The open abdomen: why and when should the abdomen be left open? (Part 1 of 3)

Pretorius JP, MBChB, MMed(Surg), FCS(SA) Critical Care **Liebenberg C**, MBChB **Piek D,** MBChB **Smith M,** MBChB
Department of Critical Care, Medical School, Faculty of Health Sciences, University of Pretoria and the Steve Biko Academic Hospital
Correspondence to: Prof Jan Pretorius, e-mail: jan.pretorius@up.ac.za
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Abstract

Intra-abdominal hypertension, abdominal compartment syndrome (ACS) and poly-compartment syndrome are serious clinical problems. The key is early recognition of at risk patients so that preventative measures may be implemented. The concepts of controlled fluid resuscitation, surveillance and prophylactic operative decompression with temporary abdominal closure (TAC) are central to prevention of the events leading to multiple organ dysfunction. Where ACS develops, therapeutic operative decompression and TAC is the management of choice.

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Introduction

Although there are non-operative possibilities, the most definitive management, prophylactic or therapeutic, of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) is surgical decompression and temporary abdominal closure (TAC), implying non-suture of the abdominal fascia.[1] The open abdomen has become an increasingly common occurrence in light of the evolution and overall benefits of the concepts of "damage control" and "goal directed" resuscitation in intensive care.[2] The superiority of an open abdomen approach in reducing the incidence of multiple organ failure and the resulting morbidity and mortality in high risk patients has been substantiated. It is now time to embrace this practice as the standard of care where indicated, bearing in mind the fact that primary closure of the abdomen when possible and safe is optimal. One must not forget that the decision to maintain an open abdomen is an action of necessity and a dangerous and temporary measure, and that primary fascial closure should be prompt and planned from the outset. So the pertinent questions have become: why and when should the abdomen be left open?

The approach to the open abdomen is an evolving concept and our enthusiasm for this strategy should not be dampened by the complexities of managing such an exasperating problem

In this article, part one in a series of three, we will address intra-abdominal hypertension, abdominal compartment syndrome and polycompartment syndrome, and answer the questions: why and when should the abdomen be left open? In the two follow up articles, we will discuss the management of the open abdomen, examining effective short-term and long-term TAC techniques, and the complications of open abdomens, such as enteroatmospheric fistulae.

Intra-abdominal pressure, hypertension and compartment syndrome

The recognition of IAH and ACS as the cause of significant morbidity and mortality among the critically ill has increased dramatically over the last decade.[3-8] This has necessitated standardising working definitions. The World Society of Abdominal Compartment Syndrome (WSACS), an international consensus group, has been pivotal in the advancement of knowledge in this area.[9] Further information can be found on their website at www.wsacs.com. Some of their consensus definitions are used in this article.

Measuring intra-abdominal pressure

In any case of acute abdomen, it is inappropriate, perhaps bluntly negligent, not to monitor intra-abdominal pressures, especially in an intensive care environment.

Intra-abdominal pressure (IAP) is the pressure measured within the abdominal cavity. It is expressed in mmHg and is measured at end expiration in the supine position with the abdominal muscles relaxed. Most commonly, a CVP hydromanometer is used to obtain measurements by connecting it to the urinary catheter. Then, 25 ml of sterile saline is instilled into the bladder and *urinary bladder pressures (UBP)* are measured. The intravesical pressure is considered an accurate reflection of the IAP, as the abdominal contents are fluid in character and relatively non-compressible, and thus behave according to Pascal's law.[10,11]

It is important to note that these measurements are made in cmH_2O, and must be converted to mmHg (mmHg = cmH_2O/1.36). Aseptic technique should be maintained and transducers should be zeroed at the midaxillary line if the patient is supine, or at the symphysis pubis if the patient is lying on his or her side. Normal IAP is 5–7 mmHg in critically ill adults.[12] Using the mean arterial pressure (MAP) and the intra-abdominal pressure (IAP), one can calculate the abdominal perfusion pressure (APP):

MAP – IAP = APP

The APP must be greater than 60 mmHg to ensure adequate abdominal organ perfusion. A sufficient APP is, in fact, the desired end point and should be monitored at all times.

