

# Intra-abdominal hypertension and abdominal compartment syndrome in burns, obesity, pregnancy, and general medicine

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## Abstract

Intra-abdominal hypertension (IAH) is an important contributor to early organ dysfunction in trauma and sepsis. However, relatively little is known about the impact of intra-abdominal pressure (IAP) in general internal medicine, pregnant patients, and those with obesity or burns. The aim of this paper is to review the pathophysiologic implications and treatment options for IAH in these specific situations. A MEDLINE and PubMed search was performed and the resulting body-of-evidence included in the current review on the basis of relevance and scientific merit. There is increasing awareness of the role of IAH in different clinical situations. Specifically, IAH will develop in most (if not all) severely burned patients, and may contribute to early mortality. One should avoid over-resuscitation of these patients with large volumes of fluids, especially crystalloids. Acute elevations in IAP have similar effects in obese patients compared to non-obese patients, but the threshold IAP associated with organ dysfunction may be higher. Chronic elevations in IAP may, in part, be responsible for the pathogenesis of obesity-related co-morbid conditions such as hypertension, pseudotumor cerebri, pulmonary dysfunction, gastroesophageal reflux disease, and abdominal wall hernias. At the bedside, measuring IAP and considering IAH in all critical maternal conditions is essential, especially in preeclampsia/eclampsia where some have hypothesized that IAH may have an additional role. IAH in pregnancy must take into account the precautions for aorto-caval compression and has been associated with ovarian hyperstimulation syndrome. Recently, IAP has been associated with the cardiorenal dilemma and hepatorenal syndrome, and this has led to the recognition of the polycompartment syndrome. In conclusion, IAH and ACS have been associated with several patient populations beyond the classical ICU, surgical, and trauma patients. In all at risk conditions the focus should be on the early recognition of IAH and prevention of ACS. Patients at risk for IAH should be identified early through measurements of IAP. Appropriate actions should be taken when IAP increases above 15 mm Hg, especially if pressures reach above 20 mm Hg with new onset organ failure. Although non-operative measures come first, surgical decompression must not be delayed if these fail. Percutaneous drainage of ascites is a simple and potentially effective tool to reduce IAP if organ dysfunction develops, especially in burn patients. Escharotomy may also dramatically reduce IAP in the case of abdominal burns.

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Intra-abdominal hypertension (IAH) is an important contributor to early organ dysfunction in emergency general surgery, trauma, and sepsis patients. However, relatively little is known about the impact of intra-abdominal pressure (IAP) disturbances in general internal medicine and pregnant patients and those with obesity and burns. Although inciting pathologies are innumerable, the human pathophysiologic responses to inflammation and injury are similar, and thus the paucity of knowledge concerning IAH/ACS in other populations likely represents a lack of awareness on the part of caregivers and researchers. The aim of this paper is to review the pathophysiological implications, diagnosis, and treatment options for IAH and abdominal compartment syndrome (ACS) in the above-mentioned specific situations.

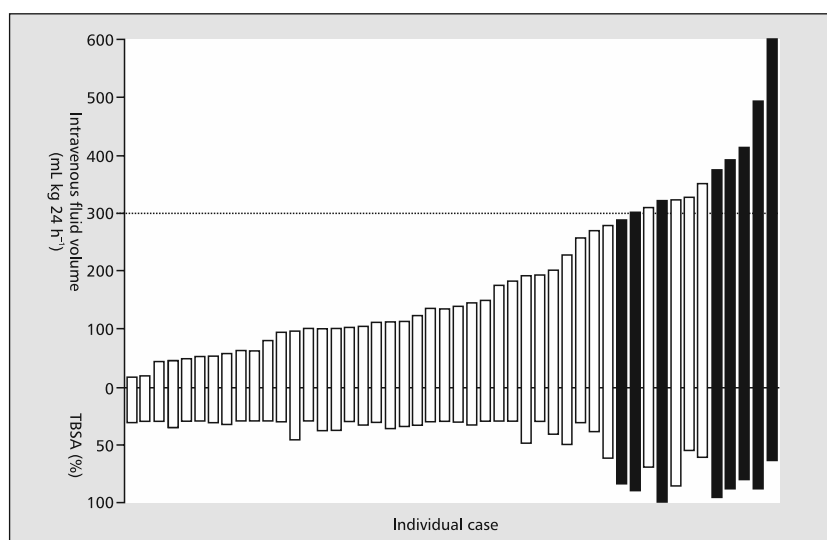
## METHODS

A MEDLINE and PubMed search was performed using the search terms (“abdominal compartment syndrome” or “abdominal hypertension” or “abdominal pressure”) and (“pregnancy” or “obstetrics” or “burns” or “obesity” or “heart failure” or “renal failure” or “endoscopy” or “ovarian hyperstimulation syndrome” or “eclampsia” or “polycompartment syndrome” or “cardiorenal syndrome” or “peritoneal dialysis”). The identified abstracts were screened and selected on the basis of relevance, methodology, and scientific merit. Full text articles of the selected abstracts were used to supplement the authors’ expert opinion and experience. The

bibliographies of the selected papers were also reviewed for other relevant citations. The resulting references were included in the current review that focuses on IAH in burns, obesity, pregnancy, and general internal medicine. Each topic will be discussed separately, hereafter, related to practical clinical questions on definitions, epidemiology, pathophysiology, diagnosis, and prevention and treatment, and concluding with some key messages for the reader. This “extended” review may also be supplemented by a book chapter on the same topic in the Core Critical Care Series [1].

