BMJ Best Practice

Abdominal compartment syndrome

Straight to the point of care

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Summary

Abdominal compartment syndrome is most commonly due to excessive fluid resuscitation (>5 L in 24 hours) or massive blood transfusion (>10 units in 24 hours).

Clinical signs are non-specific and appear late. Classical findings are of increased airway pressure, decreased urine output, and a tense abdomen.

Diagnosis depends on proactive monitoring of intra-abdominal pressure (IAP) in patients with risk factors.

Medical options to decrease IAP include evacuation of intra-luminal contents, optimisation of fluid balance, correct body positioning, adequate analgesia, and, in severe cases, neuromuscular blockade.

Definitive treatment is surgical abdominal decompression; reserved for patients in whom medical interventions fail.

Fatal if left untreated. Even with treatment, mortality is high.

Definition

Abdominal compartment syndrome (ACS) is a sustained IAP over 20 mmHg, with or without an abdominal perfusion pressure below 60 mmHg, that is associated with new organ dysfunction or failure.[1]

Epidemiology

Historically, the prevalence of ACS was most frequently studied in the context of trauma, where it has been shown to occur in 1% of all general trauma admissions and 5% to 15% of trauma ICU admissions.[2] However, improvement in resuscitation and operative strategies in trauma patients has significantly reduced the prevalence of ACS.[3] Currently, in patients with known risk factors, intra-abdominal hypertension can be found in approximately 25% of ICU admissions with almost 3% having ACS.[4] Mortality rate is high, ranging from 25% to 75%, due to the presence of multi-organ failure and severe underlying injuries.[2] The mortality associated with raised intra-abdominal pressure itself is attributable to ACS.[2]

Aetiology

Intra-abdominal hypertension and ACS share the same aetiology, and the clinical distinctions are points along a continuum. The underlying conditions that produce elevated intra-abdominal pressure (IAP) are divided into primary and secondary causes.

Primary causes are due to decreased abdominal compliance, presence of an intra-abdominal or retro-peritoneal injury, or a pathological process. These are less common than the secondary causes.

- Decreased abdominal compliance: processes that decrease abdominal compliance (i.e., the elasticity of the abdominal wall and diaphragm), such as severe obesity, burns with abdominal wall eschars, and severe ventilator dyssynchrony with use of accessory muscles, can significantly increase IAP.[5]
- Intra-abdominal infection/inflammation: infections leading to generalised peritonitis can cause intensive inflammation of the peritoneal surfaces and gut. Significant fluid resuscitation and surgical intervention is often required, which can result in gut oedema and the generation of peritoneal fluid. This is particularly prominent in the paediatric population, where infectious enterocolitis is a significant risk factor for the development of ACS in children.[6] The inflammatory response in severe acute pancreatitis leads to ACS in approximately 40% of cases, with a significant increase in mortality.[7] [8]
- Haemoperitoneum: this can be produced by a ruptured abdominal aortic aneurysm, arterial or venous trauma, or ruptured hepatic tumours. These patients require fluid resuscitation and massive transfusion and will therefore also have gut oedema and ascites. A haemoperitoneum can produce IAPs high enough to cause ACS. However, because fluid resuscitation is instigated early, haemoperitoneum is more commonly seen in combination with ascites and gut oedema.
- Ileus: any process that decreases or impairs the normal transit of bowel contents (paralytic, mechanical, or pseudo-obstructive ileus) can produce accumulation of luminal contents leading to bowel distension and an increase in IAP.
- Pneumoperitoneum: this can arise from progression of a pathophysiological process, such as peptic ulcer disease or diverticulitis, that leads to a perforated viscus. It is also intentionally induced prior to laparoscopy, but ACS will only develop if the upper pressure limit is set inappropriately high.
- Liver cirrhosis: patients with higher amounts of ascites at baseline are at higher risk of developing ACS if abdominal pressure is increased by another cause.

Secondary causes are due to tense ascites or oedema of an otherwise normal bowel.

- Excess fluid resuscitation is the most common cause of ACS, usually with a significant crystalloid component.[9] In this context 'excess' is defined as more than 3 L of intravenous fluid resuscitation within a 24-hour period. This level of resuscitation is frequently required in patients with sepsis, severe trauma, bleeding, burns, or coagulopathies.[10] [11]

- Massive blood transfusion protocols (>10 units in 24 hours) are also common causes; these are usually given to patients with severe traumatic injury or post-traumatic coagulopathy.

In practice, many primary causes require fluid resuscitation and/or massive transfusion, which further increase gut oedema and the generation of peritoneal fluid. These patients therefore have mixed primary and secondary causes for increased IAP.

Pathophysiology

The abdominal compartment is bound inferiorly by the pelvic floor, circumferentially by the abdominal wall, and superiorly by the diaphragm. Although the diaphragm anatomically divides the chest and abdomen, it is not a rigid barrier and therefore allows pressures to be transmitted from the abdomen to the torso. In normal circumstances, the intra-abdominal pressure (IAP) is 5 to 7 mmHg. However, certain physiological states are associated with a higher IAP, without adverse consequences. This may be seen in pregnant patients and those with a BMI of 30 or above.[2] [12] The abdominal compartment is non-expandable, and a rise in IAP therefore impairs blood flow to intra-compartmental tissues. The pathogenesis of ACS evolves in several stages:

- An underlying process increases IAP, impairing arterial blood flow to intra-abdominal tissues.
- A threshold pressure is reached at which venous resistance starts to increase, causing venous congestion and a further increase in intra-compartmental pressure. This is a self-propagating cycle.
- When the IAP is greater than 20 mmHg, capillary perfusion falls, leading to tissue ischaemia. Ischaemia causes capillary leak, leading to increased extravascular fluid loss, a further increase in intra-abdominal volume, and further elevation of IAP. This cycle is also self-propagating.
- Decreased capillary perfusion results in impaired function of organs within the abdominal compartment. Liver and renal function are impaired. Urine output falls, progressing from oliguria to anuria.
- Expansion of abdominal volume displaces the diaphragm, decreasing pulmonary compliance and increasing the airway pressure required to maintain tidal volume.
- Elevated IAP impairs venous return to the heart, decreasing cardiac output and impairing systemic perfusion.
- Impaired cardiac output and increased intrathoracic pressure from elevated IAP can decrease cerebral venous outflow and decrease cerebral perfusion pressure.[13]
- Inadequate tissue perfusion leads to multi-organ failure and death.

Classification

Classification according to cause

Primary

- Due to decreased abdominal compliance (i.e., the elasticity of the abdominal wall and diaphragm), presence of an intra-abdominal or retro-peritoneal injury, or a pathological process.

