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REVIEWS

Respiratory effects of increased intra-abdominal pressure

Aspects respiratoires du syndrome du compartiment abdominal

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KEYWORDS
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Abdominal hypertension;
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Definitions;
Etiology;
Risk factors

Abstract There has been an exponentially increasing interest in intra-abdominal hypertension (IAH). Comparison of the published data however is difficult due to the lack of consensus definitions. This review will focus on the available literature from the last years. A Medline and Pubmed search was performed using "intra-abdominal pressure" (IAP), "IAH", and "abdominal compartment syndrome" (ACS) in combination with "cardiac", "cardiovascular", "organ function", "respiratory" or "pulmonary" as search items. The aim was to find an answer to two questions: first, "Is it not time to pay attention to IAP in the critically ill?" And second, "what is the cardiovascular and respiratory impact of increased IAP?"Although the number of studies published on this topic is steadily increasing and confirms the pathophysiologic implications of IAH on end-organ function within and outside the abdominal cavity it remains difficult to compare the literature data because measurement methods and definitions used are not uniform. Therefore the World Society on Abdominal Compartment Syndrome (WSACS – www.wsacs.org) recently published a consensus definitions report. Provocative data have been published regarding the interactions between the abdominal and thoracic compartments especially in patients with capillary leak and fluid overload. In conclusion we can state that the answer is that it is now time to pay attention to IAP in the critically ill, but it is also time for standardized IAP measurement methods and multicenter randomized interventional studies.
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MOTS CLÉS
Pression abdominale ;
Hyperpression

Résumé L'intérêt des cliniciens pour le syndrome du compartiment abdominal est croissant depuis quelques années. La comparaison et l'analyse des données publiées sur ce thème sont toutefois difficiles en raison de l'absence de définitions consensuelles concernant ce syndrome. Cette revue fait le point sur la littérature disponible sur ce sujet à partir d'une

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abdominale ;
Insuffisance respiratoire ;
Insuffisance cardiaque

recherche bibliographique faite à partir des données disponibles dans « Medline ». Les mots clés utilisés pour la recherche des données ont été : « intra-abdominal pressure », « intra-abdominal hypertension », « abdominal compartment » en association avec « cardiac », « cardiovascular », « organ function », « respiratory » or « pulmonary ». L'objectif de cette mise au point a été ensuite d'essayer de répondre à deux questions : Est-il temps de se préoccuper de la pression abdominale chez les patients de réanimation ? Quel est l'impact de l'augmentation de la pression abdominale sur les fonctions cardiaques et respiratoires ? Même si le nombre d'études publiées sur ce sujet augmente, il reste difficile de comparer les données de la littérature car les méthodes de mesures et les définitions utilisées ne sont pas homogènes. Cette absence d'homogénéité a conduit la société mondiale du syndrome du compartiment abdominal (WSACS – www.wsacs.org) à publier des définitions consensuelles sur ce syndrome. Des données marquantes sont également disponibles sur les interactions entre les compartiments thoraciques et abdominaux en particulier chez les sujets en inflation hydrosodée ou atteints de fuite capillaire. Compte tenu de ces éléments, il paraît nécessaire de ne pas négliger la mesure de la pression intra-abdominale chez les patients de réanimation. Il est également temps d'en standardiser la mesure et aussi de concevoir des études multicentriques randomisées avec objectif thérapeutique.
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Introduction

A compartment syndrome exists when the increased pressure in a closed anatomic space threatens the viability of surrounding tissue. When this occurs in the abdomen the impact on end-organ function within and outside the cavity can be devastating. The abdominal compartment syndrome (ACS) is not a disease, as such it can have many causes and it can develop within many disease processes. It is only recently that ACS received a heightened awareness. The development of intra-abdominal hypertension (IAH) and ACS are of tremendous importance in the care of critically ill, surgical and trauma patients. The impact of increased intra-abdominal pressure (IAP) on end-organ function and especially the lungs can no longer be ignored!

Historical background

As was nicely pointed out recently by Schein, the effects of elevated IAP have been known since 1863, when Marey of Paris highlighted that "the effects that respiration produces on the thorax are the inverse of those present in the abdomen" [1]. In 1890, Heinricius demonstrated that ACS was fatal to animals because of impairment of respiration, decreasing cardiac diastolic distension and hypotension. The term ACS was first used by Fietsam et al. [2] in the late 1980s to describe the pathophysiologic alterations resulting from IAH secondary to aortic aneurysm surgery. Hence the first definition of ACS was finally coined.