## INTRA-ABDOMINAL HYPERTENSION IN BURN PATIENTS PATHOPHYSIOLOGY AND EPIDEMIOLOGY OF IAH IN BURNS PATIENTS

Severely burned patients usually develop IAH/ACS within 48 hours after injury [2]. The generalized increase in capillary permeability that occurs in severe burn patients contributes to extensive edema formation and intra-peritoneal accumulation of “third-space” fluid [3]. Bowel edema and fluid translocation is further worsened by venous hypertension caused by elevated IAP [4]. This increasing volume in the abdominal cavity, however, is reduced after capillary permeability improves. Therefore, secondary IAH in burn patients generally occurs within 48 hours after injury, during the initial resuscitation period, while ACS usually occurs after the acute phase, during subsequent septic episodes [5, 6]. Figure 1 shows the relationship



**Figure 1.** Resuscitation fluid volume (within first 24 hours) and % TBSA (total burned surface area) in individual burn cases. Solid bars indicate patients with abdominal compartment syndrome. Adapted from Oda et al. [6]. All patients with ACS had received more than 300 mL kg 24h<sup>-1</sup> and had more than 50% TBSA

between the resuscitation fluid volume within 24 hours post burn, percentage total body surface area (%TBSA), and complicating ACS.

The exact incidence of IAH and ACS among patients with burns is unknown, largely because it depends on the severity of burn injury, the method and frequency of IAP measurement, the exact definition used to define IAH, and the duration of IAP monitoring [7, 8]. A recent systematic review showed that the prevalence of ACS and IAH in severely burned patients is 4.1–16.6% and 64.7–74.5%, respectively [9]. The mean mortality rate for ACS in burn patients is 74.8%. The risk of ACS is higher in burned patients with a higher percentage of TBSA burned; however, patients with a lower burned TBSA may develop IAH/ACS as well [6]. ACS typically occurs when resuscitation volumes are greater than  $275 \text{ mL kg}^{-1}$  during the first 24 hours or TBSA burned is larger than 60% [6].

The effects of IAH/ACS in patients with severe burns are multifactorial. Raised IAP can lead to organ dysfunction and can affect all organ systems. The use of excessive fluid resuscitation in combination with increased capillary permeability as a result of the systemic inflammatory response to burn injury makes these patients particularly vulnerable to the development of IAH and ACS and cardiovascular, respiratory, and renal system dysfunction [5]. In severe burn patients, the kidneys are especially vulnerable to elevated IAP-related injury [10]. Markers for IAP-related organ damage might be superior to IAP measurement itself [9]. Thus, clinicians must accurately monitor patient fluid balance in the resuscitation period. Since an elevated IAP affects renal blood flow, urinary output is an unreliable index of the preload and intravascular volume resulting in the loss of an important physiologic parameter. Moreover, ACS as well as abdominal decompression for ACS increases susceptibility to multiple organ dysfunction syndrome (MODS) for severe burn patients and may also induce acute lung injury. The mortality rate of patients developing ACS is 50–80%, even when treated.

### **PREVENTION AND DIAGNOSIS OF IAH AND ACS IN BURN PATIENTS**

Secondary IAH in burn patients generally seems to occur within 48 hours after injury, during the initial resuscitation period. After this, when patients reach the 'polyuric or diuretic phase' (i.e., flow phase), the IAH/ACS risk decreases. However, if patients develop sepsis, the risk for IAH/ACS increases again and those not progressing spontaneously to the flow phase may need intervention [11]. Burn patients are also at risk of tertiary or recurrent ACS any time they require aggressive resuscitation as, for instance, after any overly aggressive burn excision [3, 4].

IAH/ACS should be suspected in all patients with severe burns. IAP measurement should therefore be per-



**Figure 2.** A severely burned (87% TBSA, 2<sup>nd</sup> degree) male, 20 hours post burn. Although elasticity of the abdominal wall was not impaired, a tense abdomen was observed, and the IAP elevated to 44 mm Hg

formed every 2 to 4 hours throughout the resuscitation period in burn patients with more than 20% TBSA. IAH exists when IAP exceeds 12 mm Hg and can result in early organ damage. Significant concern exists when the IAP exceeds 20 mm Hg, especially with new organ dysfunction as this defines the ACS, which is associated with a high mortality rate. One should pay attention to the fact that IAH/ACS might occur in patients without circumferential 3<sup>rd</sup> degree burns of their trunk. Burn patients with smoke inhalation may also be at risk of fluid sequestration [12]. In such patients, measurement of extravascular lung water (EVLW) may be helpful [13, 14]. Figure 2 shows a case in which although the elasticity of the abdominal wall was not impaired, a tense abdomen was observed with IAP elevated to 60 cm H<sub>2</sub>O. Recognizing IAH before ACS develops is very important as this may allow one to initiate medical management options.

