A new concept: the polycompartment syndrome – Part 2

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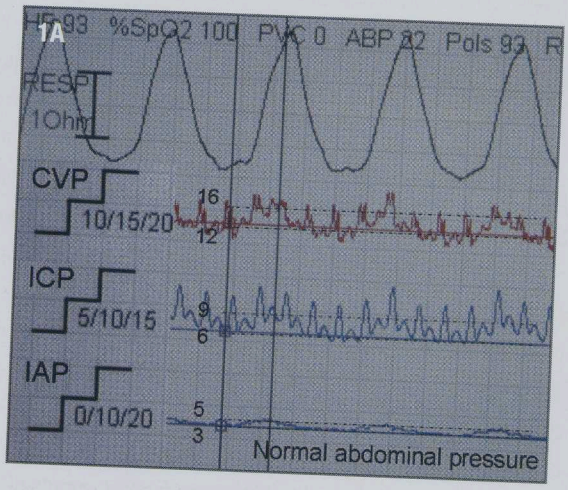
A compartment syndrome (CS) exists when the increased pressure in a closed anatomical space threatens the viability of surrounding tissue. Within the body, there are four compartments, namely the head, chest, abdomen and extremities. Our discussion of the polycompartment syndrome began in the previous issue of International Journal of Intensive Care. The first article reviewed the eye (orbital) compartment syndrome, intracranial compartment syndrome, thoracic compartment syndrome, cardiac compartment syndrome and limb (extremity) compartment syndrome. In this issue we complete our review with a discussion of the hepatic compartment syndrome, renal compartment syndrome, pelvic compartment syndrome and abdominal compartment syndrome.

HEPATIC COMPARTMENT SYNDROME (HCS)

Within the capsule of the liver itself, local haematoma formation caused by trauma or bleeding diathesis (oral anticoagulants, liver cirrhosis, etc) may have an adverse affect on tissue perfusion causing a local hepatic compartment syndrome (HCS).



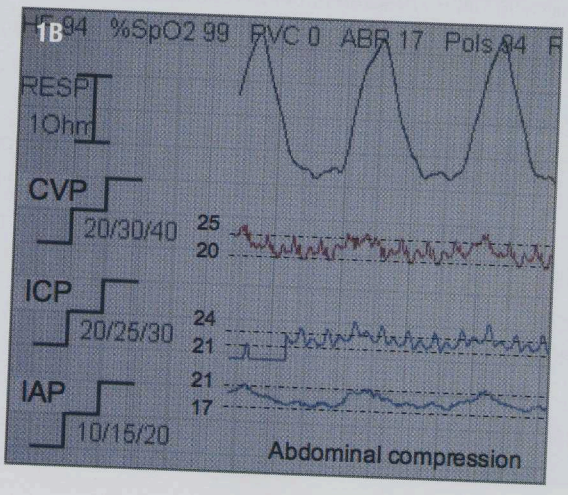


Figure 1. Simultaneous tracings of respiration (RESP), central venous pressure (CVP), intracranial pressure (ICP) and intraabdominal pressure (IAP) in a patient with combined head and abdominal trauma. The patient was mechanically ventilated via BiPAP mode with a RESP of 20 breaths per minute; inspiratory pressure was set at 32 cmH_2O with a PEEP of 5 cmH_2O and the paper tracing speed was set at 6.25 mm/s. The respiratory in- and end-expiratory variations in the pressure tracings can be observed.
Adapted from Malbrain ML, Wilmer A. The polycompartment syndrome: towards an understanding of the interactions between different compartments! *Intensive Care Med* 2007; **33**: 1869–1872.
Panel 1A. Screenshot taken from bedside Philips IntelliVue monitor during normal (baseline) IAP of 4 mmHg.
Panel 1B. Screenshot taken from bedside Philips IntelliVue monitor during increased IAP of around 19 mmHg (abdominal compression with velcro belt for prevention of incisional hernia).

