The Abdominal Compartment Syndrome Following Aortic Surgery

I. M. Loftus*1 and M. M. Thompson2

1Department of Surgery, Leicester University, Leicester, U.K.
2Department of Vascular Surgery, St. George’s Hospital, London, U.K.

Background: Multi-organ failure is a leading cause of death following aortic surgery, especially in the emergency setting. Intra-abdominal hypertension is an important factor in the development of multi-organ failure. Prevention, early recognition and prompt treatment of abdominal hypertension and the abdominal compartment syndrome may reduce mortality following aortic surgery.

Methods: A descriptive review of the literature from a Medline search.

Results and Conclusions: The abdominal compartment syndrome is the result of diverse physiological effects caused by increased intra-abdominal pressure. The syndrome has been most widely described in trauma victims, but occurs in patients following aortic surgery, particularly following ruptured aneurysm repair. Preventative therapy should be instituted to minimise its development in patients at risk, and monitoring of intra-abdominal pressure may allow prompt treatment of this condition.

Key Words: Abdominal compartment syndrome; Intra-abdominal hypertension; Aortic aneurysm.

Introduction

A compartment syndrome is defined as a “condition in which increased tissue pressure in a confined anatomic space, causes decreased blood flow leading to ischaemia and dysfunction” and “may lead to permanent impairment of function”.1 It is important to recognise the abdomen as a compartment with the potential to cause life threatening systemic and local manifestations. The abdominal compartment syndrome (ACS) has been described most often following trauma,2 but more recently has been recognised to occur in other surgical patients,3 the critically ill4 and patients on the intensive care unit.5

Raised intra-abdominal pressure (IAP) is defined as an abdominal pressure above atmospheric pressure. As the pressure increases, a spectrum of physiological effects is observed. This is referred to as intra-abdominal hypertension and may develop in to ACS. The ACS lacks a uniformly accepted definition though is generally identified as an abdominal pressure of greater than 20 mmHg in the presence of organ dysfunction.

In some of the early reports of ACS, raised IAP was recognised in patients following aortic surgery. However, there have been very few formal studies to investigate the effect of raised IAP in vascular patients. Multi-organ failure is a common problem following ruptured aortic aneurysm repair, and is the leading cause of post-operative mortality.6 It seems likely that raised IAP, particularly if it develops into ACS, significantly contributes to the onset of multi-organ failure. A recent large study in patients following ruptured aneurysm surgery has attempted to identify factors that contribute to raised IAP and post-operative mortality.7 It recognised the importance of forming a laparostomy early in the post-operative management of patients at risk of developing the ACS.

Method

A Medline and Pubmed search of the English language literature were performed using the terms “Abdominal compartment syndrome”, “intra-abdominal hypertension”, “abdominal pressure” and
“aortic aneurysm”. No other specific exclusion criteria were used.

Historical Perspective

The effects of raised intra-abdominal pressure (IAP) have been known for over a century. Marey (1863) and Burt (1870) hypothesised a reciprocal relationship between intra-thoracic pressure and IAP, and obtained pressure measurements in animals that supported a link between respiratory function and IAP. In 1890, Henricius demonstrated in animal models that a rise in abdominal pressure was fatal, due to impaired respiratory function. Subsequent investigations demonstrated the profound effect of IAP on organ function. Emerson associated a rise in abdominal pressure with reduced venous return to the heart, leading to cardiovascular failure, that was later shown to be exacerbated by haemorrhage. Similarly, raised IAP caused a progressive decline in urine output due to a reduction in renal plasma flow and glomerular filtration rate in parallel with increased renal venous pressure and IAP.

In 1940, Ogilvie suggested that closure of the abdomen under tension should be avoided, and described a technique using Vaseline impregnated canvas or cotton cloth sutured to the edges of the wound. He then applied pinch grafts to the granulating wound and performed delayed repair of the resulting ventral hernia. Baggot supported this hypothesis in 1951, associating a high post-operative mortality in infants with congenital abdominal wall defects, and adults with acquired defects, with closure of the abdomen under tension. He ascribed this to respiratory dysfunction and recommended delayed abdominal closure, with the temporary placing of loose sterile dressings over the wound.

Despite these early reports, the importance of IAP was largely ignored until a resurgence of interest in the 1980s and 1990s. Kron and co-workers coined the term ACS. They observed the effect of abdominal decompression in three of four patients with oliguria and abdominal distension following aneurysm repair. All three improved rapidly and made uneventful recoveries. The fourth patient was not de-compressed and died from renal failure. In an animal model they associated an early rise in abdominal pressure with impaired renal function independent of blood pressure or cardiac output. Subsequently, they described the pathophysiological effects of IAP in the clinical setting and used pressure measurements as an indication for abdominal decompression. This group then measured IAP using a bladder catheter, and associated an acute elevation in IAP above 30 mmHg with oliguria in 11 patients. Re-exploration and de-compression resulted in immediate diuresis in seven patients, but the four treated conservatively all developed renal failure and died.