Key to the prevention of ACS is the early recognition and treatment of IAH. Many burn physicians lack awareness of the deleterious effects of raised IAP and do not regularly measure it [15]. As burn patients are at high risk of developing IAH and ACS early in their ICU stay, routine measurement of IAP is warranted. Judicious use of fluids and avoidance of fluid over-resuscitation is the key element in the prevention of secondary ACS [16]. Percutaneous drainage of abdominal fluid collection may reduce IAP, particularly in the burn patient [17, 18]. Moreover, the choice of resuscitation fluid among critically ill patients with burns may have a clinical importance. Randomized studies have shown that hypertonic lactated saline (HLS) or plasma-based resuscitation requires less fluid and is associated with a lower risk of IAH and ACS [16]. Indeed, the use of plasma and hypertonic lactated resuscitation may prevent IAH or ACS. Despite the

fact that colloids decrease resuscitation volume needs, no benefit in preventing IAH has been proven [9]. In order to assess correctly the fluid status of the burned patient, transpulmonary thermodilution monitoring and measurement of volumetric indices like global end-diastolic volume (GEDV) and EVLW may be warranted in combination with functional hemodynamics [13, 14, 19–21]. It must be stated, however, that avoidance of burn shock and post-burn renal failure was a major advance of the 20<sup>th</sup> century. Moreover, as much as one does not want to induce IAH/ACS, physicians taking care of burn patients must remember that they still need to resuscitate severe burns. At present, however, how to do this remains an art rather than a science. Indeed, the fact is that over recent decades more fluids are being given to burn patients has led to the introduction of the concept of “fluid creep”, a phenomenon which may also be attributed to “opioid creep”.

Urinary output is often cited as an easy-to-use resuscitation target in burn resuscitation. Although this may be true for some patients, when IAH and ACS develop, oliguria may no longer be related to fluid depletion, and increasing resuscitation volumes may further worsen the problem of IAH [22].

#### **MANAGEMENT OF IAH AND ACS IN BURN PATIENTS**

Non-operative and percutaneous interventions may be applied before surgical decompression is considered. Nasogastric decompression, the use of neuromuscular blocking agents, and the removal of excess fluid by ultrasound-guided percutaneous drainage, or by a combination of continuous veno-venous hemofiltration (CVVH) with ultrafiltration and/or diuretics, are simple and possibly effective tools to reduce IAP [23]. An escharotomy of the trunk to improve abdominal wall compliance should be performed early, especially in the presence of 3<sup>rd</sup> degree burns [24, 25]. Although a midline laparotomy may make wound management more difficult in abdominal burn patients, it remains very effective in reducing IAP.

Regardless of surgical decompression, it is important to continue to measure IAP postoperatively in order to recognize recurrent IAH and/or ACS. The open abdomen after a laparotomy requires a temporary abdominal closure technique. The presence of abdominal burns may pose specific challenges to the management of the open abdomen with regard to infectious complications. The presence of significant protein loss via an open abdomen needs to be considered [26]. Early enteral and/or parenteral nutrition is of the utmost importance in these hypercatabolic patients, although recent literature results may advocate the opposite in ICU patients [27]. However, strong emphasis needs to be placed on the tremendous morbidity and high mortality of an open abdomen in patients with burns. In addition, early

use of percutaneous u/s guided drainage of peritoneal fluid can help prevent the development of IAH.

#### **INTRA-ABDOMINAL HYPERTENSION AND OBESITY**

##### **EPIDEMIOLOGY OF INTRA-ABDOMINAL HYPERTENSION IN OBESITY**

Obesity has reached epidemic proportions in the United States, as 30% of American adults are obese, defined as a body mass index (BMI) of 30–34 kg m<sup>-2</sup> (with BMI calculated as weight divided by height<sup>2</sup>). Morbid obesity is defined by a BMI of 35 kg m<sup>-2</sup> or greater. A similar trend of increasing BMI has been observed in many countries around the world. An elevated waist circumference defines central obesity. The metabolic syndrome is characterized by three or more of the following: central obesity, elevated triglyceride concentrations, lower high-density lipoprotein cholesterol levels, elevated blood pressure, and high fasting glucose concentrations. Some have introduced the term “syndrome X” [28].

Central obesity (metabolic syndrome) with an increased waist circumference and sagittal abdominal diameter has been shown to be associated with an increase in IAP [28]. Obesity has repeatedly been found to be associated with IAH [29–31]. Lambert et al. found the mean IAP in obese subjects with a mean BMI of 55 ± 2 kg m<sup>-2</sup> to be 8.8 ± 0.5 mm Hg, compared to the control group of lean females with a mean IAP of 0 ± 0.9 mm Hg [32]. Varela et al. found that, among 62 morbidly obese patients with a mean BMI of 49 ± 10 kg m<sup>-2</sup>, 77% had an IAP of 7–14 mm Hg [33]. Such data illustrates the direct relationship between obesity and IAP. While the exact pathogenesis of elevated IAP among patients with obesity is currently not known, Lambert et al. hypothesize that this association may be due to a direct mass effect of intra-abdominal adipose tissue [32]. Recent studies have found that obesity leads to chronic elevation of IAP and that IAP is positively correlated with BMI [29]. In many patients IAP is 12 mm Hg or higher, putting them at risk of acute organ dysfunction. The “normal” values of IAP in the obese patients should therefore be considered between 7 and 14 mm Hg [34, 35]. A possible explanation for higher pressures in the obese is that there could be a direct effect from the intra-abdominal adipose tissue itself on the measurement of IAP. The relation of chronically elevated IAP with chronic organ dysfunction is attracting increasing attention. This includes evidence that IAP elevation correlates with diminished pulmonary function and with systemic hypertension [36–38].

As baseline IAP is higher in morbidly obese patients, the influence of IAH and its possible dynamics of inducing organ dysfunction should certainly be considered. Although

there are a number of chronic diseases possibly related to elevated IAP, higher than usual IAP levels may be required in morbidly obese patients before organ dysfunction develops. However, the physiologic reserve may also be lower in obese patients.

