Abdominal pressure in the critically ill
Manu L.N.G. Malbrain, MD

Gut dysfunction resulting in increased gut permeability and subsequent bacterial translocation may play an important role in the development of multiple organ system failure in the critically ill within this concept. Intra-abdominal pressure is an important parameter and prognostic indicator of the patient’s underlying physiologic status. Initially thought to affect primarily trauma and surgical patients, intra-abdominal hypertension and the abdominal compartment syndrome have recently also been identified in about 20% of critically ill medical patients. Even slightly increased intra-abdominal pressure, as low as 10 mm Hg, has deleterious effects on end-organ function, impairing neurologic, cardiac, respiratory, gastrointestinal, hepatic, and renal homeostasis. Rapid restoration of volume status and abdominal decompression is essential to preserve organ function, although massive fluid overload may trigger a vicious cycle which by itself may cause intra-abdominal hypertension. The traditional filling pressures are unreliable indices of preload, necessitating the use of new markers, such as right ventricular end diastolic volume index or intrathoracic blood volume index to assess volume status and resuscitate these patients correctly. New techniques, such as intravesical or intragastric pressure monitoring combined with intramucosal pH, together with an high clinical index of suspicion, help the intensivist make a correct diagnosis, adjust treatment, and decide on early decompressive laparotomy with temporary abdominal closure.

Many intensive care physicians believe that the abdomen, especially the gut and bacterial translocation, is the site of origin of multiple organ dysfunction syndrome. Intra-abdominal pressure (IAP) is an important parameter of underlying abdominal problems, and may reflect the patient’s physiologic status [1]. Intra-abdominal pressure is easy to measure at the bedside with the standardized intravesical recording method. In the past it has mainly been used by surgeons as an indicator for intra-abdominal hypertension (IAH), the abdominal compartment syndrome (ACS), and as a guide to perform a second look laparotomy. The measurement of IAP is not a new concept, but only recently have its importance and therapeutic implications for the intensive care unit (ICU) physician become apparent. It was probably Wendt, as early as 1876 [2], who first described the association between IAH and renal impairment. However, until the early 1920s there was a poor understanding of the concept of IAP [3]. Numerous human and animals studies were not published until the 1940s, and, more recently, the 1970s and 1980s. These studies have shown various deleterious effects of raised IAP on the cardiovascular and respiratory system and on every organ concealed within the abdominal cavity [1,3–7]. Bradley and Bradley [4] suggested that the abdomen and its contents should be considered relatively noncompressive and primarily fluid in character, behaving in accordance to Pascal’s law. Although initially thought to affect mostly trauma and surgical patients, IAH and ACS have since the late 1980s and 1990s also been identified in medical patients. Only since then have ICU physicians become aware of the deleterious effects on organ function of very slight rises in IAP (as low as 10 mm Hg) [1,3,7–11]. In the future IAP will, therefore, be increasingly used as part of routine monitoring in the ICU, and maybe as an independent prognostic factor. Before adding IAP to our armamentarium of monitoring variables we must better understand the measurement technique, the incidence of IAH in different ICU patient populations, the pathophysiologic implications on cardiovascular, neurologic, respiratory, renal, and visceral functions, and the possible therapeutic options. This review focuses on data obtained from recent literature, and summarizes the results of our own clinical investigations during the past few years, with a focus on the medical aspects. It is beyond the scope of this review to discuss the different surgical treatment options for ACS.

Measurement technique
Because the abdomen acts as a fluid compartment, IAP can be measured in nearly every part of it: intraperitoneal, via bladder, uterus, inferior vena cava, rectum, or

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**Abbreviations**

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<tr>
<th>Abbreviation</th>
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<tr>
<td>ACS</td>
<td>abdominal compartment syndrome</td>
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<td>ALI</td>
<td>acute lung injury</td>
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<td>ARDS</td>
<td>acute respiratory distress syndrome</td>
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<td>BMI</td>
<td>body mass index</td>
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<td>Cdyn</td>
<td>dynamic compliance</td>
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<td>CO</td>
<td>cardiac output</td>
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<td>CVP</td>
<td>central venous pressure</td>
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<td>FRC</td>
<td>functional residual capacity</td>
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<td>IAH</td>
<td>intra-abdominal hypertension</td>
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<td>IAP</td>
<td>intra-abdominal pressure</td>
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<td>ICP</td>
<td>intracranial pressure</td>
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<td>ICU</td>
<td>intensive care unit</td>
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<td>LIP</td>
<td>lower inflection point</td>
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<td>PCWP</td>
<td>pulmonary capillary wedge pressure</td>
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<td>PEEP</td>
<td>positive end expiratory pressure</td>
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<td>Pflex</td>
<td>lower inflection point</td>
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<td>pH</td>
<td>intramucosal pH</td>
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<td>RVEDVI</td>
<td>right ventricular end diastolic volume index</td>
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<td>SVRI</td>
<td>systemic vascular resistance index</td>
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<td>V-P</td>
<td>volume-pressure</td>
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<td>ZEEP</td>
<td>zero end expiratory pressure</td>
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stomach [3,9,10]. Over the years various direct and indirect measurement methods have been suggested. Direct measurement by cannulation of the peritoneal cavity with a metal cannula or a wide-bore needle attached to a saline manometer or pressure transducer has been used historically and experimentally, but has no advantages over more accessible and simple techniques.

Indirect measurements include inferior vena cava pressure measured by a central venous line if the tip is in the inferior vena cava or by femoral vein cannulation, but correct validation of this technique has not been performed on humans; moreover, a recent study [12] comparing superior vena cava pressure with common iliac venous pressure in various conditions of IAP and positive end expiratory pressure (PEEP) showed that the difference between common iliac venous pressure and superior vena cava pressure was not affected by IAP. The most likely explanation is the differing anatomy and experimental model used to induce increased IAP in canine studies. In humans both common iliac venous pressure and superior vena cava pressure increase as IAP increases [12]. Rectal pressures have also been studied and correlate well with direct measurement [13].