Where elevated and increasing IAP is probable a system such as the closed AbViser® AutoValve IAP Monitoring Device (Wolfe Tory Medical, Inc) will be useful for more frequent and accurate measurements. There are several such devices or systems on the market to measure the IAP.

Intra-abdominal hypertension (IAH) is regarded as a sustained or repeated pathological elevation of IAP > 12 mmHg. It is graded according to severity (Table 1).[12]

Table I: Grading of IAH

Grade	Pressure
Grade 1	12–15 mmHg
Grade 2	16–20 mmHg
Grade 3	21–25 mmHg
Grade 4	> 25 mmHg

Abdominal compartment syndrome (ACS) occurs when the increased pressure in the abdomen threatens the viability of intra-abdominal organs. It is regarded as a sustained IAP of > 20 mmHg (with or without APP < 60 mmHg) that is associated with new organ dysfunction or failure[8,11] and may be classified as *primary, secondary* or *recurrent ACS.*

Primary ACS is associated with injury or disease in the abdominopelvic region, and frequently requires early surgical intervention. *Secondary ACS* is associated with conditions that do not arise in the abdominopelvic region. *Recurrent ACS* follows previous surgical or medical treatment of primary or secondary ACS.[8,11]

Risk factors for IAH and ACS[12]

- **Diminished abdominal wall compliance**
  - Acute respiratory failure.
  - Abdominal surgery with primary fascial closure.
  - Major trauma and burns.
  - Prone positioning.
- **Increased intraluminal contents**
  - Gastroparesis.
  - Ileus.
  - Colonic pseudo-obstruction.
- **Increased abdominal contents**
  - Haemo- and pneumoperitoneum.
  - Ascites and liver dysfunction.
- **Capillary leak and fluid resuscitation**
  - Acidosis (pH < 7.2), hypotension, hypothermia (core temperature < 33 °C), polytransfusion (> 10 U blood/24 hours), massive fluid resuscitation (> 5 l/24 hours).
  - Coagulopathy (platelets < 55,000/mm^3, or APTT > 2 x normal, or PTT < 50%, or INR > 1.5).
  - Major trauma and burns.
  - Oliguria, sepsis and damage control laparotomy.

Causes of increased intra-abdominal pressure

- **Retroperitoneal causes**
  - Pancreatitis.
  - Retroperitoneal or pelvic bleeding.
  - Contained abdominal aortic aneurysm rupture.
  - Aortic surgery.
  - Abscess.
  - Visceral oedema.
- **Intraperitoneal causes**
  - Intraperitoneal bleeding.
  - Abdominal aortic aneurysm rupture.
  - Acute gastric dilatation.
  - Bowel obstruction.
  - Ileus.
  - Mesenteric venous obstruction.
  - Pneumoperitoneum.
  - Abdominal packing.
  - Abscess.
  - Visceral oedema secondary to resuscitation (SIRS).
- **Abdominal wall causes**
  - Burn eschar.
  - Repair of gastroschisis or omphalocoele.
  - Reduction of large hernias.
  - Pneumatic anti-shock garments.
  - Laparotomy closure under tension.
  - Abdominal binders.
- **Chronic causes**
  - Central obesity.
  - Ascites.
  - Large abdominal tumours.
  - Peritoneal dialysis.
  - Pregnancy.