### ***PATHOPHYSIOLOGY OF IAH IN OBESE PATIENTS***

An increase in IAP can be either acute, as in ACS, or chronic, as with the development of morbid obesity or possibly pregnancy. Acutely elevated IAP has deleterious cardiovascular, renal, pulmonary, and splanchnic consequences and may require emergent abdominal decompression. In contrast, the chronic elevation of IAP, seen in morbid obesity, has been thought to contribute to other disease processes including hypertension, proteinuria, pseudotumor cerebri (PTC), pulmonary complications, gastro-esophageal reflux disease (GERD), urinary stress incontinence, venous stasis and abdominal wall hernias. However, the evidence in support or against these associations is limited.

An acute increase in IAP may produce an increase in central venous pressure and systemic vascular resistance, and a decrease in venous return, cardiac output, visceral blood flow, and renal blood flow [30, 39]. Thus, in the setting of chronic IAP and morbid obesity, recent studies have suggested that obesity-induced IAH contributes to systemic arterial hypertension and many other serious conditions [29, 32, 33, 40, 41]. In contrast, a normal IAP appears to offer a protective effect against systemic hypertension. However the pathogenesis is currently unknown and other studies have not demonstrated this correlation.

Morbidly obese individuals have significantly higher IAP and a higher prevalence of PTC [39]. The relationship between abnormally high IAP and the development of PTC has been examined in animal models and clinical cases showing that acutely increasing IAP to 25 mm Hg above baseline causes a significant increase in intracranial pressure [39, 42, 43]. PTC may result from increased pleural and intrathoracic pressures found in obese patients, which obstructs cerebral venous outflow via the jugular system.

Morbidly obese individuals are at a higher risk of experiencing pulmonary complications. It has been demonstrated that both forced expiratory volume and forced vital capacity are inversely correlated to IAP, thereby suggesting that an elevated IAP may result in pulmonary restrictive disorders.

Gastro-esophageal reflux disease (GERD) is prevalent among the morbidly obese. In the setting of obesity-related IAH, the resistance gradient between the stomach and the gastro-esophageal junction changes: gastro-esophageal junction resistance is higher in obese subjects, compared to the lean control. However, neither Lambert nor Varela found GERD to be associated with elevated IAP [32, 33]. In fact,

although counter-intuitive, Lambert et al. found GERD to be associated with a lower mean IAP in morbidly obese patients.

### ***MANAGEMENT OF IAH IN OBESE PATIENTS***

Management of IAH is similar in obese patients and should correspond to the established guidelines for management of IAH/ACS [44]. However, this management is more challenging, and may be associated with higher complication rates. Although percutaneous drainage of intra-abdominal fluid collections may be more difficult, and open abdomen management in morbidly obese patients may pose more practical problems, in essence, the strategies are no different from non-obese patients.

Chronic IAH has been postulated to cause incisional hernias in obesity. Studies on this topic however report no correlation between IAP and the presence of an incisional hernia [30]. However, most of these studies are too small to statistically analyze.

### ***INTRA-ABDOMINAL HYPERTENSION DURING PREGNANCY***

#### ***PATHOPHYSIOLOGY OF IAH DURING PREGNANCY***

In the second and the third trimester of pregnancy, the uterus occupies a major part of the abdominal cavity, and in the supine position breathlessness and decreased blood pressure (“supine hypotension syndrome”) are often seen [45, 46]. These symptoms are due to restriction of diaphragmatic motion and compression of the inferior vena cava. IAP is usually only slightly elevated, except in some rare conditions like ovarian hyperstimulation syndrome (OHSS) [47]. Furthermore, the symptoms are alleviated in the lateral, sitting, or standing positions. Due to hormonal influences during pregnancy the abdominal wall is slowly stretched, increasing its compliance, which reduces the potential for an increase in IAP caused by the expanding uterus. As a result, end-organ dysfunction as seen in critically ill patients with acute primary or secondary IAH is rare because the body has time to adapt to the slowly increasing IAP levels during the pregnancy. However, if IAP increases acutely due to other reasons (e.g., haemorrhage or pneumoperitoneum during laparoscopy), perfusion of the uterus and the foetus may be severely compromised [48].

The expansion of intra-abdominal contents in the form of fluid or tissue is the fundamental cause of increasing IAP leading to the pathophysiology of IAH and ACS. Pregnancy, with a growing fetus and the increases in intra-abdominal fluid and tissue, could be considered a perfect storm for the development of IAH/ACS. In fact, several studies have demonstrated significant increases in IAP, particularly during the third trimester of pregnancy [46, 49–52]. However, IAP levels that would be expected to cause end-organ com-

promise in most patients seem to be well tolerated in the gravid female. As mentioned above, the gradual onset of IAP with pregnancy allows for adaptation to pressures that might otherwise cause harm [45]. One mechanism for this adaptation is thought to be the development of collateral blood flow from the lower extremities and abdomen [53]. It has been generally accepted that these adaptations nullify the risk for the development of IAH/ACS in pregnancy. However with an ever-increasing understanding of the subtle and significant physiologic effects of elevated IAP we are beginning to recognize the possibility that IAH/ACS may be a direct cause or contributing factor to several common disease processes in pregnancy that can affect both the mother and the fetus. These include pre-eclampsia; eclampsia; hemolysis, elevated liver enzymes, and low platelet counts (HELLP) syndrome; fetal hypertension and fetal bladder; and urinary tract dysfunction [51, 54–62]. Despite adaptation for elevated IAP in pregnancy, risk factors for IAH/ACS do still exist but may be difficult to quantify given that pregnancy introduces a unique physiology, which therefore introduces unique risk factors and IAH related disease processes. These conditions may thus require unique monitoring endpoints. It is still unclear whether the incidence of preeclampsia and IAH in first pregnancies is different from that in multiparous women.