Measurement of IAP by an indwelling catheter in the urinary bladder has been suggested as the method of choice or gold standard [14,15]. In a recent study, Yol et al. [16] compared bladder pressure with direct insufflation pressure during laparoscopic cholecystectomy in 40 patients, and found a very good correlation between the 2 measurements (R = 0.973, P <0.0001). The bladder acts as a passive conduit, and intrinsic bladder pressure does not rise when its volume is between 50 and 100 mL. Since most patients in the ICU have a central venous or arterial line connected to a pressure transducer and a Foley bladder catheter in place, it takes less than 5 minutes to calculate the IAP. As originally described, the Kron technique disrupts for each IAP measurement what is normally a closed sterile system, placing the patient at increased risk of urinary tract infection or sepsis and subjecting healthcare providers to the risk of needle stick injuries and exposure to blood and body fluids [14]. Iberti et al. [15] reported the use of a closed system drain and transurethral bladder pressure monitoring technique, using a 20 gauge needle for each IAP measurement, thus subjecting healthcare workers to needle stick injuries. Cheatham and Safcsak [17] reported a revision of Kron’s original technique that is safer, less invasive, more efficient (repeated measurements possible) and cost effective (Fig. 1). The authors claim that, using this revised technique, the cost of performing intravesicular pressure measurements is reduced to <$10 per day and required nursing time is reduced to <1 minute per measurement; most important, the risk of urinary tract infection, sepsis, and needlestick injury is minimized. Furthermore, this tech-

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**Figure 1. Revision of the original Kron method for intravesicular pressure measurement**

A standard intravenous infusion set is connected to 1000 mL of normal saline, two stopcocks, a 60 mL Luer-Lok syringe, and a disposable pressure transducer. An 18-gauge plastic intravenous infusion catheter is inserted into the culture aspiration port of the Foley catheter and the needle is removed. The infusion catheter is attached to the first stopcock with arterial pressure tubing after being flushed with saline and “zeroed” at the level of the symphysis pubis (or the midaxillary line when the patient is in complete supine position). The Foley catheter is clamped immediately distal to the culture aspiration port. The stopcocks are turned “off” to the patient and pressure transducer, and 50 mL of saline is aspirated from the intravenous bag. The first stopcock is turned “on” to the patient and 50 mL of saline are instilled into the bladder. The stopcocks are turned “off” to the syringe and the intravenous tubing. After equilibration, the patient’s IAP is then measured at end-expiration on the bedside monitor. To verify correct measurement, gentle compression of the abdomen should give instant variations on the IAP reading in form of oscillations. If a damped signal is noted, momentary release of the clamp on the Foley catheter will ensure that all air is flushed, and IAP can be measured again. After a correct reading is obtained, the clamp is removed, the bladder allowed to drain, and the volume of saline used is subtracted from the patient’s urine output for that hour.

From [17], with permission.
nique allows repeated IAP measurement with the same equipment.

The IAP can also be measured by means of a nasogastric or gastrostomy tube. This method can be used when the patient has no Foley catheter in place, when there is trauma to the bladder, or when intravesical pressures are not reliable, as in cases with low intrinsic bladder compliance or a pelvic hematoma compressing the bladder. In these situations bladder pressure may lead to an overestimation of IAP. The procedure described above (in the legend to Fig. 1) can be applied by means of a nasogastric or gastrostomy tube, as studied by Collee [18]. Sugrue et al. [19] assessed the accuracy of an intragastric method of measuring IAP by means of the balloon of a gastric tonometer, comparing their results with simultaneous intravesical pressure recordings in 9 patients that underwent laparoscopic cholecystectomy. They found a very good correlation between both IAP measurement methods. Intra-abdominal pressure measured by means of a tonometer balloon allows a continuous trend to be obtained without interfering with urinary output estimation. Unfortunately, simultaneous intramucosal pH (pHi) and IAP measurements are not (yet) possible. A possible disadvantage of intragastric recording is the effect on interpretation of IAP values by the Migrating Motor Complex; however, these can easily be identified. Recording the “diastolic” value of IAP at end-expiration can solve this problem.

Normal and pathologic values
The normal values of IAP are subatmospheric to 0 mm Hg; values above 12 mm Hg are considered IAH. Postabdominal surgery IAP usually ranges from 3 to 15 mm Hg. When IAP is greater than 10 mm Hg, cardiac output (CO) drops; IAPs above 15 mm Hg compromise renal and splanchnic perfusion, IAPs above 20 to 25 mm Hg increase peak alveolar pressures, and, finally, one talks about ACS when IAP is increased above 20–30 mm Hg, an emergency situation. Abdominal compartment syndrome is a pathologic state caused by an acute increase in IAP (>20 mm Hg) that adversely affects cardiac, pulmonary, and renal function, that also affects all other intra-abdominal organs, and that can cause serious wound complications in surgical patients [2,8,9]. Recent studies showed deleterious effects on organ function of only slight rises in IAP, as low as 10 mm Hg [20••].

Etiologic factors and predisposing conditions
The etiologic factors can be divided into four categories [10,20••]:

1. Surgical or postoperative: postoperative hemorrhage (eg, aorta suture leakage), any abdominal surgery (eg, reduction of a massive parietal or diaphragmatic hernia), abdominal closure under excessive tension, postoperative peritonitis or intra-abdominal abscess, postoperative edema (eg, extensive dissections in cancer patients), laparoscopic surgery with inflation of intra-abdominal air, postoperative ileus or acute gastric dilatation

2. Post-traumatic: external compression from military antishock trousers, burns and multiple trauma, intraperitoneal or retroperitoneal bleeding, or visceral edema post fluid resuscitation

3. Medical: peritoneal dialysis, peritonitis or intra-abdominal abscess, edema or ascites secondary to massive fluid resuscitation (eg, septic shock), ascitic cirrhosis with decompensation, ruptured abdominal aortic aneurysm, tension pneumoperitoneum, acute pancreatitis, intra-abdominal hemorrhage, or ileus of any origin (eg, intestinal obstruction). Volume overloading may contribute to abdominal distention and IAH in different ways: (a) dilating and engaging abdominal vessels, (b) generating ascites, (c) intestinal swelling with elongation of mesenteric veins and lymphatic system causing outflow obstruction and tissue hypoxia and more intestinal swelling, creating a vicious cycle (blood goes in but doesn’t come out), (d) causing increased urine production and bladder distention.