The liver appears to be particularly susceptible to injury in the presence of elevated surrounding pressures, thus especially in case of intra-abdominal hypertension (IAH) or abdominal compartment syndrome (ACS). Animal and human studies have shown impaired hepatic cell function and liver perfusion, even with only moderately elevated intra-abdominal pressure (IAP) of 10 mmHg.[1,2] Furthermore, acute liver failure, decompensated chronic liver disease and liver transplantation are often complicated by IAH and the ACS.[3,4] Close monitoring and early recognition of IAH, followed by aggressive treatment, may confer an outcome benefit in patients with liver disease.

In the management of these patients, it might be useful to monitor the plasma disappearance rate (PDR) for indocyanine green (ICG) because this correlates not only with liver function and perfusion but also with IAP.[5,6] Since cytochrome P450 function may be altered in case of IAH/ACS, medication doses should be adapted accordingly. With increasing IAP, there is decreased hepatic arterial flow, decreased venous portal flow and increased portacollateral circulation, all of which exert physiological effects, such as decreased lactate clearance, altered glucose metabolism and altered mitochondrial function.

RENAL COMPARTMENT SYNDROME (RCS)

Intra-abdominal hypertension has been associated with renal impairment for over 150 years.[7] However, it is only recently that a clinically recognised relationship has been found.[8,9] Elevated IAP significantly decreases renal artery blood flow and compresses the renal vein leading to renal dysfunction and failure.[10] Oliguria develops at an IAP of 15 mmHg and anuria at 25 mmHg in the presence of normovolaemia and at lower levels of IAP in the patient with hypovolaemia or sepsis.[11,12] Renal perfusion pressure (RPP) and renal filtration gradient (FG) have been proposed as key factors in the development of IAP-induced renal failure. Thus:

RPP = MAP – RVP
where MAP = mean arterial pressure
and RVP = renal vein pressure
FG = GFP – PTP = RPP – PTP = (MAP – RVP) – RVP = MAP – 2*RVP
where GFP = glomerular filtration pressure
and PTP = proximal tubular pressure.

In conditions of increased IAP, the RVP may be substituted by IAP, or thus:

RPP = MAP – IAP
FG = MAP – 2*IAP.

Changes in IAP therefore have a greater impact upon renal function and urine production compared to changes in MAP. It should therefore not be so surprising that decreased renal function, as evidenced by development of oliguria, is

one of the first visible signs of IAH.

An increasing number of large clinical studies have identified that IAH (≥15 mmHg) is independently associated with renal impairment and increased mortality.[8,9,13,14] The aetiology of these changes is not entirely well established, though it may be multifactorial, involving reduced renal perfusion, reduced cardiac output and increased systemic vascular resistance and alterations in humeral and neurogenic factors. Within the kidney capsule itself, local haematoma formation (caused by trauma or bleeding diathesis) may have an adverse affect on tissue perfusion, causing a local renal compartment syndrome.[15,16] Some key points to remember are:

- The kidneys can be considered to be the 'canary' for evaluating the effects on end-organ function related to increased IAP.[17]
- The pre-renal azotaemia seen in IAH is unresponsive to volume expansion to normal cardiac output, dopaminergic agents or loop diuretics.[18,19]
- Renal function may be improved by paracentesis of the ascitic fluid and reduction in the IAP.[20]
- Prompt reduction of IAP has a dramatic beneficial effect on urine output in patients with primary and secondary ACS after trauma.[21–26]

PELVIC COMPARTMENT SYNDROME (PCS)

In the pelvic region, three major compartments (gluteus medius–minimus compartment, gluteus maximus compartment, and iliopsoas compartment) can be distinguished from the smaller compartment of the tensor fasciae latae muscle. Pelvic compartment syndromes are rare and a clear history of trauma is often lacking.[27–29] The PCS is often associated with drug and alcohol abuse, infections (necrotising fasciitis) and the use of anticoagulant therapy.[28] Increased pelvic compartment pressure (CP) may eventually increase IAP and affect kidney function, due to bilateral ureteral obstruction and renal failure caused by a massive intrapelvic haematoma with increased retroperitoneal pressure. Decompressive fasciotomy of the gluteal compartment is the treatment of choice.