Pathogenesis

Raised IAP can be classified as acute or chronic. The acute ACS is usually secondary to a rapid rise in intra-abdominal volume. Chronic increases in intra-abdominal volume, such as with ascites and morbid obesity, tend to lead to a more gradual increase in IAP which is compensated for by increased abdominal wall compliance. Physiological dysfunction is rarely seen with chronic increases in IAP, though the acute ACS may occur due to a rapid increase in chronically high IAP.

The acute ACS develops in a spectrum of surgical and non-surgical patients, and can be further classified with regard to the origin of the increased pressure. The increase in pressure may be due to increased intra-abdominal volume, which may be retroperitoneal or intraperitoneal, or due to problems of the abdominal wall. Retroperitoneal causes include pancreatitis, traumatic retroperitoneal or pelvic bleeding and sepsis. Intraperitoneal causes are more common, particularly traumatic or post-operative haemorrhage, bowel distension, visceral oedema and peritonitis. Laparoscopy has also been shown to have physiological effects, particularly cardio-respiratory and renal, though is not recognised as a direct cause of ACS.

External compression of abdominal contents can have the same effect upon IAP and the development of ACS. This is most commonly a consequence of tight abdominal closures, though is also recognised in burn patients with abdominal wall eschars, from the use of pneumatic shock garments, and more particularly from the closure of abdominal wall defects such as gastroschisis and large incisional hernias.

In vascular patients the ACS may occur following free intraperitoneal or contained retroperitoneal aneurysm rupture due to increased intra-abdominal volume, though the development of ACS is usually multifactorial. The rise in intra-abdominal pressure is exacerbated by the degree of shock. Massive fluid resuscitation tends to increase intra-abdominal and retroperitoneal volume, both visceral and vascular, due to increased extracellular volume. A number of factors combine to increase extracellular volume, including capillary leak, ischaemia-reperfusion injury and the production/release of oxygen free radicals and vasoactive peptides. Other factors may contribute to the increased pressure, in particular increased thoracic pressure associated with
the use of positive pressure ventilation and high positive end expiratory pressure. There is a well-defined increase in respiratory workload in ACS, associated with a decrease in chest wall compliance. A relative hypervolaemia from fluid resuscitation causes abdominal wall oedema and ischaemia, leading to a loss of abdominal wall compliance. Massive resuscitation alone in the absence of abdominal pathology may be sufficient to induce a “secondary” ACS. The resultant increased abdominal pressure tends to exacerbate the situation and a vicious cycle is formed leading to multi-organ failure.

Multi-organ failure is a common cause of death following aneurysm surgery, especially ruptured aneurysm repair. This is probably secondary to a widespread “systemic inflammatory response syndrome” mediated by cytokines. Aortic cross clamping causes an ischaemia-reperfusion injury with widespread activation of inflammatory pathways. This is exacerbated by the further insults of haemorrhage, acidosis and blood transfusions. Various pro-inflammatory cytokines are associated with the development of the classical signs of inflammation, including IL-1, IL-6 and TNF-α. There is no evidence of a direct link between rising intra-abdominal pressure and cytokine levels though there is an association between increased mortality following aneurysm repair and pro-inflammatory cytokine levels. Inadequate organ perfusion is likely to be responsible for many of the sequelae of multi-organ failure.

**Measurement of Intra-abdominal Pressure**

Measurement of IAP may be performed directly or indirectly. Direct pressure measurement may be achieved by the placement of an intra-abdominal catheter during laparoscopy or laparotomy. This is not generally practical in the clinical setting, as monitoring may need to continue for several days. In aortic surgery there are concerns regarding potential introduction of infection into the peritoneal cavity by prolonged use of direct pressure catheters. Indirect measures include intra-vesical and intra-gastric catheters, rectal catheter and femoral venous cannulation.

**Urinary bladder pressure**

Kron and co-workers first described the measurement of urinary bladder pressure. The basis for their technique was the recognition that the bladder behaves as a passive diaphragm when the intra-vesical volume is between 50 and 100 ml. The bladder is drained and then filled with 50–100 ml of sterile saline. The drainage tubing is clamped beyond the aspiration port, and a needle used to connect the aspiration port to a pressure transducer, using the symphysis pubis as zero (Fig. 1). If a transducer is unavailable, a water manometer can be used, converting the reading to mmHg (1.3 cm water = 1 mmHg). In Kron’s original description of the technique, the pressure was compared with that measured through an intra-peritoneal dialysis catheter. There was a close correlation in measurements for pressures between 5 and 50 mmHg. This has been confirmed by several further studies. Recently, Fusco et al. compared directly the IAP at laparoscopy up to 25 mmHg, with bladder pressure in 37 patients. Intravesical pressure closely approximated IAP to within 3 mmHg, supporting its accuracy in the clinical setting. Furthermore, the technique is
minimally invasive and easy to use. Intra-vesical pressure measurement is seen as the “gold standard” in monitoring IAP, though falsely high readings may be obtained in patients with chronically high IAP or adhesions.33