4. Predisposing conditions: hypothermia (<33°C), acidosis (pH<7.2), rate of packed cell transfusion (>10–20 U/24 h), dilutional coagulopathy or disseminated intravascular coagulation, sepsis from any origin, overdose (epidural) anesthesia, or decompensation of cirrhosis caused by infection or hemorrhage. The main causes of IAH in a medical ICU population were decompensated cirrhosis with massive ascites and septic shock with aggressive fluid resuscitation [21]. These acute changes in IAP can be contrasted with more chronically increased IAP as seen in morbidly obese patients, pregnant patients, and patients with cirrhosis [1,3,20••].

Incidence of raised intra-abdominal pressure
We found an incidence of IAH (defined as IAP ≥12 mm Hg) of 24% in a sample of 53 medical ICU patients [21]. The mean IAP was 8.4±4.6 mm Hg (all patients), 6.3±2.8 mm Hg (IAP <12) and 14.8±2.6 mm Hg (IAP ≥12). A prospective pilot study in a sample of 405 mixed ICU patients showed an overall incidence of IAH of 17.5%. The mean IAP in 71 patients with IAH was 15.8±5.6 mm Hg versus 6.5±2.8 in those without. The incidence in emergency surgery patients was 39.4% versus 19.8% in medical patients and 6.1% (6.9±3.5) in scheduled surgical patients [22]. Other studies found that the incidence of raised IAP in the seriously ill is high, with approximately 30% of postoperative general surgery patients having IAPs greater than 20 mm Hg; this incidence is even higher after emergency surgery [3,9,20••].
Pathophysiology

Neurologic

Acute IAH causes a significant increase in intracranial pressure (ICP); cerebral perfusion pressure may decrease or remain unchanged. Increased IAP produces this effect by augmenting pleural and other intrathoracic pressures, causing a functional obstruction to cerebral venous outflow by means of the jugular venous system. This hypothesis is consistent with the Monroe-Kellie doctrine, which states that 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. There may also be an added effect secondary to reduced systemic blood pressure as a result of decreased preload and CO [23]. The same phenomenon may be responsible for the high incidence of idiopathic intracranial hypertension in morbidly obese patients who have chronically elevated IAPs. Increase in ICP has also been documented during laparoscopic surgery. The effect of IAP on ICP is especially relevant in patients with multiple head and abdominal trauma, and laparoscopy in those patients may not be as risk-free as previously reported [23]. Acute increase of IAP may be the cause of neurologic morbidity in trauma patients without obvious signs of head trauma. In an animal model of IAH, nonsurgical abdominal decompression with continuous negative abdominal pressure resulted in normalization of ICP and cerebral perfusion pressure [25•].

Cardiovascular

When IAP rises above 10 mm Hg, cardiovascular changes occur: (1) CO drops due to an increase in afterload (systemic vascular resistance increases due to mechanical compression of vascular beds) and a reduction in preload (due to drop in stroke volume and a reduction of venous return); (2) mean arterial blood pressure may initially rise due to shunting of blood away from the abdominal cavity, but thereafter normalizes or decreases (usually it remains unchanged); (3) heart rate rises or remains unchanged; (4) pulmonary capillary wedge pressure (PCWP) and central venous pressure (CVP) values rise [3,9,10,20••]. By inducing venous stasis, IAH puts the patient at risk for venous thrombosis, and decompression may result in life-threatening pulmonary embolism [20••]. We studied the effects on cardiovascular function of nonsurgical decompression (by means of either ascitic fluid evacuation, gastric suction, or rectal intubation and enemas) in 11 medical ICU patients with IAH [21]. Decompression reduced mean IAP from 15.2±2.7 to 6.2±2.7 mm Hg. This resulted in a significant decrease in CVP, PCWP, and systolic pulmonary artery pressure. Cardiac output and systolic blood pressure rose significantly, and the observed effects persisted for 12.4±3.6 hours. In another study, designed to assess the effects of acute changes in IAP on cardiovascular function, IAP was increased 8–10 mm Hg by placing perfusion bags over the abdomen and banding. Hemodynamic measurements were done at baseline and during the three stages of the study after stabilization for 30 minutes in each stage. Baseline: normal IAP and zero PEEP (ZEEP); stage 1: increased IAP (perfusion bags on the abdomen) and ZEEP; stage 2: increased IAP and PEEP (cm H2O) set at IAP (mm Hg); stage 3: normal IAP (abdominal decompression by removing perfusion bags) and same PEEP level as stage 2. Increase in IAP increased CVP and PCWP significantly, whereas pulmonary artery pressure and systemic pressure remained unchanged (Fig. 2). Cardiac output and cardiac index decreased, but systemic vascular resistance index and pulmonary vascular resistance index remained unchanged. Positive end expiratory pressure application adjusted for IAP further increased IAP and filling pressures, and these changes normalized after abdominal decompression [26]. Other studies showed similar effects of IAH on hemodynamics; these effects are reversible but are most likely to pose a risk in patients with underlying respiratory disease and limited myocardial reserve [3,5,8,9,20••,23,24]. Increased IAP acting upon the diaphragm affects intrathoracic pressures to such an extent that the transmural filling pressure of the heart is reduced and venous return impeded by a rise in right atrial pressure. Preload and venous return are even further reduced in hypovolemia, which explains why all the above mentioned effects are aggravated in cases of hypovolemia or hemorrhagic shock. “Hypervolemic” patients, on the contrary, showed increased venous return in the presence of mild IAH, suggesting a protective effect of volume resuscitation [5,6,20••,31]. The addition of PEEP to increased IAP might significantly alter hemodynamics beyond the effect of PEEP or IAP alone [32,33]. The apparent deviation from Starling’s law is caused by the coupling of CVP and PCWP pressure to the intrathoracic pressure. In the presence of IAH-induced increase in intrathoracic pressure, CVP and PCWP tend to be erroneously increased when measured against atmospheric pressure and no longer reflect the true volemic status of the patient. These important effects of IAH on CVP and PCWP makes our traditional “volumetric filling pressures” unreliable indices of preload, and recent studies suggest the use of other preload markers such as right ventricular end diastolic volume index (RVEDVI) measured by volumetric thermodilution or intrathoracic blood volume index measured by dye indicator technique [30•,34].