Worst-case scenario

A 55-year-old man involved in a traffic accident has combined traumatic brain and abdominal injuries. He is transported haemodynamically unstable to the local trauma centre emergency room and is immediately taken to the operating room after a CT scan has been obtained. A cerebral spinal fluid (CSF) catheter (balloon-tipped intracranial pressure (ICP) catheter, Spiegelberg, Hamburg, Germany) is placed because of coma related to subdural haematoma and a lesion in the left frontal lobe and the suspicion of a 'contrecoup' lesion in the right occipital region. An injury to the right lobe of the liver and infrahepatic inferior vena cava is repaired. A partial spleen rupture remaining within the capsule is treated conservatively. Haemostasis is adequate. A nasogastric tube is placed for continuous IAP monitoring (CiMON probe, Pulsion Medical Systems, Munich, Germany). The abdomen is not tense with an IAP of 5 mmHg and is primarily closed. The patient is hypothermic, remains sedated and is transferred to the surgical intensive care unit (ICU). The patient is mechanically ventilated with an Evita XL ventilator (Dräger, Lubeck, Germany) via BiPAP mode at a rate of 20 breaths per minutes, with a maximal inspiratory pressure set at 32 cmH_2O and a positive end-expiratory pressure (PEEP) of 5 cmH_2O. Baseline values for central venous pressure (CVP) and ICP are 12 mmHg and 6 mmHg, respectively. The surgeon insists on putting an abdominal Velcro belt to prevent an incisional hernia. Figure 1 shows the simultaneous tracings of respiration, CVP, ICP and IAP, with and without the Velcro belt. The inspiratory and expiratory variations in the pressure tracings show a dramatic increase in CVP to 20 mmHg, ICP to 21 mmHg and IAP to 17 mmHg when the Velcro belt is applied. The index of transmission between the different compartments can be easily calculated: the average abdomino-thoracic transmission is around 60%, while the abdomino-cranial transmission is almost 100% (Table 1). The Velcro belt is immediately removed after the surgeon leaves the ICU.

Table 1. Intercompartmental transmission*

	Baseline			Compression			Index of transmission			
	ee	ei	DRES	ee	ei	DRES	Dee	Dei	ITee	ITei
IAP (mmHg)	3	5	2	17	21	4	14	16	–	–
ICP (mmHg)	6	9	3	21	24	3	15	15	107%	94%
CVP (mmHg)	12	16	4	20	25	5	8	9	57%	56%

*In this illustrative example, different compartment pressures have been obtained from the abdominal (IAP), intracranial (ICP) and intravascular or intrathoracic (CVP) compartments at end-expiration and at end-inspiration at baseline conditions and after abdominal compression in a single patient. Abdominal compression resulted from the use of a Velcro belt. Average abdomino-thoracic transmission was around 60%, while the abdomino-cranial transmission was almost 100%.

CVP: central venous pressure; ee: end-expiratory; ei: end-inspiratory; IAP: intra-abdominal pressure; ICP: intracranial pressure; ITee: index of transmission during expiration; ITei: index of transmission during inspiration; Dee: difference between end-expiratory value during abdominal compression and baseline value; Dei: difference between end-inspiratory value during abdominal compression and baseline value; DRES: end-inspiratory – end-expiratory value.