**Gastric pressure**

An alternative indirect, non-invasive technique has been described using intra-gastric pressure measurements taken from an indwelling nasogastric tube after instilling 50–100 ml of saline into the stomach.34 The mid axillary line is used as zero with the tube connected to a pressure transducer or a manometer. Animal models have shown a poor correlation between gastric pressure and true IAP,35 but clinical studies have shown a good correlation with bladder pressure, to within 2 mmHg.36 However, few of the measurements were taken at the high pressures seen in ACS. Of the 25 patients studied, only two recorded IAP measurements of >20 mmHg. Bladder pressure monitoring has therefore gained a wider clinical acceptance than gastric pressure.

**Other methods**

Catheterisation of the femoral vein allows measurement of the pressure in the vena cava. This correlates with IAP in animal models35 but has not been studied in patients. This method is invasive and carries the risk of infection and venous thrombosis.

Common CT scan findings have been described, including increased anteroposterior to transverse abdominal diameter and tense retroperitoneal infiltration.37 Narrowing of the supra-hepatic inferior vena cava has also been described both on CT and ultrasound.38 However these were small studies and the role of CT requires further evaluation.

**Pathophysiology**

**Definitions**

The normal intra-abdominal pressure is recognised to be atmospheric or sub-atmospheric.13 The pressure varies inversely with intra-thoracic pressure during spontaneous ventilation. Raised IAP is defined as any abdominal pressure above atmospheric pressure. With sequential increases in IAP, physiological effects are observed. This is often referred to as intra-abdominal hypertension (IAH). There is no standard definition of the pressure level with which IAH is associated, though it is generally described as a level between 10 and 20 mmHg. The ACS has been defined as “a constellation of the physiological sequelae of IAH”, whereby IAH is accompanied by manifestations of organ dysfunction.39

IAH affects multiple organ systems but in particular the respiratory, cardiovascular, renal, gastrointestinal and central nervous systems. The pressure threshold above which each physiological system is affected is variable. The gastrointestinal system is affected at levels as low as 10 mmHg, while the central nervous system is unaffected until pressures of >20 mmHg. Therefore, each system will be discussed separately, with the effects summarised in Table 1.

**Cardiovascular system**

Increasing IAP above 20 mmHg progressively reduces cardiac output due to reduced preload and increased afterload on the heart.40 A recent study has shown that the reduction in cardiac output may start at a pressure as low as 15 mmHg, though at this level there is no effect on tissue blood flow.41 The main determinant of the reduced cardiac output is a reduction in venous return to the heart, related to increased resistance to blood flow in the portal vein and vena cava. The maximum point of resistance is the diaphragmatic hiatus.42 IAP also leads to increased intra-thoracic pressure,

<table>
<thead>
<tr>
<th>System</th>
<th>IAP</th>
<th>10–15 mmHg</th>
<th>16–25 mmHg</th>
<th>&gt; 25 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td></td>
<td>Reduced preload and increased afterload. Reduced cardiac output</td>
<td>Reduced contractility. Gross reduction in cardiac output</td>
<td></td>
</tr>
<tr>
<td>Renal</td>
<td></td>
<td>Oliguria</td>
<td>Marked intestinal and hepatic ischaemia</td>
<td>Anuria</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Slight intestinal and hepatic ischaemia</td>
<td>Minimal effect</td>
<td>Bowel infarction</td>
<td>Hepatic failure</td>
</tr>
<tr>
<td>CNS</td>
<td></td>
<td></td>
<td></td>
<td>Increased intracranial pressure</td>
</tr>
</tbody>
</table>
further contributing to the reduced flow in the inferior and superior vena cava, and causing a degree of cardiac compression. Cardiac tamponade decreases the end diastolic ventricular volume, with the stroke volume further reduced by an increase in systemic vascular resistance caused by arteriolar vasoconstriction. There is an increase in the heart rate and contractility which only partially compensates.\textsuperscript{31,43}

Any degree of hypovolaemia will exacerbate the diminished cardiac output, reducing the preload further. Volume expansion will tend to minimise the process and some advocate volume loading prior to abdominal decompression if required.\textsuperscript{23} However, monitoring fluid balance can become difficult since the increase in intrathoracic pressure tends to increase central venous and pulmonary arterial wedge pressures, giving an erroneous reading of central filling. In a porcine model, a raised IAP of 25 mmHg significantly decreased the cardiac index with elevation of the wedge, pulmonary arterial and pleural pressures. However, the transarterial (wedge pressure–pleural pressure) decreased with increasing IAP, and fluid resuscitation returned the cardiac index to normal.\textsuperscript{31} Cardiovascular parameters therefore require careful interpretation in patients with ACS. Anaesthetic agents can also exacerbate the cardiovascular dysfunction, along with positive end-expiratory pressure ventilation.\textsuperscript{44}