Secondary

- Due to tense ascites or oedema of otherwise normal bowel. This is the most common form.

Recurrent

- Recurrence of abdominal compartment syndrome (ACS) after treatment for either primary or secondary ACS.

Case history

Case history #1

A 20-year-old man presents in severe haemorrhagic shock due to multiple blunt trauma following a motor vehicle crash. He is taken to the operating room for emergency surgery, where he is found to have a completely shattered spleen and a 3-cm liver laceration. He undergoes a splenectomy and damage-control laparotomy, and the abdominal wound is closed only at the skin with a running suture. He is transferred to the surgical ICU for ongoing resuscitation, but over the next 8 hours is noted to have decreasing urine output, worsening hypotension that is becoming refractory to fluid boluses, and poor oxygenation. Physical examination reveals poor peripheral perfusion, moderate abdominal distension, but no signs of acute wound infection.

Case history #2

A 70-year-old woman with known cirrhosis presents to the emergency department with severe hypotension and respiratory failure due to sepsis from a urinary tract infection. She undergoes aggressive fluid resuscitation that continues in the medical ICU. In the fourth hour of resuscitation she is noted to have oliguria, hypoglycaemia, and worsening metabolic and respiratory acidosis. Physical examination reveals a tense, distended abdomen with clinically apparent ascites. Her extremities are warm and well perfused.

Approach

ACS is most frequently seen in critically ill ICU patients. The diagnosis of ACS can be divided into 3 components: identification of patients at risk; recognition of clinical signs associated with the transition from intra-abdominal hypertension to ACS; and proactive measurements to confirm the suspected diagnosis.

Identifying risk factors enables earlier detection of intra-abdominal hypertension and ACS.[18] The classical clinical findings are of increased airway pressure, decreased urine output, and a tense abdomen on physical examination. However, these findings are non-specific and often only clinically apparent once severe ACS is present. It is recommended that early and protocolised intra-abdominal pressure (IAP) monitoring be done in all those who present with two or more risk factors for intra-abdominal hypertension (IAH) or ACS.[1] Continuous IAP monitoring should be initiated as soon as intra-abdominal hypertension or ACS diagnosis is suspected. Factors involved in determining the frequency of IAP monitoring include the baseline pressure measurement, the ability to alter aetiological and risk factors, and the evolution of the clinical course.

History and examination

The key to diagnosis is to start monitoring IAP early in patients who are at risk. The main aim of the history is to identify these patients. Risk factors include excessive fluid resuscitation (>3 L in 24 hours), massive blood transfusion (>10 units in 24 hours), recent intra-abdominal infection/inflammation (especially peritonitis and pancreatitis), haemoperitoneum, ileus, pneumoperitoneum, loss of abdominal domain, diminished abdominal compliance, and a history of cirrhosis. The term 'loss of abdominal domain' refers to a situation in which more of the viscera are located outside the abdominal cavity than inside because the abdominal cavity is unable to accommodate all of the abdominal contents within its fascial boundaries.

The main clinical sign is a tense, distended abdomen. Patients also have difficulty maintaining minute ventilation and oxygenation due to transmission of pressure to the torso. Later signs include oliguria progressing to anuria, hypotension, and an increase in peak airway pressure.

Monitoring of IAP

Protocolised monitoring of IAP is the only way to establish a diagnosis and should be done in all patients with two or more risk factors for IAH or clinical signs of ACS.[1] If baseline IAP is elevated, serial IAP measurements are performed.[1] The pressure should be monitored at least every 3 to 4 hours, but more frequent measurements are required if the pressure is elevated or the clinical condition is rapidly changing. This gives clinicians the opportunity to intervene early to prevent disease progression.

The abdominal perfusion pressure is the difference between the mean arterial BP and the IAP. It must be 60 mmHg or greater for organ perfusion to be adequately maintained. The diagnosis is established as follows:[1]

- IAP greater than 12 mmHg indicates intra-abdominal hypertension, which is graded by severity: grade I (12 to 15 mmHg), grade II (16 to 20 mmHg), grade III (21 to 25 mmHg), and grade IV (>25 mmHg).
- IAP greater than 20 mmHg (grade III or IV), with or without an abdominal perfusion pressure below 60 mmHg, with new-onset organ failure or dysfunction indicates ACS.
- In paediatric patients, the exact IAP that signals the transition from intra-abdominal hypertension to ACS is not currently known, but an IAP greater than 10 mmHg with new-onset organ dysfunction

has been proposed for children.[8] However, further research is needed to fully establish a threshold.

IAP is measured using a pressure transducer positioned in the bladder.[19] [20] [21] The bladder is catheterised and 25 mL of sterile saline is instilled into the bladder. The catheter tubing is clamped, and a needle is inserted via the specimen collection port proximal to the clamp or via a needleless side port and attached to a calibrated pressure transducer (zeroed at the level of the mid-axillary line). An increase in the measured pressure with gentle palpation of the abdomen confirms a good fidelity of pressure transduction. To ensure accuracy and reproducibility, the pressure should be measured at end-expiration with the patient completely supine, as slight changes in the elevation of the head of the bed can significantly increase IAP measurements.[2] [22] [23] Abdominal muscle contractions should be absent, which can be achieved with the use of sedatives and, in rare instances, pharmacological paralysis. Also, the utilisation of positive end expiratory pressure does not seem to affect measured IAP.[24]

Modifications of this system allow continuous measurement.[25] [26] [27] Devices are now commercially available that allow for measurement of trans-vesicular pressure without the use of a needle puncture and the associated risks of needle sticks.[28] IAP can be assessed via other intra-abdominal vantage points including the inferior vena cava, the rectum, or the abdominal cavity itself.[29] [25] However, these methods are not routinely recommended.

Monitoring of organ function

Patients develop impaired organ function, which must be monitored. It is important to monitor the adequacy of perfusion, alterations in acid-base status, and organ function. Unexplained or worsening acidosis, impaired pulmonary function, and alterations in renal function are the most common manifestations of organ dysfunction seen with ACS. The following investigations are required:

- Serum electrolytes, urea, and creatinine are required to monitor renal function and associated electrolyte balance.
- Arterial blood gas measurements are required to monitor acid-base disturbances. Acidosis is common, which is usually metabolic or mixed metabolic and respiratory.
- BP should be monitored regularly; invasive monitoring may be required. It should be noted that an increase in IAP can lead to unreliability of measurements of central venous pressure or pulmonary artery occlusion pressure.
- Oxygen saturation should be monitored in all patients.
- Direct measurement of peak airway pressure is possible in mechanically ventilated patients; increased peak airway pressure is a late sign.