Pathophysiology

Endothelial dysfunction, particularly of the *microvasculature,* is central to the pathophysiological development of a compartment syndrome (Figure 1). In critically ill patients, irrespective of aetiology, it is increasingly understood that inflammation and pro-oxidative stress results in disruption of endothelial homeostasis. The functional and structural alterations of the endothelium lead to break down of vascular integrity and functioning, which may precede gross capillary leak and deterioration in end-organ function.[13]

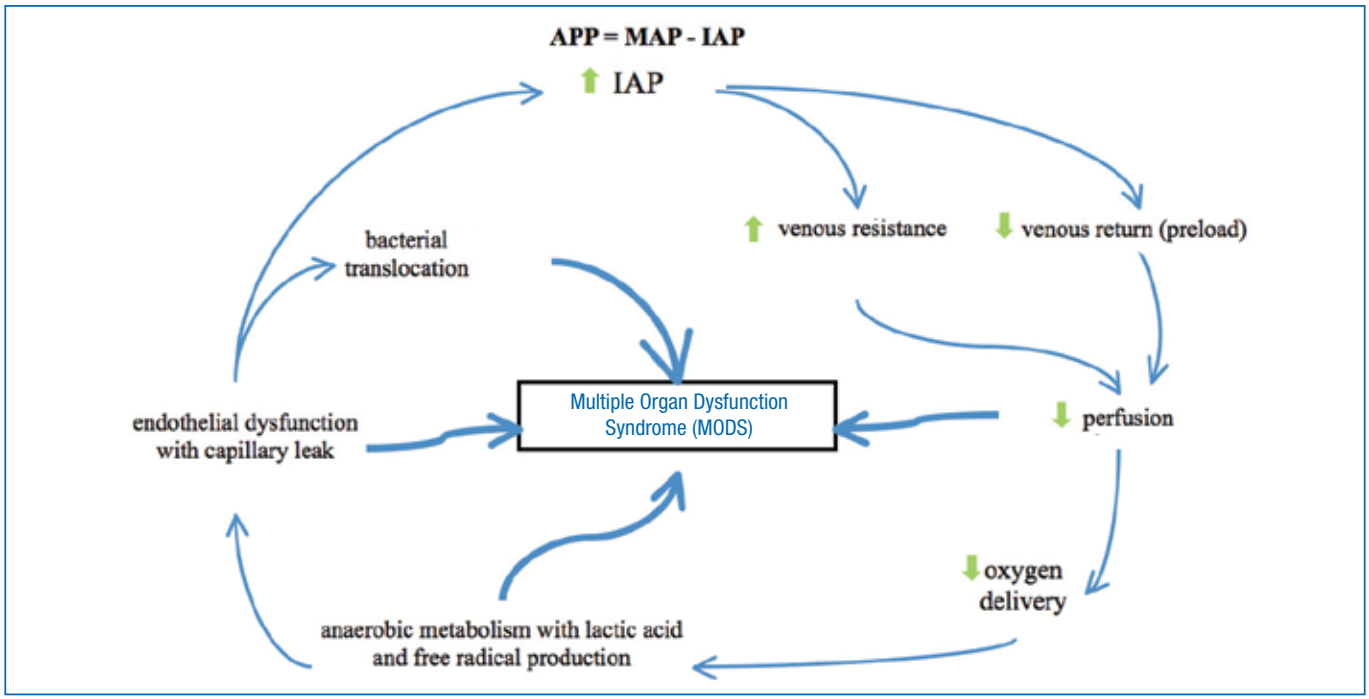

Figure 1: Pathophysiology of compartment syndrome

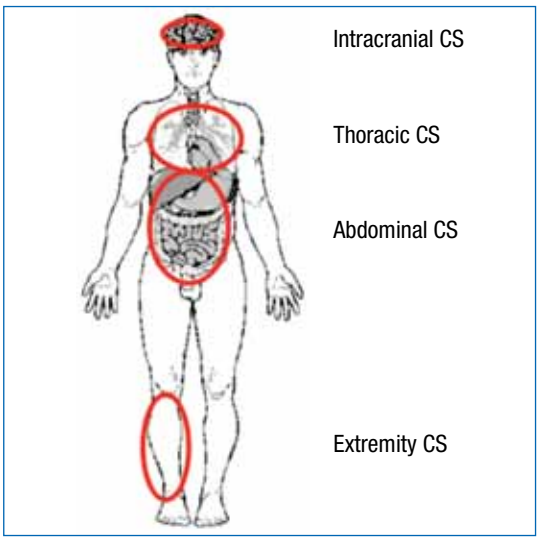

Figure 2: The four major compartments: intracranial, thoracic, abdominal, and the extremities[15]