Despite the limited understanding of IAH in maternal care, even less is known regarding its effects on the fetus [45]. Several animal studies have confirmed that the mammalian fetus *in utero* is subject to transmitted IAP [57, 58, 63]. IAH was found to decrease uterine blood flow and induced a resultant compensatory fetal hypertension [64] similar to that seen during laparoscopy even with inert gasses rather than CO<sub>2</sub>. In a gravid rabbit model, Karnak *et al.* found that intra-amniotic pressure (IAMNP) was linearly related to IAP as defined by  $IAMNP = IAP \times 0.8 + 2.0$ . The IAP and IAMNP were measured through catheters inserted respectively into both the intraperitoneal and intraamniotic cavities at 20 days gestation [58]. Given the increasing tolerance for laparoscopic surgery in gravid females, further research in this area is of critical importance.

Adapting to a growing fetus requires significant physiologic changes in the female body. These include normally induced relaxation of ligamentous tissue to accommodate the growing fetus; overall increase in cardiac output by 50% and an eight to ten fold increase in uterine blood flow [65, 66]. In addition, an adaptation that may protect against the effects of increased IAP occurs through the development of collateral blood flow that develops in anatomic regions less impacted by increased IAP including the vertebral and epidural vascular system [53]. However, variation in the development of these collateral vessels increases the risk of supine hypotensive syndrome caused by compression

of the inferior vena cava and may also increase risk for the effects of IAH/ACS.

### **INDICATIONS FOR IAP MEASUREMENT DURING PREGNANCY**

Although there is a dearth of literature on the effects of increased IAP in pregnancy, it is intuitive that pregnant patients are uniquely at risk of IAH/ACS due to the development of both acute and chronic IAP elevation [45]. While, there is no evidence to suggest that monitoring IAP is necessary in an uncomplicated pregnancy, as with other chronically adaptive IAP processes like obesity, chronic liver failure with ascites, and tumors, IAP monitoring and clinical decision-making should be based on trended pressures and clinical presentation rather than utilizing standard pressure measurements [35]. Patients with significant increases in IAP and concomitant organ failure should be considered for medical and/or surgical IAH/ACS management. To not consider IAH/ACS in such critical situations could be life threatening for both mother and fetus.

The clinician must be aware that body position affects IAP. Chun *et al.* found that IAP measurements were significantly higher in the fully supine position compared with a leftward position of 10° [46]. The authors hypothesized that the weight of the gravid uterus might have direct impact on the bladder, thereby falsely elevating the IAP measurement when fully supine.

Questions arise as to the validity of IAP measurements per WSACS guidelines in a pregnant patient after the 2<sup>nd</sup> trimester. Current guidelines describe IAP measurement in the fully supine position at end-expiration [23, 67]. Such a maneuver in pregnancy, however, could be detrimental, and we cannot recommend it [45].

### **RELATION OF IAH WITH PREECLAMPSIA AND ECLAMPSIA**

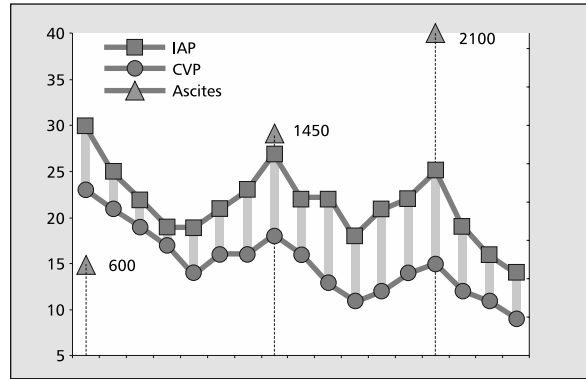
Preeclampsia, part of a spectrum of hypertensive disorders of pregnancy, is defined as the development of arterial hypertension and proteinuria after 20 weeks gestation [45, 68], and is associated with significant maternal morbidity and death [68, 69]. Two dramatic case reports have described overt ACS as a complication of preeclampsia/eclampsia/HELLP (H — hemolysis, EL — elevated liver enzymes, LP — low platelet count) syndromes requiring urgent life-saving interventions [54, 55]. The diagnosis of peripartum ACS in these cases was challenging not only due to the lack of well-established normative pregnant values of IAP, but also because of the overlap of signs and symptoms between ACS and severe preeclampsia such as oliguria, and nonspecific abdominal pain [35, 54]. Sugerman has recently hypothesized that IAH plays a central role in initiating the multi-system cascade of diminished perfusion and inflam-

mation associated with the various clinical manifestations of preeclampsia [51].

Toxemia of pregnancy occurs in 5 to 14% of pregnancies and represents a spectrum of obstetrical disease beginning with preeclampsia and including eclampsia and HELLP syndrome [70–72]. Numerous theories have attempted to explain this disease process without a clear unifying theory. It has been suggested that increased IAP may be an important trigger to this type of physiology through an IAP-induced reduction of renal blood flow which, in turn, activates the renin-angiotensin axis resulting in systemic hypertension and decreased placental blood flow. A relationship is further suggested in that most toxemia occurs in the third trimester when IAP is most elevated. In addition, we speculate that the cure for toxemia, namely fetal delivery, further reflects this relationship as delivery also results in significant reduction in IAP. However, due to a lack of clear research in this area, these conclusions have yet to be verified.