On the second post-operative day, IAP increases to 12 mmHg and ICP to 17 mmHg. The patient develops acidosis, remains hypothermic and develops worsening anaemia with a coagulopathy. Mechanical ventilation becomes more difficult and PEEP is increased to 12 cm H_2O. A low flow pressure-volume (PV) loop is constructed and identifies a best PEEP of 13 cmH_2O. Figure 2 shows the effect of applying the Velcro belt on the IAP, PV loop and lower inflection point. A second-look laparotomy shows bleeding from the ruptured spleen and a splenectomy is performed. To avoid development of ACS, the patient's abdomen is left open and a plastic intravenous bag (so-called 'Bogota bag') is sewn to the patient's skin as a temporary abdominal closure. The patient is then transferred back to the surgical ICU for re-warming and ongoing resuscitation. In the ICU, the patient remains hypotensive with elevated arterial lactate levels and low urinary output. His heart rate (HR) is regu-

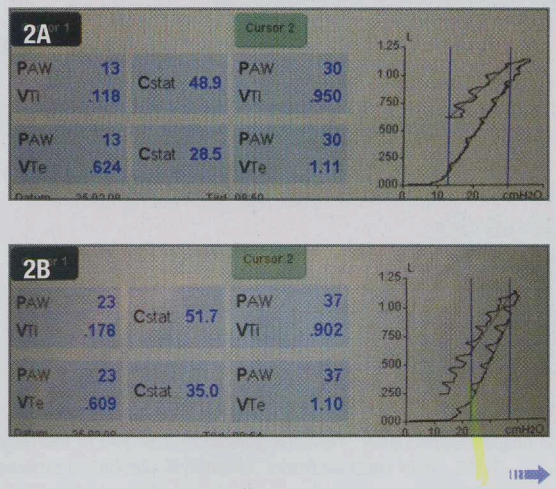


Figure 2. Low flow pressure volume (PV) loop in a patient with combined head and abdominal trauma. The patient was mechanically ventilated via BiPAP mode at 24 breaths per minute, with inspiratory pressure at 32 cmH_2O and a PEEP of 12 cmH_2O. The low flow PV loop manoeuvre was performed with an Evita XL ventilator (Dräger, Lubeck, Germany). The flow was set at 4 L/min, the maximal pressure alarm at 55 cmH_2O and the tidal volume alarm at 1200 mL. **Panel 2A.** Screen shot taken from low flow PV loop obtained with Evita XL during baseline IAP of around 12 mmHg. The tidal volume was 650 mL, hence the dynamic compliance was calculated as 32.5 ml/cmH_2O. The static compliance obtained with the low flow PV loop was 48.9 mL/cmH_2O, and the lower inflection point was 13 cmH_2O. **Panel 2B.** Screen shot taken from low flow PV loop obtained with Evita XL during increased IA of around 24 mmHg (with abdominal compression with velcro belt for prevention of incisional hernia). The tidal volume was 430 mL, hence the dynamic compliance was calculated as 21.5 mL/cmH_2O. The static compliance obtained with the low flow PV loop was 51.7 mL/cmH_2O, and the lower inflection point was 23 cmH_2O.

Table 2. The cranial and abdominal compartments

	Cranium	Abdomen
Organ (s)	Brain	Abdominal organs, small and large bowel
Fluid (s)	Cerebrospinal fluid	Ascites
Enclosure	Skull	Abdominal cage
Lesions	Tumour, haematoma	Blood, oedema, ascites, air, tumour
Pressure	ICP	IAP
Perfusion	CPP = MAP – ICP	APP = MAP – IAP

ICP: intracranial pressure; IAP: intra-abdominal pressure; CPP: cerebral perfusion pressure MAP: mean arterial pressure; APP: abdominal perfusion pressure.

lar at 150 beats per minute (bpm). A volumetric PiCCO catheter (Pulsion Medical Systems, Munich, Germany) is placed to guide the patient's management.

