Abdominal compartment syndrome

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Purpose of review

This review will set forth the new consensus definitions for intra-abdominal pressure, intra-abdominal hypertension, and the abdominal compartment syndrome from the World Congress on the Abdominal Compartment Syndrome in December 2004. The review will explore the challenges in diagnosis, pathophysiology, and recent concepts in the treatment of abdominal compartment syndrome.

Recent findings

Intra-abdominal pressure greater than 12 mm Hg may exert adverse physiologic sequelae, progressing to intra-abdominal hypertension and full-blown abdominal compartment syndrome as intra-abdominal pressure increases. The first challenge is to recognize that abdominal compartment syndrome may be a potential problem in critically ill patients. Intra-abdominal pressure monitoring is essential for this. Continuous monitoring of intra-abdominal pressure and abdominal perfusion pressure adds real-time measurements and can be performed by way of the stomach or bladder. Intra-abdominal hypertension occurs in approximately 35% of patients in the intensive care unit, and abdominal compartment syndrome in approximately 5%.

Summary

Massive resuscitation is increasingly recognized as a major contributor to abdominal compartment syndrome. Prophylactic decompression and temporary abdominal closure have important roles in preventing tertiary or recurrent abdominal compartment syndrome. Failure to recognize and treat intra-abdominal hypertension will result in increased risk of renal impairment, visceral and intestinal ischemia, respiratory failure and death.

Keywords

abdominal compartment syndrome, abdominal decompression, intra-abdominal pressure

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Dr Sugrue owns a patent on the intravesical continuous three-way catheter and has sold this to Wolfe Tory Medical.

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Abbreviations

ACS	abdominal compartment syndrome
APP	abdominal perfusion pressure
IAH	intra-abdominal hypertension
IAP	intra-abdominal pressure
TAC	temporary abdominal closure

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Introduction

The past two years have seen an exponential increase in knowledge relating to abdominal compartment syndrome (ACS). This chapter will outline some recent developments. Of note was the inaugural World Conference on the Abdominal Compartment Syndrome held in Australia in December 2004.

Definitions

In December 2004, World Congress on the Abdominal Compartment Syndrome was held, with 170 leaders from around the world setting the stage for future understanding of this complex evolving physiologic phenomenon. Here are the consensus definitions from the meeting [1].

Intraabdominal pressure

Intraabdominal pressure (IAP) is the pressure concealed within the abdominal cavity. IAP varies with respiration. Normal IAP is approximately 5 mm Hg, but it can be nonpathologically increased in the obese. IAP should be expressed in mm Hg (1 mm Hg = 1.36 cm H_2O) and measured at end-expiration with the patient in the supine position, and abdominal muscle contractions should be absent. The transducer should be zeroed at the level of the midaxillary line The gold standard for direct IAP measurement is direct needle puncture and transduction of the pressure within the abdominal cavity (e.g., during peritoneal dialysis or laparoscopy). The gold standard for intermittent indirect IAP measurement is transduction of the pressure within the bladder. The gold standard for continuous indirect IAP measurement is a balloon-tipped catheter in the stomach or a continuous bladder irrigation method. Abdominal perfusion pressure (APP) = mean arterial pressure − IAP.

Intra-abdominal hypertension

Intra-abdominal hypertension (IAH) is defined by either one or both of the following: (1) an IAP of 12 mm Hg or greater, recorded by a minimum of three standardized measurements conducted 4 to 6 hours apart; (2) an APP of 60 mm Hg or less, recorded by a minimum of two standardized measurements conducted 1 to 6 hours apart. IAH is graded as shown in Table 1.

Abdominal compartment syndrome

Abdominal compartment syndrome is defined as the presence of an IAP of 20 mm Hg or greater with or without APP below 50 mm Hg, recorded by a minimum of three standardized measurements conducted 1 to 6 hours apart and single or multiple organ system failure that was not

Table 1. Grading of intra-abdominal hypertension

Grade	Intraabdominal pressure (mm Hg)
I	12–15
II	16–20
III	21–25
IV	>25

previously present. In contrast to IAH, ACS should not be graded because it is an all-or-nothing phenomenon.

