

Abdominal compartment syndrome

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Summary

Abdominal compartment syndrome (ACS) is a condition associated with significant morbidity and mortality. This article includes a case summary of a patient who developed ACS, after which the pathophysiology, diagnosis and management of this important condition are discussed.

CASE HISTORY

A 26-year-old previously fit and well man was a pedestrian involved in a road traffic accident. His injuries included rib fractures, a ruptured spleen and an unstable pelvic fracture. He was admitted to the intensive care unit following external fixation of his pelvis, laparotomy and splenectomy. Estimated blood loss was over 4 litres, which was replaced with a combination of blood, fresh frozen plasma and platelets. At the end of the case the patient had a metabolic acidosis, a mild coagulopathy, mild anaemia and was hypothermic. In view of these findings the patient remained ventilated.

Overnight the coagulopathy, anaemia and hypothermia were corrected. However his ventilatory requirements increased, with peak inspiratory pressure rising from 22 to 38cmH₂O in order to maintain acceptable tidal volumes. His abdomen was noted to be tense and distended. In addition his base excess remained greater than -8 and his urine output remained less than 0.5ml.kg⁻¹.h⁻¹ despite fluid resuscitation.

Abdominal compartment syndrome was confirmed by measurement of his intra-abdominal pressure. This was measured via an intra-vesical catheter (see below), giving a reading of 28cmH₂O. Following discussion with the surgical team he was taken for decompressive laparotomy (laparostomy). On opening the abdomen, peak inspiratory pressures fell immediately. No active bleeding was found but large intra-abdominal and retro-peritoneal haematomas were identified. The intra-abdominal haematoma was evacuated. Primary abdominal closure was not possible. Temporary closure was achieved using part of a sterile opened intravenous fluid bag, stitched into the defect in the abdominal wall.

The patient was then returned to the ICU, still ventilated, but requiring greatly reduced ventilatory support. On day 7 he underwent closure of his abdominal wound. Two days later he underwent internal fixation of his pelvic fractures and was discharged home two weeks later.

DEFINITIONS

The potential adverse consequences of raised intra-abdominal pressure (IAP) have been recognised for many years.¹ However the term abdominal compartment syndrome (ACS) was not coined until 1984, when Kron et al published

a series of post-operative patients in whom they measured IAP.² Recently the definitions of intra-abdominal hypertension (IAH) and ACS have been standardised.³

Intra-abdominal hypertension (IAH) is defined as an intra-abdominal pressure of 12mmHg or more.

Abdominal compartment syndrome (ACS) is defined as an intra-abdominal pressure (IAP) above 20mmHg with evidence of organ dysfunction or failure.

ACS is further classified as primary, secondary or recurrent.

Primary ACS is associated with conditions within the abdomino-pelvic region.

Secondary ACS includes conditions that do not originate within the abdomen, such as sepsis and capillary leak, major burns or other conditions that require massive fluid resuscitation.

Recurrent ACS is a condition in which ACS recurs after treatment of primary or secondary ACS.

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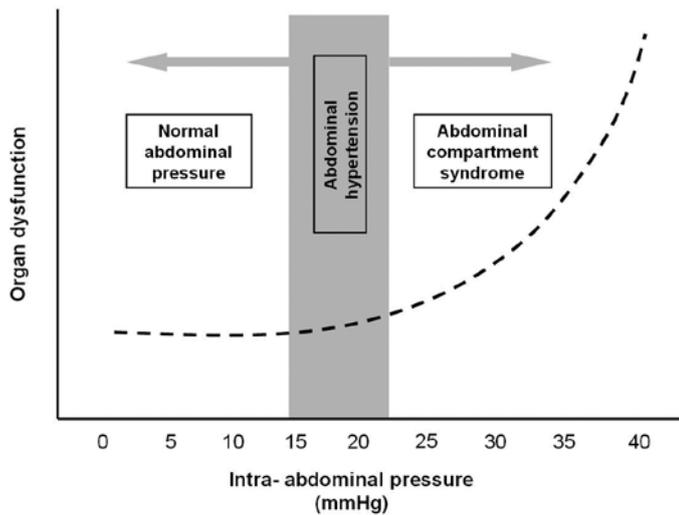


Figure 1. The relationship between intra-abdominal pressure and organ dysfunction.