The initial haemodynamic profile was consistent with profound intravascular volume depletion as a result of the patient's haemorrhagic shock, with a low cardiac index (CI) of 2 L/min/m^2, low global end-diastolic volume (GEDVi) of 534 mL/m^2, low global ejection fraction (GEF) of 12%, high pulse pressure variations (PPV) of 25%, low CVP of 9 mmHg, and low urine output (UOP) of 10 mL/h. Aggressive resuscitation, using crystalloid, colloids (Voluven®) and blood products (8 units of packed red blood cells, 8 units of fresh frozen plasma and 1 unit of platelets), was performed to both increase the patient's intravascular volume and correct the patient's coagulopathy. Over the next several hours, multiple boluses of crystalloid, colloid and packed red blood cells were administered. Initially, the patient responded appropriately to volume administration with increased CI, decreased HR and PPV, increased GEDVi and GEF, and a trend towards improved UOP. It is important to note, however, that the patient's ICP and IAP began to increase again, while dynamic respiratory compliance decreased suggesting the development of ACS. This patient should undergo immediate decompressive laparotomy to relieve the significantly elevated ICP and IAP and restore adequate cerebral perfusion pressure (CPP) to the brain and abdominal perfusion pressure (APP) to the abdominal organs. This will also greatly improve venous return to the heart, thereby improving cardiac function. The mortality from ACS is directly correlated with the rapidity with which decompressive laparotomy is performed. Delays of even 30-60 minutes can make the difference between life and death for these patients. Diuretics are contraindicated as this will only worsen systemic perfusion. Pharmacological paralysis, by reducing thoracic and abdominal wall tension, may decrease IAP temporarily, but does not treat the underlying problem of ACS.

Given the severity of the patient's physiological derangements, a decision was made to re-open his abdomen in the ICU. With abdominal decompression, dramatic improvements in CI, HR, GEF, GEDVi, ICP, CPP, APP, respiratory compliance, and UOP were realized. As a result of the reduced intrathoracic pressure (ITP) and IAP, the patient's CVP also dropped significantly, confirming that intracardiac filling pressure measurements cannot be used to guide resuscitation in this patient population. The patient was further resuscitated towards volumetric and functional haemodynamic parameters, regained diuresis and recovered further uneventfully.

Table 3. Consensus definitions*

- IAP is the steady-state pressure concealed within the abdominal cavity
- APP = MAP – IAP
- FG = GFP – PTP = MAP – 2*IAP
- IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line
- The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline
- Normal IAP is approximately 5–7 mmHg in critically ill adults
- IAH is defined by a sustained or repeated pathologic elevation of IAP greater than or equal to ≥ 12 mmHg
- IAH is graded as follows:
  Grade I: IAP 12–15 mmHg
  Grade II: IAP 16–20 mmHg
  Grade III: IAP 21–25 mmHg
  Grade IV: IAP > 25 mmHg
- ACS is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure
- Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that often requires early surgical or interventional radiological intervention
- Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region
- Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

ABDOMINAL COMPARTMENT SYNDROME (ACS)

In analogy with the head, the abdomen can be considered as a closed box (like the skull), with partially rigid sides (spine and pelvis) with an anchorage above (costal arch) and partially flexible sides (abdominal wall and diaphragm), filled with organs (like the brain), such as the small and large intestines, liver, kidneys and spleen and perfused by the mesenteric arteries, with a mesenteric and venous capacitance blood volume. The abdominal organs are surrounded by a third space filled with peritoneal fluid, like the CSF (Table 2). In real life, things are complicated by the movable diaphragm, the shifting costal arch, the contractions of the abdominal wall, and the intestines that may be empty or filled with air, liquid or a faecal mass.