Primary abdominal compartment syndrome
Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or angioradiologic intervention, or a condition that develops after abdominal surgery (such as abdominal organ injuries that require surgical repair or damage control surgery, secondary peritonitis, bleeding pelvic fractures, or other cause of massive retroperitoneal hematoma, liver transplantation). Patients who undergo an initial trial of nonoperative management for solid organ injuries who subsequently experience ACS are included in the primary ACS category. Former synonyms include 'abdominal,' 'surgical,' and 'acute.'

Secondary abdominal compartment syndrome
Secondary ACS includes conditions that do not originate from the abdomen (such as sepsis and capillary leak, major burns, and other conditions requiring massive fluid resuscitation) yet result in the signs and symptoms commonly associated with primary ACS. Former synonyms include 'extra-abdominal,' 'medical,' and 'subacute.'

Tertiary or recurrent abdominal compartment syndrome
Tertiary or recurrent ACS is a condition in which ACS develops after prophylactic or therapeutic surgical or medical treatment of primary or secondary ACS (e.g., persistence of ACS after decompressive laparotomy or development of a new ACS episode after definitive closure of the abdominal wall after previous use of a temporary abdominal wall closure). Former synonyms include 'chronic' and 'open.'

To differentiate between localized and systemic IAH/ACS, the bladder-to-gastric pressure difference should be measured. A localized problem is present when this difference exceeds 10 mm Hg.

Prevalence of intra-abdominal hypertension and abdominal compartment syndrome

The prevalence of IAH is variable, depending on the threshold used to define it and the population studied. A recent multicenter group performed a prospective study of IAH in a mixed intensive care unit (ICU) population [2]. In this study, 265 consecutive patients (mean Acute Physiology and Chronic Health Evaluation II score 17.4) admitted for more than 24 hours in one of the 14 participating ICUs were monitored until death, until hospital discharge, or for a maximum of 28 days. Medical patients accounted for 46.8% of all study patients, whereas elective surgery, emergency surgery, and trauma patients accounted for 27.9%, 16.6%, and 8.7%, respectively. IAH was present when the mean value of the two daily IAP measurements was greater than 12 mm Hg. ACS was diagnosed when an IAP greater than 20 mm Hg was associated with at least one organ failure.

On admission, 32.1% of the population had IAH, and 4.2% had ACS. Importantly, unlike the occurrence of IAH at day 1, the occurrence of IAH during ICU stay was an independent predictor of mortality. Independent predictors of IAH at day 1 were liver dysfunction, abdominal surgery, fluid resuscitation with more than 3500 ml during the 24 hours before inclusion, and ileus. Previously we identified that grade 2 IAH (16–20 mm Hg) occurs in more than 30% of patients undergoing emergency surgery [3]. Despite increasing reporting of ACS and IAH in the literature, it is often ignored [4,5].

New trends in monitoring intra-abdominal pressure measurement

There have been significant developments in IAP monitoring. Balogh *et al.* [6] prospectively validated the technique of continuous IAP monitoring and showed that this new method has almost a perfect agreement with the reference standard of Kron *et al.* [7] of intermittent intravesical IAP measurements. There are many obvious advantages of the described continuous IAP monitoring. First, it does not require a major change in the present practice apart from the use of three-way urinary catheters. This method abandons the cumbersome steps of draining, clamping of the catheter, and filling with 50 ml of normal saline. The monitoring is continuous and does not interfere with the urinary flow through the drainage port of the catheter. The continuous IAP monitoring is less labor intensive and time consuming compared with the standard intermittent measuring technique.

Continuous IAP measurement has several potential advantages to exploit in the future. Increasingly, Signal Interpretation and Monitoring will become a more powerful tool for physiologic monitoring [8•]. Continuous measurement of the IAP makes possible to monitor the APP both intermittently and continuously [9–11].

