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The polycompartment syndrome: a concise state-of-the-art review

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Abstract

A compartment syndrome is defined as an increase in the compartmental pressure to such an extent that the viability of the tissues and organs within the compartment are threatened. The term describes a syndrome and not a disease, and as such there are many diseases and underlying pathophysiological processes that may lead to such a scenario. The aim of this review is to give a state-of-the-art overview on the current knowledge on different compartment syndromes and how they may interact. Suggested definitions are included. There are four major compartments in the human body: the head, chest, abdomen, and the extremities. Initially, the term multicompartment syndrome was suggested when more than one compartment was affected. But this led to confusion as the term multi- or multiple compartment syndromes is mostly used in relation to multiple limb trauma leading to compartment syndrome requiring fasciotomy. Only recently was the term 'polycompartment syndrome' coined to describe a condition where two or more anatomical compartments have elevated pressures. When more than one compartment is affected, an exponential detrimental effect on end-organ function to both immediate and distant organs can occur. Within each compartment, the disease leading towards a compartment syndrome can be primary or secondary. The compliance of each compartment is the key to determining the transmission of a given compartmental pressure from one compartment to another. The intra-abdominal pressure helps to explain the severe pathophysiological condition occurring in patients with cardiorenal, hepatopulmonary and hepatorenal syndromes. Initial treatment of a compartment syndrome should be focused on the primary compartment and is based on three principles: lowering of compartmental pressure, supporting organ perfusion, and optimisation and prevention of specific adverse events. Clinicians need to be aware of the existence of the polycompartment syndrome and the interactions of increased compartmental pressures between compartments.

Key words: abdominal pressure, intracranial pressure, intrathoracic pressure, abdominal compartment, renal compartment, extremity compartment, thorax compartment, compartment syndrome, polycompartment, compliance

A compartment syndrome is defined as an increased pressure in a closed anatomic space, which threatens the viability of enclosed and surrounding tissue [1]. Within the body there exist four major compartments, namely the head, chest, abdomen, and extremities. Within each of these compartments individual organs may be affected by compartment syndromes. Table 1 summarises the different compartments and related pathophysiologic and clinical implications of compartment syndromes in these locations. As a result of differing anatomy and physiology within each location, compartment syndromes may manifest in a multitude of ways, some of which remain relatively poorly understood. The abdominal compartment has unique effects because it is geographically situated 'up-stream' from the extremities and 'down-stream' from the chest. Therefore, it may influence the physiology and pathophysiology of each of these other compartments.

A therapeutic conflict is a situation where each of the possible therapeutic decisions carries some potential harm. The presence of a compartment syndrome often plays a role when we are dealing with these dilemmas. In high-risk critically ill patients, the decision about fluid administration in particular should be done within this context. Therapeutic conflicts pose the greatest challenge for protocolised cardiovascular management in anaesthetised and critically ill patients, where our decisions can make a significant difference to outcome [2, 3].

Scalea et al. first introduced the term 'multiple compartment syndrome' in a study of 102 patients with increased intra-abdominal (IAP), intrathoracic (ITP), and intracranial pressure (ICP) after severe traumatic brain injury [4]. They suggested that different compartments within the body are not isolated and independent entities, but instead should be considered closely interconnected. The term multi- or multiple compartment syndrome is now mostly used in relation to multiple limb trauma leading to compartment syndrome, thus necessitating fasciotomy. However, the term *polycompartment syndrome* (PCS) was only recently coined in order to avoid confusion [5]. Organ-organ interactions and resulting polycompartment syndromes may occur more frequently than clinicians report.

The purpose of this state-of-the-art review was to provide a critical overview of current literature examining the pathophysiology, clinical presentation/epidemiology, and management of the polycompartment syndrome, beginning with an illustrative case [6–8].

Illustrative case: polycompartment syndrome with secondary limb compartment syndrome and primary head and abdomen compartment syndromes

A 23-year-old man was involved in a high-speed car crash. He was hypotensive at the scene and during the 40 minute transport to hospital. After an Emergency Department assessment, he underwent an exploratory laparotomy, which included a splenectomy, and peri-hepatic packing. His abdomen was left open with a Bogota bag. ICP monitoring was conducted as a result of a severe traumatic brain injury (TBI) and a massive blood transfusion was given during the first 24 hours of the intensive care unit (ICU) management.

After 24 hours in the ICU, primary abdominal fascial closure was performed. He subsequently developed Grade IV (IAP above 25 mm Hg) intra-abdominal hypertension (IAH) with a sustained IAP of 28 mm Hg, consistent with a primary abdominal compartment syndrome (ACS) [9]. Both lower limbs, which had not sustained primary injury, were noted to be swollen and tense on palpation, and the anterior and posterior extremity compartment pressures were 42 and 38 mm Hg respectively, indicating secondary limb compartment syndrome. At this time the IAP was 38 mm Hg. In response to these findings, abdominal decompression was performed, together with complete bilateral lower limb fasciotomies. During limb surgery, ischaemic and necrotic muscle was found in the lateral compartment, and an extensive debridement was undertaken. While abdominal decompression initially reduced both IAP and ICP, ICP subsequently rose and remained high until the patient's eventual recovery four weeks later. The increased ICP resulted from a combination of primary (related to TBI) and secondary (related to ACS) insults, while the extremity compartment syndrome developed secondary to ACS.

This case also nicely demonstrates that open abdomen treatment does not always prevent the re-occurence of an abdominal compartment syndrome.

PATHOPHYSIOLOGY

Although an increased compartment pressure will increase venous resistance and decrease perfusion pressure in the implicated compartment, it may also affect other compartments (Fig. 1). The resulting impact on organ function and viability can be devastating. What follows is a discussion on the four major compartments and the different syndromes that can occur. The cause of a compartment syndrome may be *primary* (e.g. tumours and trauma), but most often it will be a *secondary* insult. This typically occurs after massive fluid resuscitation in the setting of trauma and the well-described lethal triad (coagulopathy, hypothermia, acidosis), or during sepsis with poor source control and ongoing capillary leak as suggested previously [10–13].

HEAD

INTRACRANIAL COMPARTMENT SYNDROME

The intracranial contents are confined within a rigid bony cage. According to the Monroe-Kellie doctrine, any

