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# When and how should we measure intra-abdominal pressure?

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A sustained increase in intra-abdominal pressure (IAP) may result in abdominal compartment syndrome (ACS). This is a well documented complication in critically ill patients, but there appears to be a reluctance to routinely measure IAP in patients at high risk of developing intra-abdominal hypertension (IAH) and ACS. This may be due to a lack of clinical skills or perceived complexity of the procedure.

## Incidence and prognosis of IAH/ACS

IAH is defined as IAP >12 mmHg. IAH is graded from I to IV according to severity, with grade IV being an IAP ≥25 mmHg.

ACS is defined as an IAP >20 mmHg associated with acute organ failure.[1]

Malbrain *et al*.[2] looked at the incidence and prognosis of IAP in a mixed population of critically ill patients (265 consecutive patients) in a multi-centre study. On admission, 67.9% of patients had a normal IAP (<12 mmHg), 32.1% had IAH (IAP >12 mmHg), and 12.9% of the patients with IAH had ACS. The prevalence of ACS for the group was 4.2% (1 in 25 patients). Independent predictors for IAH were liver dysfunction, abdominal surgery, fluid resuscitation and ileus. The mortality rate was significantly higher in the group with IAH compared with the group who did not have IAH (38.8% v. 22.2%, $p$=0.0005).

## Pathophysiology and clinical manifestations

IAH affects regional blood flow and impairs tissue perfusion. This decreased tissue perfusion is linked to the development of systemic inflammatory response syndrome (SIRS) and multiorgan failure. Kimball[3] has referred to IAH and ACS as 'ARDS' of the gut and describes the phenomenon as 'another deadly cousin in the family of systemic inflammatory response diseases'.

The clinical manifestations of ACS are related to the consequences of IAH on organ function. The triad includes increased peak airway pressures, decreased urine output and a tense abdomen. The onset of ACS can only be detected by measuring IAP.

## Detection of IAH and ACS

Despite ACS being recognised as a condition that critically ill patients are at risk of developing, its proactive detection by measuring IAP is not common practice. A survey of intensive care units in the UK[4] revealed that 25% of units do not measure IAP. Of the units that do measure IAP, 93.2% measured IAP when there was a suspicion that ACS was developing. This retroactive response does not assist in the early identification and management of IAH and ACS.

The World Society of the Abdominal Compartment Syndrome (WSACS) consensus guidelines recommend that IAP should be routinely measured in all critically ill patients who are at risk for developing IAH and ACS.[1] On admission to the ICU, all patients should be screened for IAH/ACS risk factors. New onset of organ failure or progressive organ failure warrants IAP measurement. In addition, IAP should be routinely measured if the patient has two or more risk factors.

The risk factors for IAH/ACS are:

- Diminished abdominal wall compliance
  - Acute respiratory failure (especially with elevated intrathoracic pressures)
  - Abdominal surgery with primary fascial closure or tight closure
  - Major trauma or burns
  - Prone positioning, head of bed >30°
  - High BMI, central (abdominal) obesity
- Increased intra-luminal contents
  - Ileus
  - Gastroparesis
  - Colonic pseudo-obstruction
- Increased abdominal contents
  - Ascites/liver dysfunction
  - Haemoperitoneum/pneumoperitoneum
- Capillary leak/fluid resuscitation
  - Acidosis (pH <7.2 kPa)
  - Hypotension
  - Hypothermia (core temperature <33°C)
  - Polytransfusion (>10 units of blood/24 h)

- Coagulopathy (platelets < $55\times10^9$/l or prothrombin time >15 seconds or partial thromboplastin time >2 × normal or international normailsed ratio >1.5)
- Massive fluid resuscitation (>5 l/24 h)
- Pancreatitis
- Oliguria
- Sepsis.

## Measurement

The monitoring of IAP should be part of the assessment of critically ill patients who are at risk of developing IAH/ACS. This assessment should have a standardised protocol.

Clinical estimation of IAP using palpation for abdominal tenseness has been shown to be inaccurate.[5] Measuring IAP indirectly via the urinary bladder is currently considered the gold standard.[1,6] Factors to be taken into account are that this is an indirect measurement of IAP and it may interfere with the urinary catheter drainage system.[6,7] Measurement of IAP using the indirect bladder technique may not be accurate if there is gastric distension, pelvic pathology or a neurogenic bladder.[7] Contraindications to measuring IAP using the bladder technique include pelvic fracture, haematuria or neurogenic bladder.[2]

Reproducibility is necessary for practitioner confidence. The value obtained is unlikely to be utilised or acted upon if there is uncertainty about its accuracy.

The bladder technique for measuring IAP indirectly was originally described by Kron and colleagues.[8] This technique involves disconnecting the Foley urinary catheter and instilling saline into the bladder. The urinary drainage bag is then reconnected and clamped distal to the culture aspiration port. A 16G needle is inserted into this culture port and connected to a manometer or pressure transducer. The disadvantages of this technique are that it places the patient at increased risk for urinary tract infection, and exposes the health care providers to the risk of needle-stick injury.[9] The aspiration port membrane becomes leaky (due to repeated puncturing) and may result in the replacing of the urinary catheter. This intermittent method also increases the 'hassle factor'.

Cheatham and Safcsak[10] revised Kron's original technique to form a closed system where repeated measurements could be taken. A 'ramp' consisting of 3 stopcocks in-line is inserted in the drainage tubing connected to a Foley urinary catheter. The first stopcock has a standard infusion set connected to a normal saline vacolitre; a 60 ml syringe is connected to the second stopcock; and the third stopcock is connected to a pressure transducer.[9]

Miller and colleagues at Groote Schuur Hospital have recently modified this method by replacing the ramp with a single stopcock (Lopez Valve, Pediatric; ICU Medical, CA, USA – Ref. 011-M9000-P) inserted in the drainage tubing near the hub of the Foley urinary catheter. A standard water manometer is primed with normal saline and then attached to the stopcock. The correct amount of saline is then injected into the bladder using a 60 ml luer-tip syringe which is attached to the injection port of the manometer line. Table I sets out a detailed 'how to' description of this technique.