Pathophysiology

Intra-abdominal pressure is primarily determined by the volume of the viscera and the intra-compartment fluid load. The abdominal cavity pressure-volume curve has been studied in animals. Postmortem evaluation of human pressure-volume curves may not be reliable because of the post-mortem loss of abdominal wall compliance. In general, the abdominal cavity has a great tolerance to fluctuating

volumes, with little rise in IAP [12]. The compliance of the abdominal cavity can be seen at laparoscopy, wherein it is possible to instill as much as 5 liters of gas into the peritoneal cavity without exerting any significant influence on IAP. In a previous evaluation of IAP during laparoscopy we have found that the mean volume of gas required to generate a pressure of 20 mm Hg was 8.8 ± 4.3 l [13]. Adaptation can occur over time, and this is seen clinically in patients with ascites, large ovarian tumors, and, of course, pregnancy. Chronic ACS occurs in some morbidly obese patients, with significantly increased IAP, predisposing to chronic venous stasis, urinary incontinence, incisional hernia, and intracranial hypertension [14,15].

The causes of acutely increased IAP are usually multifactorial. Common causes are as follows:

(1) Trauma and intra-abdominal hemorrhage;
(2) Abdominal surgery;
(3) Retroperitoneal hemorrhage;
(4) Peritonitis, usually secondary or tertiary (pancreatitis, recurrent abscess);
(5) Laparoscopy and pneumoperitoneum;
(6) Repair of large incisional hernia;
(7) Abdominal banding with postoperative Velcro belt to prevent incisional hernia;
(8) Massive fluid resuscitation defined as more than 5 liters of fluid in a 24-hour period;
(9) Ileus, whether paralytic, mechanical, or pseudo-obstructive.

Whereas trauma patients constitute one of the commonest subsets of patients to experience intra-abdominal hypertension and the ACS, it was postoperative aortic surgery patients that Fietsam *et al.* [16] referred to in coining the term ACS [16].

Effect of raised intra-abdominal pressure on individual organ function

Whereas intra-abdominal hypertension has a global affect on the body, with increasing IAH, leading to ACS, it tends to affect one system first, usually the renal or gastrointestinal system. This section will discuss the selective affects of IAH.

Renal

Renal dysfunction in association with increased IAP has been recognized for more than 100 years, but only recently have its effects on large series of patients been reported.

In 1945, Bradley and Bradley [17], in a study of 17 volunteers, demonstrated that there was a reduction in renal plasma flow and glomerular filtration rate in association with increased IAP. In 1982, Harman *et al.* [18] showed that as IAP increased from 0 to 20 mm Hg in dogs the glomerular filtration rate decreased by 25%. At 40 mm Hg, the dogs were resuscitated and their cardiac output returned to normal; however their glomerular filtration rate and renal blood flow did not improve, indicating a local effect on renal blood flow. The situation in seriously ill patients may, however, be different, and the exact cause of renal dysfunction in the ICU is not clear because of the complexity of critical illness. We found that out of 20 patients with increased IAP and renal impairment, 13 already had impairment before the IAP increased [19].

The most likely direct effect of increased IAP is an increase in the renal vascular resistance, coupled with a moderate reduction in cardiac output. Pressure on the ureter has been ruled out as a cause, given that investigators have placed ureteric stents with no improvement in function [20]. Other factors that may contribute to renal dysfunction include humeral factors and intraparenchymal renal pressures. The concept of renal decapsulation, on the basis of raised intrarenal pressure, was popular some decades ago but now is rarely practiced.

The absolute value of IAP required to cause renal impairment has not been established. Some authors have suggested that 10 to 15 mm Hg is a critical cutoff point [21,22]. Maintaining adequate cardiovascular filling pressures in the presence of raised IAP also seems to be important [23].