PATHOPHYSIOLOGY

Intra-abdominal pressure reflects the relationship between intra-abdominal volume and abdominal compliance, and has a direct relationship with intra-thoracic pressure. The relationship between abdominal pressure and volume is non-linear. Abdominal compliance is affected by speed of increase in abdominal volume, as evidenced by the slight changes in IAP that occur during pregnancy. Normal IAP is estimated to be around 6mmHg. Chronic IAH can occur in some obese patients and patients with a large volume of ascites.

The causes of acutely raised IAP are often multifactorial.

Common causes of raised IAP are:

- Trauma and intra-abdominal or retroperitoneal haemorrhage
- Abdominal surgery (including laparoscopy intraoperatively)
- Peritonitis
- Repair of large incisional hernias
- Massive fluid resuscitation (>5 litres per 24 hours).

Small changes in IAP are usually well tolerated but as IAH worsens, it will eventually affect regional blood flow and impair tissue perfusion. This is often related to the systemic inflammatory response and multiorgan failure and the organs involved include the renal, cardiovascular, respiratory, gut, central nervous and immune systems. These will be discussed in turn.

Renal effects of ACS

Impairment of renal function seen in ACS is caused by an increase in renal vascular resistance, caused by renal vein compression. Reduction in cardiac output may be another causative factor. The exact IAP required to cause renal impairment is not known but some authors suggest a level of above 15mmHg. Impaired urine output has been noted in 65% of acutely injured patients with an IAP over 25mmHg and in 100% of those with an IAP above 35mmHg.⁴ Co-morbidities affecting renal function clearly have an additive effect. In addition to controlling IAH, maintaining cardiovascular filling pressures are important to limit renal dysfunction.

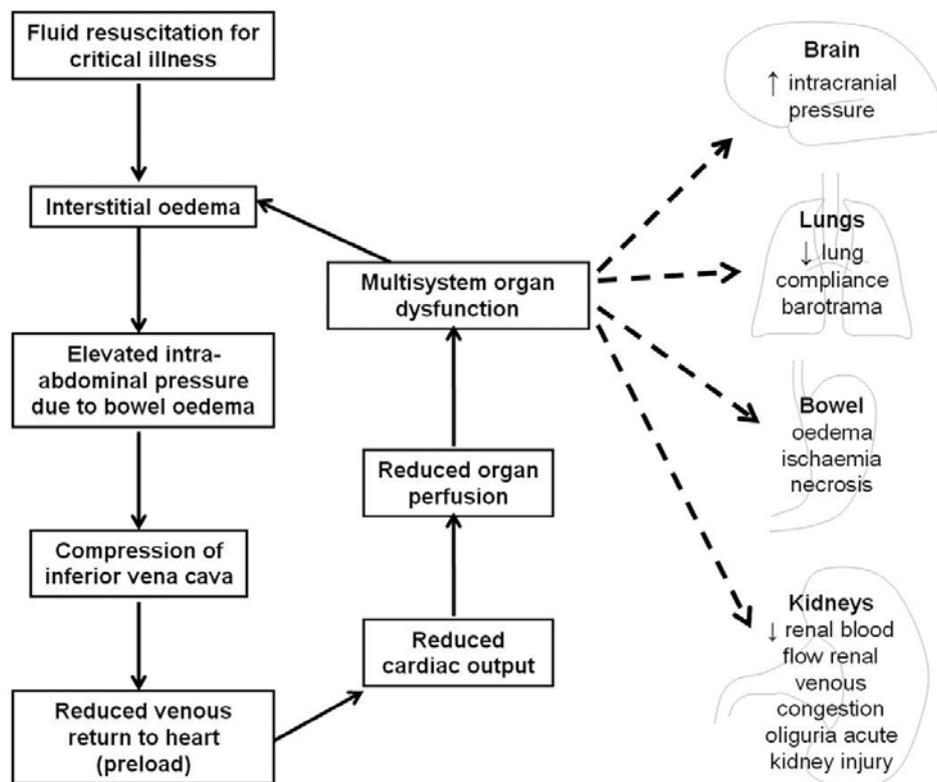


Figure 2. Overview of the effects of raised abdominal pressure on organ function.