Definitions

The World Society of Abdominal Compartment Syndrome (WSACS – www.wsacs.org) was founded in 2004 to serve as a peer-reviewed forum and educational resource for all healthcare providers as well as industry that have an interest in IAH and ACS. The mission of the society is to foster education, promote research and thereby improve survival of patients with IAH and ACS. Recently the first consensus definitions report of the WSACS has been published [3]. Table 1 summarizes these consensus definitions.

Recognition of ACS

Clinical awareness

Despite an escalation of the medical literature on the subject, there still appears to be an under-recognition of the syndrome. The results of several surveys on the physician's knowledge of IAH and ACS have recently been published [4]. The bottom line is that there is still a general lack of clinical awareness and many ICUs never measure IAP. No consensus exists on optimal timing of measurement or decompression. In a recent editorial Ivatury [5] states that: "One potential exegesis of this widespread under-appreciation of these syndromes may be related to our rapidly evolving understanding of their patho-physiology. Our knowledge is no longer restricted to experimentally sound (isolated IAH) concepts, but is elevated to a true clinical phenomenon (IAH as a "second-hit" after ischemia–reperfusion)." To fill this void a new survey has been launched. The survey can be accessed via www.wsacs.org/survey.htm.

Etiology

The ACS can be diagnosed when there is increased IAP with evidence of end-organ dysfunction. While multiple causes of acute cardiopulmonary, renal, hepatosplanchnic or neurologic deterioration exist in the intensive care unit, it is important that we recognize the IAP as being an independent risk factor for this organ function deterioration. Hence the timely recognition of the underlying risk factors and predisposing conditions that lead to IAH and ACS is extremely important. Indications for IAP monitoring should be based on the presence/absence of these risk factors. Many conditions are reported in association with IAH/ACS, and they are summarized in Table 2.

Table 1 Consensus definitions [3]

Definition 1	IAP is the steady-state pressure concealed within the abdominal cavity
Definition 2	APP = MAP – IAP
Definition 3	FG = GFP – PTP = MAP – 2 × IAP
Definition 4	IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line
Definition 5	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml of sterile saline
Definition 6	Normal IAP is approximately 5–7 mmHg in critically ill adults
Definition 7	IAH is defined by a sustained or repeated pathologic elevation of IAP ≥ 12 mmHg
Definition 8	IAH is graded as follows: Grade I: IAP 12–15 mmHg Grade II: IAP 16–20 mmHg Grade III: IAP 21–25 mmHg Grade IV: IAP > 25 mmHg
Definition 9	ACS is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure
Definition 10	Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention
Definition 11	Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region
Definition 12	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

ACS: abdominal compartment syndrome; APP: abdominal perfusion pressure; FG: filtration gradient; GFP: glomerular filtration pressure; IAH: intra-abdominal hypertension; IAP: intra-abdominal pressure; MAP: mean arterial pressure; PTP: proximal tubular pressure.

Table 2 Etiology, risk factors and predisposing conditions for IAH

Related to diminished abdominal wall compliance
Mechanical ventilation, especially fighting with the ventilator and the use of accessory muscles
Use of PEEP or the presence of auto-PEEP
Basal pleuroneumonia
High body mass index
Pneumoperitoneum
Abdominal (vascular) surgery, especially with tight abdominal closures
Pneumatic anti-shock garments
Prone and other body positioning
Abdominal wall bleeding or rectus sheath hematomas
Correction of large hernias, gastroschisis or omphalocoele
Burns with abdominal eschars
Related to increased intra-abdominal contents
Gastroparesis/gastric distention/Ileus/Colonic pseudo-obstruction
Abdominal tumor
Retroperitoneal/ abdominal wall hematoma
Enteral feeding
Related to abdominal collections of fluid, air or blood
Liver dysfunction with ascites
Abdominal infection (pancreatitis, peritonitis, abscess, …)
Hemoperitoneum
Pneumoperitoneum
Related to capillary leak and fluid resuscitation
Acidosis[a] (pH below 7.2)
Hypothermia[a] (core temperature below 33 °C)
Coagulopathy[a] (platelet count below 50,000 per mm^3 OR an activated partial thromboplastin time (APTT) more than two times normal OR a prothrombin time (PTT) below 50% OR an international standardized ratio (INR) more than 1.5)
Polytransfusion/trauma (> 10 units of packed red cells/ 24 hours)
Sepsis (as defined by the American–European Consensus Conference definitions)
Severe sepsis or bacteremia
Septic shock
Massive fluid resuscitation (> 5 l of colloid or > 10 l of crystalloid/24 hours with capillary leak and positive fluid balance)
Major burns

[a] The combination of acidosis, hypothermia and coagulopathy has been forwarded in the literature as the deadly triad [82,83].