Elevation in the central venous pressure has a secondary effect upon blood pressure, tending to lead to a systemic hypertension.\textsuperscript{45} This is thought to be due to a reduction in cerebral venous drainage, subsequent increased intracranial pressure and a central nervous system mediated response to raise systemic blood pressure.\textsuperscript{46}

\textbf{Respiratory system}

Respiratory dysfunction has been shown to occur at intraabdominal pressures as low as 15 mmHg, with progressive deterioration at higher pressures.\textsuperscript{31} There are a number of mechanisms involved. The increase in IAP causes the diaphragm to rise, reducing the intrathoracic volume. This leads to a decrease in lung capacity and compliance. The rise in intrathoracic pressure increases pulmonary vascular resistance, further contributing to the reduction in gas exchange. Recent work suggests that, as well as alterations in lung compliance, there is a decrease in chest wall compliance.\textsuperscript{27} With increasing IAP in the ACS, respiratory dysfunction is manifest by progressive hypventilation and respiratory failure. Work in a porcine model suggested that this occurred earlier when the subject had suffered from a haemorrhagic insult despite resuscitation.\textsuperscript{47} Abdominal decompression has been shown to rapidly reverse the respiratory failure in ACS.\textsuperscript{13}

\textbf{Renal dysfunction}

Renal dysfunction is a well-recognised feature of the ACS, with gradual increases in pressure leading to incremental decreases in urine output. Renal impairment with increased IAP may occur in up to 20% of all patients following laparotomy,\textsuperscript{3} and greatly increases the risk of post-operative mortality. An IAP of 20 mmHg leads to oliguria, and a pressure of >30 mmHg is associated with anuria.\textsuperscript{13,14} The deterioration in renal function is probably multifactorial but can be divided into pre-renal and renal factors. The reduction in cardiac output decreases renal perfusion, but correction of the cardiac output does not fully resolve the renal impairment. Raised IAP leads to compression of the renal vein, increased renal vascular resistance and a decrease in renal perfusion. Furthermore, direct compression of the renal parenchyma contributes to the vascular resistance. These factors combine to impair glomerular filtration, and increase renal production of renin, aldosterone and anti diuretic hormone. Consequently, there is a further increase in renal vascular resistance and sodium and water retention. In a porcine model, compression of the renal vein alone was sufficient to significantly reduce renal blood flow, impair glomerular filtration and induce protein leak.\textsuperscript{48} The same authors have subsequently provided data from a similar model to suggest that renal parenchymal compression is less important than renal vein compression.\textsuperscript{49}

Volume expansion, diuretics and dopamine provide very limited benefit, and early abdominal decompression is essential to promote rapid reversal of the dysfunction.

\textbf{Visceral dysfunction}

Increased IAP leads to a reduction in mesenteric and hepatic arterial flow, decreased flow in the hepatic microcirculation and portal vein and reduced blood flow to the intestinal mucosa. Diebel demonstrated, in a porcine model, reduced flow in the hepatic artery and hepatic microvasculature at a pressure of only 10 mmHg, with further reductions on incremental increases in IAP.\textsuperscript{50} An IAP of 20 mmHg produced a 45% reduction in hepatic artery flow and a 65% drop in portal venous flow. This reduction in blood flow impaired intestinal oxidation, causing an acidosis and the release of free radicals. Bowel tissue oxygenation
has been shown to drop with a pressure of 15 mmHg. Gastric pH has been used as a measure of gut ischaemia in ACS. Ivatury studied 70 patients with penetrating abdominal trauma and found that reduced gastric pH was an early sign of IAH and was reversed by early abdominal decompression. Maynard and co-workers monitored gastric pH in patients following ruptured abdominal aortic aneurysm repair. This group showed that a drop, rather than the absolute pH, provided the most sensitive predictor of outcome. Recently, near-infrared spectroscopy has been described as a non-invasive accurate measure of mesenteric and systemic perfusion in ACS.

Work with colour labelled microspheres in a porcine model demonstrated a significant reduction in tissue blood flow in the spleen, pancreas, oesophagus and gastric mucosa at an IAP of 20 mmHg. Others, using transit time flowmetry and colour labelled microspheres again in a porcine model, have suggested that, although tissue blood flow may be impaired at low pressures, a pressure as high as 40 mmHg is required to cause severe organ damage.

The effect of raised IAP on organ perfusion is exacerbated by high positive end expiratory pressure ventilation, and haemorrhage despite adequate resuscitation. Friedlander measured superior mesenteric artery blood flow in pigs using a Doppler flow probe with incremental increases in IAP. Above 20 mmHg, the effect on blood flow was worse in those animals subjected to a haemorrhage of 20% of circulatory volume despite adequate resuscitation. These data supported the concept of early abdominal decompression in ACS, since normalising cardiac output alone failed to improve mesenteric blood flow.