**OVARIAN HYPERSTIMULATION SYNDROME AND OTHER GYNECOLOGIC CONDITIONS IN RELATION TO IAH**

Ovarian hyperstimulation syndrome (OHSS) is an increasingly common complication of ovulation induction for assisted reproduction [61, 62]. The mechanism is not entirely understood but is a response to exogenous administration of human chorionic gonadotropin (HCG) [73]. It is hypothesized that the use of HCG in ovulation induction triggers a systemic inflammatory response resulting in increased capillary permeability and ovarian neoangiogenesis. Significant third space fluid accumulation with pleural, pericardial, and abdominal effusions may occur as a result of capillary leak (ovarian neoangiogenesis) and along with visceral edema, increased IAP may result [61, 62]. In its most severe form, massive and rapid accumulation of abdominal ascites results in overt ACS [73]. Management includes intensive care unit admission with IAP monitoring and paracentesis to relieve IAH or ACS, especially if respiratory failure occurs [62, 73]. Figure 3 and Table 1 show the evolution of IAP in a patient with OHSS after staged paracentesis. During staged evacuation, the compliance of the abdominal wall gradually increased from 55 to 191 mL cm H<sub>2</sub>O (Table 1). The compliance can be calculated by the



**Figure 3.** Evolution of IAP and CVP in a patient with OHSS. Evolution of intra-abdominal pressure (IAP) and central venous pressure (CVP) in a patient with ovarian hyperstimulation syndrome (OHSS) after gradual staged evacuation of ascites. Note the improvement of abdominal wall compliance over time with larger ascites volumes needed to result in the same drop in IAP (time markers per 4 hours)

change in intra-abdominal volume divided by the change in IAP (or thus  $\Delta IAV/\Delta IAP$ ). As assisted reproduction increases in prevalence, it becomes imperative to recognize this relatively common complication and to consider the potential role of IAH in its pathophysiology.

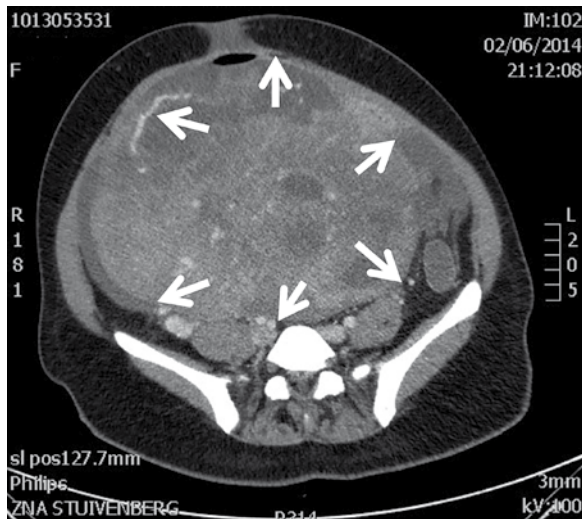
A wide range of additional gynecologic pathology can be associated with IAH/ACS related by the propensity to cause intra-abdominal mass effects. Meig’s syndrome, ovarian mucinous cystadenoma and other solid ovarian tumors for example, are associated with hydrothorax and ascites. Definitive therapy usually requires surgical removal of the tumor itself [74]. Other reported conditions associated with IAH, such as ovarian tumors, are shown in Figure 4 [75].

**MANAGEMENT OF IAH DURING PREGNANCY**

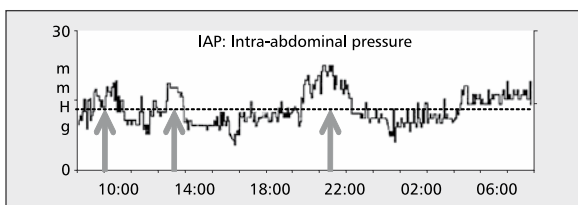
There remains a significant lack of research and evidence-based recommendations for the management of IAP in pregnancy. IAP is elevated in pregnancy and most women develop adaptive mechanisms that minimize the impact of this pressure [46]. However, IAH may combine with other risk factors to either cause or exacerbate several common obstetrical/gynecological disease processes. Due to baseline increases in IAP, all critically ill obstetrical/gynecologic patients should undergo vigilant IAP monitoring.

**Table 1.** Evolution of IAP and calculation of abdominal wall compliance in a patient with OHSS (see also Fig. 3)

Day	IAP before (mm Hg)	IAP After (mm Hg)	$\Delta IAP$ (mm Hg)	Ascites amount (ml)	Cab (mL cm H <sub>2</sub> O <sup>-1</sup> )
1	30	19	11	600	55
2	27	18	9	1450	161
3	25	14	11	2100	191



**Figure 4.** Abdominal compartment syndrome in a patient with an ovarian mucinous cystadenoma. A 38-year-old woman presenting with a tensely distended abdomen developed hypotension, respiratory distress, and oliguria based on the diagnosis of an ovarian tumor complicated by abdominal compartment syndrome. Contrast enhanced CT of the abdomen shows an inhomogeneous mass of 20 by 15 cm, expanding to all directions (arrows) and compressing the surrounding vascular structures



**Figure 5.** Effect of peritoneal dialysis on IAP measured continuously via the stomach. Continuous IAP tracing in a patient on peritoneal dialysis. The IAP was measured via a balloon-tipped nasogastric tube connected to a Spiegelberg monitor (Spiegelberg, Hamburg, Germany). Dialysis fluid was instilled into the abdominal cavity at 9:00; 13:00 and 21:00 (large arrows). When the abdomen was filled, the IAP increased from baseline values of 9 mm Hg up to 22 mm Hg and this resulted in difficult ventilation with a drop in tidal volume and increased end tidal  $\text{CO}_2$  values. The dotted line shows the IAH grade I threshold of 12 mm Hg