Diagnosis

Clinical and radiologic examination

The abdominal perimeter or girth or clinical examination cannot be used as a surrogate for IAP since this is far from accurate with a sensitivity and positive predictive value of around 40–60% [6,7]. Radiologic investigation with plain radiography of the chest or abdomen, abdominal ultrasound or CT-scan are also insensitive to the presence of increased IAP.

Measurement of IAP

Since the abdomen and its contents can be considered as relatively non-compressive and primarily fluid in character, behaving in accordance to Pascal's law, the IAP measured at one point may be assumed to represent the IAP throughout the abdomen [8,9].

In the strictest sense, normal IAP ranges from 0 to 5 mmHg [10]. Certain physiologic conditions, however, such as morbid obesity [11,12], ovarian tumors, cirrhosis or pregnancy, may be associated with chronic IAP elevations of 10–15 mmHg to which the patient has adapted with an absence of significant patho-physiology. In contrast, children commonly demonstrate low IAP values [13].

Different indirect methods for estimating IAP are used clinically because direct measurements are considered to

be too invasive [8,14]. These techniques include rectal, uteral, gastric, inferior vena caval and urinary bladder pressure measurement. Only gastric and bladder pressures are used clinically. Over the years bladder pressure has been forwarded as the gold-standard indirect method (Fig. 1). The bladder technique has achieved the most widespread adoption worldwide due to its simplicity and minimal cost [8,9]. However considerable variation is noted between the different techniques used, and recent data suggest to instill minimal volumes (10–25 ml) into the bladder for priming [15–17].

Recently, new measurement kits, either via a FoleyManometer (Holtech Medical, Kopenhagen, Denmark, at www.holtech-medical.com), an AbViser-valve (Wolfe Tory Medical, Salt Lake City, UT, USA, at www.wolfetory.com) or a balloon-tipped stomach catheter (Spiegelberg, Hamburg, Germany, at www.spiegelberg.de and Pulsion Medical Systems, Munich, Germany, at www.pulsion.com) [9] have become commercially available.

Abdominal perfusion pressure (APP) measurement

Analogous to the widely accepted and clinically utilized concept of cerebral perfusion pressure, calculated as mean arterial pressure (MAP) minus intracranial pressure, APP, calculated as MAP minus IAP, has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation [18–21].

$$\mathrm{APP} = \mathrm{MAP} - \mathrm{IAP}$$

APP, by considering both arterial inflow (i.e. MAP) and restrictions to venous outflow (i.e. IAP), has been demonstrated to be statistically superior to either parameter alone in predicting patient survival from IAH and ACS [21]. A target APP of at least 60 mmHg has been demonstrated to correlate with improved survival from IAH and ACS.

Pathophysiologic implications

IAH affects multiple organ systems in a graded fashion. In order to better understand the clinical presentation and management of disorders of IAH, one must understand the physiologic derangements within each organ system separately [22]. It is beyond the scope of this review to give a concise and complete summary of the pathophysiologic implications of raised IAP on end-organ function within and outside the abdominal cavity [23,24]. We will only discuss some key-messages related to each organ that will affect daily clinical practice. Afterwards we will more extensively discuss the impact on the heart and the lungs since this is the topic of this review.

Neurologic function

Acute IAH causes an increase in intracranial pressure due to augmentation in pleural pressure. Cerebral perfusion pressure will decrease due to a functional obstruction of cerebral venous outflow caused by the increased intrathoracic pressure due to the cephalad displacement of the diaphragm in combination with a reduced systemic blood pressure as a result of decreased preload and cardiac output.