Cardiovascular effects of ACS

Increased IAP reduces cardiac output by decreasing preload (compression of the inferior vena cava and hepatic vein) and by decreasing left ventricular compliance, secondary to increased intra-thoracic pressures. Increased intra-thoracic pressure results in increased central venous, right atrial and pulmonary wedge pressures, despite the decreased cardiac output. Significantly, monitoring these pressures could give the impression of over-filling when the patient may be hypovolaemic.

Respiratory effects of ACS

The effects of IAH on respiration are largely mechanical. Diaphragmatic elevation and splinting causes a reduction in ventilation by decreased thoracic volume and decreased lung compliance. Compressive atelectasis eventually results in increased ventilation-perfusion mismatch and hypoxia. If the patient is breathing spontaneously they will develop rapid shallow breathing. In the ventilated patient it will become increasingly difficult to achieve adequate ventilation. Patients are likely to become hypoxic with a rising CO_2 .

Other effects of ACS

Other potential adverse effects of IAH include visceral hypoperfusion with secondary bacterial translocation, impaired abdominal wound healing, worsening of raised intra-cerebral pressure in trauma patients and possibly an increased cytokine response.⁵

DIAGNOSIS OF ACS

Clinical examination alone is insensitive for the detection of IAH. Early detection requires proactive measurement of intra-abdominal pressure in patients at risk.

Indications for considering monitoring IAP include:⁶

- Any patient whose abdomen appears clinically to be distended and firm and who has a poor urine output, low BP and difficulty with ventilation.
- Patients with open or blunt abdominal trauma.
- Patients with burns and massive fluid resuscitation.

MEASUREMENT OF IAP

The standard measurement technique of IAP is by use of a Foley bladder catheter. Fifty millilitres of saline should be instilled into the bladder. Intra-vesical pressure can then be estimated by measuring the vertical height of the column of fluid when the catheter tubing is held vertical and the patient is supine. Alternatively, the collection tubing from the catheter is clamped after instilling saline into the bladder and then intra-vesical pressure can be measured via a needle inserted aseptically into the aspiration port, proximal to the clamp, which is then attached to a pressure manometer or transducer. The level of the pubic symphysis is taken as zero for both these techniques.

MANAGEMENT OF ACS

The definitive treatment of ACS is to achieve a maintained reduction in IAP. Aggressive non-operative intensive care support is critical to prevent the complications of ACS. This involves careful management of cardiovascular and respiratory impairment, maintenance of



Figure 3. A; Equipment for measurement of intra-abdominal pressure. This consists of a length of clear tubing with 1cm markings connected between the end of the catheter and the tube leading to the collection bag. B; After instillation of 50ml sterile water into the bladder, the vertical height of fluid within the tubing is measured from a set point (either the symphysis pubis or the greater trochanter of the femur, as here). This intravesical pressure is an accurate estimate of the intra-abdominal pressure.

intravascular volume, whilst avoiding over generous fluid resuscitation and possibly commencement of renal replacement therapy, if facilities are available. Mechanical ventilation with deep sedation and muscle relaxation is usually required unless abdominal decompression can be undertaken. Simple measures to decrease IAP such as nasogastric decompression and avoidance of constipation must not be overlooked. In some circumstances abdominal collections may be able to be drained percutaneously.