Radiological investigations

Abdominal CT scan

- This is frequently used to identify underlying intra-abdominal pathological conditions. It is not used to assess a rise in IAP. However, a careful interpretation of the CT study can reveal associated signs of raised IAP. The key sign is 'circularisation' of the transverse abdominal contour, as defined by increased ratio of the antero-posterior:transverse diameter, due to the increased pressure. If this sign is found, measurement and monitoring of IAP should be considered to provide a definitive diagnosis.[30] [31] [32] [33]

Abdominal ultrasound

- This is used to identify underlying intra-abdominal pathological conditions and, increasingly in the ICU setting, to assess intravascular volume. If the ultrasound shows the presence of intra-abdominal fluid, renal vein compression, or inferior vena cava compression, measurement and monitoring of IAP should be considered to exclude intra-abdominal hypertension and ACS.[34]

History and exam

Key diagnostic factors

presence of risk factors (common)

- Key risk factors include excessive fluid resuscitation (>5 L in 24 hours), massive blood transfusion (>10 units in 24 hours), recent abdominal infection (especially peritonitis), haemoperitoneum, and ileus.

abdominal distension (common)

- A tense, distended abdomen is the most important clinical sign.

oliguria (common)

- Urine output is decreased due to impaired renal blood flow and decreased urine production.

increased respiratory effort (common)

- Patients have difficulty maintaining minute ventilation and oxygenation due to a decrease in pulmonary compliance produced by transmission of abdominal pressure to the torso.
- Atelectasis can also occur.

hypotension (common)

- Increased intra-abdominal pressure compresses the inferior vena cava and impairs venous return to the heart, leading to decreased pre-load and cardiac output.

Risk factors

Strong

excessive fluid resuscitation (>3 L in 24 hours)

- Oncotic and hydrostatic effects of large-volume fluid resuscitation lead to increased amounts of extracellular and extravascular fluid, causing gut oedema and ascites. Both increase intra-abdominal pressure.

massive blood transfusion (>10 units in 24 hours)

- Hydrostatic effects of massive blood transfusion lead to increased amounts of extracellular and extravascular fluid, causing gut oedema and ascites. Both increase intra-abdominal pressure.

decreased abdominal compliance

- Abdominal compliance is determined by the elasticity of the abdominal wall and diaphragm. Conditions such as severe obesity, burns with abdominal wall eschars, and severe ventilator dyssynchrony with use of accessory muscles can decrease abdominal compliance, which can significantly increase IAP.[14]

intra-abdominal infection/inflammation

- Intra-abdominal infections, primarily those causing generalised peritonitis, can lead to an intensive inflammatory response in the peritoneal surfaces and the gut.
- Significant fluid resuscitation and surgical intervention are often required, both of which increase gut oedema and the generation of peritoneal fluid.
- The inflammatory response in severe acute pancreatitis leads to ACS in approximately 40% of cases, with a significant increase in mortality.[7] [8]

haemoperitoneum

- Patients require fluid resuscitation and massive transfusion, and will therefore also have gut oedema and ascites.
- Haemoperitoneum can produce intra-abdominal pressures high enough to cause ACS. However, because fluid resuscitation is instigated early, haemoperitoneum in combination with ascites and gut oedema is more common.

Weak

ileus

- Any process that decreases or impairs the normal transit of bowel contents can produce accumulation of luminal contents leading to distension of the bowel and an increase in intra-abdominal pressure.

pneumoperitoneum

- Can arise from perforation of a peptic ulcer or diverticulum, or it can be iatrogenic prior to laparoscopy. In the case of perforation, peritoneal inflammation increases, with consequent intestinal oedema and peritoneal fluid production. In the iatrogenic case, induced pneumoperitoneum generally only leads to ACS when the upper pressure limit is set inappropriately high.

loss of abdominal domain

- The term 'loss of abdominal domain' refers to a situation in which more of the viscera are located outside the abdominal cavity than inside because the abdominal cavity is unable to accommodate all of the abdominal contents within its fascial boundaries.
- Patients who are undergoing a repair of large ventral hernia with previous loss of domain are at risk for developing increased abdominal pressure after hernia closure.[15]

comorbid cirrhosis

- Patients with advanced cirrhosis may have large volumes of ascitic fluid present at baseline and are at increased risk if intra-abdominal pressure is increased by another cause.

retroperitoneal haematoma

- Mass effect from the haematoma causes an effective loss of intra-abdominal volume, with a resultant increase in IAP. It has been described in patients who have spontaneous retroperitoneal bleeds from anticoagulation and from endovascular procedures that cause bleeding.[16] [17]

Investigations

1st test to order

Test	Result
trans-bladder measurement of intra-abdominal pressure • Performed by treating physician at bedside. Baseline measurements should be performed in patients with clinical signs of raised intra-abdominal pressure, or two or more risk factors for intra-abdominal hypertension.[1] • Should be monitored at least every 3 to 4 hours. More frequent monitoring required if pressure is elevated or clinical course is changing rapidly. • Intra-abdominal pressure (IAP) >12 mmHg indicates intra-abdominal hypertension, graded as follows: grade I (12 to 15 mmHg), grade II (16 to 20 mmHg), grade III (21 to 25 mmHg), grade IV (>25 mmHg).[1] • Abdominal perfusion pressure is the difference between mean arterial BP and IAP. Must be 60 mmHg or greater for organ perfusion to be adequately maintained. • IAP >20 mmHg with or without abdominal perfusion pressure <60 mmHg, with new-onset organ dysfunction or failure is diagnostic for ACS. • In paediatric patients, the exact IAP that signals the transition from intra-abdominal hypertension to ACS is not currently known, but an IAP >10 mmHg with new-onset organ dysfunction has been proposed for children.	**elevated**
oxygen saturation • Decreased pulmonary compliance or atelectasis can lead to hypoxia.	**normal or decreased**
serum urea and creatinine • Required to monitor renal function and associated electrolyte balance. • Ratio of urea to creatinine is usually >20:1, a classical indicator of impaired renal perfusion. • Renal failure, due to impaired renal perfusion, is very common.	**elevated**
arterial blood gases • Metabolic acidosis due to organ ischaemia is the most common abnormality. • Decreased pulmonary compliance or atelectasis can lead to a respiratory component with hypoxia and hypercapnia.	**metabolic acidosis or mixed metabolic and respiratory acidosis**