Hepatic function

The liver appears to be particularly susceptible to injury in the presence of elevated IAP. Animal and human studies have shown impairment of hepatic cell function and liver perfusion even with only moderately elevated IAP of

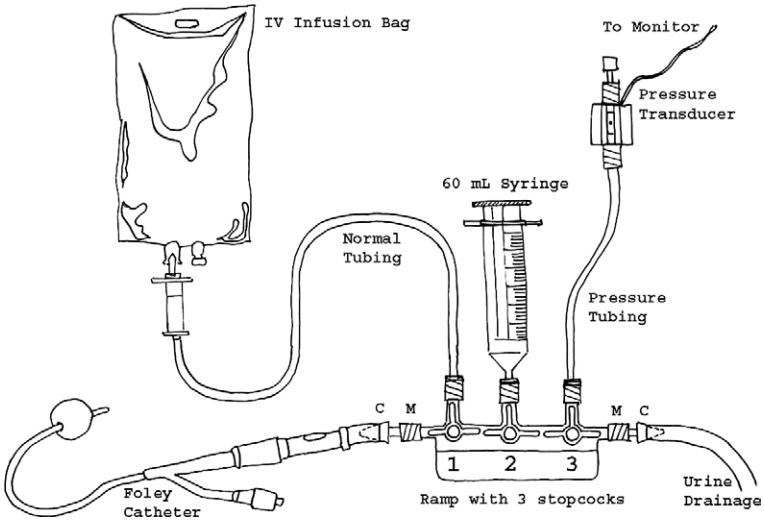

Figure 1 Using a patent Foley catheter with clamped drainage tube, 10–25 ml sterile water are infused into the bladder via 3 three-way stopcocks placed in series between the Foley catheter and the urine drainage tubing. By turning the stopcocks "open" and "close" to either the saline infusion bag or the pressure transducer the bladder can be primed and the bladder pressure as estimate for IAP can be measured afterwards. The bladder pressure measured with a transducer correlates well with directly measured IAP (reprinted with permission [8]).

10 mmHg [25,26]. Furthermore acute liver failure, decompensated chronic liver disease and liver transplantation are frequently complicated by IAH and the ACS [27,28]. In the management of these patients it might be useful to measure the plasma disappearance rate for indocyaninegreen as this correlates not only with liver function and perfusion but also with IAP [29,30]. With increasing IAP there is, decreased hepatic arterial flow, decreased venous portal flow and increase in the portacollateral circulation, this all exerts physiological effects with decreased lactate clearance, altered glucose metabolism and mitochondrial function.

Renal function

IAH has been associated with renal impairment for over 150 years [1]. It is only recently however that a clinically recognized relationship has been found [31–34]. The etiology of renal impairment in IAH is not entirely well established, however it may be multifactorial: reduced renal perfusion, reduced cardiac output and increased systemic vascular resistance and alterations in humeral and neurogenic factors.

Gastrointestinal function

IAH has profound effects on splanchnic organs, causing diminished perfusion, mucosal acidosis and setting the stage for multiple organ failure [35]. The pathologic changes are more pronounced after sequential insults of ischemia–reperfusion and IAH. It appears that IAH and ACS may serve as the second insult in the two-hit phenomenon of the causation of multiple organ dysfunction syndrome [36,37]. Recent clinical studies have demonstrated a temporal relationship between ACS and subsequent multiple organ failure [35,38,39]. In animals ACS provokes cytokine release, and neutrophil migration resulting in remote organ failure. In humans ACS results in splanchnic hypoperfusion that may occur in the absence of hypotension or decreased cardiac output. This ischemia and reperfusion injury to the gut serves as a second insult in a two-hit model of multiple organ failure where the lymph flow conducts gut-derived pro-inflammatory cytokines to remote organs. IAP inversely correlates with pHi [40–42]. IAP inversely correlates with plasma disappearance rate for indocyaninegreen [29]. IAH triggers a vicious cycle leading to intestinal edema, ischemia, bacterial translocation and finally multiple organ dysfunction syndrome [43–45].

Abdominal wall and endocrine function

Increased IAP has been shown to reduce abdominal wall blood flow by the direct, compressive effects leading to local ischemia and edema [46]. This can decrease abdominal wall compliance and exacerbate IAH [47]. Abdominal wall muscle and fascial ischemia may contribute to infectious and non-infectious wound complications (e.g. dehiscence, herniation, necrotizing fasciitis) often seen in this patient population.

