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Understanding abdominal compartment syndrome

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Abdominal compartment syndrome (ACS) has evolved from an inconsistently reported, poorly understood phenomenon in patients after emergency abdominal surgery to an established syndrome that contributes to organ dysfunction in different types of critically ill patients [1]. ACS is a syndrome and not a disease, and as such it occurs in conjunction with many disease processes, either due to the primary illness or in association with treatment interventions. Indeed ACS has been reported to occur in medical patients and non-abdominal surgery patients alike, as well as in pediatric ICU patients [2, 3].

A number of studies have documented the impact of increased intra-abdominal pressure (IAP) both in animal and human studies, and several interventions aimed at reducing IAP have been evaluated. Although current definitions may suggest that increased IAP is only associated with organ dysfunction when reaching the level of ACS, all these studies have found that even at IAP levels between 12 and 20 mmHg—the range defined as intra-abdominal hypertension (IAH)—organ function may already be impaired, albeit often clinically unnoticeably. The incidence of IAH varies and is around 20–30 % on admission, while as many as 50–70 % of patients (depending on the condition) may develop IAH during the first week of ICU stay [4]. As ACS can be prevented and treated, it is reasonable to assume that the syndrome will be less prevalent in the future, while IAH will be encountered more often.

In a recent systematic review, Holodinsky et al. described 25 risk factors associated with IAH and 16 with ACS [5]. These can be roughly categorized in three categories, which may be more helpful at the bedside to identify patients at risk (Table 1). Especially noteworthy is the potential role of fluid resuscitation in the development of IAH and ACS. Recognizing the pivotal role of fluid resuscitation in the pathogenesis of IAH and ACS supplies the clinician with a target for preventive measures. Large volume resuscitation with crystalloids should be avoided in patients with or at risk of ACS [6].

Increased IAP induces organ dysfunction through two major pathways. Firstly, the pressure effect of increased IAP is transmitted directly to another body compartment and secondly, increased IAP affects systemic hemodynamics. This is exemplified by increasing intra-thoracic pressure by IAP transmitted directly to the thoracic cavity (upward displacement of the diaphragm). Importantly, the

Table 1 When to suspect IAH and ACS

Abdominal catastrophes	Severe organ dysfunction	Fluid balance
Trauma, peritonitis, acute pancreatitis, ruptured abdominal aortic aneurysm	Metabolic Respiratory Renal Hemodynamic	>3000–4000 mL in 24 h window
Often post-surgery		

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IAP can also impact the pressure in more distant body cavities such as the intracranial cavity and the brain within. In this case IAP may increase intracranial pressure (ICP) by inducing venous stasis.

Measuring and monitoring the IAP is the key to diagnosis and management of patients with IAH and ACS. IAP is determined by two elements—the intra-abdominal volume and compliance of the abdominal wall [7]. These may both be changed in patients with IAH, but do not necessarily contribute equally to IAH. It is important to appreciate that both of these essential elements may contribute to the pathogenesis of IAH since the interventions aiming to decrease IAP usually affect either one of the two. Baseline IAP may be higher in obese patients, and muscle activity should be absent for reliable IAP measurement.

As per consensus ACS is defined as an increased IAP (greater than 20 mmHg) and the onset of new organ dysfunction [8]. It may be difficult to differentiate between organ dysfunction caused by ACS and organ dysfunction caused directly by the underlying disease. In an attempt to differentiate between the two possible reasons for organ dysfunction, the temporal association between the onset of IAH/ACS and the dynamics of organ dysfunction may help to elucidate the contribution of IAP to organ dysfunction. Although it may be difficult to determine whether organ dysfunction is caused by IAH at lower levels of IAP (12–20 mmHg), it is reasonable to claim that organ dysfunction occurring in a time-dependent manner at an IAP greater than 20–25 mmHg allows the assumption of causal relation.

Until recently decompressive laparotomy was considered as the only treatment of choice for ACS. Currently, however, several non-surgical options are available [9]. Of these, percutaneous drainage of intra-abdominal collections has emerged as having a great potential to reduce IAP when increased intra-abdominal volume contributes to IAH [10].

Apart from interventions aimed at reducing IAP, the impact of IAH on overall management of the patient should be considered. IAH may be associated with enteral feeding intolerance, and gastric residuals should be checked frequently. Changes in abdominal wall (and notably diaphragmatic) compliance will impact the respiratory system [11], and mechanical ventilation should be adapted accordingly [12]. Parameters used for hemodynamic monitoring will also be affected; IAH causes an increase in intrathoracic pressures such as central venous pressure and pulmonary artery occlusion pressure, but also affects dynamic parameters such as stroke volume variation and pulse pressure variation [13]. Passive leg raising may also be false negative when IAP is high as venous return to the heart is impeded.

As fluid resuscitation is one of the key iatrogenic contributors to IAH, fluids should be used judiciously in IAH and ACS. Although fluids may indeed improve macrocirculation parameters in patients with IAH, the microcirculation in IAH may still be impaired; rather than trying to improve the cardiac output above minimal safe levels, decreasing IAP should be of primary concern [13]. In this context diuresis may be an unreliable parameter guiding fluid administration, as oliguria is very frequent in patients with IAH/ACS and fluid loading will only lead to further increases in IAP, and compromised kidney function [14].

In conclusion, understanding ACS requires knowledge of the basic mechanisms behind the syndrome as well as the pathophysiology of resulting organ dysfunction not limited to abdominal organs. This allows the clinician to choose the optimal treatment strategy to reduce IAP with a minimum of side effects. Furthermore, in order to manage the patients with ACS appropriately the clinician should consider the negative effects of increased IAP on hemodynamics and ventilation. Most importantly, understanding ACS implies acknowledging the importance of IAH and treating this condition before ACS develops.

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Compliance with ethical standards

Conflicts of interest Jan De Waele has served as a consultant to KCI and Smith and Nephew, and is a former president of WSACS—the Abdominal Compartment Society. Manu LNG Malbrain is founding president and current treasurer of WSACS—the Abdominal Compartment Society. He is also member of the medical advisory board of Pulsion Medical Systems (Maquet Getinge group) and consults for ConvaTec, KCI, Smith and Nephew, Spiegelberg, and Holtech Medical.

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