Other tests to consider

Test	Result
peak airway pressure • Direct measurement of peak airway pressure is possible in mechanically ventilated patients. • Increased peak airway pressure is a late sign.	**normal or elevated**
abdominal CT scan • Frequently used to identify underlying intra-abdominal pathological conditions. • Not used to assess intra-abdominal pressure, but a careful interpretation of CT study can reveal circularisation of the transverse abdominal contour due to increased pressure as an incidental finding. • If found, measurement of intra-abdominal pressure should be considered.[30] [31] [32]	**'circularisation' of transverse abdominal contour**
abdominal ultrasound • Used to identify underlying intra-abdominal pathological conditions and, in ICU, to assess intravascular volume. • May show intra-abdominal fluid, renal vein compression, or inferior vena cava compression. • If found, measurement of intra-abdominal pressure should be considered.[34]	**intra-abdominal fluid, compression of renal veins and/or inferior vena cava**

Emerging tests

Test	Result
measurement of intra-abdominal pressure via vena cava, rectum, or abdominal cavity • Can be used to assess intra-abdominal pressure.[25] [29] However, not routinely recommended.	**elevated**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Shock	• Shock can be clinically indistinguishable from ACS and significant overlap exists, because patients develop ACS from excess fluid resuscitation given to treat hypovolaemic or septic shock.	• Intra-abdominal pressure (IAP) is normal.
Acute tubular necrosis	• Acute tubular necrosis is clinically indistinguishable from ACS, although may have a history of exposure to nephrotoxic agents. • This condition can also occur as a complication of ACS.	• The IAP is normal. • Urinalysis may show granular casts. • Renal biopsy shows characteristic features.
Acute renal failure	• Renal failure is clinically indistinguishable from ACS. • The condition presents with oliguria in many of the same patient populations that are at increased risk for ACS. • It can also occur as a complication of ACS.	• The IAP is normal. • Renal ultrasound shows dilated renal calyces (suggesting obstruction) or reduced corticomedullary differentiation. • Renal biopsy reveals intra-renal causes.
Adult respiratory distress syndrome	• Patients have cough with expectoration of frothy pulmonary oedema. • Basilar or diffuse rales are present on chest auscultation. • Patients generally require higher levels of oxygen and/or PEEP to maintain oxygen saturation >90% than patients with ACS.	• IAP is normal or mildly elevated (due to pressure transmission from torso to abdomen). • Chest x-ray shows contusion and new bilateral interstitial infiltrates suggestive of pulmonary oedema.

Criteria

Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines (World Society of the Abdominal Compartment Syndrome)[1]

An intra-abdominal pressure (IAP) greater than 12 mmHg indicates intra-abdominal hypertension, graded as follows:

- Grade I (12 to 15 mmHg)

- Grade II (16 to 20 mmHg)
- Grade III (21 to 25 mmHg)
- Grade IV (>25 mmHg).

A sustained IAP greater than 20 mmHg, with or without an abdominal perfusion pressure below 60 mmHg, with new-onset organ dysfunction or failure is diagnostic of ACS. [World Society of the Abdominal Compartment Syndrome] (http://www.wsacs.org)

In paediatric patients, the exact IAP that signals the transition from intra-abdominal hypertension to ACS is not currently known, but an IAP greater than 10 mmHg with new-onset organ dysfunction has been proposed for children.[8] However, further research is needed to fully establish a threshold.

Approach

Intra-abdominal hypertension should ideally be detected and treated before abdominal compartment syndrome (ACS) develops. A series of medical manoeuvres exists to reduce intra-abdominal pressure (IAP), and these should always be employed regardless of whether the patient presents with intra-abdominal hypertension or ACS. These include:

- Treatment of the underlying cause.
- Supportive care with monitoring of IAP, analgesia, and correct body positioning.
- Optimisation of fluid balance. This involves avoidance of excessive fluid resuscitation, the use of colloids and hyper-osmotic agents and 25% human albumin, and treatment with diuretics.
- Evacuation of intra-luminal contents. Small bowel contents can be evacuated using nasogastric (NG) gastroprokinetic agents and limitation of enteral feeding. Large bowel contents can be evacuated by a rectal tube, coloprokinetic agents, enemas, and, as a last resort, colonoscopic decompression.

Adherence to a defined algorithm of early surveillance and intervention has led to improved outcomes in patients with intra-abdominal hypertension/ACS.[35] The emphasis on the above strategies will depend on the underlying cause. For example, if the patient has received excessive fluid resuscitation, aggressive management of fluid balance will be pursued. However, if the underlying cause is ileus, evacuation of intra-luminal contents will be pursued.

The definitive treatment of ACS, surgical decompression, should only be employed if and when medical therapy proves to be inadequate, either by paracentesis or by surgery. Some patients may require mechanical ventilation to overcome the effects of pressure transmission to the torso from the abdomen. Neuromuscular blockade is an extreme measure that can be used to reduce abdominal wall compliance. Patients with renal failure may require dialysis or haemofiltration to treat their renal failure and restore neutral fluid balance.

Treatment of underlying cause

A primary cause such as abdominal bleeding or trauma, packing, intra-abdominal infection/inflammation, ileus, pneumoperitoneum, or bowel ischaemia should be managed; most of these conditions require surgical intervention, which may already have been performed prior to the development of ACS.

Secondary causes are almost always related to excessive fluid resuscitation and massive blood transfusions, which require optimisation of fluid balance. Escharotomies may need to be performed in patients with thermal injuries.

Supportive care

All patients require supportive care with regular monitoring of IAP and oxygen saturations. Adequate analgesia and sedation should be provided. In addition to providing pain relief, this helps to relax the abdominal musculature and improves abdominal compliance.

Although important in other critical care bundles, elevated head of bed may worsen abdominal compliance; therefore, lowering bed tilt to less than 30° should be considered in patients with suspected poor abdominal compliance. IAP may decrease if the patient is placed flat (supine position), or even placed into the reverse Trendelenburg's position (patient flat, head up, feet down). Reverse Trendelenburg may specifically mitigate diaphragmatic displacement and improves lung compliance. It may also improve abdominal compliance. Some patients may not tolerate a reverse Trendelenburg's position, due to a drop in mean arterial pressure or cardiac output.[10] Tight or constrictive clothing places pressure on

the abdomen and should be removed. Any bandages that increase the tension on the abdominal cavity should also be removed where possible.

The need for organ support should be assessed in all patients. Some patients require mechanical ventilation to overcome the effects of pressure transmission from the abdomen to the thorax. Abdominal perfusion pressure should be maintained at or above 60 mmHg, which may require the use of vasopressors.

Optimisation of fluid balance

Positive fluid balance, if present, should be corrected to return the patient to neutral fluid balance. Careful assessment of volume status and cardiac function is required.

