscopic decompression should be considered.[1,2,5,7] Whilst restricting enteral intake may be required, restarting enteral feeding at the earliest opportunity is advocated to help maintain bowel mucosal integrity, which in turn reduces the alteration of bowel flora and bacterial translocation.[1] Aggressive fluid resuscitation is practised in the initial stages of disease management as haemodynamic stability is sought. Depending on the disease process, however, third space losses (as seen in acute pancreatitis) can lead to the intra-abdominal accumulation of fluid. Pharmacological diuresis with furosemide in conjunction with renal replacement therapy or paracentesis may be beneficial in reducing the IAP. Whilst the evidence for this is poor, consideration of their use is recommended as a treatment of last resort.[5,7]

Surgical management of ACS

Surgical decompressive laparotomy and temporary maintenance of an open abdomen is the only definitive treatment for ACS. A midline incision from the xiphoid sternum to the pubic symphysis, extending through the rectus sheath and peritoneum, is made. The immediate release of pressure causes a dramatic and instantaneous improvement in visceral perfusion and organ function.[2,7,9] The IAP does not fully normalize immediately but a significant reduction is seen.[12] Whilst it has the potential to be lifesaving, decompressive laparotomy and an open abdomen are associated with significant morbidity and should only be used when medical management has failed.[7] Reperfusion injury, massive protein losses, hypercatabolic state, fascial retraction, large ventral hernia, frozen abdomen, infection and enterocutaneous fistulae can all ensue, complicating an already fragile clinical scenario. The risk and severity of complications exponentiate the longer the abdomen is left open.[7,12] Continuous patient monitoring is necessary, particularly at the time of decompression. The sudden reduction in IAP causes a marked release of anaerobic metabolites into the circulation, which have accumulated as a by-product of tissue hypoperfusion and venous stasis. This consequent reperfusion injury can be more catastrophic to the patient than the elevated IAP, with cardiac arrhythmias and even death resulting.[2,9] Rapidly infusing intravenously crystalloid fluids as the scalpel penetrates through and decompresses the abdomen can limit the systemic reperfusion injury and maintain haemodynamic stability.[11] Prophylactic antibiotics are not recommended.[13]

The abdomen should be left 'open' following surgical decompression. Temporary abdominal closure techniques are employed to allow the continued release of excess pressure. When the IAP normalizes, healing occurs either by secondary or tertiary intention. Subsequent closure of the abdominal fascia is possible in 70–90% of patients depending on the temporary abdominal closure technique used and the duration in which the abdomen is left open. Studies advocate rapid fascial closure, ideally within 7 days, to reduce the morbidity associated with an inability to appose the rectus fascia together.[7] The WSACS recommends abdominal fascial closure to happen during the same hospital admission as that for the ACS.[5] Poor cosmesis and function due to the development of ventral hernias are frequently faced by patients who fully recover from ACS once the abdomen is closed.

Various temporary measures to facilitate abdominal closure have been reported in the literature. These include skin only closure, the Bogota bag, biological and synthetic mesh, and negative pressure therapy.[5,7,9,12] Until recently, the use of one over another was down to institution and surgeon preference. Negative pressure wound therapy using a vacuum-assisted closure (VAC) device, for example the ABTHERA system, is now