Renal

It has been suggested that critical IAP necessary to initiate renal dysfunction is 10–15 mm Hg for oliguria and 20–25 mm Hg for anuria [3,9,10]. The etiology of renal impairment with decrease in renal blood flow, glomerular filtra-
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Figure 2. Effects of abdominal compression and decompression on cardiovascular and respiratory function in seven mechanically ventilated patients

(A,B) Central venous pressure and pulmonary capillary wedge pressure measurements were done at baseline and during the three stages of the study after stabilization for 30 minutes in each stage. Baseline: normal IAP and zero PEEP; stage 1: increased IAP (perfusion bags on the abdomen) and zero PEEP; stage 2: increased IAP and PEEP (cm H₂O) set at IAP (mm Hg); stage 3: normal IAP (abdominal decompression by removing perfusion bags) and same PEEP level as stage 2. Increase in IAP with 8±1.9 mm Hg resulted in a 1.7±1.2 mm Hg increase in CVP and a 2.3±1.4 mm Hg increase in PCWP (baseline vs stage 1), application of 14.9±3.6 cm H₂O PEEP resulted in a 3.7±2.5 mm Hg increase in CVP and a 3.1±1.7 increase in PCWP (stage 1 vs stage 2), and abdominal decompression by a 7.9±2.3 mm Hg decrease in IAP resulted in a 2.6±1.2 mm Hg decrease in CVP and a 3±1.4 decrease in PCWP (stage 2 vs stage 3), making CVP and PCWP unreliable markers of preload in patients with IAH who are mechanically ventilated with PEEP above 10 cm H₂O. (C,D) Effects of abdominal compression, followed by PEEP application and decompression on oxygenation and dynamic compliance. An 8±1.9 mm Hg increase of IAP caused a significant drop in dynamic compliance (Cdyn) from 40±10.2 mL/cm H₂O to 30.6±9.4 and a drop in PaO₂ from 87±13.7 mm Hg to 80.8±12.4 (baseline vs stage 1). Application of 14.9±3.6 cm H₂O PEEP resulted in a significant increase in Cdyn from 30.6±9.4 to 41.7±7 and PaO₂ from 80.8±12.4 to 107.1±28.3 (stage 1 vs stage 2). Abdominal decompression by 7.9±2.3 mm Hg resulted in a further rise in Cdyn from 41.7±7.1 to 45.3±9.1 and PaO₂ from 107.1±28.3 to 129.5±29.1 (stage 2 vs stage 3). BL, baseline; Cdyn, dynamic compliance; CVP, central venous pressure; IAP, intra-abdominal pressure; PCWP, pulmonary capillary wedge pressure; PEEP, positive end expiratory pressure; ZEEP, zero end expiratory pressure.

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Although some other reports did not show improvement in renal function after decompression [21,35•].

Respiratory

Intra-abdominal pressures above 15–20 mm Hg increase peak and plateau alveolar pressures. The rise in pressure on the diaphragm pushes both hemidiaphragms upward and causes a pattern of restrictive lung disease with a drop in functional residual capacity (FRC) and all other lung volumes [3,9,10]. Pulmonary vascular resistance is elevated and ventilation-perfusion abnormalities may occur. This results in diminished compliance, causing difficult ventilation and weaning. The respiratory system may be divided into the chest wall and the lung. Since the diaphragm is coupled to the abdominal wall,
any increase in IAP may therefore affect chest wall and lung mechanics, increasing the possibility for the development of atelectasis and reduction in FRC [36]. Calculation of volume-pressure curves has shown in animal and human studies that abdominal and subsequently chest wall compliance goes up after abdominal decompression, and this correlates well with the volume recruited [36,37].

We studied the effects of acute changes in IAP on respiratory function and found that an 8±1.9 mm Hg increase of IAP resulted in a significant increase in peak, plateau, and mean alveolar pressures whereas dynamic compliance (Cdyn) dropped significantly [26]. Application of 14.9±3.6 cm H2O PEEP resulted in a further significant increase alveolar pressures, whereas Cdyn returned to baseline values. Abdominal decompression by 7.9±2.3 mm Hg resulted in a significant decrease in alveolar pressures, whereas Cdyn improved further, together with PaO2 and PaO2/FiO2 ratio (Fig. 2). These results are in accordance with those from other studies [36,38••], which also found an increase in Cdyn, PaO2 and PaO2/FiO2 ratio after abdominal decompression.

The American-European consensus conference tells us to set PEEP above the lower inflection point (LIP, Pflex), to avoid high airway pressures, to keep plateau pressures between 30 and 35 cm H2O, to minimize oxygen toxicity by accepting lower PaO2 values whenever FiO2 exceeds 0.65, and, finally, to use low tidal volumes between 6 and 9 mL/kg. However, these strategies are difficult to apply in patients with IAH [39]. Until now only one randomized clinical trial, by Amato et al. [40•], showed a difference in mortality by using these lung-protective strategies. This study has been the subject of much debate and criticism; nevertheless, the basic idea of the study, to compare high PEEP low tidal volume versus low PEEP high tidal volume, is interesting. The main problems with this and other studies are: (1) patients were not divided into groups depending upon whether the underlying etiology for acute respiratory distress syndrome (ARDS) was surgical, secondary, or extrapulmonary versus medical, primary, or pulmonary, and (2) exact IAP values were not measured. Recent data have shown that lung and chest wall mechanics are quite different between these two groups [36,38,41,42•]. Ranieri et al. [36] showed in a very nice study that patients with ARDS had quite different respiratory mechanics: surgical patients had stiffer chest walls than medical patients, probably because of abdominal distention and decreasing compliance with increasing volume, implying alveolar overdistention, whereas medical patients had increasing compliance with increasing lung volume, suggesting ongoing alveolar recruitment. Compliance went up after decompression in the patients with IAH, and this correlated well with the volume recruited. The study also showed that the optimal PEEP level that correlates well with the LIP is overestimated in patients with IAH, and that the maximal tidal volume that correlates well with the upper inflection point is underestimated when calculated with the routinely used respiratory system volume-pressure curve compared with calculation by lung volume-pressure curve. The only drawback in this study was that FRC and the exact IAP values were not measured. In another interesting study, Gattinoni et al. [38••] showed that the localized character of parenchymal involvement in primary ARDS results in decreased lung but normal chest wall compliance, whereas secondary ARDS presents with preserved lung but decreased chest wall compliance, and that PEEP allows marked recruitment of lung units only in secondary but not in primary ARDS. As a result, the application of PEEP in pulmonary ARDS may cause overdistention of already open lung units, making these patients more prone to ventilator-associated lung injury than patients with extrapulmonary ARDS and IAH. The same phenomenon may be responsible for the change in respiratory mechanics in morbidly obese patients and for beneficial effects on respiratory mechanics and oxygenation parameters when ARDS patients are put in the prone or upright position [39,41,42•,43•]. Large volume sigh breaths can further recruit lung units, and this occurs mainly in extrapulmonary ARDS [42•]. In an interesting animal model, Mure et al. [43•] demonstrated that the prone position improves pulmonary gas exchange to a greater degree in the presence of abdominal distension. Other animal data also show interesting results regarding fluid balance and IAP, suggesting that large positive fluid balances result in increased IAP and lowered chest wall compliance [37]. This is an interesting twist to the “dry versus wet” debate with respect to fluid therapy in ARDS. Other studies showed that prior hemorrhage and resuscitation caused an earlier decline in cardiopulmonary function in the setting of IAH [44]. So, the truth may be somewhere between the catch-phrases “Dry lungs are happy lungs” and “Keep them dry, watch them die”; however, in view of the changes in chest wall mechanics by increased IAP, it may be preferable not to fluid-overload our ARDS patients [45].