Table 1A. The four compartments: pathophysiology and monitoring

	Head			Chest		Abdomen				Extremities
	Brain	**Orbita**	**Eye**	**Thorax**	**Heart**	**Abdomen**	**Liver**	**Kidney**	**Pelvis**	
Primary physiologic parameter	Intracranial pressure (ICP)	Orbital compartment pressure (OCP)	Intra-ocular pressure (IOP)	Intra thoracic pressure (ITP)	Filling pressure (CVP, PAOP)	Intra-abdominal pressure (IAP)	Perihepatic pressure, upper IAP (UIAP)	Peritubular pressure (PTP), renal venous pressure (RVP)	Pelvic compartment pressure, lower IAP (LIAP)	Extremity compartment pressures (ECP)
Secondary parameter	Cerebral perfusion pressure (CPP) = MAP – ICP, compliance	Orbital perfusion pressure (OPP) = MAP – OCP	Ocular perfusion pressure (OPP) = = MAP – IOP	Peak, plateau and mean airway pressure, lung and chest wall compliance	Coronary perfusion pressure (CoPP) = DBP – PAOP = DBP – ITP	Abdominal perfusion pressure (APP) = MAP – IAP	Liver perfusion pressure = = MAP – UIAP	Renal perfusion pressure (RPP) = MAP - RVP	Pelvic perfusion pressure = = MAP – LIAP	Peripheral arterial perfusion pressure, tissue perfusion pressure (TPP) = capillary pressure – ECP
CP measurement	Fluid filled ventriculostomy, airfilled balloon-tipped catheter, parenchymal solid state microchip transducer	Orbital tissue tension manometry (not possible in clinical practice)	Ocular tissue tension manometry	Oesophageal pressure measurement via a balloon-tipped catheter; surrogate parameters: Palv (Ppeak, Pplat, Pmean)	via deep venous or Swan-Ganz catheter	Via bladder (FoleyManometer, AbViser valve) Via stomach (Spiegelberg, Pulsion Medical Systems)	Estimation of UIAP via stomach (Spiegelberg, Pulsion Medical Systems)	Estimation of PTP and RVP via bladder (Foley-Manometer, AbViser valve)	Estimation of LIAP via bladder (Foley-Manometer, AbViser valve)	via a needle connected to a fluid-filled pressure transducer system
Syndrome	Intracranial hypertension (ICH): ICP > 15 mm Hg	Intra-orbital hypertension: OCP > 20 mm Hg	Intra-ocular hypertension: IOP > 17 mm Hg	ITP > 15 mm Hg	CVP > 20 mm Hg PAOP > 25 mm Hg	Intra-abdominal hypertension (IAH): IAP ≥ 12 mm Hg	Perihepatic hypertension: UIAP > 12 mm Hg	Renal hypertension: PTP or RVP > 12 mm Hg	Pelvic hypertension: LIAP > 12 mm Hg	Extremity hypertension: ECP > 15 mm Hg
	Intracranial compartment syndrome (ICS) – cerebral herniation: ICP > 25mm Hg	Orbital compartment syndrome (OCS): OCP > 30 mm Hg	Ocular compartment syndrome (OCS): IOP > 30 mm Hg	Thoracic compartment syndrome (TCS): ITP > 25 mm Hg	Cardiac compartment syndrome (CCS) – cardiac tamponade	Abdominal compartment syndrome (ACS): IAP > 20 mm Hg	Liver compartment syndrome (LCS): UIAP > 20 mm Hg	Kidney compartment syndrome (KCS): PTP or RVP > 20 mm Hg	Pelvic compartment syndrome (PCS): LIAP > 20 mm Hg	Extremity compartment syndrome (ECS): ECP > 30 mm Hg
Aetiology	Primary (tumour, sub- or epidural haematoma,…) Secondary (auto-PEEP, hypoxia or hypercarbia, hypertension, ventilation, seizures, ,…) Postoperatively (oedema, mass lesion,…)	Intrinsic (tumour) Extrinsic (traumatic retrobulbar haemorrhage) Combination (fluid resuscitation, burn injury)	Intrinsic (glaucoma) Extrinsic (posttraumatic glaucoma) Combination (fluid resuscitation, burn injury)	Post cardiac surgery, spontaneous mediastinal or pleural haemorrhage, tumour, COPD with dynamic hyperinflation, tension pneumothorax	Trauma, tumour, spontaneous bleeding, fluid resuscitation	Primary IAH: associated with injury or disease in the abdomino-pelvic region Secondary IAH: does not originate from the abdomino-pelvic region Recurrent IAH: chronic state of IAH	Primary: associated with injury or disease in the liver Secondary: mostly related to massive fluid overload	Primary: associated with injury or disease in the kidney Secondary: mostly related to massive fluid overload	Primary: associated with injury or disease in the pelvis Secondary: mostly related to massive fluid overload	Crush injury Trauma with fractures Bleeding disorders Burns

Table 1B. The four compartments: implications and management

	Head			Chest		Abdomen				Extremities
	Brain	**Orbita**	**Eye**	**Thorax**	**Heart**	**Abdomen**	**Liver**	**Kidney**	**Pelvis**	
Potential implication	Brain death	Blindness, eye muscle paralysis	Blindness	Cardiopulmonary collapse	Cardiac collapse, electromechanical dissociation	Multiple organ dysfunction	Liver failure	Kidney failure	Kidney failure (retroperitoneal haematoma)	Extremity loss
Therapeutic intervention	Lower ICP: CSF drainage	Lower orbital compartment pressure (OCP)	Lower IOP	Lower ITP	Evacuate pericardiac effusion	Lower IAP: ascites drainage, medical management	Lower UIAP: ascites drainage, medical management	Lower PTP or RVP: ascites drainage, medical management	Lower LIAP: ascites drainage, medical management	Lower ECP: optimize fluid management
	Increase CPP: vasopressors, fluids	Increase orbital PP	Increase OPP	Escharotomy, chest tube	Pericardiac tube,	Increase APP: vasopressors, fluids, escharotomy	Increase liver PP: vasopressors, fluids	Increase kidney PP: vasopressors, fluids	Increase pelvic PP: vasopressors, fluids	Increase TPP
Rescue therapy	Open compartment	Open compartment	Open compartment	Open compartment	Open compartment	Open compartment	Open compartment	Open compartment	Open compartment	Open compartment
	Decompressive craniectomy	Orbital decompression	Ocular decompression	Decompressive sternotomy	Decompressive pericardiectomy, pericardiotomy	Decompressive laparotomy	Decompressive laparotomy, hepatic decapsulation	Decompressive laparotomy, renal decapsulation	Decompressive laparotomy, gluteal fasciotomy	Decompressive fasciotomy
Importance	Adaptation of ventilatory support essential, recognition life saving, and avoidance of vegetative state (postanoxic encephalopathy)	Recognition of syndrome can be eye saving	Recognition of syndrome can be eye saving	Recognition of syndrome can be life saving; Adaptation of ventilatory support essential	Recognition of syndrome can be life saving	Prevention of bacterial translocation and MODS can be life saving	Prevention of bacterial translocation and MODS can be liver and life saving	Recognition of syndrome can be kidney saving	Recognition of syndrome can be kidney saving	Recognition can be limb saving
Effect on (upstream)	IOP, cardio-respiratory function (SVR, PVR)	Eye, eye muscles	–	Lung (Palv), ICP, IAP, CVP, PAOP	Lung, ICP	All other compartments (Lung, ICP, ITP, CVP, PAOP,…), limbs	All other compartments (Lung, ICP, ITP, CVP, PAOP,…), HARS, HAPS	All other compartments (Lung, ICP, ITP, CVP, PAOP,…), CARS	All other compartments (Lung, ICP, ITP, CVP, PAOP,…)	Kidney (rhabdomyolysis), lungs, heart
Affected by (downstream)	intracardiac pressures (CVP, PAOP), ITP, PEEP and IAP	ICP, ITP, PEEP, IAP	ICP, ITP, PEEP, IAP	IAP (abdomino-thoracic transmission 50% on average), mechanical ventilation (PEEP)	ITP, IAP (abdomino-thoracic transmission 50% on average), mechanical ventilation (PEEP)	ITP, mechanical ventilation (PEEP)	ITP, mechanical ventilation (PEEP)	ITP, mechanical ventilation (PEEP)	ITP, mechanical ventilation (PEEP)	IAP (diminished venous return)

APP — abdominal perfusion pressure; CARS — cardio-abdominal renal syndrome; CP — compartment pressure; CPP — cerebral perfusion pressure; CSF — cerebrospinal fluid; CVP — central venous pressure; ECP — extremity compartment pressure; HAPS — hepato-abdominal pulmonary syndrome; HARS — hepato-abdominal renal syndrome; IAP — intra-abdominal pressure; ICP — intracranial pressure; IOP — intra-ocular pressure; ITP — intrathoracic pressure; LIAP — lower intra-abdominal pressure; MODS — multiple organ dysfunction syndrome; OCP — orbital compartment pressure; OPP — orbital or ocular perfusion pressure; PAOP — pulmonary artery occlusion pressure; PEEP — positive end-expiratory pressure; PTP —peritubular pressure; RVP — renal venous pressure; TPP — tissue perfusion pressure; UIAP — upper intra-abdominal pressure

increase in volume within the intracranial compartment (e.g. an expanding traumatic intracranial haematoma) leads to a reciprocal decrease in the volume of cerebrospinal fluid and intracranial venous blood volume [14]. When compensation mechanisms are exhausted, an increase in ICP with a corresponding decrease in cerebral perfusion pressure (CPP), defined as mean arterial pressure (MAP) minus ICP, may result [14].

$$CPP = MAP - ICP$$

Treatment options for intracranial hypertension (ICH) either focus on lowering ICP (by evacuation of cerebrospinal fluid or a variety of other techniques aimed at decreasing brain tissue oedema) or raising CPP (by maintaining an adequate MAP with fluids or vasopressors) [15, 16]. However, fluid therapy used to support CPP may exacerbate visceral oedema, promote ascites, and increase IAP, which in turn may increase intrathoracic (ITP), internal jugular venous, and intracranial pressures (possibly through decreased intracranial venous drainage via the jugular veins) [16]. Therefore, in patients with severe TBI, treatment decisions that may result in a vicious cycle, increasing pressures in various compartments, should be carefully considered [4].

