MONITORING INTRABDOMINAL PRESSURE

Something Old, Something New, Something Borrowed…

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Systematic interest in intraabdominal pressure began about two decades ago. Since than, the amount of scientific data regarding this problem have been risen exponentially. This happened due to several reasons: description of pathophysiological pathways which link intra-abdominal hypertension (IAH) to multiple organ dysfunction syndrome refinement of measurement techniques, emergence of national, international and worldwide scientific organizations dedicated to IAH and the dramatic increase in body of published papers dealing with this topic. The critical mass of this accumulation was reached and the explosive dissemination of data bursted.

Intra-abdominal hypertension is defined as sustained or repeated pathological elevation in intra-abdominal pressure (IAP) ≥ 12 mmHg. Abdominal compartment syndrome (ACS) is defined as sustained IAP ≥ 20 mmHg associated with new organ dysfunction. Three types of ACS were described: *primary* ACS, caused by a disease in the abdomino-pelvic area, *secondary* ACS, caused by conditions which do not originate in this region, usually associated with sepsis/systemic inflammatory response syndrome and/or aggressive fluid resuscitation, and *recurrent* ACS.

Measurement of intra-abdominal pressure changed over time regarding techniques and indications, as well. Measurement techniques changed from urinary, gastric or rectal catheter to intravascular (inferior vena cava) evaluation, from large volume to low volume bladder instillation, and special devices were developed and marketed, as a response of companies to increasing interest.

Indications for intra-abdominal pressure monitoring were changed, also. Initially indicated in case of abdominal trauma or severe abdominal emergencies (abdominal aorta aneurysm repair, e.g.), than indicated in intra-abdominal/extra-abdominal sepsis, nowadays intra-abdominal pressure monitoring is recommended in nearly all types of critical illness (trauma, surgical and medical). Measurement of IAP should be routine as monitoring of blood pressure, heart rate, temperature, respiratory rate, peripheral oxygen saturation, central venous pressure, and urinary output.

What caused this dramatic change of indications? There are several responsible factors: the documented association between IAH and mortality, the description of pathophysiological consequences of IAH and ACS, the association with aggressive fluid resuscitation and/or sepsis and shock, the influence on treatment strategy and the documented interrelation with other compartment syndromes. Let's talk a little bit about them!

Figures of IAH prevalence among critically ill patients are amazing (Table I). What is sticking? First, the similar figures between septic patients and the general population of critically ill. This means that, not only septic patients, but all critically ill patients are prone to develop IAH. Secondly, the high incidence of IAH in the medical

critically ill. In contrast with intuitive expectations, incidence of ACS in medical critically ill patients is higher than in surgical patients. Moreover, Vidal et al (2008) demonstrates that IAP is an independent predictor of mortality [1].

The pathophysiological consequences of IAH are well described: decrease in cardiac output, venous return and arterial blood pressure, increase in central venous pressure and pulmonary artery occlusion pressure, increase in airway pressure, inadequate ventilation with hypoxia and hypercarbia, splahnic hypoperfusion, decrease in urine output and metabolic/mixed acidosis. When the physiological consequences impair function, ACS is present and respiratory, cardio-vascular, renal and digestive compromise may occur.

Table I
Prevalence of IAH in critically ill and septic patients

IAP	Critically ill [2]			Septic [3]		
	All	Surgical	Medical	All	Surgical	Medical
≥12 mm Hg	58.8%	65%	54.4%	58%	67%	52.1%
≥15 mm Hg	28.9%	27.5%	29.8%	29%	25.2%	27.6%
≥20 mm Hg + organ dysfunction	8.2%	5%	10.5%	6%	4.1%	9.3%

IAP=intra-abdominal pressure, pts=patients

IAH and sepsis are intrinsically linked. In sepsis failure to early optimize tissue perfusion may lead to cellular hypoxia, ongoing systemic inflammation and multiple organ dysfunction. But IAH / ACS leads to tissue perfusion compromise and may mimic or augment septic shock. Indeed, patients with septic shock have the highest reported prevalence of IAH / ACS in all critically ill patients. IAH results in intra-/ extra-abdominal organ dysfunction due to pressure induced vascular compression / hypoperfusion and immune mediated organ dysfunction. IAH may act as the second insult in the two-event model of multiple organ dysfunction syndrome [4].

