The Polycompartment Syndrome

Orbital Compartment Syndrome – OCS

Acute orbital compartment syndrome is a rare but treatable complication of increased pressure within the confined orbital space, intra-ocular pressure (IOP). The increased IOP may cause pressure-related decreased ocular perfusion pressure (OPP) similar to that caused by mass lesions or Graves disease. The condition presents with recognisable physical findings (eye pain, reduced ocular motility, pro-optosis, diplopia) and progressive visual deficit. Recognition and prompt treatment may prevent blindness. A recent study in burn patients showed that increased IOP was significantly (p= 0.015) associated with the amount of fluids given during the first 24 hours (37.2 ± 14.4 L vs 24.6 ± 12.3 L) and with the presence of periorbital burns. Emergent orbital decompression resulted in a drop in IOP from 59.4 ± 15.9 mmHg to 28.6 ± 8.2 mmHg. Conditions that can be associated with OCS are infection, inflammation, spinal surgery, optic nerve sheath compression (tumour or meningeoma), vascular problems with ophthalmic artery or retinal vein occlusion, traumatic asphyxia syndrome, bleeding diathesis or even after orbital extravasation of X-ray contrast material.
**Intracranial Compartment Syndrome – ICS**

A unique feature of the brain is that the intracranial contents are confined within a rigid bony cage. Because the volume of the cranial cavity is limited by its bony casing, any change in the size of any intracranial compartment leads to a reciprocal change in the size of the remaining compartments leading to alterations in cerebral perfusion pressure (CPP) and intracranial pressure (ICP). Many studies have been published regarding the best treatment options for ICH either focusing on lowering ICP (by diuretics or evacuation) or raising CPP (by maintaining correct MAP with fluids or vasopressors). However fluid therapy used to support CPP may cause retroperitoneal and visceral oedema, ascites accumulation and increased IAP, which in turn can further increase ICP. Therefore in patients with severe traumatic brain injury, treatment decisions may result in a vicious cycle that increases pressures in various compartments.

The effects of IAP and ITP on ICP have not been extensively studied to date, and remain a challenging area for laboratory and clinical investigators. In the study by Scalea, 78 patients had an ICS and underwent a decompressive craniectomy (DC), resulting in a significant decrease in ICP from 24 to 14 mmHg. The other 24 patients had a multiple CS and underwent both a decompressive craniectomy and a decompressive laparotomy (DL). The combination of DC and DL in these 24 patients led to a decrease in ICP from around 32 to 14 mmHg after DC and from 28 to 19 mmHg after DL (the effect being different depending on whether DC or DL was performed first). After DL, the IAP decreased from 28 to around 18 mmHg and so did mean airway pressure from 37 to 27 cm H$_2$O. The authors concluded that increased ICP can result from primary traumatic brain injury as well as from increased IAP, which has been documented before. Patients with PolyCS received significantly more fluids during the first 7 days of ICU stay, around 63 ± 21 L vs 40 ± 13 L (p< 0.001). They also stayed longer in the ICU, about 25 ± 13 days vs 17 ± 12 days (p= 0.01) and in the hospital, 29 ± 16 days vs 21 ± 14 days (p= 0.05). While there was a trend towards higher mortality in these patients (42% vs. 31%), it did not reach statistical significance. PolyCS should therefore be considered in multiple injured patients with increased ICP that does not respond to therapy.