However, despite these theoretical concerns, the effects of IAP and ITP on ICP have not been extensively studied to date, and remain a challenging area for fundamental and clinical investigators [17–24]. Remarkably, patients with polycompartment syndrome receive significantly more fluids during the first week of ICU stay and predictably their ICU/hospital admission is prolonged [4]. While there was also a trend towards higher mortality in these patients (42% vs 31%), statistical significance was not reached. PCS should be considered in multiple injured patients with increased ICP who do not respond to conventional therapy [4].

ORBITAL COMPARTMENT SYNDROME

Acute orbital compartment syndrome (OCS) is a rare complication of increased pressure within the orbit. An increased intra-orbital pressure may cause decreased orbital perfusion pressure by a mechanism similar to that occurring with CPP. Because intra-orbital pressure cannot be measured directly, intraocular pressure (IOP) can be used as a surrogate, indirect estimation (as pressures within the orbit are directly transmitted to the eye). Ocular/orbital perfusion pressure (OPP) can be calculated as follows:

$$OPP = MAP - IOP$$

Orbital compartment syndrome presents with typical signs and symptoms (eye pain, reduced ocular motility, diplopia and pro-optosis) resulting in progressive visual deficits and is mostly seen in relation to retrobulbar haematomas or trauma [25]. Recognition and prompt treatment is of paramount importance to prevent permanent blindness. A study in burn patients reported that increased IOP corresponded with fluid administration volume given during the first 24 hours of hospitalisation and with the presence of peri-ocular burns [26]. Other conditions associated with orbital compartment syndrome include infection, inflammation, spinal surgery, vascular problems with ophthalmic artery or retinal vein occlusion, optic nerve sheath compression, traumatic asphyxia syndrome and bleeding diathesis or disseminated intravascular coagulopathy as seen in sepsis [25]. Orbital compartment syndrome has even been described after orbital extravasation of X-ray contrast material [25]. Intra-orbital compartment syndrome needs to be differentiated from intra-ocular compartment syndrome as seen with (secondary) glaucoma, especially in trauma patients [27].

OCULAR COMPARTMENT SYNDROME

The eye is situated in the orbital compartment and any pressure increase within this compartment, even if the eye itself is not affected, will result in an increased intra-ocular pressure as discussed above. However, primary intra-ocular compartment syndrome can also occur in relation to increased IOP without intra-orbital compartment syndrome, as seen with primary (e.g. narrow angle glaucoma, tumours) or secondary (e.g. post-traumatic) glaucoma. Intra-ocular hypertension is defined as IOP > 17 mm Hg while ocular compartment syndrome occurs at IOP above 25–30 mm Hg.

CHEST

THORACIC COMPARTMENT SYNDROME

Thoracic compartment syndrome results as an accumulation of air, fluid or blood in the chest and has traditionally been described in trauma. It has also been described in patients undergoing cardiac surgery, where in the setting of substantial myocardial oedema, mediastinal haematomata, noncardiogenic pulmonary oedema, or acute ventricular dilatation, sternal closure may precipitate cardiac tamponade leading to haemodynamic instability or even collapse [28, 29]. Although it has been described in the setting of thoracic trauma damage control procedures, thoracic compartment syndrome is rare in patients with thoracic trauma due to the limited survival when injuries are significant enough to result in massive intrathoracic tissue oedema after resuscitation. Tension pneumothorax or haemopneumothorax (with increased ITP) occurs most often in patients with polytrauma or following an iatrogenic injury related to insertion of a central venous catheter, diagnostic or therapeutic procedure. However, traumatic cardiac tamponade

can be seen as a primary thoracic compartment syndrome (see below). In the ICU, increased ITP is most commonly related to sepsis, capillary leak, aggressive fluid resuscitation, positive pressure ventilation with high PEEP (positive end-expiratory pressure) or dynamic hyperinflation, pneumothorax, COPD with auto-PEEP, diminished chest wall compliance (e.g. morbid obesity or circumferential chest burns), lung fibrosis and ARDS [30]. Rising ITP, reflected as an increase in mean or peak inspiratory pressures during thoracic wall closure, may serve as an early warning that a patient is at risk for thoracic compartment syndrome [6]. Increased ITP (normally < 5–7 mm Hg) can be measured by a balloon-tipped catheter positioned in the lower third of the oesophagus and will affect the lungs, heart and brain by limiting venous return (Fig. 1)[31]. Since increased ITP, like raised IAP, is most commonly related to excessive fluid resuscitation, both frequently coexist [32, 33]. Important issues to consider include:

1. PEEP should be set to counteract ITP and IAP while at the same time avoiding over-inflation of already well-ventilated regions of lung [34, 35]. The fact that PEEP is expressed in cm H_2O (with a conversion factor of 1.36 to mm Hg) takes into account the average abdomino-thoracic index of transmission of around 50% [31, 34]

$$Best\ PEEP\ (cmH_2O) = IAP\ (mm\ Hg)$$

2. During lung protective ventilation, plateau pressures should be limited to transmural or transpulmonary plateau pressures (**$Pplat_{tm}$**) below 30 to 35 cm H_2O, otherwise end-tidal CO_2 will increase [36].

$$Pplat_{tm} = Pplat - ITP = Pplat - IAP/2 < 35\ cm\ H_2O$$

3. Increased ITP and IAP decrease lymphatic drainage and facilitate lung oedema as discussed elsewhere in this article [37, 38]. Therefore, monitoring of extravascular lung water index (EVLWI) may be beneficial in guiding de-resuscitation [39, 40].
4. IAP increases with inspiration (IAP_{ei}) and decreases with exhalation (IAP_{ee}) during spontaneous respiration and mechanical ventilation. Therefore, ΔIAP and the abdominal pressure variation (APV), expressed as a percentage, may indirectly predict abdominal wall compliance (Fig. 2) [5, 41].

$$\Delta IAP = IAP_{ei} - IAP_{ee}$$

$$APV = \Delta IAP/IAP_{mean}$$

CARDIAC COMPARTMENT SYNDROME

Within the thorax, cardiac tamponade may be considered as a specific compartment syndrome. By definition,