Monitoring of IAP may impact on treatment strategy. Early recognition and treatment of IAH may improve outcome. For instance, IAH increases central venous pressure, but decreases venous return, which may lead to unrecognized and untreated hypovolemia. By the other hand, IAH may be the consequence of aggressive fluid resuscitation or, better said, „volume overdose“. Measurement of IAP may help to better integrate data about fluid load and optimize treatment.

In severe acute pancreatitis IAP monitoring should begin at hospital admission. Once IAH is detected an algorithm-based treatment should guide therapy in order to decrease IAP [5]: percutaneous drainage of cytokines rich peritoneal fluid, early renal replacement therapy or decompressive laparotomy. In order to avoid disadvantages associated with an open abdomen a new method of decompression recommends a subcutaneous linea alba fasciotomy preserving skin as a cover [6].

Since 2007 the *polycompartment syndrome* (PCS) was described [7,8], which associates increased pressure in different body compartments. Compartment syndromes may be: within the head - *intracranial compartment syndrome (CS)* (traumatic or hemorrhage intracranial hypertension), and *orbital CS* (glaucoma or trauma); within the thorax – *thoracic CS* (tension pneumothorax), or *cardiac CS* (pericardial tamponade); within the abdomen - *abdominal CS* (trauma and sepsis), or *hepatic*, or *renal,* or *pelvic CS* (trauma); within extremities - *extremity CS* (crush injuries) (Table II). The PCS

describes not only the physiological consequences of increased pressure on the surrounding tissues, but also the interactions between different body compartments. The PCS is associated with trauma and results in increased morbidity and mortality. Since 1995 interactions between intraabdominal and intracranial pressures were described. Scalea et al [9] published 2007 a study of 102 patients with severe head injury. In 24 patients combined decompressive craniotomy and decompressive laparotomy resulted in significant decrease in intracranial pressure, documenting the interactions between intra-abdominal and intracranial pressure and the effects of specific interventions.

Table II
The four compartments (adapted from Cheatham [8])

	Head	**Chest**	**Abdomen**	**Extremities**
Syndrome	Intracranial CS	Thoracic CS	Abdominal CS	Extremity CS
Potential implications	Brain death	Cardio-resp. collapse	MODS	Extremity loss
Primary physiological parameter	Intracranial pressure (ICP)	Intrathoracic pressure (ITP)	Intra-abdominal pressure (IAP)	Extremity compartment pressure (CP)
Secondary parameter	Cerebral perfusion pressure (CPP)	Peak/mean airway pressure	Abdominal perfusion pressure (APP)	Peripheral arterial perfusion pressure
Therapeutic interventions	↓ ICP: CSF drainage ↑ CPP: fluids vasopressors,	↓ ITP: escharotomy, chest tube	↓ IAP: ascites ↑ APP: vasopressors, fluids	↓ CP: drainage
Resuscitative plan	Decompressive craniectomy	Decompressive sternotomy	Decompressive laparotomy	Decompressive fasciotomy
Importance	Adaptation of ventilatory support essential	Recognition of syndrome can be life saving	Prevention of bacterial translocation and MODS can be life saving	Recognition may be limb saving

CS=compartment syndrome, MODS=multiple organ dysfunction syndrome

Kimball et al [10] concluded in a survey of intensive care physicians published in 2006, that while urgent, aggressive treatment for other compartment syndromes (increased intracranial pressure, tension pneumothorax, pericardial tamponade, extremity compartment syndromes) is standard of care, most intensive care practitioners fail to treat intra-abdominal hypertension and the abdominal compartment syndrome with the same urgency, if at all.

In conclusion, measurement of IAP is an old tool. What is new is the recognition of its importance for guiding therapy and for patient outcome. As in case of sepsis, recognition of IAH / ACS should initiate an algorithm-based approach. Something old, something new, something borrowed…

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