The term, ACS, was first used by Fietsam *et al.* in the late 1980s to describe the pathophysiological alterations resulting from intra-abdominal hypertension (IAH) secondary to aortic aneurysm surgery: 'In four patients that received more than 25 liters of fluid resuscitation increased IAP developed after aneurysm repair. It was manifested by increased ventilatory pressure, increased central venous pressure, and decreased urinary output. This set of findings constitutes an abdominal compartment syndrome caused by massive interstitial and retroperitoneal swelling… Opening the abdominal incision was associated with dramatic improvements…'[19]

The World Society on Abdominal Compartment Syndrome (WSACS, www.wsacs.org) was founded in 2004 to serve as a peer-reviewed forum and educational resource for all healthcare providers as well as industry with an interest in IAH and ACS. Recently, the first consensus definitions have been published.[30,31] Table 3 summarises these consensus definitions: a sustained increase in IAP equal to or above 12 mmHg defines IAH, whereas ACS is defined by a sustained IAP above 20 mmHg with new-onset organ failure.

Table 4. Risk factors for the development of IAH and ACS

Related to diminished abdominal wall compliance
- Mechanical ventilation, especially fighting with the ventilator and the use of accessory muscles
- Use of positive end-expiratory pressure (PEEP) or the presence of auto-PEEP
- Basal pleuroneumonia
- High body mass index
- Pneumoperitoneum
- Abdominal (vascular) surgery, especially with tight abdominal closures
- Pneumatic antishock garments
- Prone and other body positioning
- Abdominal wall bleeding or rectus sheath haematomas
- Correction of large hernias, gastroschisis or omphalocoele
- Burns with abdominal eschars

Related to increased intra-abdominal contents
- Gastroparesis
- Gastric distention
- Ileus
- Volvulus
- Colonic pseudo-obstruction
- Abdominal tumour
- Retroperitoneal/abdominal wall haematoma
- Enteral feeding
- Intra-abdominal or retroperitoneal tumour
- Damage control laparotomy

Related to abdominal collections of fluid, air or blood
- Liver dysfunction with ascites
- Abdominal infection (pancreatitis, peritonitis, abscess, etc)
- Haemoperitoneum
- Pneumoperitoneum
- Laparoscopy with excessive inflation pressures
- Major trauma
- Peritoneal dialysis

Related to capillary leak and fluid resuscitation
- Acidosis* (pH < 7.2)
- Hypothermia* (core temperature < 33°C)
- Coagulopathy* (platelet count < 50000/mm3)

OR an activated partial thromboplastin time (APTT) > 2 times normal
OR a prothrombin time (PTT) < 50%
OR an international standardised ratio (INR) > 1.5

- Polytransfusion/trauma (> 10 units of packed red cells/24 h)
- Sepsis (American–European Consensus Conference definition)
- Severe sepsis or bacteraemia
- Septic shock
- Massive fluid resuscitation (> 5 L of colloid or >10 L of crystalloid/24 h with capillary leak and positive fluid balance)
- Major burns

The combination of acidosis, hypothermia and coagulopathy has been forwarded in the literature as the 'deadly triad'.[46,47]

Monitoring of IAP

Since the abdomen and its contents can be considered as relatively non-compressive and primarily fluid in character, behaving in accordance to Pascal's law, the IAP measured at one point may be assumed to represent the IAP throughout the abdomen.[32,33] Intra-abdominal pressure increases with inspiration (diaphragmatic contraction) and decreases with expiration (diaphragmatic relaxation).

In the strictest sense, a normal IAP ranges from 0 to 5 mmHg.[34] However, certain physiological conditions, such as morbid obesity,[35,36] ovarian tumours, cirrhosis or pregnancy, may be associated with chronic IAP elevations of 10–15 mmHg to which the patient has adapted with an absence of significant pathophysiology. In contrast, children commonly demonstrate low IAP values.[37] The clinical importance of any IAP must be assessed in view of the baseline steady-state IAP for the individual patient.

Measurement of IAP

Intra-abdominal pressure can be directly measured with an intraperitoneal catheter attached to a pressure transducer. During CO_2-insufflation in laparoscopic surgery, the IAP is measured directly via the Verres needle.