**Thoracic Compartment Syndrome – TCS**

Thoracic compartment syndrome has traditionally been described in adult and paediatric patients undergoing cardiac surgical procedures. In the setting of substantial myocardial oedema, acute ventricular dilatation, mediastinal haematoma or noncardiogenic pulmonary oedema, sternal closure may precipitate cardiac tamponade physiology leading to haemodynamic instability or collapse. Theoretically TCS could also occur in patients with trauma, however is rarely seen due to the limited survival of patients whose injuries were significant enough to result in massive tissue oedema after resuscitation from thoracic trauma. In the ICU, increased ITP is seen most commonly in relation to sepsis, capillary leak, fluid resuscitation, positive pressure ventilation with high PEEP or dynamic hyperinflation, pneumothorax, COPD with auto-PEEP, diminished chest wall compliance (e.g. morbid obesity or eschars), lung fibrosis and ARDS. The most important strategy to prevent TCS or decrease the ITP and to facilitate closure is the limitation of resuscitation fluid therapy through the use of hypertonic saline or colloid solutions. The rising ITP, mean or peak inspiratory pressure during thoracic wall closure may serve as an early warning that the patient is at risk for TCS. The increased ITP (normal < 5–7 mmHg) can be measured via a balloon-tipped catheter positioned in the lower third of the oesophagus will exert its effect on the lungs, the heart and the brain (by limiting venous return). Since increased ITP is most commonly related to futile fluid resuscitation, IAP and ITP go hand in hand. Some key-issues to remember are:

- Best PEEP should be set to counteract ITP and IAP whilst in the same time avoiding over-inflation of already well-aerated lung regions
  
  - Best PEEP (cmH$_2$O) = IAP (mmHg)

- During lung protective ventilation, the plateau pressures should be limited to transmural plateau pressures below 35cmH$_2$O
  
  - $P_{platm} = P_{plat} - ITP = P_{plat} - IAP/2 < 35$ cm H$_2$O

- Increased ITP and IAP increase lung edema, within this concept monitoring of extravascular lung water index (EVLWI) seems warranted.

**Cardiac Compartment Syndrome – CCS**
Within the thorax, the heart can develop an isolated CS also called cardiac tamponade. Cardiac tamponade occurs when there is accumulation of fluid or air in the pericardium caused by trauma, haemorrhage, infection or tumour causing impaired filling of the ventricles and decreased cardiac output (CO). As little as 250 mL of fluid can cause acute cardiac tamponade whereas under chronic conditions greater amounts of fluid can accumulate as the cardiovascular system can slowly adjust. The same effect on the heart can occur via transmission of increased ITP either directly as seen with TCS or indirectly as seen with ACS, due to the cephalad movement of the diaphragm. In case of increased ITP or IAP coronary perfusion pressure (CoPP) is lowered: CoPP = DBP – PAOP = DBP – ITP. The increase in ITP will also result in a difficult preload assessment because traditional filling pressures will be erroneously increased. When ITP or IAP rise above 10-12 mmHg CO drops due to an increase in afterload (systemic vascular resistance) and a decrease in preload and left ventricular compliance. Tachycardia may develop, mean arterial blood pressure will decrease and a pulsus paradoxus may occur. Cardiovascular dysfunction and failure (low CO, high SVR) are common in conditions of increased ITP or IAP. Finally, hepatomegaly (backward failure) may develop in chronic cases, so that cardiac tamponade may have a distant effect on other organs.

Some key-issues to remember are:

- Our understanding of traditional haemodynamic monitoring techniques and parameters, however, must be re-evaluated in conditions of increased ITP or IAP since pressure-based or “barometric” estimates of intravascular volume as pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP) are erroneously increased.
  - The clinician must be aware of the interactions between ITP, IAP, PEEP, and intracardiac filling pressures
  - Misinterpretation of the patient's minute-to-minute cardiac status may result in the institution of inappropriate and potentially detrimental therapy
  - Transmural (tm) filling pressures, calculated as the endexpiration value (ee) minus the ITP better reflect preload:
    - CVPtm = CVPee – ITP
    - PAOPtm = PAOPee – ITP
  - A quick estimate of transmural filling pressures can also be obtained by subtracting half of the IAP from the endexpiratory filling pressure
    - CVPtm = CVPee – IAP/2
    - PAOPtm = PAOPee – IAP/2
  - “Volumetric” estimates of preload status such as right ventricular end diastolic volume index (RVEDVi) or global end diastolic volume index (GEDVi), are especially useful in conditions of the changing ventricular compliance due to elevated ITP.
  - The cardiovascular effects are aggravated by hypovolemia and the application of PEEP, whereas hypervolemia has a temporary protective effect.