The conundrum of fluid resuscitation is that adverse outcomes may be associated with both inadequate and excessive fluid administration. This is especially true of crystalloids. Oedema resulting from massive fluid resuscitation in the presence of capillary leak is regarded as the mechanism for the development of IAH. This links over-resuscitation with multiple organ failure and death.[12]

IAH as a consequence of compromised and leaking capillaries is, in itself, a detrimental positive feedback cycle. The resulting decrease in abdominal perfusion pressure leads to decreased oxygen delivery to tissues, causing a rise in anaerobic metabolism, lactic acid production and free radical formation. The state of the already compromised and leaking microvasculature worsens, exacerbating the existing IAH. Furthermore, the direct forces arising from an increase in compartment pressure, be it from capillary leak or other compressive causes such as pneumo- or haemoperitoneum, increase venous resistance and decrease perfusion pressure, thus compounding the cycle. Worsening mucosal breakdown then leads to bacterial translocation and multisystem organ failure (MSOF).

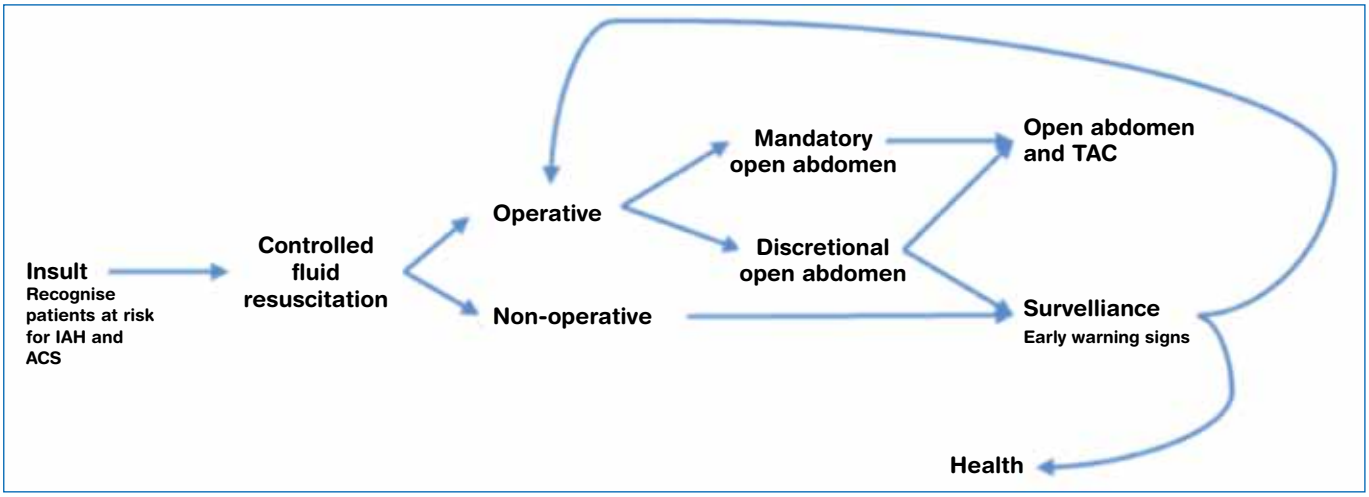

Figure 3: Management pathway of IAH and ACS

It is important to realise that these pathophysiological effects of inflammation on endothelial functioning are not confined to a single compartment, but occur throughout the body, giving rise to the concept of *poly- or multicompartment syndrome*. An increase in compartment pressure will exert a direct force on the original compartment and its contents, as well as affecting adjacent and distant compartments via direct pressure or resulting pathophysiology. Thus, though there are many compartments within the body, they are not independent entities isolated from one another but are intricately connected. The impact on end organ function and viability, both within and outside of the original compartment, may be devastating (Figure 2). [14]

To illustrate this relationship, IAP elevation causes immediate increases in intracranial pressure (ICP), internal jugular pressure (IJP), central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP).