Cardiovascular

Increased IAP reduces cardiac output as well as, increasing central venous pressure, systemic vascular resistance, pulmonary artery pressure, and pulmonary artery wedge pressure [19,23]. It should be remembered, however, that because of the associated rise in intrapleural pressure, some of the rises seen in central venous pressure may not reflect the intravascular volume and may be misleading when the patient's volume status is assessed. Cardiac output is affected mainly by a reduction in stroke volume, secondary to a reduction in preload and an increase in afterload. This is further aggravated by hypovolemia. Paradoxically, in the presence of hypovolemia, an increase in IAP can be temporarily associated with an increase in cardiac output. The normal left atrial/right atrial pressure gradient may be reversed during raised IAP [24]. It has been identified that venous stasis occurs in the legs of patients with abdominal pressures above 12 mm Hg [25]. In addition, studies in patients undergoing laparoscopic cholecystectomy show up to a fourfold increase in renin and aldosterone levels [26]. One of the most comprehensive reviews on the cardiovascular effects of AIH has just been written by Cheatham [27•].

Respiratory

Both animal and human experiments have shown that IAP exerts a significant effect on pulmonary function. In association with increased IAP, there is diaphragmatic stenting, exerting a restrictive effect on the lungs with

reduction in ventilation; decreased lung compliance; increase in airway pressures; and reduction in tidal volumes. These changes can occasionally be seen during laparoscopy, wherein lung compliance has been shown to be reduced once the IAP exceeds 16 mm Hg. Respiratory changes related to increased IAP are aggravated by increased obesity and other physiologic conditions such as severe hemorrhage. There is also some adverse effect on the efficiency of gas exchange. Often patients with raised IAP are acidotic, and whereas this may initially be metabolic in origin, the effect of raised IAP adds a respiratory component.

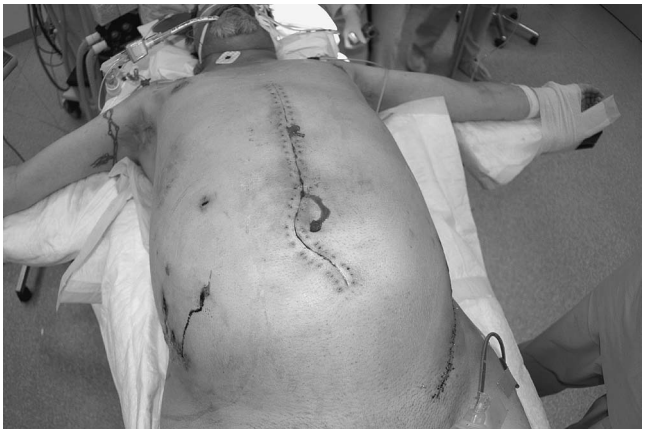
In critically ill patients receiving ventilation, the effect on the respiratory system can be significant, resulting in reduced lung volumes, impaired gas exchange, and high ventilatory pressures. Hypercarbia can occur, and the resulting acidosis can be exacerbated by simultaneous cardiovascular depression as a result of raised IAP. The effects of raised IAP on the respiratory system in the ICU can sometimes be life threatening, requiring urgent abdominal decompression. In patients with true ACS undergoing abdominal decompression, there is a remarkable change in intra-operative vital signs. I should like to point out, however, that these patients are a minority rather than a majority of patients with increased IAP and ACS. One could argue that a patient should never be allowed to get to this stage. Monitoring of vital signs and acid–base status is vital in this patient. A typical example of a tight-looking patient with an ACS is shown in Figure 1. You can see the abdomen is about to pop!

Visceral perfusion

Interest in visceral perfusion has increased with the popularization of gastric tonometry, and there is an association between IAP and visceral perfusion as measured by gastric pH [13]. This was confirmed in 18 patients undergoing laparoscopy, in whom a reduction of 11 to 54% in blood flow was seen in the duodenum and stomach, respectively, at an IAP of 15 mm Hg [28]. Animal studies suggest that reduction in visceral perfusion is selective, affecting intestinal blood flow before, for example, adrenal blood flow [29]. We have demonstrated in a study of 73 post-laparotomy patients that IAP and pHi are strongly associated, suggesting that early decreases in visceral perfusion are related to levels of IAP as low as 15 mm Hg [19]. Increasing IAPs may result in visceral hypoperfusion and secondary bacterial translocation as well as affecting wound healing. Both abnormal pHi and IAP predicted the same adverse outcome with increased risk of hypotension, intra-abdominal sepsis, renal impairment, a need for repeat laparotomy, and death. It is important to measure IAP to increase awareness of its potential adverse effects on the gut. The indications for IAP monitoring are as follows:

(1) Postoperative patients (abdominal surgery);
(2) Patients with open or blunt abdominal trauma;
(3) Mechanical ventilated ICU patients with other organ dysfunction as assessed by daily Sequential Organ Failure Assessment score;
(4) Patients with a distended abdomen and signs and symptoms consistent with abdominal compartment syndrome: oliguria, hypoxia, hypotension, unexplained acidosis, mesenteric ischemia, elevated intracranial pressure.

Figure 1. Patient with grossly distended abdomen and abdominal compartment syndrome



Patient following trauma with secondary intraperitoneal sepsis, grossly distended abdomen and impending wound dehiscence for a re-laparotomy.

General support

The precise management of IAP remains somewhat clouded by many published anecdotal reports and uncontrolled series. Aggressive nonoperative intensive care support is critical to prevent the complications of ACS. This involves careful monitoring of the cardiorespiratory system and aggressive intravascular fluid replacement, especially if this is associated with hemorrhage [30]. Excessive fluid resuscitation, however, will actually add to the problem [31]. Simple measures such as nasogastric decompression are, of course, mandatory. Some possible nonsurgical options are these:

(1) Paracentesis;
(2) Gastric suctioning;
(3) Rectal enemas and suctioning;
(4) Gastroprokinetics (cisapride, metoclopramide, domperidone, erythromycin);
(5) Colonoprokinetics (prostigmine);
(6) Furosemide either alone or in combination with human albumin 20%;
(7) Continuous venovenous hemofiltration with aggressive ultrafiltration;
(8) Continuous negative abdominal pressure;
(9) Sedation;

Figure 2. The open abdomen with a fistula

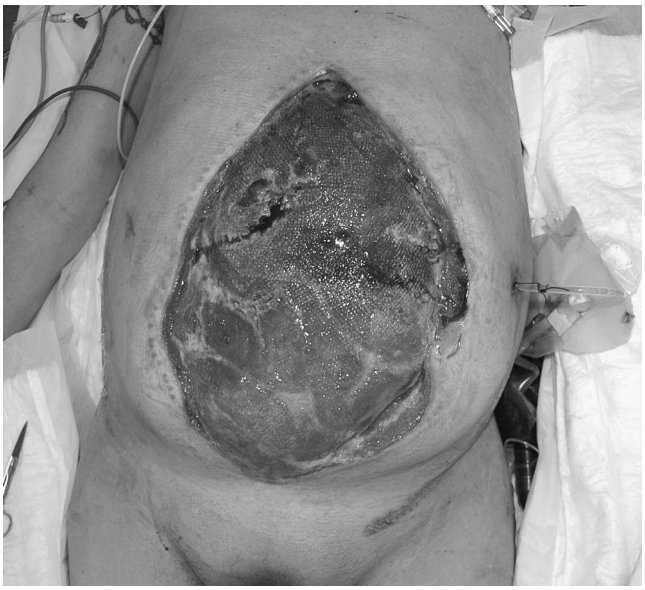
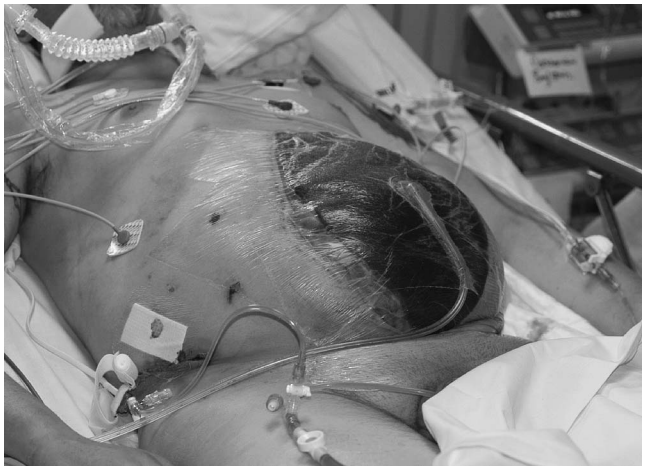


Figure 3. Patient with a vacuum-assisted closure dressing in place, controlling abdominal secretions on low suction



The healthy granulation tissue seen after vacuum-assisted closure dressing on the patient previously shown (Fig. 1) following management of intraabdominal sepsis.