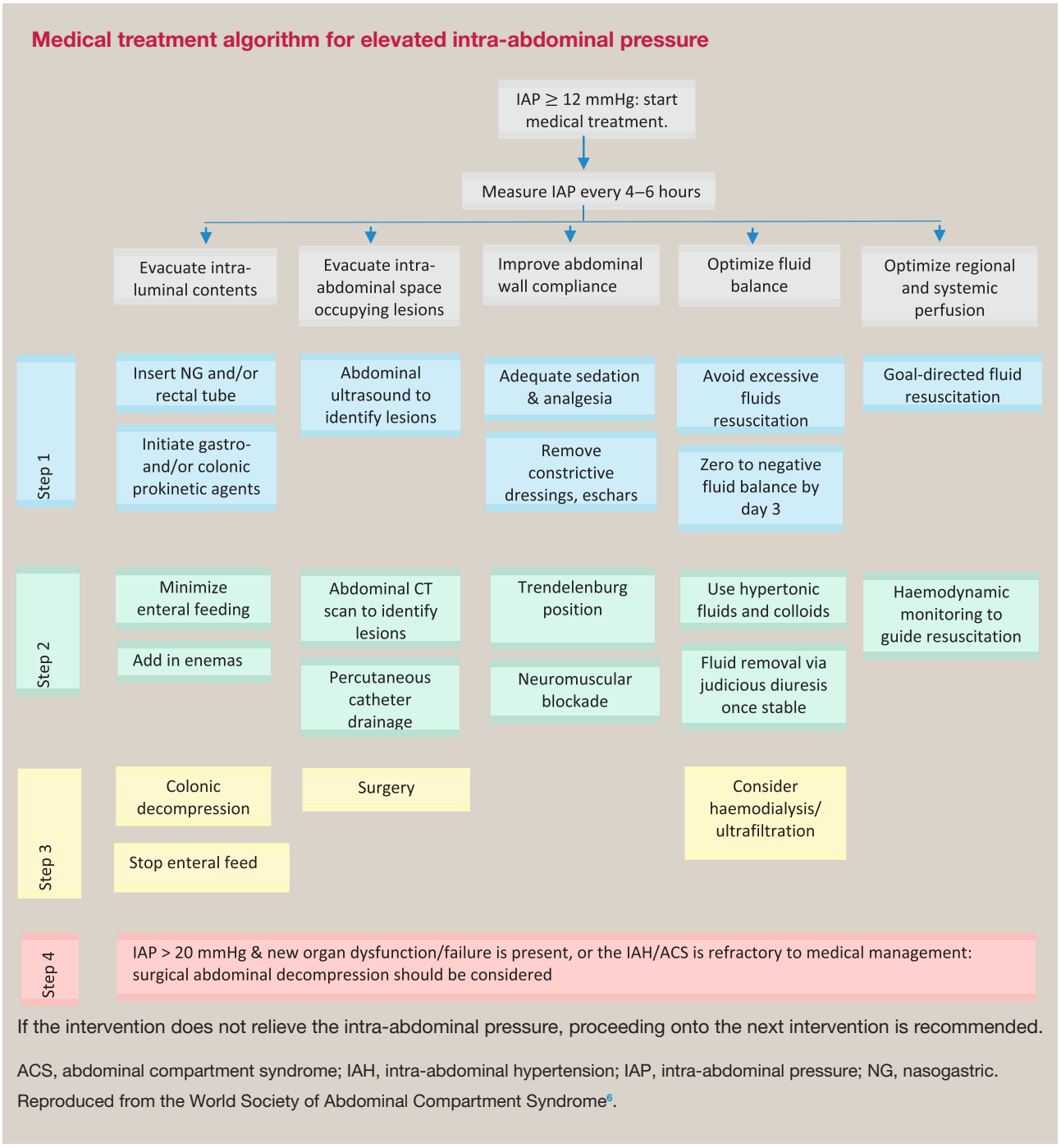


Figure 3

the gold standard technique, however its preference is based on low grade evidence.[5] A perforated plastic sheet is positioned over the abdominal viscera, with a sponge placed between the fascial edges. An airtight seal is established and pierced by a suction drain that is connected to a negative pressure pump.[7] Negative pressure stimulates the formation of granulation tissue, gradually approximates the wound edges together, removes slough and abdominal fluid and reduces the size of the wound. Particular care must be taken when using negative pressure wound therapy to prevent enterocutaneous fistulae from developing. This can be avoided through the use of low pressures and careful insertion of the fenestrated plastic sheet (which is 'bowel friendly').[2,12] Some authors have reported the use of biological meshes for abdominal closure. The WSACS, however, does not support their routine use.[5] The Bogota bag – which is crafted from a 3-litre sterile irrigation bag – is a sterile plastic sheet which is sewn to the abdominal skin or fascia. It has the advantage of being inexpensive and easily allows inspection of the bowel and abdominal contents, however managing fluid losses is problematic with this system.[9] Newer technologies such as the fasciotens have also

come to market. The fasciotens applies vertical traction to the abdominal wall musculature, preventing muscle retraction and facilitating abdominal closure.[14]

Recurrent ACS can occur despite surgical decompression if the underlying pathology responsible for the increased IAP is not addressed or if the increase in intra-abdominal volume produced by the open abdomen is inadequate to reduce the IAP. Continuous tentative patient monitoring is therefore required.[7,13]

Conclusion

A high index of suspicion for ACS should be maintained when managing critically unwell patients with abdominal distension who display elevated ventilatory pressures, oliguria and other new organ dysfunction. It is hoped that the WSACS consensus guidelines will progress research in this field and improve patient outcomes associated with this rare, but life-threatening, condition. ◆

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Practice points

- Early detection and intervention of raised intra-abdominal pressure (IAP) is crucial to improving outcomes, thus regular and accurate assessment of IAP in the critically ill patient is necessary
- Medical management is aimed at reducing intra-abdominal volume and enhancing abdominal wall compliance. Excessive fluid resuscitation should be avoided, intraluminal contents should be reduced (e.g. insertion of a nasogastric or rectal tube and the use of pro-kinetic drugs should be considered), intra-abdominal fluid collections should be drained, and adequate sedation and analgesia should be administered
- Decompressive laparotomy remains the definitive treatment for abdominal compartment syndrome (ACS) where medical management has failed, but is not without morbidity. Early abdominal wall closure should be the aim (ideally within 7 days), where the clinical picture allows
- The underlying cause of the ACS (e.g. trauma, pancreatitis, infection) must be managed in order to prevent recurrence