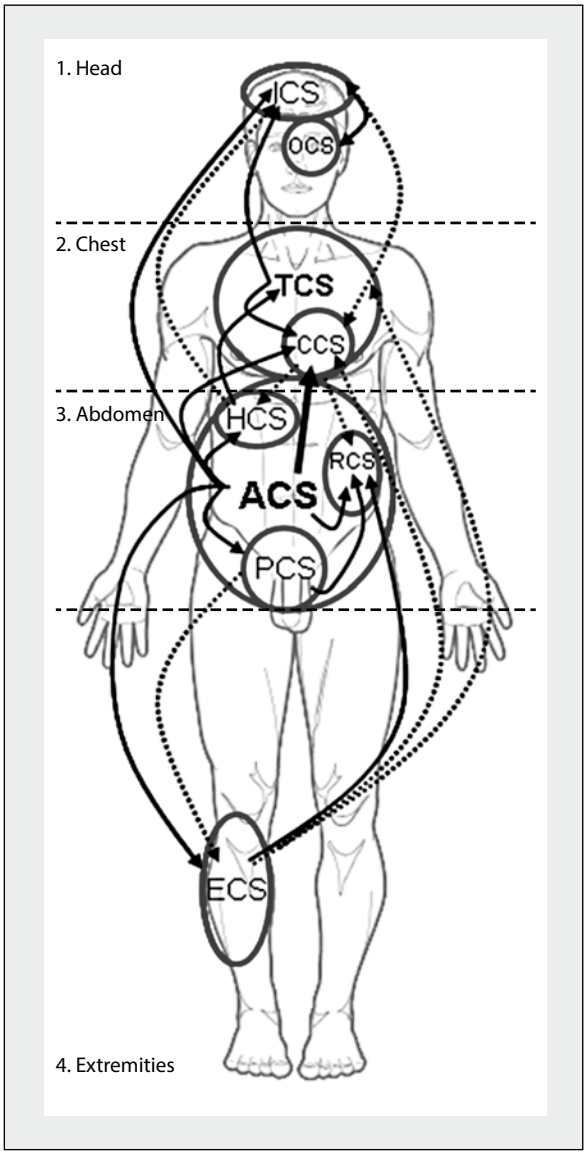

Figure 1. Interactions between the four main body compartments (head, chest, abdomen and extremities). Arrows indicate interactions between different compartments. The head contains the intracranial, intra-orbital and intra-ocular compartments. The chest contains the thorax, lungs and heart, each of which can develop a compartment syndrome. The abdomen contains the abdominal, hepatic, renal, and pelvic compartments. Solid lines show direct effects by transmission of mechanical pressure forces. Dotted lines show indirect distant effects between compartments (see text for explanation, adapted from [7]). ACS — abdominal compartment syndrome; CCS — cardiac compartment syndrome; ECS — extremity compartment syndrome; HCS — hepatic compartment syndrome; ICS — intracranial compartment syndrome; RCS — renal compartment syndrome; OCS — orbital compartment syndrome; PCS — pelvic compartment syndrome; TCS — thoracic compartment syndrome

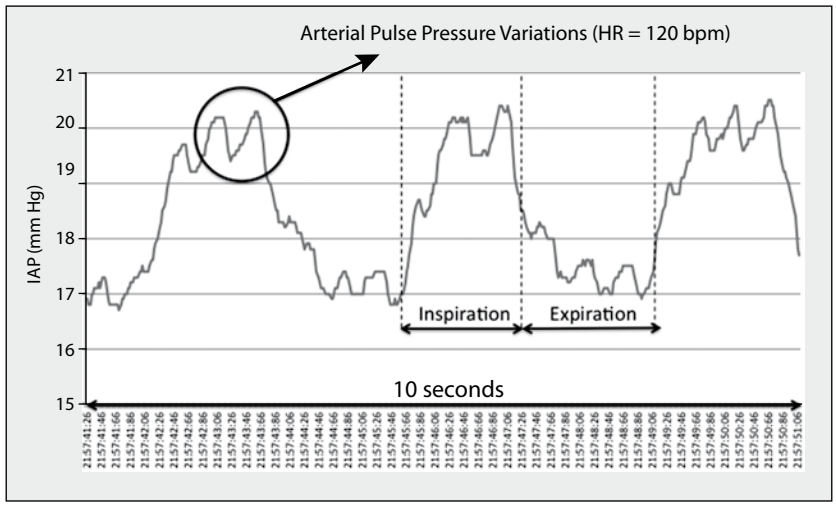

Figure 2. Respiratory variations of intra-abdominal pressure (IAP) are an indirect estimation of abdominal wall compliance. A raw data tracing of continuous IAP monitoring recorded with balloon-tipped nasogastric probe connected to CiMON monitor (Pulsion Medical Systems, Munich, Germany) obtained in a mechanically ventilated patient showing breath-to-breath variations in IAP (ΔIAP). Ventilator settings were: inspiratory positive airway pressure (IPAP) = 25 cm H_2O and positive end-expiratory pressure (PEEP) = 10 cm H_2O. The tracing shows the respiratory variations in IAP as well as the arterial pulse pressure variations (because of the vicinity of the heart to the nasogastric probe) at a heart rate (HR) of 120 beats per minute, highlighted by the circle. Mean IAP was 18.5 mm Hg with IAP = 17 mm Hg at end expiration (IAP_{ee}) and IAP = 20 mm Hg at end inspiration (IAP_{ei}), resulting in a ΔIAP (defined as $IAP_{ei} - IAP_{ee}$) = 3 mm Hg. The abdominal pressure variation (APV) can be calculated as ΔIAP divided by mean IAP (i.e. 3/18.5 = 16.2 %). Higher APV values for a given ventilator setting correspond to lower abdominal wall compliance. The thoraco-abdominal index (TAI) of transmission can then be calculated as ΔIAP divided by (Pplateau minus PEEP) or thus 3/15 = 20%

cardiac tamponade occurs as a result of an accumulation of fluid or air in the pericardium, usually as a result of trauma, haemorrhage, infection, or tumour [42]. The result is impaired filling of the ventricles and decreased cardiac output (CO). Strikingly, as little as 250 mL of fluid can cause acute cardiac tamponade, whereas under chronic conditions much greater amounts of fluid can accumulate as the cardiovascular system compensates with the slower, gradual accrual [43].

A similar condition arises when either ITP directly (in the case of thoracic compartment syndrome), or IAP indirectly (in the case of abdominal compartment syndrome) compresses the cardiac chambers. The latter is due to an upward movement of the diaphragm. In the case of increased ITP or IAP, coronary perfusion pressure (CoPP) is lowered:

$$CoPP = DBP - PAOP = DBP - ITP$$

with DBP = diastolic blood pressure and PAOP = pulmonary artery occlusion pressure.

Increased ITP also results in a more difficult preload assessment because of falsely increased invasively measured filling pressures [30, 44]. When the ITP or IAP rises above values of 10–12 mm Hg, CO drops due to an increased afterload (systemic vascular resistance), a decreased preload (diminished venous return, and falsely increased barometric filling pressures), and left ventricular compliance (due to the tamponade effect of increased ITP) [31, 45–48], as discussed below. This often manifests with tachycardia, a decreased MAP, and pulsus paradoxus (or an increased pulse pressure variation). Cardiovascular dysfunction and failure (low CO, high SVR) are common in conditions of increased ITP or IAP [49, 50]. All these can result in right ventricular dysfunction with secondary tricuspid valve regurgitation. Right ventricular function is predominantly load-dependent and successful response to intensive medical therapy predicts good outcome in heart failure patients [44, 51]. Eventually, the increased right-sided pressure and heart failure result in hepatic congestion, leading to hepatic fibrosis and cirrhosis. In addition to the more direct effects of cardiac tamponade, further distant effects on other organs may manifest (Fig. 1). Important issues to consider include:

1. Invasively measured haemodynamics (through a pulmonary artery catheter [PAC]) are difficult to interpret in conditions of increased ITP or IAP. This is because pressure-based or 'barometric' estimates of intravascular volume expressed as PAOP and central venous pressure (CVP) are increased. The importance of this is reflected in a patient's volume status being misinterpreted, potentially resulting in the institution of inappropriate and potentially detrimental therapy [5, 52].
2. Transmural (tm) filling pressures, involving the end-expiration value (ee) and ITP, may better reflect true preload status [34, 36]:

$$CVP_{tm} = CVP_{ee} - ITP$$

$$PAOP_{tm} = PAOP_{ee} - ITP$$

3. The index of transmission (IT) of alveolar pressures onto the vasculature allows for the calculation of CVP_{tm} and can be estimated as suggested by Teboul et al. [53]. The IT is higher in patients with good respiratory compliance (such as those with emphysema) and low in those with poor compliance (ARDS, lung fibrosis):

$$IT = (CVP_{ei} - CVP_{ee})/(Pplat - PEEP)$$

$$CVP_{tm} = CVP_{ee} - IT \times PEEP$$

4. A quick estimate of transmural filling pressures can also be obtained by subtracting half of the IAP from the end-expiratory filling pressure, as the average abdomino-thoracic index of transmission is 50% (illustrated in Fig. 3) [54]:

$$CVP_{tm} = CVP_{ee} - IAP/2$$

$$PAOP_{tm} = PAOP_{ee} - IAP/2$$

5. 'Volumetric' preload estimates such as right ventricular end diastolic volume index (RVEDVi), or global end diastolic volume index (GEDVi), are useful alternatives to pressure-based measurements in conditions of increased ITP [48, 55–58]. Correction of the GEDVi in relation to the global ejection fraction (GEF) can be used to fine-tune the true preload status in this setting [59].
6. The haemodynamic effects of IAH are aggravated by hypovolemia and the application of PEEP [60-64], whereas hypervolemia has only a temporary protective effect since it can aggravate tissue oedema, thus triggering a vicious cycle [23].
7. Increased IAP leads to an increase in the ITP, and provides an explanation for the pulse pressure variation and stroke volume variation increase (with PPV preferred over SVV) [65]. Our traditional threshold for fluid responsiveness therefore needs to be adapted in patients with IAH [66]. Fluid responsiveness in IAH is defined as a PPV above 20% rather than the usual 12% threshold. Systolic pressure variation should not be used in the setting of increased IAP or ITP because the increase seen in IAH and ACS only reflects a ΔUp phenomenon [67, 68].
8. Increased IAP can lead to a false negative passive leg raising test since venous return from the legs and

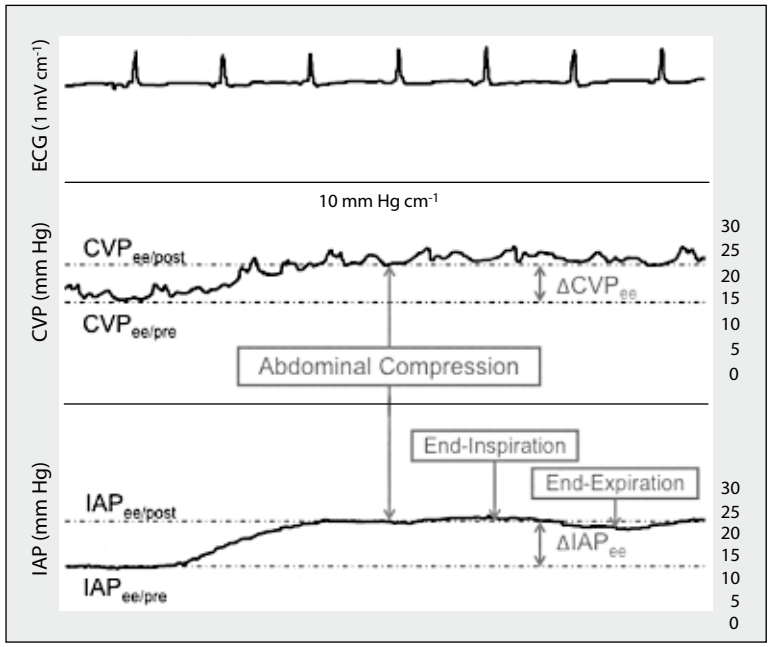

Figure 3. Calculation of abdomino-thoracic index (ATI) of transmission at the bedside. Simultaneous ECG, CVP and IAP tracing before (pre) and during (post) abdominal compression with a Velcro belt used to prevent incisional hernia. The abdomino-thoracic index of transmission can be calculated as follows:
The change in endexpiratory CVP: $\Delta CVP_{ee} = CVP_{ee/post} - CVP_{ee/pre} = 22 - 15 = 7$ mm Hg
The change in endexpiratory IAP: $\Delta IAP_{ee} = IAP_{ee/post} - IAP_{ee/pre} = 23 - 12 = 11$ mm Hg
The abdomino-thoracic index of transmission = $\Delta CVP_{ee}/\Delta IAP_{ee} = 7/11 = 63\%$

splanchnic pool may be impeded, causing a smaller endogenous fluid challenge [69, 70]

LIMBS AND EXTREMITIES

Limb, or extremity, compartment syndrome is a condition in which the pressure in a closed muscle compartment increases to a level that reduces capillary blood perfusion below the level necessary for tissue viability [6]. Permanent loss of function and muscle contraction may occur. Extremity compartment syndrome can be measured by a needle connected to a fluid-filled pressure transducer system. Normal compartment pressure (CP) values should be < 20 mm Hg [6]. This technique can be used to guide the need for surgical intervention.

Tissue perfusion pressure =
= Arteriolar (capillary) pressure — extremity CP

Extremity compartment syndrome is more common in obese patients where it mostly results from fractures following trauma (especially of the tibia), tight plaster casts, muscle contusions, bleeding disorders, burns (with eschars), venous obstruction, and arterial occlusion with post-ischaemic swelling [6]. Extremity compartment syndrome will result in muscle compression and rhabdomyolysis, which may cause hypovolemia, acute kidney injury (AKI), coagulopathy, acute lung injury (ALI), and shock [6]. Therefore, extremity compartment syndrome can also contribute to distant effects on other organs (Fig. 1). In the case of established extremity compartment syndrome, the only definitive treatment is decompressive fasciotomy, and muscle debridement in a case of necrosis [6, 71]. Initially, fluid resuscitation can be used to counteract the deleterious effects of extremity compartment syndrome on distant organ function. However, this may eventually also lead to a vicious cycle with increased tissue oedema and a further rise in compartmental pressures.

Apart from its influence on other distant organs, extremity compartment pressures may be influenced by increased IAP. ACS or pelvic compartment syndrome both diminish venous return from the extremities, promoting further limb swelling. Monitoring of high-risk patients should be undertaken. Wall et al. defined categories of high-risk patients warranting invasive monitoring [72]:

— Males aged > 35 years with fractures of the tibia and/or the radius/ulna
— High-energy injuries (open fractures and/or severe soft tissue injuries)
— Soft tissue injuries in males aged > 35 years with a bleeding disorder or receiving anticoagulants
— Crush injuries
— Prolonged limb compression (i.e. following drug overdose)
— High-risk patients should be assessed for extremity compartment syndrome at least every 4 h for a minimum of 24 h after the precipitating injury

ABDOMEN

HEPATIC COMPARTMENT SYNDROME

As a result of the liver being an encapsulated organ, local haematoma formation caused by trauma or bleeding diathesis (oral anticoagulants, liver cirrhosis) may compromise tissue perfusion by causing hepatic compartment syndrome [73, 74]. Local hepatic compartment syndrome, or increased upper abdominal compartment pressures, has been described following major liver surgery (trauma, tumour resection, liver transplantation) [75–78]. Furthermore, the liver appears to be particularly susceptible to injury in the presence of elevated surrounding pressures, especially in a case of IAH or ACS [79]. The causes are probably multifactorial and related to altered haemodynamics, damage to the microcirculation, and the presence of interstitial oedema. Animal and human studies have shown impairment of hepatic cell function and liver perfusion, even with only a moderately elevated IAP of 10 mm Hg [79, 80]. Plasma disappearance rate for indocyaninegreen may be a useful parameter, as it correlates not only with liver function and perfusion but also with IAP [81–83]. Increased IAP leads to decreased hepatic arterial flow, decreased venous portal flow, and an increase in portal-systemic collateral circulation, causing decreased lactate clearance, altered glucose metabolism, and altered mitochondrial function [76, 80].