In addition to direct organ damage, the reduction in blood flow to the bowel may increase bacterial translocation, increasing the risk of sepsis and multiorgan failure. In a rodent model of increased IAP, a pressure of 25 mmHg for 60 min reduced mesenteric blood flow by nearly 40% and led to bacterial translocation, predominantly to the mesenteric lymph nodes. The abdominal wall was also affected, with localised ischaemia and oedema leading to an increased risk of wound complications.

Central nervous system

Initially described in animal studies, the ACS is now recognised in humans to cause increased intracranial pressure, probably by impairment of cerebral venous outflow. The rise in intracranial pressure is mirrored by a reduction in cerebral perfusion pressure, independent of cardio-respiratory function and is reversed by abdominal decompression. In a recent prospective but non-randomised study in an intensive care setting, moderate to severely head-injured patients with stabilised intracranial hypertension were studied to assess the effect of raised IAP. Increased IAP caused a significant rise in intracranial pressure and intrathoracic pressure, supporting the theory of impaired cerebral venous outflow.

In summary, different IAPs have a variable effect upon different physiological systems. A pressure of >20 mmHg has a profound effect upon most organ systems, and sequential insults such as haemorrhage and fluid resuscitation can elicit physiological effects at lower pressures. This has important clinical considerations, particularly in the management of the critically ill surgical patient.

Clinical Considerations

As discussed previously, the ACS exists when IAH is associated with organ dysfunction. Immediate treatment on recognition of the ACS must include optimal fluid resuscitation in an intensive care setting. Anaesthetic paralysis followed by urgent decompressive laparotomy is the mainstay of intervention. In certain situations the risk of ACS should be identified during surgery and a formal lararostomy considered at that time. This can be achieved using a variety of techniques that will be discussed.

There are no large-scale studies of the ACS. The largest published series described 34 patients from the Vanderbilt University trauma centre over a 13-year period. Various investigations have shown that IAH increases morbidity and mortality, but there are very few studies documenting the outcome of ACS without intervention. In the absence of a randomised clinical trial, we rely on anecdotal evidence and audit regarding the role of decompressive laparotomy. However, in a review by Saggi and colleagues, it was suggested that “ACS without expedient decompression is uniformly fatal”. Of the 11 reports they studied, the overall rate of improvement in organ function was 93% following decompression, with an overall survival of 59%.

Most of the literature regarding ACS reports the experience with trauma victims, particularly since the advent of damage-control laparotomy. The advent of “damage-control” surgery in the critically ill trauma victim, often acidicotic and hypothermic, has increased the incidence and recognition of ACS. This is partly due to an increase in the number of such victims salvaged and the sequela of the salvage surgery.
such as abdominal packing and massive fluid resuscitation. The concept of the staged laparotomy is that rapid damage limitation, such as haemorrhage control, stapling of gastrointestinal injuries without resection and drainage of pancreatic or biliary injuries without immediate reconstruction, increases patient survival by reducing the duration on the operating table and the chance of hypothermia, acidosis and coagulopathy. Moore has described five critical decision-making phases of staged laparotomy, through patient selection, intraoperative reassessment, physiologic resuscitation in the surgical intensive care unit, return to the operating room for definitive procedures and abdominal wall reconstruction (Table 2). The theory behind these stages could be applied to the haemodynamically unstable patient with a ruptured aneurysm in whom a risk of ACS could be predicted.

Ertel and co-workers published one of the largest series of ACS in trauma patients. In a partly retrospective and partly prospective series of 311 patients with severe abdominal and/or pelvic trauma who underwent “damage control” laparotomy on the day of admission, 17 patients or 5.5% developed ACS. This proportion was higher if only the severely injured were considered. All underwent primary fascial closure and required emergency decompressive laparotomies. There was a rapid and dramatic improvement in all of the physiological parameters measured. The mortality for the study group as a whole was 22.8%; six of the 17 patients with ACS died, two from respiratory failure, two from haemorrhagic shock and two from multi-organ failure. Of 15 patients who underwent mesh closure of the abdomen because of excessive abdominal tension, no patient developed ACS. The authors concluded that primary fascial closure was critical for the development of ACS after damage control laparotomy.

Meldrum et al. reported a prospective study of 145 severely injured patients requiring laparotomy and ITU admission, of whom 21 (14%) developed ACS. Liver injuries were the most common source of intra-abdominal haemorrhage (57%) and 67% required packing. The ACS, defined in this series as a bladder pressure of >20 mmHg in combination with physiological dysfunction, developed soon after the initial laparotomy (mean 27 ± 4 h). Decompression resulted in improvement in cardio-respiratory and renal function. On the basis of this study, the authors supported a grading system previously described by Burch and colleagues, recommending abdominal decompression for all patients with an abdominal pressure of greater than 25 mmHg (Table 3).