However, different indirect methods for estimating IAP are used clinically because direct measurements are considered to be too invasive.[32,38] These techniques include rectal, uteral, gastric, inferior vena caval and urinary bladder pressure measurements. Only gastric and bladder pressures are used clinically. Over the years, bladder pressure has been forwarded as the gold-standard indirect method and measurement kits have become available: FoleyManometer (Holtech Medical, Copenhagen, Denmark) or an AbViser-valve (Wolfe Tory Medical, Salt Lake City, Utah, USA).

The IAP can also be measured continuously via a balloon-tipped stomach catheter that recently became available (Spiegelberg, Hamburg, Germany and Pulsion Medical Systems, Munich, Germany).[33,39] This avoids the problems associated with the creation of a hydrostatic fluid column and allows continuous IAP and APP measurement.

Measurement of APP

Abdominal perfusion pressure can be calculated in a similar way to the widely accepted and clinically used concept of calculating CPP. Cerebral perfusion pressure is calculated as CPP = MAP – ICP, where MAP = mean arterial pressure and ICP = intracranial pressure.

Thus, abdominal perfusion pressure (APP) is calculated as:

APP = MAP – IAP

where MAP = mean arterial pressure
and IAP = intra-abdominal pressure.

This calculation has been proposed as a more accurate predictor of visceral perfusion and a potential end-point for resuscitation by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP).[40–43]

Which patient?

Although the prevalence and incidence of IAH in critically ill patients is considerable,[44,45] routine IAP measurement in all patients admitted to the ICU is currently rarely performed and probably not indicated. The ACS can be diagnosed when there is increased IAP with evidence of end-organ dysfunction. Although there are many causes of acute cardiopulmonary, renal, hepatosplanchnic or neurological deterioration in the ICU, it is important that we recognise the IAP as being an independent risk factor for this organ function deterioration.

The WSACS has provided a list of risk factors associated with IAH and ACS (Table 4);[46,47] if two or more risk factors are present, baseline routine IAP monitoring is advised.[31,48] Massive volume resuscitation after a 'first hit' for any reason (burns, trauma, pancreatitis, haemorrhagic shock, etc) can lead to increased IAP, particularly post-operatively or in a septic patient. The 'second hit' probably results from the effects of 'capillary leak', shock with ischaemia–reperfusion injury and the release of cytokines combined with massive increases in total extracellular volume.[49]

What technique?

According to the WSACS consensus guidelines, IAP should be measured at end-expiration in the complete supine position, after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line at the iliac crest after an instillation volume of maximal 20–25 mL.[30] An intermittent technique may be used for screening, while in some patients, a continuous technique may be preferable, for example, when the APP is used as a resuscitation end-point, or in patients with impending ACS requiring urgent abdominal decompression.

What frequency?

When an intermittent method is used, measurements should be obtained at least every 4 to 6 hours. In patients with evolving organ dysfunction, this frequency should be increased up to hourly measurements.

When to stop IAP measurement?

Measurement of IAP can be discontinued when the risk factors for IAH are resolved or the patient has no signs of acute organ dysfunction, and IAP values have been below 10–12 mmHg for 24–48 hours. If there is recurrent organ dysfunction, IAP measurement should be reconsidered.

What about IAP measurement in children?

Some studies have been performed regarding IAP measurement in children.[37,50] The transvesical route can be used safely in children, but obviously the instillation volume is important in this population. Davis *et al.* found that 1 mL/kg produces reliable IAP values when compared to higher volumes.[37] Normal IAP values are lower in children (3–5 mmHg) and the thresholds defining IAH (9 mmHg) and ACS (16 mmHg) are also lower compared to adults.

What about IAP measurement in awake patients?

Intra-abdominal pressure measurement is most often performed in sedated patients in whom muscle contractions are absent. When measuring IAP in awake patients, specific attention should be made that no muscle contractions are present, e.g. during forced expiration with auto-PEEP, in a patient with chronic obstructive pulmonary disease. Adequate pain medication should be administered, especially after abdominal surgery, as even placing the patient in supine position may induce abdominal pain and muscle contractions, leading to falsely elevated IAP readings.