RENAL COMPARTMENT SYNDROME

The association between IAH and renal impairment has been known for more than 150 years [84]. However, the exact pathophysiological interplay between IAP and acute kidney injury has only been studied intensively in recent years [77, 85, 86]. Elevated IAP significantly decreases renal blood flow and causes renal venous hypertension through pressure transduction, leading to renal dysfunction and failure [87, 88]. Oliguria develops at IAP >15 mm Hg and values > 25 mm Hg are associated with anuria, even in the presence of normovolemia [89, 90]. As expected, in the setting of hypovolemia or sepsis, renal function is more easily compromised [89, 90]. Renal perfusion pressure (RPP) and renal filtration gradient (FG) have been proposed as key factors in the development of IAP-induced renal failure:

$$RPP = MAP - RVP$$

Where RVP = renal vein pressure

$$FG = GFP - PTP = RPP - PTP = (MAP - RVP) - RVP = \\ = MAP - 2 \times RVP$$

Where GFP = glomerular filtration pressure and PTP = proximal tubular pressure

The above equations are, however, probably too simplistic since they don't take into account the autoregulatory effect of the afferent arterioles in the kidney. Autoregulation will keep renal blood flow (RBF) relatively constant, with a MAP between 75 and 200 mm Hg. In addition, these equations do not take into account a varying degree of vasoconstriction that may be present in the kidneys, depending on the underlying pathology leading to ACS. Nevertheless, in conditions of increased IAP, the RVP may be substituted by IAP, and the above equations become:

$$RPP = MAP - IAP$$

$$FG = MAP - 2 \times IAP$$

As stated before, these equations have limitations related to their inability to account for vasoconstriction and autoregulation [91]. However, theoretically the changes in IAP may have a greater impact on renal function and urine production than changes in the MAP. Therefore, it should not be surprising that decreased renal function, as evidenced by the development of oliguria, is one of the first signs of IAH. The kidneys may be considered the 'canary in the coalmine' for IAH [86]. An increasing number of observational studies have identified that IAH (IAP ≥ 12 mm Hg) is independently associated with renal impairment and increased mortality [77, 85, 92, 93]. The exact pathophysiological mechanisms of this are not yet fully elucidated, but may be multifactorial and include reduced renal blood flow, reduced CO, increased SVR, and alterations in neurohumoral factors [86]. Within the capsule of the kidney itself, local haematoma formation (caused by trauma or bleeding diathesis) may have a further adverse effect on tissue perfusion, causing a local renal compartment syndrome [94–96].

PELVIC COMPARTMENT SYNDROME

Three major compartments can be identified in the pelvic region, including the gluteus medius-minimus, gluteus maximus, and iliopsoas compartments. These are distinguished from the smaller compartment of the tensor fasciae latae muscle. Pelvic compartment syndrome is rare, and a clear history of trauma is often lacking [97–99]. It is often associated with drug and alcohol abuse, infections (necrotising fasciitis), and the use of anticoagulant therapy, especially low molecular weight heparin in the setting of diminished kidney function in the elderly [98, 100]. Increased pelvic compartment pressures may eventually increase IAP and affect kidney function due to bilateral ureteral obstruction, or by the same effects as described above in renal compartment syndrome. Measurement of upper and lower abdominal compartment pressures may provide helpful information [75]. Moreover, the presence of a massive intrapelvic haematoma, causing increased retroperitoneal pressure, can precipitate renal failure [101]. Decompressive fasciotomy of the gluteal compartment is probably the treatment of choice [6].

ABDOMINAL COMPARTMENT SYNDROME

Similarly to the head and the Monroe-Kellie doctrine, the abdomen can be considered as a closed box with partially rigid (spine, pelvis and costal arch) and partially flexible sides (abdominal wall and diaphragm) [1, 102]. Since the abdominal cavity can be considered as a relatively non-compressible and primarily fluid-containing compartment, behaving in accordance with Pascal's law, the IAP measured at one point can be assumed to represent the IAP throughout the entire abdomen [103–105]. In normal conditions, IAP ranges from 0 to 5 mm Hg [106, 107]. However, certain physiological conditions such as morbid obesity, ovarian tumours, cirrhosis, or pregnancy, may be associated with chronic IAP elevations of 10–15 mm Hg [1, 107–109].

The gold standard method for measuring IAP involves the use of a transurethral Foley catheter connected to a manometer (Holtech Medical, Charlottenlund, Denmark), or coupled to an AbViser valve (ConvaTec Medical, formerly Wolfe-Tory, UT, USA), or any other 'home-made' system that includes a pressure-measuring technique [103]. Continuous IAP measurement may be performed via a balloon-tipped catheter placed into the stomach (Spiegelberg, Hamburg, Germany or CiMON, Pulsion Medical Systems, Munich, Germany) [110–112]. Continuous IAP monitoring has the additional benefit of showing real-time interactions between the different compartments [5].

The term ACS was first used by Fietsam et al. in the late 1980s to describe the pathophysiologic alterations resulting from IAH secondary to aortic aneurysm surgery [113]. The World Society on the Abdominal Compartment Syndrome (WSACS – www.wsacs.org) was founded in 2004 to serve as a peer-reviewed forum and educational resource for all clinicians with an interest in understanding IAH and abdominal compartment syndrome. The first consensus definitions were in 2006 and 2007 [1, 114], and these were updated in 2013 [9]. Table 2 summarises the latest consensus definitions, while the new 2013 consensus definitions (including polycompartment syndrome) are listed in Table 3. According to these definitions, a polycompartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures [9]. Table 4 lists

Table 2. Consensus definitions. Definitions regarding intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) according to the 2006 and 2013 WSACS guidelines update (adapted from Malbrain et al. [1] and Kirkpatrick et al. [9])

Def	2006 definitions	Def	2013 definitions
1	IAP is the steady-state pressure concealed within the abdominal cavity	1	IAP is the steady-state pressure concealed within the abdominal cavity
2	APP = MAP – IAP	2	APP = MAP – IAP
3	FG = GFP – PTP = MAP – 2 * IAP		REJECTED
4	IAP should be expressed in mm Hg 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	3	IAP should be expressed in mm Hg 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
5	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline	4	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline
6	Normal IAP is approximately 5–7 mm Hg in critically ill adults	5	IAP is approximately 5–7 mm Hg and around 10 mm Hg in critically ill adults
7	IAH is defined by a sustained or repeated pathologic elevation of IAP $\geq$ 12 mm Hg	6	IAH is defined by a sustained or repeated pathologic elevation of IAP $\geq$ 12 mm Hg
8	IAH is graded as follows: Grade I: IAP 12–15 mm Hg Grade II: IAP 16–20 mm Hg Grade III: IAP 21–25 mm Hg Grade IV: IAP > 25 mm Hg	7	IAH is graded as follows: Grade I: IAP 12–15 mm Hg Grade II: IAP 16–20 mm Hg Grade III: IAP 21–25 mm Hg Grade IV: IAP > 25 mm Hg
9	ACS is defined as a sustained IAP ≥ 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction/failure	8	ACS is defined as a sustained IAP ≥ 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction/failure
10	Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention	9	Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention
11	Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region	10	Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region
12	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS	11	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

Table 3. New additional 2013 consensus definitions

Def	New 2013 consensus definitions
12	A poly-compartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures.
13	Abdominal compliance quantifies the ease of abdominal expansion, is determined by the elasticity of the abdominal wall and diaphragm, and is expressed as the change in intra-abdominal volume per change in intra-abdominal pressure in L (mm Hg)$^{-1}$.
14	An open abdomen (OA) is any abdomen requiring a temporary abdominal closure due to the skin and fascia not being closed after laparotomy. The technique of temporary abdominal closure should be explicitly described.
15	The open abdomen is classified with the following grading system: **1 – No Fixation** 1A: clean, no fixation 1B: contaminated, no fixation 1C: enteric leak, no fixation **2 – Developing Fixation** 2A: clean, developing fixation 2B: contaminated, developing fixation 2C: enteroatmospheric/cutaneous fistula, developing fixation **3 and 4 – Frozen Abdomen** 3: frozen abdomen, no fistula 4: frozen abdomen with enteroatmospheric/cutaneous fistula
16	Lateralisation of the abdominal wall refers to the phenomenon whereby the musculature and fascia of the abdominal wall, best seen by the rectus abdominis muscles and their enveloping fascia, move laterally away from the midline over time.