We studied the efficacy of the recommended lung-protective ventilation strategies in seven patients with secondary acute lung injury (ALI)/ARDS and IAH, as well as the effect of PEEP-adjustment for IAP on oxygenation, compliance, and peak and plateau pressures [46]. When lung-protective strategy is aimed at peak inspiratory pressure <25–30 cm H2O, it was impossible to lower FiO2, to augment PEEP, or to alter the respiratory rate. Therefore, in this study of seven patients with ALI/ARDS and raised IAP, we were confronted with the fact that the lung-protective mecha-
nisms suggested in the literature were difficult to achieve or to apply. After PEEP-adaptation according to IAP, oxygenation-parameters dramatically improved but at the expense of raised peak, plateau, and mean alveolar pressures; however, this occurred without an increased risk for early barotrauma. We suggest that the definition of ARDS be further refined to distinguish between patients with ARDS with or without an abdominal component (increased IAP) that might influence chest wall mechanics. Our data, as well as the studies byGattinoni et al., Ranieri et al., Pelosi et al., and Mure et al. [36,38,41–43•,46], clearly demonstrate that not all ARDS patients are the same in terms of lung mechanics, that underlying etiologies and comorbidities are important, that determination of upper inflection point and LIP as settings for peak inspiratory pressure (and correlated maximal tidal volume) and PEEP is not as simple as it seems at the bedside, that IAP may affect chest wall mechanics, that abdominal decompression has beneficial effects on respiratory mechanics and oxygenation, and that there might be a possible correlation between Pflex and IAP.

In order to sort out the last hypothesis and to continue the search for appropriate ventilation in what is termed ARDS we performed total respiratory system mechanics and constructed 193 volume-pressure curves (super syringe technique) in 22 patients with ALI (Fig. 3) [47]. We found a very good correlation between IAP (calculated at ZEEP and converted to cm H2O) and Pflex (cm H2O) for the whole group of patients (193 measurements in 22 patients, R2 = 0.754, P < 0.0001) and this correlation was even better in the patients with secondary ALI/ARDS (137 measurements in 15 patients, R2 = 0.829, P < 0.0001). As suspected, the correlation was worse in patients with primary ALI/ARDS (56 measurements in 7 patients, R2 = 0.488, P < 0.0001). Correlation was also better in the subgroup with IAP ≥ 12 mm Hg (R2 = 0.718 vs 0.311). The mean Pflex for all measurements was around 12 cm H2O. This simple strategy is suggested for determination of best PEEP in patients with ALI/ARDS and IAP ≥ 12 mm Hg instead of the more time-consuming, not generally accepted, and not risk-free calculation of Pflex with the super syringe method.

**Visceral and hepatic**

When IAP is raised > 15 mm Hg, regional blood flow to all intra-abdominal organs is diminished due to a diminished splanchnic perfusion, resulting in tissue hypoxia and intestinal swelling [3,9,10,20••]. Elongation of mesenteric veins due to intestinal swelling causes tissue hypoxia and triggers a vicious cycle, generating more intestinal swelling and subsequent hypoxia. Increasing IAPs may, therefore, result in visceral hypoperfusion and secondary bacterial translocation and multiple organ dysfunction.
dysfunction syndrome, and may affect wound healing [48–53]. Increased IAP is associated with reduction of hepatic flow, a decreased mesenteric arterial blood flow, intestinal mucosal blood flow, and arterial perfusion to the stomach, duodenum, intestine, pancreas, and spleen. Adrenal blood flow remains preserved in most cases.

In patients with cirrhosis and portal hypertension, acute changes in IAP did not change the hepatic venous pressure gradient but markedly modified splanchnic and systemic hemodynamics [7,8]. Acute elevation of IAP may have deleterious effects by decreasing CO and hepatic blood flow and increasing blood flow through the portocollateral vessels (azygos system). Opposite changes occur after abdominal decompression. These results suggest that acute elevations of IAP may have deleterious effects in patients with cirrhosis and reduction of high IAP, as occurs after paracentesis in patients with tense ascites, may be beneficial. Whether or not raised IAP triggers bleeding from esophageal varices remains uncertain. Lymphatic duct flow is diminished when IAP rises, reducing the transport of peritoneal fluid into the thoracic lymphatic system.