Ivatury and co-workers investigated the incidence of ACS in patients with penetrating abdominal trauma. They studied a combination of gastric pH and bladder pressures in 70 consecutive patients with life threatening injuries. They identified ACS in 23 patients, and revealed that the incidence was significantly higher in those patients who had undergone primary fascial closure compared with those who underwent mesh closure (52 vs 24%). The mortality was higher in those patients with IAH and those who underwent primary fascial closure.

Further support for prompt decompressive laparotomy or prophylactic mesh closure following severe trauma has been provided by the retrospective review of Eddy, who identified a 68% mortality in 34 patients with ACS over a 12-year period. Morris reported a 15% incidence of ACS in a retrospective analysis of 107 patients who underwent staged laparotomy and packing, but more recently Offner has suggested the incidence is higher, demonstrating a 34% incidence over a 5 year period.

The ACS and Aneurysm Surgery

The ACS can develop in the post-operative course of elective and ruptured aneurysms, though is thought to be more common in the latter. All of the published studies in the field of aortic surgery are small. However, the metabolic consequences of aneurysm repair are well documented, and some of the changes may be attributable to raised IAP. The mortality from

<table>
<thead>
<tr>
<th>Stage</th>
<th>Management priority</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Patient selection and abbreviated laparotomy.</td>
</tr>
<tr>
<td>II</td>
<td>Intraoperative reassessment and choice of closure.</td>
</tr>
<tr>
<td>III</td>
<td>Physiologic restoration in the surgical intensive care unit including management of ACS (± decompression).</td>
</tr>
<tr>
<td>IV</td>
<td>Definitive surgical procedures.</td>
</tr>
<tr>
<td>V</td>
<td>Abdominal wall reconstruction.</td>
</tr>
</tbody>
</table>

Table 2. Moore’s staging of the damage control laparotomy. This could be applied to ruptured aortic aneurysm surgery in an unstable patient.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Bladder pressure (mmHg)</th>
<th>Recommended action</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>10–15</td>
<td>Maintain normovolaemia</td>
</tr>
<tr>
<td>II</td>
<td>16–25</td>
<td>Hypovolaemic resuscitation ± paralysis ± decompression</td>
</tr>
<tr>
<td>III</td>
<td>26–35</td>
<td>Abdominal decompression</td>
</tr>
<tr>
<td>IV</td>
<td>&gt;35</td>
<td>Abdominal decompression and re-exploration</td>
</tr>
</tbody>
</table>

Table 3. Meldrum’s Grading of the Abdominal Compartment System, with respect to trauma care. This could be applied to the post-operative management of patients following aortic surgery.
ruptured aneurysm surgery remains in the region of 50%, with multi-organ failure a common cause of death.

Many of the features recognised in trauma may occur in patients with ruptured aneurysms, such as closure of the wound against tension, visceral swelling, retroperitoneal haematoma and massive haemorrhage with large volume fluid resuscitation. Compromised physiological function in this elderly age group may contribute to the development of ACS.

The report by Kron and colleagues, in which the first description of the technique for bladder pressure measurement was described, referred to four cases of ACS occurring in patients with aortic aneurysms. The first case was a “leaking” abdominal aneurysm. Subsequent cases were a young patient with Marfan’s syndrome who underwent elective repair of a thoraco-abdominal aneurysm, a ruptured infrarenal aneurysm and a man with a rapidly expanding infrarenal aneurysm. Emergency decompression was performed in three patients whose renal function improved immediately, but in the fourth patient who was not explored, renal failure persisted until death. The authors recommended urinary bladder pressure monitoring in all patients undergoing major surgery and mandatory emergency decompression for those with a pressure of >25 mmHg.

Fietsam reported a 4% incidence of ACS following ruptured aneurysm repair with primary closure. They observed increased ventilatory pressure, increased central venous pressure, and decreased urinary output associated with massive abdominal distension in four patients, caused by interstitial and retroperitoneal swelling. The syndrome developed in all four patients within 24 h and all patients received more than 251 of fluid resuscitation perioperatively. Decompressive laparotomies were performed in the Intensive Care Unit with placement of a mesh. In two additional patients, the abdominal incision was left open with mesh. Opening the abdominal incision was associated with dramatic improvements in all of the physiological parameters. The authors conclude that some patients with ruptured abdominal aortic aneurysm do not tolerate the closure of the abdominal wall, and prompt recognition and exploration or delayed closure may affect the outcome in some select cases.

In a prospective longitudinal study published soon after that of Fietsam, the IAP of 42 patients admitted to an intensive care unit after abdominal aortic surgery was monitored. Renal impairment occurred in 22 patients (53%), and compared to those with normal renal function they had significantly higher IAP. Ten patients were re-explored because of haemodynamic instability and oliguria, and all had an IAP of greater than 18 mmHg. Following re-exploration, there was a significant increase in urine output (115 ± 40 mL/h, p < 0.01), and decrease in IAP (10 ± 3 mmHg, p < 0.01). The authors concluded that an IAP greater than 18 mmHg following aortic surgery was a significant risk factor for the development of impaired renal function.