There is little consensus on the precise indications for and timing of surgical decompression. It is more important to correct pathophysiological abnormalities than to achieve a particular IAP.⁶ At present there are no absolute guidelines and all patients with increasing IAP and deteriorating organ function should be considered for decompression. This point is illustrated by the case history above, where surgical abdominal decompression was performed because of deteriorating renal function and compromised ventilation. Prompt intervention is beneficial – early surgical decompression may be associated with reversal of organ deterioration in 80% of patients, but despite this the mean survival rate is only 53%.⁷ With increasing awareness of this condition, surgical decompression for abdominal compartment syndrome is increasingly undertaken and would appear to have a beneficial effect on organ function. However no randomised studies have been undertaken to demonstrate its effect on patient mortality.

As anaesthetists, if the surgeon is struggling to achieve closure and you suspect that the intra-abdominal pressure is high, consider measuring the IAP during abdominal closure or, at least, before the patient is transferred from theatre. The anaesthetist has an important role in preventing a surgeon from closing an abdomen under excessive pressure.

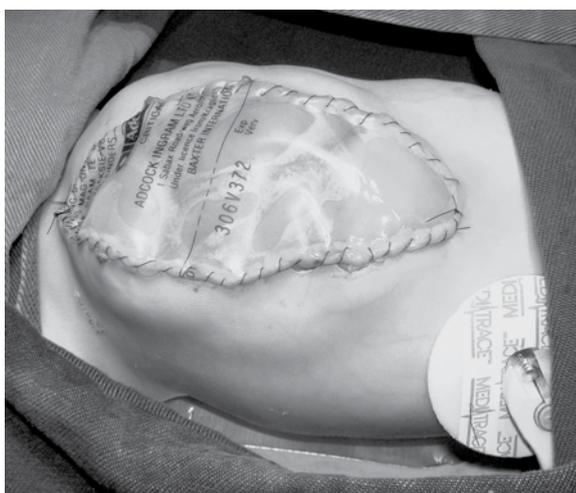


Figure 4. Closure of a neonate's abdomen with a Bogota bag, after surgery to correct gastroschisis.

Anaesthetising a patient for abdominal decompression can be hazardous. It may be difficult to achieve adequate ventilation when transferring to the theatre. When the abdomen is decompressed there may be a sudden fall in blood pressure (despite a rise in cardiac output) and intravenous fluid resuscitation is likely to be required. The immediate increase in lung compliance can lead to hyperventilation, so ideally end-tidal CO₂ or blood gases should be monitored.

CONCLUSION

ACS is a syndrome associated with high morbidity and mortality. IAP monitoring should be used in at risk patients. This can be simply and reliably estimated by use of saline instilled in a bladder catheter. As there are no current guidelines regarding the role for and timing of decompressive laparotomy, each case will require individual assessment and discussion between the intensive care and surgical teams.

FURTHER READING

Cheatham ML, Safcsak K. Is the evolving management of intra-abdominal hypertension and abdominal compartment syndrome improving survival? *Crit Care Med* 2010; **38**: 402–7.

REFERENCES

- Emerson H. Intra-abdominal pressures. *Arch Intern Med* 1911; **7**: 754–8.
- Kron IL, Harman PK, Nolan SP. Measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. *Ann Surg* 1984; **199**: 28–30.
- Malbrain ML, Cheatham ML, Kirkpatrick A et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. *Intensive Care Med* 2006; Sep 12 [Epub ahead of print].
- Meldrum DR, Moore FA, Moore EE, Francoise RJ, Sauaia A, Burch JM. Prospective characterisation and selective management of the abdominal compartment syndrome. *Am J Surg* 1997; **174**: 667–73.
- Rezende-Neto JB, Moore EE, Melo de Andrade MV et al. Systemic inflammatory response secondary to abdominal compartment syndrome: stage for multiple organ failure. *J Trauma* 2002; **53**: 1121–8.
- Surgue M. Abdominal compartment syndrome. *Curr Opin Crit Care* 2005; **11**: 333–8.
- Surgue M, D'Amour S. Review of publications identifying outcomes after decompression in patients with ACS. *J Trauma* 2001; **51**: 419 (Letter).

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