(10) Curarization;
(11) Body positioning;
(12) Botulinum toxin into internal anal sphincter.

Surgical management

As yet, there are few guidelines for exactly when surgical decompression is required in the presence of raised IAP. Some studies have stated that abdominal decompression is the only treatment and that it should be performed early to prevent ACS [32]. This is an overstatement and is not supported by level 1 evidence [33].

The indications for abdominal decompression are related to correcting pathophysiologic abnormalities as much as achieving a precise and optimum IAP. For example, if gas exchange is being increasingly compromised with collapse of the lung bases, or ventilatory pressures are increasing, abdominal decompression should be strongly considered. Similarly, if cardiovascular or renal function is being compromised and raised IAP is suspected, then decompression should be considered early. Unfortunately, visceral hypoperfusion is very difficult to predict, apart from gastric tonometry, and guidelines for surgical intervention would have to rely on levels of IAP that have been shown to correlate with visceral ischemia.

The approaches to abdominal decompression also vary. Temporary abdominal closure (TAC) has been popularized as a mechanism to reverse many of the sequelae of increased IAP. The theoretical benefits of abdominal decompression and TAC are therefore attractive, and some authors have advocated the prophylactic use of TAC to decrease postoperative complications and facilitate planned re-exploration. However, it may be hard to justify this approach until a subgroup of high-risk patients can be more accurately identified. Burch *et al.* [32] have stated that abdominal decompression can reverse the sequelae of the ACS. IAP levels have been advocated as a guide to closure of the abdominal wall, especially in children. However, the existing literature currently has few prospective studies. Wittman *et al.* [34,35], in two separate studies in 1990 and 1994, prospectively evaluated outcomes in 117 and 95 patients, respectively. A multi-institutional study of 95 patients concluded that a staged approach to abdominal repair, with TAC, was superior to conventional techniques for dealing with intra-abdominal sepsis. Torrie *et al.* [36] retrospectively reported their experience with

Table 2. Approach to surgical dressing and management

Technique	Control of abdominal contents	Active removal of exudate	Quantify 3rd space losses	Promotion of granulation	Achieves skin closure	Achieves fascial closure	Cost
Bogota bag	+	–	–	–	–	–	+
Wittman patch	+	–	–	–	–	+	+++
Prosthetic mesh	+	–	–	–	–	(+)	++
Vacuum pack	+	+	–	–	±	–	+
TNP therapy	+	+	+	+	+	+	++++

TNP, topical negative pressure.

64 patients (median Acute Physiology and Chronic Health Evaluation II score 21) undergoing TAC and found the mortality to be 49%.

The main indications for performing TAC include these: abdominal decompression both prophylactic and therapeutic; facilitate re-exploration in abdominal sepsis; and inability to close the abdomen. One must remember, however, that the open abdomen is not without its morbidity and complications as can be seen with the patient with a fistula in Figure 2.

There are a variety of dressing and closure options. The vacuum-assisted closure dressing is one, but it should be used at a relatively low pressure (<50 mm Hg) to avoid fistula formation (Fig. 3). It has the disadvantage, however, of being expensive (Table 2).

Conclusion

Increasingly, IAH and ACS will be diagnosed and not just thought of as curiosities [37]. The challenge lies not in identifying predictors of ACS but in optimizing treatment, including identifying patients who need decompression and when this should be done. The newly formed Society of the Abdominal Compartment Syndrome will act as a portal for discussion, clinical trials, and research.

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