In the prospective study following laparotomy by Sugrue, 25 out of 88 had aortic surgery of which two were emergencies. Four elective and one emergency aneurysm repair developed raised IAP (>20 mmHg) but none underwent decompressive laparotomy. The results were not broken down any further and so it is difficult to establish the individual outcome of these patients.

In a study by Oelschlager, the records of 23 surviving ruptured aneurysm patients were reviewed. In a group of eight patients who underwent delayed closure, there was a trend towards increased survival with fewer patients developing multi-organ failure. Significant improvements in oxygenation were observed compared to those who underwent primary fascial closure.

As discussed earlier, gastric intramucosal pH has been suggested as the most reliable indicator of the adequacy of tissue oxygenation and degree of splanchnic ischaemia following ruptured aneurysm repair. Though bladder pressures were not studied, the observed drop in gastric pH may have reflected a rise in IAP. This potential association deserves further investigation.

There has been considerable debate regarding the timing of, and criteria for, decompression. During Moore’s phases II and III of the staged laparotomy (Table 2) there is often a delicate balance between effective tamponade of bleeding and the untoward physiological effects of the ACS. However, survival rates seem to be much higher when a liberal approach is adopted. In Meldrum’s series, where decompression was performed for a bladder pressure of >20 mmHg, the survival was 71%. However, when parameters of severe respiratory or renal compromise were used as indicators for decompression, the survival was only 37.5%. Some authors have called for a randomised trial to settle this issue, though the results of early and liberal decompression seem convincing. In some cases this can be performed safely on the intensive care unit if the patient is unfit for transfer to the operating theatre.

Most recently, Rasmussen and co-workers compared the outcome of patients who needed mesh-based closure with those who underwent standard abdominal closure following ruptured aneurysm.
repair. The study comprised 90 patients who underwent primary closure and 45 who had mesh closure. They determined that patients who had mesh closure had greater blood loss and fluid resuscitation, more prolonged hypotension, more profound acidosis and hypothermia and, not surprisingly, a higher mortality than those who underwent primary closure. Importantly, those who underwent immediate mesh closure (n = 35) had less multiple organ failure and a lower mortality rate than those who had a decompression and mesh closure after initial primary closure (n = 10). They recommend early mesh closure in Fig. 2. Suggested management plan for patients with raised IAP, or at risk of developing the ACS following aortic surgery.
patients after ruptured aneurysm repair, citing the following predictors of poor outcome:

1. A haemoglobin of less than 10 g;
2. Preoperative cardiac arrest;
3. Systolic blood pressure of <90 mmHg for more than 18 min;
4. More than 3.5 l of fluid resuscitation per hour of the operation;
5. Temperature less than 33°C;

The study is limited by its retrospective nature and the lack of direct measures of abdominal pressure, but provides a useful argument for a formal prospective study.

It seems clear, even in the absence of randomised controlled trials, that prompt recognition of raised IAH and emergency abdominal decompression can reverse the organ dysfunction associated with the ACS and improve survival. Decompression should be seen as a lifesaving procedure. Subsequently, definitive surgery needs to be planned to achieve abdominal closure. In the patient at risk of developing the ACS following ruptured aneurysm repair, delayed primary closure should be considered. Routine measurement of bladder pressure should be performed and used as a guide to the suitability of primary closure. Urgent decompression should be considered in any patient with an abdominal pressure over 20 mmHg, or at lower pressures associated with worsening organ dysfunction (Fig. 2).

**Abdominal Wall Closure and Reconstruction**

As discussed previously, primary closure should be delayed if a patient is recognised to be at risk of developing raised IAP. Furthermore, decompressive laparotomy should be performed as a matter of urgency in patients following aneurysm surgery with an abdominal pressure of greater than 20 mmHg. Bladder pressure monitoring should become standard practice, especially following ruptured aneurysm repair. The threshold for performing decompression may be lower in the presence of deteriorating organ function. Various methods have been described for the subsequent wound management.

The gap between the wound edges must be covered to prevent excessive fascial retraction, the formation of a large hernia and peritoneal contamination. The use of alternative closure devices allows coverage of the intraperitoneal organs without undue tension. Such devices include towel clips, the silastic “Bogota bag” and the use of a mesh. Towel clips can be applied at 2 cm intervals to the skin edge, a moist gauze or pack applied and secured with a self-adhesive plastic sheet. If the bladder pressure starts to rise, successive towel clips can be removed in the intensive care unit to reduce the pressure. The “Bogota” bag can be fashioned from a sterile Foley irrigation bag cut along the seams and either sewn or stapled to the skin or fascia. A transparent non-adhesive drape can be stapled to the skin allowing direct observation of the intraperitoneal organs. Some authors have advocated the use of adhesive drapes applied directly over omentum and the wound edges, with no mesh, sutures or staples. This may prevent further damage to the wound edges. The use of a vacuum closure system (the vacuum pack) has been advocated for temporary management of the open abdomen.