Abdominal compartment syndrome predominantly affects the renal, hepatic, gastrointestinal, endocrine, cardiac, pulmonary and central nervous systems.

Table II: Guidelines for fluid therapy

Resuscitation	To specific end points	SaO_2 > 90% Monitor DO2 MAP > 65 mmHg Stroke volume variation < 13% Pulse pressure variation < 5 mmHg
Replacement	According to measured losses and constituents	0-150 ml = 100% 150-300 ml = 80% 300-500 ml = 60% > 500 ml = 50%
Maintenance	According to age and ideal body weight	1st 20kg = 1,500ml plus 20 ml/kg thereafter if age < 50 years or 15 ml/kg if age > 50 years

DO2: oxygen delivery; MAP: mean arterial pressure; SaO_2: oxygen saturation

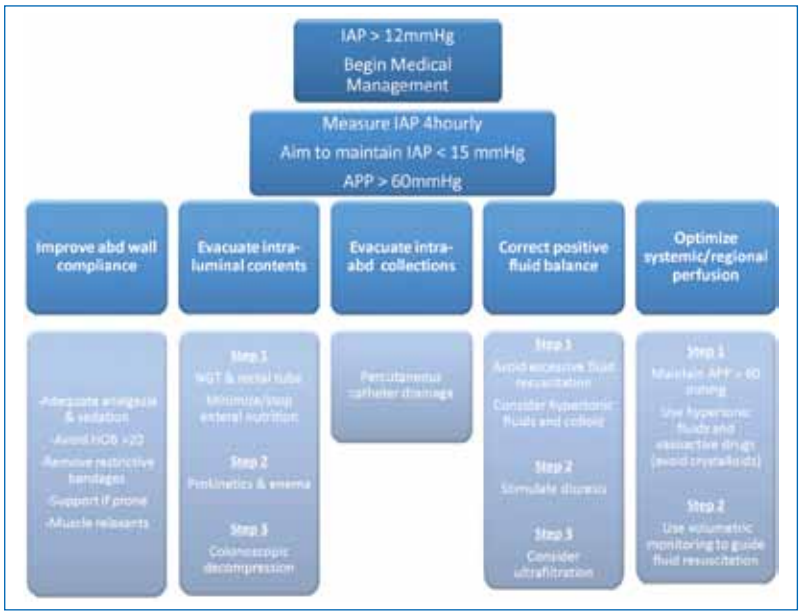

Figure 4: Non-operative management of IAH[11]

Prevention and management of IAH and ACS

There are two important elements in the care of ACS:

- Management of IAH where ACS has not yet developed; and
- Therapeutic operative decompression of ACS with TAC.

Management of IAH where ACS has not yet developed

Concepts critical to the prevention of the development of ACS include the control of resuscitation fluids, surveillance and monitoring and, if ACS is inevitable, consideration of prophylactic operative decompression with TAC. As mentioned previously, there are a host of conditions and circumstances commonly associated with the development of IAH and ACS. It is important to recognise at risk patients and start preventative measures immediately.

The first concept to be aware of is the judicious administration of resuscitation fluids.[15] The volume and type of fluid infused should be administered according to appropriate guidelines or protocols with the goal of achieving specific end points.[16,17]

When IAH is diagnosed, it is not necessary to move directly to prophylactic operative decompression with TAC to preclude ACS from developing. There are numerous non-operative measures which can be useful in the resolution of IAH and prevention of ACS (Figure 4).