Diebel et al. [49] assessed blood flow to the small bowel in an animal model, using three techniques: ultrasonic flow probe (to measure mesenteric artery blood flow), laser Doppler flow probe (to measure direct intestinal mucosal blood flow), and determination of intramucosal pH (to measure regional intestinal mucosal perfusion). While systemic perfusion indices (mean arterial pressure, PCWP, and CO) were kept unchanged during the study period with the infusion of lactated Ringer’s solution, the authors observed a severe progressive decrease in mesenteric and mucosal blood flow as IAP was increased, particularly at levels above 20 mm Hg (30–40% decrease at IAP levels 10–20 mm Hg, and 40–70% at levels 20–40 mm Hg). Although actual correlation coefficients were not reported, it appears that the three techniques are similar. In a comparable animal model, the same authors documented that slight increases in IAP (10 mm Hg) resulted in dramatic reductions in hepatic arterial blood flow (39%) [50]. It was only at IAP levels of 20 mm Hg that decreases in portal venous flow (34%) and microcirculatory blood flow (29%) became apparent. The vascular resistance of the intestine, by regulating the inflow of blood into the portal circulation, and of the hepatic arterial system, is the major determinant of liver blood flow. In IAH, however, IAP can become the major factor for determining mesenteric vascular resistance, and thus hepatic blood flow can be reduced on a purely mechanical basis. Impairment in visceral perfusion with increased IAP may be more likely in the hypovolemic patient. This study clearly demonstrated that the deleterious effects of IAH can occur at relatively low levels of IAP of about 10 mm Hg. The same group continued their initial observations in another animal model (23 rodents) and demonstrated the occurrence of decreased mesenteric blood flow and loss of intestinal barrier function (as indexed by bacterial translocation) in the presence of increased IAP at levels of 20–25 mm Hg for 60 minutes [51]. Regardless of the relative importance of bacterial translocation into the portal venous blood and intestinal lymphatics in the subsequent development of multiple organ failure, this study demonstrates that the deleterious effects of IAH can occur in a relatively short time.

A prospective study of gastric tonometry in patients with IAH showed a decrease of pHi with increased IAP and thus suggests ischemia [54]. Abnormal pHi readings (<7.32) were present in 49.3% of patients (39.7% on ICU admission). Raised IAP (>20 mm Hg) was present in 38.4% (28.8% on admission); IAPs above 15 mm Hg were present in 42.5%. Compared with patients with normal pHi, abnormal pHi patients were 11.3 (95% CI 3.2–43.5) times more likely to have increased IAP (>20 mm Hg), with an adjusted odds ratio of 1.4 (95% CI 0.4–5.1). With a cutoff at 15 mm Hg, the crude odds ratio for abnormal pHi was 14.7 (95% CI 4–57.7). Both abnormal pHi and IAP predict the same adverse outcomes with increased risk of hypotension, shock, intra-abdominal sepsis, renal impairment, need for relaparotomy, and death. Because about 70% of all abnormal pHi and IAP readings were obtained on admission, there seems to be a need for more refined management of these patients in the operating room before they arrive in the ICU. Global indices of tissue perfusion such as blood pressure, pulse rate, arterial pH, and lactate may not reflect aberrations of regional perfusion. Both IAP and pHi are strongly correlated and both may better reflect early problems in regional perfusion of intra-abdominal organs. Cases of fatal intestinal ischemia during laparoscopic surgery have also been reported, probably due to increased IAP and visceral ischemia. Ivory et al. [55•] conducted a retrospective study of 70 patients admitted to a level I trauma center with life-threatening abdominal trauma; all had IAP measurements. Patients with IAH (IAP >25 mm Hg) were treated with bedside or operating room laparotomy. Injury severity was similar between patients who had mesh closure (n = 45) and those who had fascial suture (n = 25). The overall incidence of IAH was 32.9%, and 22.2% in the mesh group versus 52% in the fascial suture group. Mortality, multiple organ dysfunction syndrome points, lactate, and base deficit were significantly worse in the IAH group. Tonometry was performed in 42 patients (60%), and of these 11 (18.3%) also had IAH, of whom 8 (72.7%) had abnormal pHi. In patients with IAH, pHi improved after abdominal decompression in 75%. The authors conclude that IAH
is frequent after major abdominal trauma and it may cause gut mucosal acidosis long before the onset of clinical ACS. Uncorrected, IAH may lead to splanchnic hypoperfusion, ACS, distant organ failure, and death. Because this was a retrospective study that examined only patients with arbitrarily defined “life-threatening abdominal trauma” the incidence of 32.9% lacks meaning. The authors did not demonstrate whether IAH was an independent risk factor for multiple organ failure or death (no multivariate analysis done); neither did they define the level of IAH that requires decompression, because all patients with IAP>25 mm Hg had “prophylactic” decompression. All these changes are exacerbated by hemorrhage or hypovolemia but have also been reported in cases where CO and systemic vascular resistance were kept within the normal range, suggesting a direct affect on all intra-abdominal organs [56,57]. Optimizing cardiac function alone during even short periods of even moderate levels of IAH may be inadequate to prevent adverse effects on splanchnic perfusion [57].

In recent years, increasing numbers of surgeons and intensivists have become aware of ACS, defined as an acute rise in IAP over 20 to 25 mm Hg and first described by Fietsam [58], who reported four patients who developed oliguria, hypoxia, hypercapnia, high peak inspiratory pressures, and a tense distended abdomen. The abdominal compartment syndrome is nowadays considered an emergency for which the open abdomen approach with temporary abdominal closure is the only treatment of choice [10,14,20••,59].

**The graded pathophysiologic response to increased intra-abdominal pressure**

Many authors allude to the biphasic cardiovascular response to elevated IAP. During moderate IAP elevation blood is squeezed from the splanchnic pool to the thoracic compartment, suddenly increasing venous return and CO [10]. With ongoing increased IAP, CO drops as the abdominal capacitance vessels are emptied. This initial “autotransfusion” effect of increased IAP is classically observed during the application of military anti-shock trousers. Comorbidities may also play an important role in aggravating the effects of raised IAP, such as pre-existing chronic renal failure, massive hemorrhage, hypovolemia, or pre-existing cardiomyopathy. In most cases it is the acuity of increase in IAP that is important and not necessarily the absolute increase [10,20••,23].