A variety of mesh materials may be used to achieve temporary closure (Fig. 3). An alternative to a single mesh sheet is a velcro-like closure mesh or artificial bur, which allows successive reapproximation of the fascial layers until closure and in theory reduces the risk of fistula and hernia development.

The wound above the artificial burr is sealed with a “wound shield” that acts as a barrier against exogenous contamination in the intensive care unit. A similar mesh device incorporates a zip rather than velcro. Once the patient is stable, definitive closure must be achieved. This may simply involve removal of towel clips or mesh followed by primary fascial reapproximation. However, in the presence of continued intraperitoneal oedema or fascial retraction, delayed primary closure may not be possible. Successive approximation or “silo reduction” may achieve this aim.

![Fig. 3. Mesh closure of the abdomen following emergency decompression in a patient with the ACS following ruptured aortic aneurysm repair.](image-url)
Occasionally severe retraction or fascial necrosis may occur. In such cases, the wound may be left to granulate directly over the viscera, or preferably over an absorbable mesh with omental cover. This is contraindicated in the presence of sepsis. A partial thickness skin graft can be applied to the granulating wound (Fig. 4). The resultant large ventral hernia can be repaired at a later date with a further delayed attempt at fascial closure, employing lateral fascial relaxing incisions, or the use of a non-absorbable polypropylene mesh.

**Conclusion**

The ACS is a condition that develops from progressive rises in IAP, resulting in multiple organ dysfunction. It is well recognised following major trauma, but, this also occurs following aortic surgery, particularly ruptured aortic aneurysm repair. Improving the outcome in such patients depends upon prevention of the ACS, early recognition of increasing IAP and urgent intervention to decompress the tense abdomen. Delay may result in multi-organ failure and death. The measurement of IAP is simple and non-invasive, and should be a routine component of physiological monitoring in patients following ruptured aneurysm repair. We would consider emergency decompression in the presence of an abdominal pressure greater than 20 mmHg, and possibly lower pressures with evidence of impending organ failure. However, prospective studies are required to further study the link between abdominal pressures, cytokine levels, the development of the ACS and clarify the timing of decompression.
Intra-abdominal hypertension and abdominal compartment syndrome.

A century later, isn’t it time to pay attention?

R. J. Ayers, J. R. Martin, and A. J. White

Department of Surgery,The University of Texas Southwestern Medical Center at Dallas, Dallas, TX 75390-8834, USA

Summary

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are complications in the critically ill patient. Many studies have been performed with controversial results. The real extent of the problem is not clearly understood. The present review will focus on the clinical consequences of IAH and ACS and the development of new methods to prevent the development of ACS.

I. Introduction

IAH was first described in 1947 in a patient with a large abdominal aortic aneurysm. The patient was found to have an increased intra-abdominal pressure (IAP) of 30 mm Hg. The increased IAP was associated with decreased cardiac output and mortality. The incidence of IAH and ACS has increased dramatically in the past 10 years due to advances in surgical techniques, longer hospital stays, and the use of inotropic agents. ACS occurs when the IAP is sustained above 30 mm Hg for an extended period of time, leading to organ dysfunction and multi-organ failure. The incidence of ACS is estimated to be 10% in critically ill patients. The incidence of ACS is also increasing due to the increasing incidence of abdominal surgery and the use of inotropic agents.

II. Pathophysiology

IAH and ACS are caused by an increase in IAP due to decreased blood flow to the abdominal organs. The decreased blood flow to the abdominal organs leads to decreased oxygen delivery and tissue hypoxia. The decreased oxygen delivery leads to increased lactic acidosis, decreased cardiac output, and increased mortality. The decreased tissue oxygen delivery leads to decreased tissue oxygenation and increased risk of organ failure. The decreased tissue oxygenation leads to decreased myocardial contractility, decreased pulmonary function, and increased mortality.

III. Clinical Consequences

IAH and ACS are associated with increased mortality and morbidity. The increased mortality is due to organ failure and multi-organ failure. The increased morbidity is due to decreased functional capacity, decreased quality of life, and decreased survival. The increased mortality and morbidity are due to decreased tissue oxygenation and decreased oxygen delivery to the abdominal organs. The decreased tissue oxygenation and decreased oxygen delivery lead to decreased functional capacity, decreased quality of life, and increased mortality.

IV. Prevention

Prevention of IAH and ACS is the best strategy to decrease mortality and morbidity. The prevention of IAH and ACS involves the use of prophylactic interventions, such as abdominal binder placement, nasogastric tube placement, and the use of inotropic agents. The prophylactic interventions decrease the incidence of IAH and ACS and decrease mortality and morbidity.

V. Conclusion

IAH and ACS are complications in critically ill patients. The real extent of the problem is not clearly understood. The present review will focus on the clinical consequences of IAH and ACS and the development of new methods to prevent the development of ACS.

References


Accepted 13 November 2002