Limb or Extremity Compartment Syndrome – ECS

The ECS is a condition in which the CP within the closed muscle compartment increases to a level that reduces capillary blood perfusion below the level necessary for tissue viability. Permanent loss of function and contracture may occur. The extremity CP can be measured via a needle connected to a fluid-filled pressure transducer system. Normal CP should be below 20 mmHg and should be used to guide the need for surgical intervention.

- Tissue PP = capillary pressure – extremity CP

The clinical findings are characterised by the 5 Ps: Pain, Pressure (tension on muscles), Paraesthesia, Paresis and Peripheral pulse alterations (diminished or prolonged capillary refill above 5 seconds). A crush injury can be caused by the patient's own weight in case of unconsciousness related to poisoning, drug overdose, strenuous exercise or during prolonged anaesthesia, especially if the patient has a high body mass index (BMI). External causes of increased extremity CP are mainly related to trauma with fractures (especially of the tibia) and tight
plaster casts, muscle contusions, bleeding disorders, burns (with eschars), venous obstruction, arterial occlusion with post ischaemic swelling, all causing muscle compression and further crush injury. This will result in muscle compression and rhabdomyolysis, which may cause hypovolemia, acute kidney injury and failure, coagulopathy, acute lung injury (ALI) and shock. Hence the increased extremity CP may have a distant effect on other organs. Besides aggressive fluid resuscitation, the only definitive treatment if CP rises above 30 mmHg is decompressive fasciotomy with muscle debridement in case of necrosis. Increased IAP related to ACS or Pelvic CS can have an effect on extremity CP due to the diminished venous return from the extremities to the central circulation causing further limb swelling.

Hepatic Compartment Syndrome – HCS

Within the capsule of the liver itself, local haematoma formation caused by trauma or bleeding diathesis (e.g. oral anticoagulants, liver cirrhosis,...) may have an adverse effect on tissue perfusion causing a local hepatic compartment syndrome. The liver appears to be particularly susceptible to injury in the presence of elevated surrounding pressures, thus especially in case of IAH or ACS. Animal and human studies have shown impairment of hepatic cell function and liver perfusion even with only moderately elevated IAP of 10 mmHg. Furthermore, acute liver failure, decompensated chronic liver disease and liver transplantation are frequently complicated by IAH and ACS. 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 indocyaninegreen (ICG) as this correlates not only with liver function and perfusion but also with IAP. 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 an increase in the portacollateral circulation, which all exerts physiological effects with decreased lactate clearance, altered glucose metabolism and altered mitochondrial function.

Renal Compartment Syndrome – RCS

Intra-abdominal hypertension (IAH) has been associated with renal impairment for over 150 years. It is only recently however that a clinically recognised relationship has been found. Elevated IAP significantly decreases renal artery blood flow and compresses the renal vein leading to renal dysfunction and failure. Oliguria develops at an IAP of 15 mmHg and anuria at 25 mmHg in the presence of normovolemia and at lower levels of IAP in the patient with hypovolemia or sepsis. Renal perfusion pressure (RPP) and renal filtration gradient (FG) have been proposed as key factors in the development of IAP-induced renal failure.

\[ RPP = MAP - RVP \]
\[ FG = GFP - PTP = RPP - PTP = (MAP - RVP) - RVP = MAP - 2RVP \]
- Where GFP = glomerular filtration pressure
- And PTP = proximal tubular pressure
In conditions of increased IAP, the RVP may be substituted by IAP, or thus:

\[ RPP = MAP - IAP \]
\[ FG = MAP - 2*IAP \]

- Thus, changes in IAP have a greater impact upon renal function and urine production than will changes in MAP. It should not be surprising, therefore, 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 (>15mmHg) is independently associated with renal impairment and increased mortality. The etiology of these changes is not entirely well established, however it may be multifactorial: Reduced renal perfusion, reduced cardiac output and increased systemic vascular resistance and alterations in humeral and neurogenic factors. Within the capsule of the kidney itself, local haematoma formation (caused by trauma or bleeding diathesis) may have an adverse affect on tissue perfusion causing a local renal compartment syndrome.

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Conclusions

First suggested in 2007, the polycompartment syndrome is a constellation of the physiologic sequelae of increased compartment pressures, be it ICP, ITP or IAP. 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 the 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, while 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.

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