Excessive fluid resuscitation should be avoided, and employing a restrictive fluid resuscitation strategy has clear benefits that outweigh aggressive fluid removal in the setting of elevated IAP.[24] Colloids and hypertonic crystalloids should be used rather than isotonic or hypotonic crystalloids. To exert intravascular osmotic pressure and return fluid to the intravascular compartment, 25% human albumin can be given, although its effectiveness has not been confirmed in clinical trials.

Diuretic therapy can be successfully employed in fluid-overloaded patients whose kidneys will respond to these agents. Care must be taken to monitor pre-load to avoid exacerbating shock in critically ill patients who are hypovolaemic. However, excessive fluid loss is easier to correct than excessive fluid administration. It is better to start with a higher dose so that effective diuresis is achieved quickly. The major toxicity of diuretic agents, particularly if high doses are used, is ototoxicity. These agents should therefore be administered via infusion pump at a controlled rate.

Measurement of the fluid status with central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) can be erroneously elevated because of the transmural effect of increased IAP on these measurements. The abdomino-thoracic pressure transmission is estimated to be around 50% of the IAP. Therefore, calculating CVP = CVP-(IAP/2) and the PAOP = PAOP-(IAP/2) gives better estimates of these pressures.[36]

Evacuation of intra-luminal contents

Evacuation of intra-luminal gastric contents using an NG tube reduces pressure exerted by accumulation of gastric contents. An NG tube is usually already present in ICU patients and should be strongly considered. NG decompression is more likely to decrease pressure when gastric distension is from fluid, rather than air (more compressible).

Small bowel contents can be evacuated using an NG tube, gastroprokinetic agents, and limitation of enteral feeding. Gastroprokinetic agents (e.g., erythromycin) should also be considered, especially if ileus is the underlying cause. Limitation or discontinuation of enteral feeding may be considered if these measures are inadequate.

Colonic decompression may have a minor impact on IAP. As with NG decompression, removal of fluid or impacted stool is most likely to be of benefit. Coloprokinetic agents (e.g., neostigmine) and enemas should be considered if the response to tube placement is inadequate. Colonoscopic decompression may be considered as a final option.

Abdominal decompression

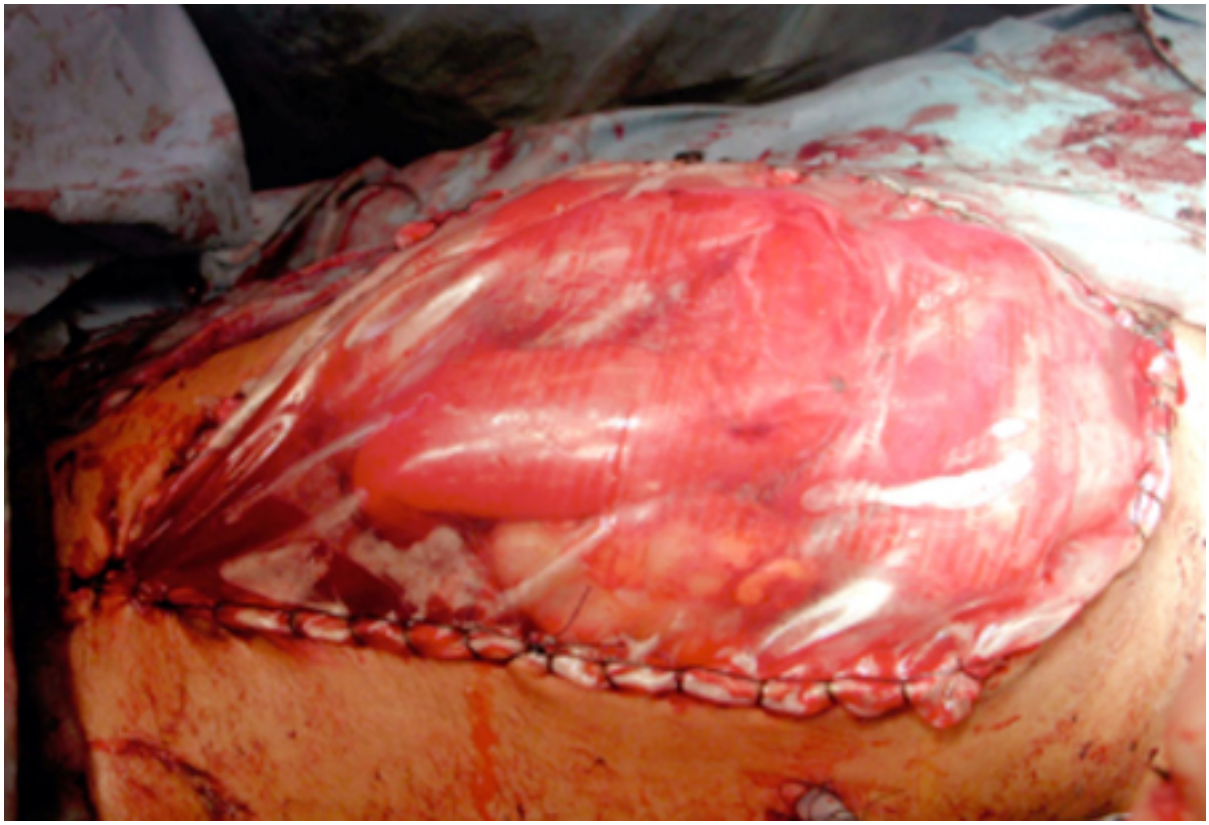
Paracentesis with percutaneous decompression has been described primarily in patients with burns. A peritoneal lavage or dialysis catheter is placed percutaneously into the peritoneal cavity and set to gravity drainage. If free fluid volumes are large, IAP is quickly lowered. Pathophysiological processes that stimulate production of free peritoneal fluid are more amenable to this approach. Bedside ultrasound may facilitate identification and drainage of fluid collections.[37] Successful percutaneous decompression has been associated with fluid drainage of >1000 mL or a decrease in IAP of >9 mmHg in the first 4 hours post-decompression.[38] If ACS persists after percutaneous drainage, then surgical treatment should be given immediately.[39]

A delay in the definitive treatment of ACS with surgical decompression often leads to a progressive increase in the risk of mortality.[24]

Open abdominal decompression is the definitive treatment of ACS but is reserved for patients in whom other interventions have failed.[10] [40] [41] [42] Hypotension, oliguria, and elevated airway pressure, if present, resolve rapidly after the procedure. Although general anaesthesia and the presence of a surgeon are required, the procedure can be safely performed at the bedside in the ICU.[39] This is an important advantage, because many patients with ACS are clinically unstable.