## Management of IAH and ACS

If the reading obtained indicates that there is IAH, there are treatment options that can reduce IAP. An IAH/ACS management algorithm has been developed by the WSACS.[1]

The medical treatment options to reduce IAP (as recommended by WSACS) focus on improving abdominal wall compliance; evacuating intra-luminal contents and abdominal fluid collections; correcting positive fluid balance; and providing organ support.[1,12]

Abdominal decompression should be considered if the patient has an IAP >20 mmHg with organ failure.[1]

### Abdominal decompression

Definitive treatment for ACS is prompt abdominal decompression by midline laparotomy incision and temporary abdominal closure. This intervention can be perceived as both dramatic and invasive by patient, family and staff. Moore *et al.*[13] recommend that both patient and family receive counselling. It is imperative that they receive adequate information regarding the management, closure methods and potential complications when the timing is appropriate. It is essential that the critical care nurse is at the patient's bedside when the sedation is lightened or interrupted in order to reassure the patient. No studies could be found on the emotional and psychological impact of awakening to an 'open' abdomen, but the assumption is frightening for both the patient and the family.

Although abdominal decompression is commonly referred to as an 'open abdomen', the term 'temporary abdominal closure' is less dramatic and a more accurate description thereof.

### ACS in the open abdomen

De Waele *et al.*[14] reviewed the English literature from 1972 to 2004 for studies that looked at the effects of decompressive laparotomy in patients with ACS. IAP was significantly lower after decompression (15.5 v. 34.6 mmHg before, $p<0.001$), but IAH persisted in the majority of patients.

Gracias *et al.*[15] conducted a retrospective review of 20 trauma patients who had an open abdomen managed with vacuum-packed dressings. These were not patients who had abdominal decompression for IAH and ACS. The authors found that the vacuum-packed

**Table I. Procedure for measurement of IAP via a urinary catheter using a water manometer and single stopcock**

1. Set up a water manometer set with 0.9% sodium chloride solution and flush the line.
2. Level/zero the water manometer at the level of the iliac crest in the mid-axillary line.[1]
3. Clean the drainage tubing proximal to the hub of the Foley urinary catheter and cut the tubing ±5 cm from the sample port and insert the stopcock. *Strict asepsis must be maintained throughout the procedure.*
4. Connect the infusion line from the water manometer to the hub of the drainage bag stopcock.
5. Attach a 60 ml luer tip syringe to the injection port of that infusion line and draw up a maximum of 25 ml of saline.[1]
6. Place the patient in a supine position.[1,11]
7. The patient may require additional sedation if there is abdominal muscle straining.[1]
8. Position the manometer stopcock so that it is 'open to the bladder' and 'open to air.'
9. Position the drainage bag stopcock, so that it is 'closed to the drainage bag' (i.e. open to the bladder and manometer).
10. Inject the saline slowly into the bladder, while pinching the infusion line just above the injection port (to ensure that the saline is injected into the bladder).
11. Wait for 30 - 60 seconds before measuring (this allows for bladder detrusor muscle relaxation).[1]
12. Unclamp the infusion line and watch the fluid column rise until it is equivalent to the pressure in the abdomen.
13. The pressure should be measured at *end*-expiration (measuring end-expiration enhances the reproducibility and accuracy of the measurements because it minimises the additional influence of thoracic pressures on the reading).[11]
14. Beware of air in the system – it can falsely elevate the reading.
15. Once the reading has been obtained, turn the drainage bag stopcock so that the fluid instilled can drain from the bladder into the urine drainage bag.
16. The system can be left in place for subsequent measurements[11] or it can be disconnected, and both the stopcock and the line can be capped with a luer lock.
17. Recommendations are that a baseline IAP is established in patients at risk and serial measurements are taken every 4 hours while the patient is critically ill.[1]
18. Re-position the patient and ensure the head of the bed is 30 - 45° (unless contraindicated).
19. IAP should be expressed in mmHg as IAH is graded in mmHg.
20. To convert from cm $H_2O$ to mmHg, use the following equation:

$$\frac{\text{IAP (cm } H_2O)}{1.36} = \text{IAP (mmHg)}$$

*Note:* IAP estimated via the urinary bladder is directly affected by the amount of fluid in the bladder. Injecting too much fluid into a non-compliant bladder will raise the intra-vesical pressure and therefore overestimate IAP.[6] Current recommendations are that a maximum of 25 ml of saline is instilled in the bladder (1 ml/kg for children up to 20 kg) for the purpose of measurement.[1]

closure technique did not prevent the occurrence of ACS. Results of the study showed that 1 in 4 patients developed ACS despite having an open abdomen. The onset of ACS occurred between 1.5 and 12 hours after the placement of the vacuum-packed dressing.

Both studies highlight that IAP monitoring is still mandatory despite abdominal decompression.

## Conclusion

IAP should be measured according to a standardised protocol in all patients at risk of developing IAH/ACS and should not be used to confirm the presence of ACS in a patient who develops a tense abdomen with clinical deterioration.

Further studies are required to compare the effects of restrictive versus non-restrictive closure devices for the management of the open abdomen.

There are commercial IAP measurement sets available; however, the revised method developed by Miller and colleagues at Groote Schuur is simple, easy to use and affordable and therefore more appropriate for the South African setting.

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