**Intra-abdominal pressure in relation to body mass index**

Sugerman et al. [60,61] showed that IAP was greater in morbidly obese (body mass index [BMI] >45) not critically ill patients, and correlated with other comorbidities such as hypoventilation, gastro-esophageal reflux, venous stasis, stress incontinence, incisional hernia, hypertension, and diabetes. Weight loss following gastric bypass decreased IAP, sagittal abdominal diameter, and obesity comorbidity. Hence it seems logical that the adverse physiologic effects related to acute increased IAP are aggravated by obesity [3,23,41]. However, we did not find a good correlation between high BMI and high IAPs in the critically ill [22]. Although there was a trend toward higher IAPs in obese patients, the differences were not statistically significant and may be due to the small sample size. The incidence of raised IAP in patients with BMI >30 was 23.5%, compared with 20.4% in those with BMI 25–30, 14% with BMI 20–25, and 7.6% in patients with BMI <20.

**Intra-abdominal pressure and body mass index as a prognostic factor**

In a medical ICU population, ICU mortality was 61.5% in 13 patients with raised IAP versus 10% in 40 patients with normal IAP [21]. In patients with IAH there was no difference in the absolute IAP value between survivors and nonsurvivors; however, there was a trend toward lower mortality the greater the absolute change in IAP after nonsurgical decompression and the greater the amount of ascites removed by paracentesis [21].

Excess body weight increases the risk of death from any cause and from cardiovascular disease in adults [61]. In the majority of population studies, the relation of BMI to mortality is a U-shaped curve, with increased risk in the lowest and highest percentiles of the distribution. In acutely ill patients, however, BMI below the 15th percentile remains an independent predictor of mortality whereas a high BMI (>85th percentile) was not significantly related to risk of mortality [62]. We prospectively studied the relation between IAP and BMI and their relation to subsequent mortality in 405 ICU patients [21]. Twenty-eight of 334 patients (8.4%) without IAH died in the ICU, versus 46 of 71 patients (64.8%) with BMI 20–25, and 7.6% in patients with BMI <20.

**Abdominal pressure in the critically ill**

Malbrain

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replacement therapy, the incidence of (abdominal) sepsis and septic shock, the cost of ICU and hospital stay, and the length of ICU stay. There was no U-shaped (concave) mortality curve associated with BMI; on the contrary, there was a trend toward lower mortality with higher BMI but none of this reached statistical significance: 25.8% in the first, 15% in the second, 16.3% in the third, and 16.2% in the fourth BMI quartile. In patients within the first BMI quartile (<22), ICU mortality was significantly higher compared with the total group of other BMI quartiles: 25.8% versus 15.8%.

Other studies have also shown that, in patients with increased IAP (>20 mm Hg) following major surgery, the following incidences of adverse outcome were observed: renal impairment 69%, oliguria 38%, abdominal sepsis 17%, and mortality 45% compared with 15%, 5%, 5% and 8% respectively in patients with normal IAP [3,9].

**Implications for the intensive care unit physician**

The data obtained from the mainly surgical literature show us the following:

1. IAP can easily be measured at the bedside with the standardized intravesical pressure recording method; intragastric continuous measurement with a modified tonometer seems promising.
2. The exact level of IAH that defines “critical IAP” remains subject to debate, but there is consensus that decompression should be performed at levels of IAP above 20–25 mm Hg; recent reviews, however, suggest that IAP as low as 10 mm Hg can cause organ dysfunction.
3. The incidence of IAH (IAP above 12–15 mm Hg) may be higher than suspected and varies according to underlying pathology but seems to be around 20% in medical patients, 30% in the surgical ICU population, and is even higher in emergency surgery patients.
4. Although overlooked for many years, there are well-established medical causes of IAH, of which massive fluid resuscitation for distributive shock is the most frequently encountered in the ICU.
5. The abdomen acts as a fluid compartment and follows the laws of Pascal.
6. Intra-abdominal hypertension has deleterious effects on all organs concealed within the abdomen and far outside, causing increased ICP, cardiovascular collapse and venous thrombosis, acute renal failure, acute respiratory failure, splanchnic hypoperfusion and abdominal wall ischemia, and finally triggering a vicious cycle which inevitably results in ACS.
7. Acute increase in IAP may be the cause of neurologic comorbidity in multiple trauma patients without obvious signs of head trauma, and abdominal decompression may normalize ICP and cerebral perfusion pressure.
8. The presence of hypovolemia, hemorrhage, and PEEP exacerbates the deleterious effects of IAH; however, it seems preferable not to fluid overload the patient with ARDS.
9. In cases of hemorrhagic shock, initial autotransfusion can be obtained by deliberately increasing IAP by putting weights on the abdomen (same effect as military antishock trousers).
10. The important effects of IAH on CVP and PCWP makes our traditional “volumetric filling pressures” unreliable indices of preload; RVEDVI measured by volumetric thermodilution and intrathoracic blood volume index measured by dye indicator technique are better preload markers when IAP is increased.
11. Not all patients with ARDS are the same with regard to underlying pathology and respiratory mechanics: lung recruitment strategies with high PEEP levels, sighs, and prone position seem to work better in patients with IAH.
12. In the search for optimal PEEP in ARDS, PEEP adjustment for raised IAP can be used, since there seems to be a good correlation between IAP and LIP (Pflex), especially in patients with secondary ARDS and IAH.
13. Patients with cirrhosis and raised IAP may benefit from paracentesis without the risk for hepatorenal syndrome.
14. There seems to be a strong correlation between IAP, pHi, and increased gut permeability (as demonstrated by bacterial translocation); this is important because the association between increased gut permeability and the subsequent development of multiple organ failure and death has been recently demonstrated by Doig 64•.
15. The abdominal compartment syndrome is a well-recognized clinical entity, is considered an emergency, and requires immediate action.
16. Intra-abdominal pressure acts as a guide for decompressive (re)laparotomy with temporal abdominal closure or nonsurgical decompression.
17. There is a good correlation between IAP and BMI in morbidly obese non-critically ill patients, a correlation that does not persist in the critically ill.
18. Intra-abdominal pressure and pHi predict the same adverse outcomes (shock, renal failure, sepsis, need for relaparotomy and death).
19. Intra-abdominal pressure seems to be an independent risk factor for mortality, comorbidities, hospital stay, and cost.