It is accomplished by performing a midline laparotomy incision. Care should be taken to avoid inadvertent injury to the bowel, which may bulge as soon as the peritoneal cavity is entered. The most restrictive abdominal layer is the fascial layer, and this should be opened generously in cephalad and caudad directions. A variety of temporary closure methods have been described, but in most cases both the fascia and the skin are left open.[43] [44] [45] A dressing must be applied to prevent dessication of the viscera. More recently there has been considerable enthusiasm for negative pressure/vacuum therapies, which have the added benefit of removing fluid that might otherwise re-accumulate and cause a recurrence of ACS. Additionally, the utilisation of negative pressure therapy may reduce the transmission of inflammatory mediators to the blododstream, potentially slowing the progression of multiorgan dysfunction.[24]

Patients undergoing decompressive laparotomy will later require permanent abdominal wound closure. Options range from delayed primary fascial closure and various flap/fascial release measures[46] [47] to mobilisation of skin flaps or split-thickness skin grafting. All of these measures are associated with a ventral hernia that requires separate, late management.[48] The utilisation of negative pressure wound therapy devices has increased in recent years and reports suggest that these devices are associated with increased fascial closure rates and decreased length of hospital stays compared with traditional temporary abdominal closures.[49] [50] However, there is potential for wound vacuum devices to cause tertiary ACS if the vacuum pressure is too high; therefore, ongoing monitoring should continue and medical management of intra-abdominal hypertension/ACS should persist after the abdomen is opened as this will facilitate the earlier closure of the fascia.



'Bogota bag' temporary abdominal closure after surgical decompression of abdominal compartment syndrome

From the personal collection of Michaela A. West, MD, PhD

Additional treatments

Neuromuscular blockade

- Complete neuromuscular blockade can be given as a last resort to patients to decrease the compliance of the abdominal wall. Patients will require airway control and mechanical ventilation.
- Sedative and neuromuscular blocking agents may decrease BP and/or cardiac output, which may be problematic in hypotensive patients who remain in shock.
- It is important to monitor the adequacy of neuromuscular blockade, especially if IAP increases while patients are receiving this treatment.[51] [52]

Dialysis

- This may effectively mobilise excess fluid and is particularly useful in the setting of acute or chronic renal failure, when diuretic therapies are not feasible. Dialysis takes hours to days to be effective, but under some circumstances net removal of even 1 to 2 litres of excess fluid can significantly lower IAP.
- Disadvantages include the need for higher flow cannulae and the complications that may arise from their insertion, such as infection. Also, dialysis requires trained personnel and specialised equipment, which, while widely available, is not immediately accessible in all ICUs.

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
all patients		
	1st	treatment of underlying cause
	plus	supportive care with monitoring
	plus	optimised fluid management
	plus	mechanical evacuation of intra-luminal contents
	adjunct	pharmacological evacuation of intra-luminal contents
	adjunct	mechanical ventilation ± vasopressors
■ no response to initial treatment	plus	percutaneous abdominal decompression
	adjunct	surgical abdominal decompression
	adjunct	neuromuscular blockade + mechanical ventilation
	adjunct	dialysis

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

all patients

1st **treatment of underlying cause**

» A primary cause such as abdominal bleeding or trauma, packing, intra-abdominal infection/inflammation, ileus, pneumoperitoneum, or bowel ischaemia should be managed; most of these require surgical intervention, which may already have been performed prior to the development of abdominal compartment syndrome.

» Secondary causes are almost always related to excessive fluid resuscitation and massive blood transfusions, which require optimisation of fluid balance. Escharotomies may need to be performed in patients with thermal injuries.

plus **supportive care with monitoring**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 2.5 to 10 mg intramuscularly every 2-4 hours when required; or 0.8 to 10 mg/hour intravenously via infusion pump

» All patients require regular monitoring of intra-abdominal pressure and oxygen saturations.

» Adequate analgesia and sedation should be provided. In addition to providing pain relief, this helps to relax the abdominal musculature and improves abdominal compliance.

» The bed tilt should ideally be <30° to improve abdominal wall compliance. Prone positioning should be avoided. The reverse Trendelenburg's position (patient placed flat, head up, feet down) improves lung and abdominal compliance, but may not be tolerated by some patients due to a drop in mean arterial pressure or cardiac output.[10]

» Tight or constrictive clothing places pressure on the abdomen and should be removed. Restrictive bandages causing tension on the abdominal cavity should also be removed where possible.

Acute

» The need for organ support should be assessed in all patients.

plus **optimised fluid management**

Treatment recommended for ALL patients in selected patient group

Primary options

» furosemide: 20-40 mg intravenously initially, followed by 10-160 mg/hour continuous infusion

OR

» bumetanide: 1 mg intravenously initially, followed by 0.5 to 2 mg/hour continuous infusion

» Excessive fluid resuscitation should be avoided, and employing a restrictive fluid resuscitation strategy has clear benefits that outweigh aggressive fluid removal in the setting of elevated IAP.[24] Colloids and hypertonic fluids should be used rather than crystalloids. To return fluid to the intravascular compartment, 25% human albumin can be given, although its effectiveness has not been confirmed in clinical trials.

» Diuretic therapy is successful in reducing fluid overload, but care must be taken to avoid exacerbating shock in critically ill patients who are hypovolaemic. It is better to start with a higher dose so that effective diuresis is achieved quickly; excessive fluid loss is easier to correct than excessive fluid administration. Diuretics should be administered via infusion pump at a controlled rate to avoid ototoxicity.

plus **mechanical evacuation of intra-luminal contents**

Treatment recommended for ALL patients in selected patient group

» Evacuation using a nasogastric tube reduces pressure exerted by accumulation of gastric and small bowel contents; removal of fluid rather than air is most likely to be of benefit.

» Colonic decompression may have a minor impact on intra-abdominal pressure. Removal of fluid or impacted stool is most likely to be of benefit.

» If the response to tube placement is inadequate, the patient may benefit from an enema.

Acute

» However, pharmacological treatment with gastro- and coloprokinetic agents to further evacuate the GI lumen is usually considered first, before enema therapy.

» Limitation or discontinuation of enteral feeding may also be considered if there is no response to tube placement and drug treatment.