If these non-operative measures prove unsuccessful, prophylactic operative decompression should be considered. Given the hazards of managing the open abdomen, it is at present not recommended to do presumptive decompression for IAH without organ dysfunction being present.[18]

Therapeutic operative decompression of ACS with TAC

If, on initial presentation, a critically ill patient requires operative management in the form of a laparotomy, the key decision becomes whether to close the abdomen or to leave it open as a laparastome. It is imperative to start measuring IAP in theatre if, and as soon as, the sheath is closed, as pressures above 12 mmHg will be a warning sign that the patient is at risk of worsening IAH. Pressures above 20 mmHg are an indication to re-open the sheath and leave the abdomen open.

Currently, therapeutic operative decompression with TAC is the accepted intervention for ACS, i.e. where organ dysfunction is present and due to IAH. In this case, abdominal decompression may be a lifesaving intervention and the appropriate TAC must be chosen for the resulting grade of open abdomen. This will be discussed in a subsequent article.

There are *mandatory indications* for leaving an abdomen open:

- Massive intestinal oedema.
- Rapid conclusion of procedure in damage control surgery.

- Need for multiple re-explorations of the abdomen.
- Fascia and abdominal wall preservation.
- Presence of bowel and/or retroperitoneal oedema protruding above the fascia.
- Pulmonary deterioration during closure.
- Haemodynamic instability with closure.

The latter two are signs of deterioration of the patient's condition as a direct consequence of increased IAP and ACS.

However, there are also *discretional indications* for leaving the abdomen open:

- Faecal contamination or peritonitis.
- Large resuscitation requirements over a short period (> 10 l of crystalloids and/or > 6 U of blood).
- Possibility of ongoing large volume resuscitation.
- "Deadly triad" of hypothermia, coagulopathy and acidosis.[19,20]
- Multiple intra-abdominal injuries.
- Packing of the abdomen.
- Subjective tight closure of the anterior abdominal wall (Figure 3).

Here, astute assessment and judgement are necessary in deciding the likelihood of the development of IAH and ACS should the abdomen be closed, as it may still be prudent to prophylactically leave the abdomen open.

The role of surveillance

Surveillance becomes critical in cases where the abdomen is closed, or if the initial condition does not require operative management. Patients should be carefully monitored for these warning signs of IAH and ACS.[21]

- **Cardiovascular system**
  - Raised central venous pressure.
  - Raised internal jugular pressure.
  - Labile blood pressure.
  - Increased pulse pressure variation.
  - Increased stroke volume variation.
  - Decreased cardiac output.
- **Pulmonary**
  - Respiratory failure.
  - Raised pulmonary artery occlusion pressure.
  - Pulmonary oedema.
  - Atelectasis.
- **Central nervous system**
  - Raised intracranial pressure.
  - Confusion.
  - Decreased level of consciousness
- **Renal**
  - Oliguria.
- **Hepatic**
  - Lactic acidosis.

Early recognition of IAH is imperative for improved patient outcomes. The most effective IAH surveillance strategy is the serial measurement of UBP. Although IAH should be considered a possibility in a noticeable percentage of critically ill or injured patients, outright ACS is still rare.

It is advised to measure UBP every 4 hours in high-risk patients, while moderate- and low-risk patients can be monitored every 6–12 hours. One should aim to maintain an IAP of less than 12 mmHg and an APP of greater than 60 mmHg.

Conclusion

There are many patients at risk for developing intra-abdominal hypertension, abdominal compartment syndrome and polycompartment syndrome. It is important to recognise these patients so that preventative measures may immediately be taken. The concepts of controlled fluid resuscitation, surveillance and prophylactic operative decompression and TAC are central to prevention. Where ACS develops, therapeutic operative decompression and TAC is the management of choice.

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