**Conclusions**

Because most of the data is obtained from anecdotal reports, animal studies, and retrospective or small prospective human studies from the surgical literature,
and because until now there is no data available from large, multicenter, randomized trials in mixed ICUs, it is clear that the clinical relevance of IAP in the general ICU population seems controversial. The concept of IAP measurement and the understanding of its physiologic implications are not new in medicine, but only recently have they been applied to the ICU population, and in the last decade increasing numbers of clinical studies have been performed on mixed ICU populations. Much work still needs to be done, but it is strongly believed that in the future IAP measurement will be part of routine monitoring in every ICU. Patients with increased IAP require our careful attention and, whenever necessary, abdominal decompression should be considered. A fancy new monitoring device capable of measuring IAP and pH need simultaneously and showing their evolution over time as a trend could be the necessary push that IAP and pH need before becoming generally accepted as part of routine monitoring in every ICU.

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References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as: • Of special interest •• Of outstanding interest


An excellent review on the topic from a surgical point of view with an emphasis on the ICU management and various surgical treatment options (damage control surgery, temporary abdominal closure, bedside decompression). The author proposes a modified monitoring method which is safer, less invasive, more efficient (repeated measurements possible), and cost effective.


The authors continued their initial animal research and confirm that increased IAP leads to increased ICP and diminished cerebral perfusion pressure. The application of an interesting nonsurgical decompression method, continuous negative abdominal pressure showed an amelioration of the intracranial disturbances in swine with IAH; however, continuous negative abdominal pressure impaired cerebral perfusion in swine with normal IAP.


27 Steinman M, da Silva LE, Coelho U, Poggetti RS, Bevilacqua RG, Birolini D, Rocha e Silva M: Hemodynamic and metabolic effects of CO₂ pneumo-


An excellent study comparing PCWP, CVP, and RVEDVI as estimates of preload in 20 patients with IAH before and after surgical decompression. Multiple regression analysis demonstrated that cardiac index correlated best with RVEDVI. In a previous study, the same authors also found a good correlation between CI and RVEDVI even at levels of PEEP up to 50 cm H₂O.


An interesting study (and one of the few prospective studies) that analyzed outcomes in 49 consecutive patients with IAH who underwent temporary abdominal closure. The study showed, a bit surprisingly, no improvement in renal function after decompression, although 10 patients experienced brisk diuresis. A possible mechanism might be the development of a renal compartment syndrome with ongoing IAH causing acute tubular necrosis, for which recovery time is longer.


The excellent study demonstrates that manifestations of pulmonary mechanics are quite different depending on the etiology of ARDS. Recruitment of alveoli by application of PEEP is possible only in secondary ARDS. In primary ARDS it may cause overdistention of already open lung units, promoting ventilator-induced lung injury. A drawback was that only 15 cm H₂O PEEP was applied and that no wealveolar recruitment maneuvers were done as suggested by Amato’s study. Nevertheless, these data may explain the diverse results of clinical trials in ARDS.


A randomized controlled clinical trial comparing protective ventilation (n = 29) with conventional ventilation (n = 24) in ARDS and the only study that showed a significant reduction in 28-day mortality and clinical barotrauma in the lung-protective group. (The recent NIH study was terminated after 600 patients, because outcome was better in the low-tidal volume group; however, data evaluation is incomplete and the study has not been published.) Lung-protective ventilation was not associated with a higher rate of survival to hospital discharge. This study has been the subject of much debate and criticism: (1) very few patients were included; despite this, six interim analyses were done (after every 5 patients recruited after the first 28); (2) the power calculation and the difference in survival used (2.5) were suspect, and the use of a reasonable estimate would have increased the number of patients required; (3) the applied statistical test (Cox’s proportional hazard model) is not appropriate for the endpoint used (28-day mortality), and the more applicable χ² test would not show statistical significance, and, finally, (4) the study could not be blinded. Despite all this, the basic idea of the study, to compare high PEEP low tidal volume versus low PEEP high tidal volume, is interesting. The main problem with this and other ARDS studies is appropriate patient selection (ARDS caused by pulmonary or extrapulmonary disease? Surgical or medical ARDS? Septic or nonseptic ARDS? Primary or secondary ARDS? Underlying disease?) and that exact IAP values were not measured.


An excellent study in which the authors tested the hypothesis that lung-protective ventilation in ARDS using low tidal volumes and high PEEP does not reverse all the atelectasis. The application of large sigh breaths had beneficial effects on lung recruitment (measured by end expiratory lung volume) that correlated well with oxygenation and lung elastance. The interesting part was that all of these changes were more pronounced in patients with secondary (abdominal) ARDS. This study adds to the growing body of evidence that all ARDS patients are not the same in terms of lung mechanics, which complicates the search for appropriate ventilation. The application of sighs in addition to current best practice seems promising, especially in patients with IAH.


In an interesting animal study, these authors demonstrate that the prone position increases PaO₂, and decreases alveolar-arterial difference in oxygen tension and ventilation-perfusion heterogeneity to a greater degree in the presence of IAH. A possible explanation might be a decrease in IAP, resulting in a concomitant decrease in pleural pressure in the prone position, thus facilitating regional ventilation in the dependent lung zones near the diaphragm.


A retrospective study of 70 patients with life-threatening abdominal trauma; all had IAP measured. Two groups of patients were compared: patients with loosely applied abdominal wall mesh closure (n = 45) and those with fascial suture closure (n = 25) after appropriate intra-abdominal exploration and treatment. The overall incidence of IAH (defined as IAP >25 mm Hg) was 32.9%, 22.2% in the mesh group versus 52% in the fascial suture group. The authors conclude that IAH is frequent after major abdominal trauma and it may cause gut mucosal acidosis long before the onset of clinical ACS. Uncorrected, it may lead to splanchic hypoperfusion, ACS, distant organ failure, and death. Loose closure of the abdomen under circumstances of severe abdominal trauma (by mesh or sterile intravenous bags) may facilitate the prevention of IAH and reduce complications. Since it was a retrospective study that examined only patients with arbitrarily defined “life-threatening abdominal trauma,” the incidence of 32.9% lacks meaning. The authors did not demonstrate whether IAH was an independent risk factor for multiple organ failure or death (no multivariate analysis done); neither did they define the level of IAH that requires decompression, because all patients with IAP >25 mm Hg had “prophylactic” decompression.