## INTRA-ABDOMINAL HYPERTENSION IN GENERAL MEDICINE

### INTRA-ABDOMINAL PRESSURE MONITORING DURING PERITONEAL DIALYSIS

IAP may be markedly increased during acute peritoneal dialysis (PD), when a significant amount of dialysis fluid, usually 2–4 L in an adult, is introduced into the peritoneal cavity. In humans, decreased cardiac output and increased pulmonary artery pressures have been reported [76, 77]. Figure 5 shows the effects of peritoneal dialysis on continuous IAP measured via a balloon-tipped catheter in the

stomach in a mechanically ventilated patient. This increase in IAP was associated with an increase in end-tidal  $\text{CO}_2$  when the abdomen was filled. For these reasons, acute PD is not commonly performed in adults in the intensive care setting, but it can be used in circumstances where other forms of acute renal replacement therapies are not available [78].

Studies comparing acute PD with veno-venous haemofiltration in patients with sepsis-induced acute renal failure have reported a significantly higher mortality with PD [79]. Although this could be due to less effective dialysis, organ dysfunction due to intermittent IAH (up to 2.5 times the baseline value) when the abdomen is filled is another possible explanation, since this may result in reduced cardiac output and blood pressure (Fig. 5). A pathophysiologic explanation may be compression of the inferior vena cava with resultant decreased venous return [76]. Such effects could be aggravated in situations of hypovolemia and positive pressure ventilation. In children, PD is usually well tolerated and it is still a common procedure in critically ill pediatric patients who develop acute renal failure.

IAH is a risk factor for abdominal wall complications (hernia acquisition and fluid leakage) in patients on chronic ambulatory peritoneal dialysis (CAPD) [80]. Besides IAP, advanced age, polycystic kidney disease, and high BMI were also independent risk factors for these complications. Automated CAPD with low daytime fill volume and pressures should be considered in all patients at risk for such complications. A strong correlation between BMI and IAP in children on PD has been noted [81], which gives a better understanding of the individual variability and of the unique relationship between IAP and intraperitoneal volume. It is therefore recommended that filling pressures during CAPD should be limited to 14 mm Hg [82].

### INTRA-ABDOMINAL HYPERTENSION IN THE HAEMATOLOGY PATIENT

Recent studies have referred to the increased incidence and consequences of IAH in haematological patients [83–85]. The causes are multiple and may also be multifactorial. Growth factor-induced capillary leak syndrome with concomitant large volume fluid resuscitation and third space sequestration, chemotherapy induced ileus, colonic pseudo-obstruction (Ogilvie's syndrome), mucositis or gastroenteritis (graft versus host disease) may produce IAH. Sepsis and infectious complications aggravating intestinal and capillary permeability favor fluid accumulation. Extramedullary hematopoiesis as seen with chronic myeloid leukemia can also result in hepatosplenomegaly [84], chronic IAH, and chronic (irreversible) pulmonary hypertension. The mechanisms of veno-occlusive disease seen after stem cell transplantation may be triggered by or related to increased IAP [85].



### **INTRA-ABDOMINAL HYPERTENSION IN GASTROENTEROLOGY**

ACS has been described in patients with toxic megacolon related to *Clostridium difficile* gastro-enteritis [86, 87]. IAH may also trigger (re)bleeding of esophageal varices in patients with end stage liver cirrhosis. These patients often require placement of a nasogastric tube to decompress the stomach after endoscopy [88]. ACS has been reported after colonoscopy complicated with perforated diverticulitis or during gastroscopy in animals [89, 90]. Ascites as a result of liver failure and or hepatorenal syndrome can increase IAH. Ascitic fluid can be safely percutaneously drained in patients with these conditions [91–93].

### **INTRA-ABDOMINAL HYPERTENSION IN RESPIRATORY CONDITIONS**

ACS has been associated with noninvasive ventilation with the head of bed (HOB) at 45° due to aerophagia [94], in relation to tension pneumothorax [95, 96], and IAH has been reported in COPD patients when measured at end-expiration during forced breathing [97]. ACS and acute kidney injury due to excessive auto-positive end-expiratory pressure have also more recently been described in a patient with severe COPD [98].

### **INTRA-ABDOMINAL HYPERTENSION IN NEUROLOGIC CONDITIONS**

Raised IAH due to constipation, ileus, or small bowel obstruction has been reported to play a role in malfunctioning ventriculo-peritoneal shunts in patients with hydrocephalus [99]. Within the concept of the polycompartment syndrome increased IAH may be an extracranial cause for increased intracranial pressure (ICP) in patients with combined cranial and abdominal trauma [100]. Scalea et al. was the first to introduce the term multiple compartment syndrome in a study of 102 patients with increased intra-abdominal (IAP), intrathoracic (ITP), and intracranial (ICP) pressures after severe brain injury [100]. These authors suggested that different compartments within the body are not isolated and independent entities but instead are closely connected. As the term multi- or multiple compartment syndrome is now mostly used in relation to multiple limb trauma needing fasciotomy, the term *polycompartment syndrome* (PCS) was proposed in order to avoid confusion [101] and is now accepted [44]. While a discussion of the PCS is beyond the scope of this paper, it is important for the clinician to understand that pressure-related interactions occur between the four major bodily compartments: the brain, thorax, abdomen, and extremities [102, 103]. Laparoscopic surgery is therefore not the first option in patients with traumatic brain injury and increased ICP [104].