ACS — abdominal compartment syndrome; APP — abdominal perfusion pressure; FG — filtration gradient; GFP — glomerular filtration pressure; IAH — intra-abdominal hypertension; IAP — intra-abdominal pressure; MAP — mean arterial pressure; OA — open abdomen; PTP — proximal tubular pressure

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

A. 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 pleuropneumonia
• High body mass index#
• Pneumoperitoneum
• Abdominal surgery, especially with tight abdominal closures
• Pneumatic anti-shock garments
• Prone and other body positioning
• Abdominal wall bleeding or rectus sheath haematomas
• Correction of large hernias, gastroschisis or omphalocoele
• Burns with abdominal eschars
• Interstitial and abdominal wall oedema
B. Related to increased intra-luminal contents
• Gastroparesis
• Gastric distension
• Ileus#
• Volvulus
• Colonic pseudo-obstruction (Ogilvie)
• Intraluminal (colonic) abdominal tumour
• Enteral feeding
C. Related to intra-abdominal collections of fluid, air, tissue or blood
• Intra-abdominal or retroperitoneal tumour
• Damage control laparotomy
• Postoperative abdominal surgery#
• Retroperitoneal haematoma
• Liver dysfunction with ascites
• Abdominal infection (pancreatitis, peritonitis, abscess,…)
• Haemoperitoneum
• Pneumoperitoneum
• Laparoscopy with excessive inflation pressures
• Major trauma (liver or spleen rupture)
• Ruptured aortic aneurysm
• Peritoneal dialysis
D. Related to capillary leak and fluid resuscitation
• Acidosis* (pH below 7.2)
• Hypothermia* (core temperature below 33°C)
• Coagulopathy* (platelet count below 50,000/mm3 OR an activated partial thromboplastin time (APTT) more than two times normal OR a prothrombin time (PTT) below 50% OR an international standardised ratio (INR) more than 1.5)
• Polytransfusion/trauma (> 10 units of packed red cells/24 hours)
• Sepsis# (as defined by the American – European Consensus Conference definitions)
• Severe sepsis# or bacteraemia
• Septic shock#
• Massive fluid resuscitation# (> 3 L of colloid or >10 L of crystalloid/24 hours with capillary leak and positive fluid balance)
• Major burns

*The combination of acidosis, hypothermia and coagulopathy has been termed in the literature 'the deadly triad' [115, 116]
#Indicates statistically significant risk factors according to a recent meta-analysis [116]

some of the potential risk factors for the development of IAH [117, 118], including obesity (odds ratio (OR) 5.10; 95% confidence interval (CI), 1.92 to 13.58), sepsis (OR 2.38; 95% CI, 1.34 to 4.23), abdominal surgery (OR 1.93; 95% CI, 1.30 to 2.85), ileus (OR 2.05; 95% CI, 1.40 to 2.98), and large volume fluid resuscitation (OR 2.17; 95% CI, 1.30 to 3.63). All were identified as independent risk factors for IAH in a recent meta-analysis [118].

Table 5. Potential treatment options for compartment syndrome

1. Improvement of compartment wall compliance
• Sedation and analgesia (not fentanyl)
• Neuromuscular blockade
• Body positioning
• Negative fluid balance
• Skin pressure decreasing interfaces
• Weight loss
• Percutaneous abdominal wall component separation
• Subcutaneous linea alba fasciotomy (SLAF)
• Escharotomies
2. Evacuation of intra-compartmental 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
• Pericardiectomy
3. Correction of capillary leak and positive fluid balance
• Albumin in combination with diuretics (furosemide)
• Antibiotics and sepsis source control
• Colloids (hypertonic albumin) instead of crystalloids
• Dobutamine (not dopamine)
• Dialysis or CVVH with ultrafiltration
• Ascorbinic acid in burn patients
4. Specific therapeutic interventions
• Targeted compartment perfusion pressure (PP > 55 mm Hg)
• External negative compartment pressure
5. Rescue therapy
• ICS: decompressive craniectomy
• ACS: decompressive laparotomy
• TCS: decompressive sternotomy
• ECS: decompressive fasciotomy
• PCS: decompressive gluteal fasciotomy
• RCS: renal decapsulation
• HCS: hepatic decapsulation
• CCS: decompressive pericardiotomy
• OCS: orbital decompression

Analogous to the widely accepted and clinically utilised concept of CPP, abdominal perfusion pressure (APP) has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP) [19, 49, 119, 120].

$$APP = MAP - IAP$$

However, to date there have been no additional outcome benefits identified that would prompt the use of APP ahead of monitoring IAP [9]. More information on ACS with regard to IAP measurement, incidence, epidemiology, risk factors, pathophysiologic implications on end organ func-

tion, medical and surgical treatment are beyond the scope of the present review and are covered in other articles [9, 102, 103, 118, 121–122].

CLASSIFICATION OF POLYCOMPARTMENT SYNDROME

The clinical importance of the diverse aspects of polycompartment syndrome prompts a proposal of a new classification that stratifies the condition into two categories according to its underlying aetiology. Analogous to the classification of IAH or abdominal compartment syndrome, it is reasonable for polycompartment syndrome to be either primary or secondary, in view of the potential effect on organ function [124].

A **primary** compartment syndrome may be defined as a pathological rise of compartmental pressures with physical tissue or organ injury (i.e. intracranial haematoma or limb fracture).

In **secondary** compartment syndrome, there is no primary injury in the affected compartment and symptoms are solely based on pressure transmission from one compartment to another (i.e. abdominal compartment syndrome that develops following a tension pneumothorax) [124]. This was illustrated above in the case with polycompartment syndrome with secondary limb compartment syndrome and primary head and abdomen compartment syndromes.

SPECIFIC CONDITIONS

1. CARDIO-ABDOMINAL-RENAL SYNDROME (CARS)

The abdominal compartment could potentially form a missing link in the pathophysiology of acute decompensated heart failure and cardio-renal syndrome [86, 91, 125–127]. Recently, our group reported that raised IAP was prevalent in patients with advanced heart failure and reduced left ventricular ejection fraction, and that this increase in IAP correlated with impaired renal function [128]. However, IAH and ACS, defined as ≥ 12 mm Hg and > 20 mm Hg respectively, are less frequent and frank ascites is rare [128]. Importantly, medical treatment resulting in a decrease in the IAP may improve renal function, and in cases of persistently high IAP, ultrafiltration has been proven beneficial [128, 129]. However, further studies are needed.

Notably, while organ dysfunction in the intensive care literature has only been described when IAP exceeds 12 mm Hg, patients with acute decompensated heart failure appear to develop worsening renal function at a lower IAP [128]. This might suggest that in this clinical setting renal reserve during increased IAP is limited. Furthermore, although the degree of renal dysfunction correlates with the degree of elevated IAP, there can be a wide range of IAPs in relation to serum creatinine levels at presentation [128]. While we can only speculate why this discrepancy exists, other mechanisms, including coexisting systemic congestion, pre-existing occult renal insufficiency, as well as drugs used during the treatment of acute decompensated heart failure, probably play a role.