» Colonoscopic decompression is a final option.

adjunct **pharmacological evacuation of intra-luminal contents**

Treatment recommended for SOME patients in selected patient group

Primary options

» erythromycin base: 250 mg intravenously every 6 hours

OR

» neostigmine: 0.5 mg intramuscularly three times daily

» Gastro- and coloprokinetic agents are usually considered if placement of nasogastric and rectal tubing is inadequate, especially if ileus is the underlying cause.

adjunct **mechanical ventilation ± vasopressors**

Treatment recommended for SOME patients in selected patient group

Primary options

» vasopressin: consult specialist for guidance on dose

OR

» noradrenaline (norepinephrine): consult specialist for guidance on dose

» Some patients require mechanical ventilation to overcome the effects of pressure transmission from the abdomen to the thorax.

» Abdominal perfusion pressure should be maintained at 60 mmHg or greater, which may require use of vasopressors.

» Consult specialist for guidance on choice of vasopressor and dose.

no response to initial treatment **plus** **percutaneous abdominal decompression**

MANAGEMENT

Acute

Treatment recommended for ALL patients in selected patient group

» Paracentesis with percutaneous decompression should be attempted first. Peritoneal lavage or dialysis catheter is placed percutaneously into the peritoneal cavity and set to gravity drainage. If free fluid volumes are large, intra-abdominal pressure (IAP) is quickly lowered. Successful percutaneous decompression has been associated with fluid drainage of >1000 mL or a decrease in IAP of >9 mmHg in the first 4 hours post-decompression.[38]

» If ACS persists after percutaneous drainage, surgical treatment should be given immediately.[39] A delay to definitive treatment with surgical decompresssion often increases the risk of mortality.[24]

adjunct **surgical abdominal decompression**

Treatment recommended for SOME patients in selected patient group

» Surgical decompression via a laparotomy is the definitive treatment but is reserved for patients in whom other interventions have failed.[10] [40] [41] Although general anaesthesia is required, the procedure can be safely performed at the bedside in the ICU.[39] This is an important advantage, because many patients with ACS are clinically unstable. It is accomplished by performing a midline laparotomy incision. In most cases both the fascia and the skin are left open and a dressing must be applied to prevent dessication of the viscera.[43] [44] [45] Patients undergoing decompressive laparotomy will later require permanent abdominal wound closure. Options include delayed primary fascial closure, various flap/fascial release measures,[46] [47] mobilisation of skin flaps or split-thickness skin grafting, and use of negative pressure wound therapy devices.[49] [50] Many patients develop a ventral hernia that requires separate, late management.[48]

adjunct **neuromuscular blockade + mechanical ventilation**

Treatment recommended for SOME patients in selected patient group

» Given as a last resort to patients to decrease compliance of the abdominal wall. Patients require airway control and mechanical ventilation.

Acute

» Sedative and neuromuscular blocking agents (e.g., pancuronium) may decrease BP and/or cardiac output, which may be problematic in hypotensive patients who remain in shock.

» It is important to monitor the adequacy of neuromuscular blockade, especially if intra-abdominal pressure increases while patients are receiving this treatment.[51] [52]

» Consult specialist for advice on choice of agent and dose.

adjunct **dialysis**

Treatment recommended for SOME patients in selected patient group

» Dialysis or haemofiltration may effectively mobilise excess fluid, and both are particularly useful in the setting of acute or chronic renal failure, when diuretic therapy is not feasible.

» Takes hours to days to be effective, but under some circumstances net removal of even 1 to 2 litres of excess fluid can significantly lower intra-abdominal pressure.

» Trained personnel and specialised equipment, while widely available, may not be immediately accessible in all ICUs.

Primary prevention

A high index of suspicion should be maintained in all patients with risk factors for elevated intra-abdominal pressure (IAP). The aim is to identify and treat intra-abdominal hypertension before it can progress to ACS. Patients at risk should be identified and protocolised monitoring of IAP instigated early. Excess fluid administration is the most common cause and can be avoided if appropriate endpoints of resuscitation are accurately assessed. Global endpoints include vital signs, cardiac output, pulmonary artery wedge pressure, lactate, and the base deficit. Local endpoints allow assessment of tissue-specific perfusion and include gastric tonometry and near-infrared spectroscopy.

Patient discussions

Patients with temporary abdominal closure will need instructions regarding local wound care if they have an open wound. Patients who have undergone decompressive laparotomy are at risk for any of the complications that are attendant on any major abdominal operation, including late infection and an increased risk for adhesive bowel obstruction. They should be instructed to seek immediate medical attention if they have a high fever, signs of GI bleeding, or severe abdominal pain. For the first 30 to 60 days, patients should avoid heavy lifting and contact athletics in view of the risk of a ventral hernia. If the fascia was not closed, lifting and activity restrictions should continue until the abdominal wall is repaired.

Monitoring

Monitoring

If patients have undergone surgical decompression, they will have ongoing requirements for local wound care. Dressings can be changed at the bedside in uncomplicated cases. Patients who required decompressive laparotomy and whose fascia could not be primarily closed will require surgical follow-up to determine the optimal means and timing of abdominal wall reconstruction.

ACS is an acute condition, and long-term monitoring is not necessary. Follow-up is, however, required if there was a primary underlying cause.

Many patients who require decompressive laparotomy develop a late ventral hernia requiring surgical correction. If raised intra-abdominal pressure can be reversed quickly, there is a window of approximately 7 to 10 days during which delayed fascial closure has a high success rate; if this window is missed, virtually all patients develop a hernia.

FOLLOW UP

Complications

Complications	Timeframe	Likelihood
acute renal failure	**short term**	**high**
The kidneys are particularly vulnerable to ischaemic injury; often some degree of renal dysfunction (elevated urea/creatinine). Exacerbation of renal dysfunction complicates management of fluid overload. Generally reversible and self-limiting, but more severe cases may require dialysis until renal function recovers. Can be a differential diagnosis as well as a complication; in this situation, intra-abdominal pressure (IAP) is normal.		
acute tubular necrosis	**short term**	**high**
The kidneys are particularly vulnerable to ischaemic injury, which can lead to acute tubular necrosis. Exacerbation of renal dysfunction complicates management of fluid overload. Generally reversible and self-limiting, but patients with more severe cases may require dialysis until renal function recovers. Can be a differential diagnosis as well as a complication; in this situation, IAP is normal.		
recurrent abdominal compartment syndrome (ACS)	**short term**	**medium**
Patients who have been treated for ACS may develop recurrent ACS. Best prevented by vigilant monitoring of IAP and early intervention if pressure increases or fails to decrease in response to definitive treatment. If recurrent ACS develops in patients not initially treated with surgical decompression, then it must be undertaken. If patients develop recurrent ACS after decompressive laparotomy, dressings should be checked and patients may require re-operation, along with more aggressive adjunctive measures. Recurrent ACS causes increased morbidity as compared with isolated single episodes of ACS, an example of the so-called 'second hit' phenomenon.[53]		
ischaemic bowel	**short term**	**medium**
Elevations of IAP can produce ischaemic injury to the abdominal viscera. Small and large intestines are most frequently involved. Physicians and surgeons should have a high index of suspicion for this complication if the IAP is very high, ACS is recognised late, or there is evidence of intestinal ischaemia at laparotomy or worsening sepsis, acidosis, or refractory shock. Treatment involves surgical exploration and resection, repair, or diversion.		
bowel perforation	**short term**	**medium**
Elevation of IAP can produce perforation of the small and large intestine.		