### **INTRA-ABDOMINAL HYPERTENSION IN RENAL CONDITIONS**

IAH is now recognized as a risk factor for the development of acute kidney injury and failure in the renal literature [10, 105, 106]. Although the effects of increased IAH are multiple, the kidney is especially vulnerable to increased IAH because of its anatomic position. While the means by which kidney function is impaired in patients with ACS is incompletely elucidated, available evidence suggests that the most important factor involves alterations in renal blood flow. IAH should be considered as a potential cause of acute kidney injury in critically ill patients; its role in other conditions, such as hepatorenal syndrome, remains to be elucidated. Because several treatment options (both medical and surgical) are available, IAH and ACS should no longer be considered irrelevant epiphenomena of severe illness or critical care [107].

### **INTRA-ABDOMINAL HYPERTENSION IN CARDIAC CONDITIONS**

Increased IAH has been related to coronary artery bypass surgery and prolonged extracorporeal circuit times [108, 109]. In chronic heart failure and cardiorenal dilemma or cardiorenal syndrome, IAH is associated with deteriorating renal function [110, 111]. Devices to induce hypothermia after cardiac arrest use a closed loop system with the instillation of cold fluids into the peritoneal cavity. For safety reasons, these devices limit the instillation of fluids based on IAH, with target pressures below 15 mm Hg.

The abdominal compartment could potentially form a missing link in the pathophysiology of acute decompensated heart failure (ADHF) and cardio-renal syndrome. It has recently been shown that raised IAH is prevalent in advanced heart failure causing reduced ejection fraction and the impairment of renal function [110]. However, IAH and ACS are less frequent and overt ascites is rare [110]. Importantly, medical treatment resulting in a decrease of IAH improves renal function and in cases of persistently high IAH, ultrafiltration might be beneficial [110, 111]. Notably, while organ dysfunction in the intensive care literature has only been described when IAH exceeds 12 mm Hg, patients with ADHF develop worsening renal function at much lower IAH levels [110]. This might suggest that the underlying reserve of the kidneys is impaired with increased IAH in this setting. It is also vital to emphasize that although the degree of renal dysfunction is probably correlated with the degree of IAH elevation, there can be a wide range of IAH in relation to serum creatinine levels at presentation [110]. Therefore, the term Cardio-Abdomino-Renal Syndrome or CARS, helps to emphasize the potentially important role of the abdominal compartment and splanchnic vasculature in the pathophysiology of ADHF and cardio-renal syndrome [112].

## SUMMARY KEY POINTS

### IAH IN BURN PATIENTS

- IAH will develop in most if not all severely burned patients, and may become rapidly fatal.
- The incidence of IAH is directly related to the burned surface (TBSA > 60%) and the amount of fluids given (> 275 mL kg<sup>-1</sup> first 24 hours).
- Always suspect IAH and routinely measure the IAP in the resuscitation period.
- Avoid over-resuscitation with large volumes and overuse of crystalloids.
- Consider the use of hypertonic solutions to minimize fluid accumulation.
- Percutaneous drainage of ascites is a simple and effective tool to reduce IAP if organ dysfunction develops. Consider diuretics and CVVH with net ultrafiltration.
- Escharotomy can dramatically reduce IAP in case of circular abdominal (but also thoracic) burns and will improve abdominal wall compliance.
- Although decompressive laparotomy is a definitive therapy, wound maintenance and infection control may then become difficult.
- Knowing the risk of ACS is important.

### IAH AND OBESITY

- Although acute elevations in IAP have similar effects in obese patients compared to non-obese patients, the threshold before organ dysfunction develops may be higher.
- Chronic elevations in IAP may, in part, be responsible for the pathogenesis of obesity-related co-morbid conditions such as hypertension, pseudotumor cerebri, pulmonary dysfunction, GERD, and abdominal wall hernias.
- Further large-scale studies are necessary to determine the relationship between chronic IAP and obesity-related co-morbidities.

### IAH AND PREGNANCY

- The IAP is chronically increased during pregnancy.
- Because the body has time to adapt end-organ dysfunction is rare.
- The IAP should not be measured in the supine position but in the left lateral decubitus at 10°.
- Laparoscopy can have deleterious effects on the fetus and should be performed with caution and only when absolutely indicated in the third trimester.
- IAH is associated with preeclampsia and eclampsia.
- Paracentesis is treatment of choice for ovarian hyperstimulation syndrome (OHSS).

### IAH IN GENERAL MEDICINE

- IAP has been increasingly recognized to play a major role in different pathologies and is emerging in every field of medicine in the last decades.
- Fill volume during peritoneal dialysis should be limited to an IAP of 14 mm Hg.
- Cardiorenal syndrome should be termed CARS, cardio-abdomino-renal syndrome instead.
- An IAP > 9 mm Hg may play a role in the deterioration of renal function in patients with congestive heart failure.
- Think of the organ-organ interactions and PCS, poly-compartment syndrome.

### CONCLUSIONS

The occurrence of IAH and ACS has been associated in many conditions beyond the classical ICU, surgical or trauma patient. The true incidence of IAH in burns, obesity, pregnancy and general medicine is high and probably underestimated. In adults but also in children, patients at risk for IAH should be identified early during the treatment and the focus should be on early recognition of IAH and prevention of ACS. Thus, IAP should be measured regularly. Appropriate actions should be taken when IAP increases above 15 mm Hg, especially in patients difficult to ventilate or those with new onset organ dysfunction. Although medical management comes first, one should not hesitate to resort to surgical decompression if this fails.

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