Absolute increases in blood or interstitial volume may not occur in every episode of acute decompensated heart failure (AHDF). This was illustrated by Chaudhry et al. [130] who demonstrated that most heart failure patients gain only 1 kg of weight before admission for ADHF. This implies that vascular redistribution is another important mechanism for elevated cardiac filling pressures. The splanchnic vasculature normally contains about 25% of the total blood volume, a large part of which can quickly be recruited to the circulatory system through elastic recoil of the splanchnic veins and sympathetically-mediated venoconstriction [131, 132]. Because of the extensive orthosympathic innervations of abdominal capacitance veins, it is probable that more blood is distributed to the effective circulation in states of increased sympathetic nerve system activation, such as ADHF.

Consequently, we propose the term *cardio-abdominal-renal syndrome* or *CARS*, to emphasise the potentially important role of the abdominal compartment and splanchnic vasculature in the pathophysiology of ADHF and cardio-renal syndrome [91].

2. HEPATO-ABDOMINAL-RENAL SYNDROME (HARS)

Acute kidney injury is a severe complication in patients with decompensating liver cirrhosis, occurring in about 20% of patients, and is associated with poor outcomes [133]. Patients with cirrhosis are susceptible to developing renal failure because of the progressive vasodilatory state (secondary hyperaldosteronism) and reduced effective blood volume. Microcirculatory damage and shedding of the glycocalyx layer results in the movement of fluid across the physiological vascular barrier resulting in the accumulation of interstitial fluid, thus contributing to the formation of IAH and ACS. Hepatorenal syndrome is initiated by portal hypertension, and may be triggered by bacterial infections, nonbacterial systemic inflammatory reactions, excessive diuresis, gastrointestinal haemorrhage, diarrhoea, nephrotoxic agents and IAH [134]. The term *HARS, hepato-abdominal-renal syndrome,* succinctly describes this pathophysiological process, as increases in the IAP may be the missing link in the development of AKI in decompensated liver failure. In patients with portal hypertension and oesophageal varices, increases in IAP may have deleterious effects on variceal haemodynamics, markedly increasing the volume, pressure, and wall tension of the varices. Increases in IAP may furthermore contribute to the progressive dilatation that precedes the rupture of the varices resulting in new oesophageal bleeding [135]. In patients with portal

hypertension and tense ascites, paracentesis may improve variceal wall stress [136] and kidney function if euvolaemia is maintained [137]. Orthotopic liver transplantation is the best current treatment and leads to a gradual recovery of renal function in the vast majority of patients.

3. HEPATO-ABDOMINAL-PULMONARY SYNDROME (HAPS)

Liver-related causes of dyspnoea in chronic liver disease are common and can be related to pulmonary or extrapulmonary problems (e.g. cirrhotic myopathy and cardiomyopathy, muscle waisting) [138]. Pulmonary causes can be subdivided into parenchymal and extraparenchymal (e.g. pleural effusions or restrictive disease from tense ascites). Parenchymal causes can be either alveolar (e.g. aspiration pneumonia, basal atelectasis), interstitial (e.g. lymphocytic interstitial pneumonia, fibrosing alveolitis, COP, or noncardiogenic pulmonary oedema from fluid overload or hepato-abdominal-renal syndrome), or vascular (e.g. alveolar haemorrhage, hepatopulmonary syndrome or portopulmonary hypertension) [139].

Hepatopulmonary syndrome is defined as an arterial oxygenation defect induced by intrapulmonary vascular dilatations associated with hepatic disease, and its prevalence is estimated to be around 20% in patients awaiting orthotopic liver transplantation. The clinical triad consists of: 1) liver disease and/or portal hypertension; 2) the presence of intrapulmonary vascular dilatations; and 3) increased alveolar-arterial gradient resulting in orthodeoxia (arterial hypoxemia in the upright position) and platypnoea (improved oxygenation in supine position). Chronic liver disease with portal hypertension is often associated with (tense) ascites. Again it is proposed that IAP may play a key role in the development of dyspnoea in patients with chronic liver disease. The new term *HAPS,* or *hepato-abdominal-pulmonary syndrome,* describes this clinical problem. Indeed, increased IAP and portal hypertension in liver cirrhosis may lead to an elevation of nitric oxide levels and activation of the renin angiotensin aldosterone system [140]. The resulting vasodilation, water and sodium retention in combination with the hyperdynamic state will lead to expansion of systemic and central blood volumes and interstitial fluid accumulation, triggering a vicious cycle.

CLINICAL MANAGEMENT

At present, in the setting of often limited evidence, we suggest that the management of patients with polycompartment syndrome should be based on three principles [141, 142]. Firstly, specific medical and surgical procedures are required to reduce compartment pressures (Table 5). These measures include improving compartment wall compliance, evacuation of intra-compartment contents, correction of capillary leak and positive fluid balance, and (surgical) rescue management. Secondly, general and organ supportive therapy (intensive care) of the critically ill patient should be tailored to individual needs. As suggested above, a major issue involves the iatrogenic injury caused by over-resuscitation with crystalloid fluids resulting in secondary compartment syndrome. Some studies have shown that the cautious administration of colloids not only seems to decrease the incidence of ACS in burn and trauma patients, but also the ACS associated complications and mortality, as well as the complications related to increased pressures in other compartments [10, 143–146]. The timing, speed, type and dose of fluid resuscitation will play a major role in future treatment and prevention of polycompartment syndrome. The motto is not to give too much fluid, but to give the right fluids judiciously. The results of the recent large randomised controlled trials showing harmful effects of hydroxyethyl starch fluids on kidney function may however complicate this clinical dilemma further [147]. Finally, optimisation and prevention of specific adverse events after surgical decompression resulting from ischaemia and reperfusion is warranted.

CONCLUSIONS

First defined in 2007, polycompartment syndrome is a constellation of physiological sequelae of increased compartmental pressures in multiple compartments of the body [4, 5]. While polycompartment syndrome is uncommon, its consequences can be significant. It may be beneficial to further classify into primary and secondary compartment syndromes (similar to ACS) and to recognise the different components of a polycompartment syndrome. Widespread use of (continuous) ICP and IAP monitoring, and in a subset of patients combining this with intermittent limb compartment pressure monitoring, will allow timely diagnosis of primary polycompartment syndrome. Given the existing guidelines on acute limb compartment syndrome, the World Society on the Abdominal Compartment Syndrome may need to consider guidelines for polycompartment syndromes in addition to abdominal compartment syndromes.

Within polycompartment syndrome, the abdomen plays a central role and the effect of IAH on different organ systems has been described, along with recommendations to monitor and compensate for these effects. Even chronic elevations of IAP seem to affect various organ systems in the body. The abdominal compartment does play a key role in the pathophysiology of acute decompensated heart failure, cardiorenal and hepatorenal syndromes. Therefore, in our opinion, the terms cardio-abdominal-renal syndrome or CARS and hepato-abdominal-renal syndrome or HARS seem to be more appropriate.

Different conditions precipitate the occurrence of polycompartment syndrome, including severe burns, massive

fluid resuscitation, severe sepsis, and/or prolonged hypotension. The ultimate treatment goal of polycompartment syndrome is not only to decrease the compartment pressure (by improving compartment wall compliance), but also to improve organ function with the ultimate goal of decreasing mortality. Decompressive craniectomy, sternotomy, pericardiotomy, fasciotomy and laparotomy are the only treatment options that have been shown to reach these goals in the past. However, less invasive techniques and medical treatment strategies show promise in achieving compartment pressure reduction and organ function improvement. This is probably where the future lies.

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