Complications	Timeframe	Likelihood
Physicians and surgeons should have a high index of suspicion for this complication if the IAP is very high, ACS is recognised late, or there is worsening sepsis, acidosis, or refractory shock. Treatment involves surgical exploration and resection, repair, or diversion.		
ventral hernia post-laparotomy	**variable**	**high**
Many patients who require decompressive laparotomy develop a late ventral hernia requiring surgical correction. If raised IAP can be reversed quickly, there is a window of approximately 7 to 10 days during which delayed fascial closure has a high success rate; if this window is missed, virtually all patients develop a hernia.		
large bowel obstruction post-laparotomy	**variable**	**medium**
Patients who have undergone decompressive laparotomy are at risk for any of the complications that are attendant on any major abdominal operation, including adhesive bowel obstruction.		
small bowel obstruction post-laparotomy	**variable**	**medium**
Patients who have undergone decompressive laparotomy are at risk for any of the complications that are attendant on any major abdominal operation, including adhesive bowel obstruction.		

Prognosis

ACS is fatal if left untreated. Even with treatment, the mortality is high. Patients who are at risk for development of ACS remain at increased risk throughout their ICU stay and even after discharge. Furthermore, patients who have undergone decompressive laparotomy may develop recurrent ACS, particularly if inciting events have not been addressed, if temporary dressings are too tight or because of progression of underlying disease states.

Response to treatment

The usual response to definitive surgical treatment is an immediate decrease in intra-abdominal pressure and concomitant improvement in organ dysfunction, haemodynamics, and acid-base derangements. Frequently, especially if treatment is instituted early, urine volumes will increase, oxygenation will improve, mean arterial pressure will rise, and patients will have a decreased need for inotropic or pressor agents.

Repeat laparotomy

Some patients may need to undergo a repeat laparotomy to inspect the viability of the viscera, especially if ACS was diagnosed late, to remove persistent blood or fluid collections, and possibly to begin to re-approximate the fascia. Worsening of acidosis or clinical course in this setting mandates re-exploration, because bowel infarction or perforation from ACS may be present.

Diagnostic guidelines

International

Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines (https://www.wsacs.org/education/436/wsacs-consensus-guidelines-summary)

Published by: World Society of the Abdominal Compartment Syndrome **Last published:** 2021

Treatment guidelines

International

Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines (https://www.wsacs.org/education/436/wsacs-consensus-guidelines-summary)

Published by: World Society of the Abdominal Compartment Syndrome **Last published:** 2021

Online resources

1. World Society of the Abdominal Compartment Syndrome (http://www.wsacs.org) *(external link)*

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Images

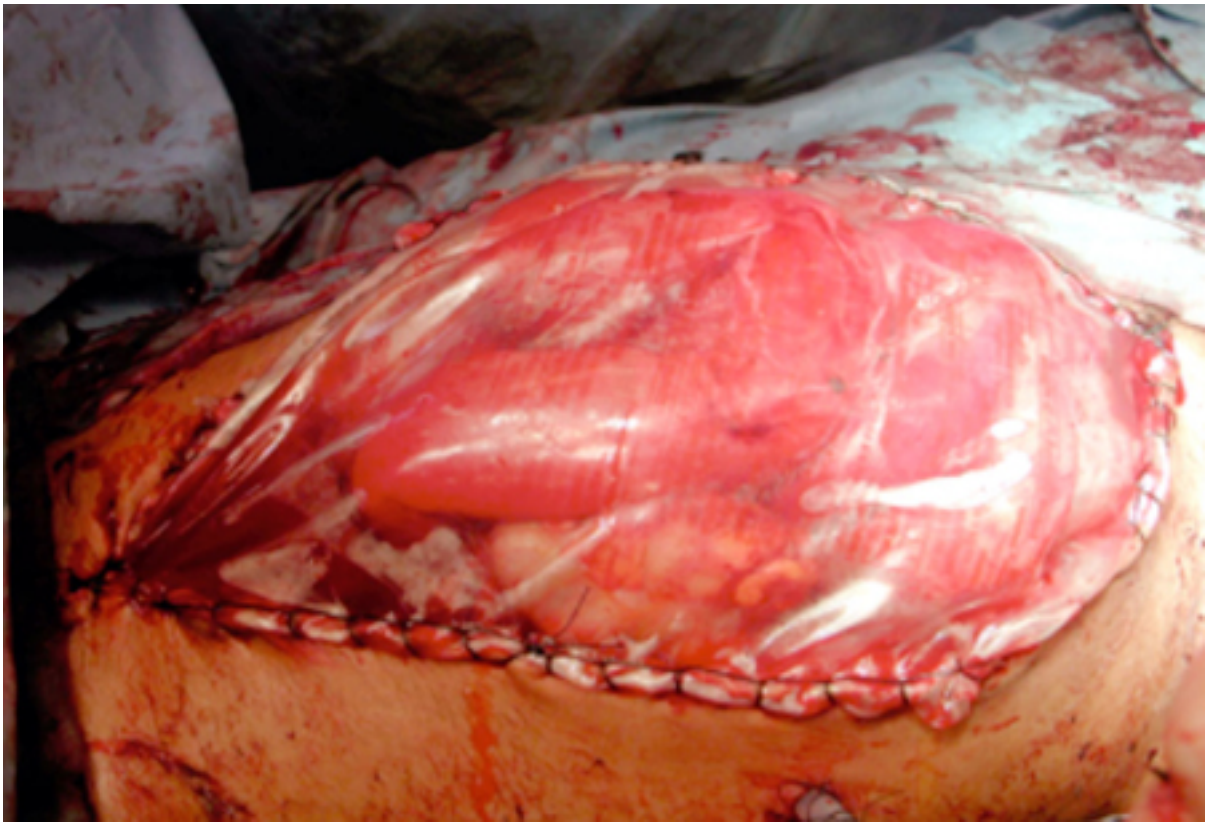

Figure 1: 'Bogota bag' temporary abdominal closure after surgical decompression of abdominal compartment syndrome

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This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

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