CLINICAL MANAGEMENT

The management of patients with polycompartment syndrome is based on three principles.[51,52] The first of these principles is the use of specific medical and surgical procedures to reduce the compartment pressure (Table 5):

- improvement of compartment wall compliance
- evacuation of intra-compartment contents
- correction of capillary leak and positive fluid balance
- specific treatments
- rescue treatments.

The second principle involves general and organ support (intensive care) of the critically ill patient, while the third principle utilises optimisation and prevention of specific adverse events after surgical decompression (ischaemia/reperfusion).

Table 5. Treatment options for compartment syndrome

Improvement of compartment wall compliance
- Sedation
- Pain relief (not fentanyl!)
- Neuromuscular blockade
- Body positioning
- Negative fluid balance
- Skin pressure decreasing interfaces
- Weight loss
- Percutaneous abdominal wall component separation
- Escharotomies

Evacuation of intra-compartment contents
- Gastric tube and suctioning
- CSF, ascites, pleural or pericardial drainage
- Rectal tube and enemas
- Chest tube and suctioning
- Endoscopic decompression of large bowel
- Colostomy or ileostomy
- CT- or US-guided aspiration of abscess
- CT- or US-guided aspiration of haematoma
- Pericardectomy

Correction of capillary leak and positive fluid balance
- Albumin in combination with diuretics (furosemide)
- Correction of capillary leak (antibiotics, source control, etc)
- Colloids (Hypertonic-Voluven® instead of crystalloids)
- Dobutamine (not dopamine!)
- Dialysis or CVVH with ultrafiltration
- Ascorbinic acid in burn patients

Specific therapeutic interventions
- Continuous negative external pressure (VAC®)
- Targeted compartment perfusion pressure

Rescue therapy
- ICS: decompressive craniectomy
- ACS: decompressive laparotomy
- TCS: decompressive sternotomy
- ECS: decompressive fasciotomy
- PCS: pelvic compartment syndrome
- RCS: renal decapsulation
- HCS: hepatic decapsulation
- CCS: decompressive pericardiotomy
- OCS: orbital decompression

**ACS: abdominal compartment syndrome; CCS: chronic compartment syndrome; CSF: cerebrospinal fluid; CT: computed tomography; ICS: intracranial compartment syndrome; ECS: exertional compartment syndrome; HCS: hepatic compartment syndrome; OCS: orbital compartment syndrome; PCS: pelvic compartment syndrome; RCS: renal compartment syndrome; TCS: thoracic compartment syndrome; US: ultrasound.*

CONCLUSION

First suggested in 2007, the polycompartment syndrome is a constellation of the physiological sequelae of increased compartment pressures, whether ICP, ITP or IAP.[53,54] Recent observations suggest an increasing frequency of this complication in all types of patients and increased compartment pressures are independently associated with morbidity and mortality. Even chronic elevations of CP seem to affect the various organ systems in the body.

In spite of this, the syndrome is still in its infant stage and remains poorly recognised and thus poorly treated in some cases. The diagnosis relies largely on CP measurement. Within the polycompartment syndrome, the abdomen plays a central role and the effect of IAH on different organ systems has been described, along with recommendations to compensate for these effects.

The ultimate goal of treatment is not only to decrease CP,

but also to improve organ function and to decrease mortality. Decompressive craniectomy, sternotomy, fasciotomy and laparotomy are the only treatment options that have been shown to reach most of these goals today. However, some less invasive techniques and some medical treatment strategies have shown promise in achieving CP reduction as well as organ function improvement. The bottom line is that futile crystalloid over-resuscitation may cause (iatrogenic) secondary ACS. In contrast, the cautious administration of colloids not only seems to decrease the incidence of ACS in burn and trauma patients, but also ACS-associated complications and mortality, as well as the complications related to increased pressures in other compartments.

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