Cardiac and respiratory effects of increased abdominal pressure

Cardiovascular function

Due to the cephalad movement of the diaphragm pleural pressure and intrathoracic pressure will increase. This will result in a difficult preload assessment because traditional filling pressures will be erroneously increased. When IAP rises above 10 mmHg cardiac output drops due to an increase in afterload and a decrease in preload and left ventricular compliance. Systemic vascular resistance increases due to mechanical compression of vasculary beds and a reduction in preload due to drop in stroke volume and a reduction of venous return [48–51]. Mean arterial blood pressure may initially rise due to shunting of blood away from the abdominal cavity but thereafter normalizes or decreases [21,52]. Table 3 schematically shows the cardiovascular effects of IAH:

- cardiovascular dysfunction and failure are common in IAH or ACS;

Table 3 Cardiovascular effects related to IAP[a]

Diaphragm elevation ↑
Pleural and intrathoracic pressure ↑
Difficult preload assessment
Pulmonary artery occlusion pressure ↑
Central venous pressure ↑
Transmural filling pressure =↘
Intrathoracic blood volume index =↘
Global enddiastolic blood volume index =↘
Right ventricular end-diastolic volume =↘
Right, global and left ventricular ejection fraction =↘
Extra vascular lung water =↗
Stroke volume variation ↗
Pulse pressure variation ↗
Systolic pressure variation ↗
Inferior vena caval flow ↓
Venous return ↓
Left ventricular compliance and contractility ↓
Downward and right shift of the Frank Starling curve
Cardiac output ↓
Systemic vascular resistance ↑
MAP ↗ =↘
Pulmonary artery pressure ↑
Pulmonary vascular resistance ↑
Heart rate ↗ =
Lower extremity hydrostatic venous pressure ↑
Venous stasis, edema, ulcers ↑
Venous thrombosis ↑
Pulmonary embolism[b] ↑
Mixed venous oxygen saturation ↓
Central venous oxygen saturation ↓

[a] Cardiovascular effects are exacerbated in case of hypovolemia, hemorrhage, ischemia, auto-PEEP or high PEEP ventilation.
[b] Upon decompression.

- accurate assessment and optimization of preload, contractility, and afterload is essential to restore end-organ perfusion and function;
- our understanding of traditional hemodynamic monitoring techniques and parameters, however, must be reevaluated in IAH/ACS since pressure-based estimates of intravascular volume as pulmonary artery occlusion pressure and central venous pressure (CVP) are erroneously increased:
  - the clinician must be aware of the interactions between intrathoracic pressure (ITP), IAP, positive end expiratory pressure (PEEP), and intracardiac filling pressures;
  - misinterpretation of the patient's minute-to-minute cardiac status may result in the institution of inappropriate and potentially detrimental therapy;
  - Transmural™ filling pressures, calculated as the endexpiration value (ee) minus the ITP better reflect preload [49]:

$$\mathrm{CVP}^{\mathrm{TM}} = \mathrm{CVPee} - \mathrm{ITP}$$

$$\mathrm{PAOP}^{\mathrm{TM}} = \mathrm{PAOPee} - \mathrm{ITP}$$

  - a quick estimate of transmural filling pressures can also be obtained by subtracting half of the IAP from the end expiratory filling pressure:

$$\mathrm{CVP}^{\mathrm{TM}} = \mathrm{CVPee} - \mathrm{IAP}/2$$

$$\mathrm{PAOP}^{\mathrm{TM}} = \mathrm{PAOPee} - \mathrm{IAP}/2$$

  - the surviving sepsis campaign guidelines targeting initial and ongoing resuscitation towards a CVP of 8-12 mmHg [53] and other studies targeting a MAP of 65 mmHg [54] should be interpreted with caution in case of IAH/ACS to avoid unnecessary over- and under resuscitation!
- volumetric estimates of preload status such as right ventricular end diastolic volume index or global end diastolic volume index, are especially useful because of the changing ventricular compliance and elevated intrathoracic pressure [51,55-58];
- functional dynamic hemodynamic parameters should be used to assess volume responsiveness [59];
- the cardiovascular effects are aggravated by hypovolemia and the application of PEEP [60-64], whereas hypervolemia has a temporary protective effect [65].

Pulmonary function

The interactions between the abdominal and the thoracic compartment pose a specific challenge to the intensive care unit physicians [66]. Both compartments are linked via the diaphragm and on average a 50% (range 25-80%) transmission of IAP to the intrathoracic pressure has been noted in previous animal and human studies [51]. Patients with primary ACS will often develop a secondary acute respiratory distress syndrome (ARDS) and will require a different ventilatory strategy and more specific treatment than a patient with primary ARDS [67,68]. The major problem lays in the reduction of the functional residual capacity (FRC). Together with the alterations caused by secondary ARDS this will lead to the so-called "baby-lungs" Fig. 2 schematically illustrates the respiratory effects of IAH. Figs. 3 and 4 show the effect of increased IAP during venti-

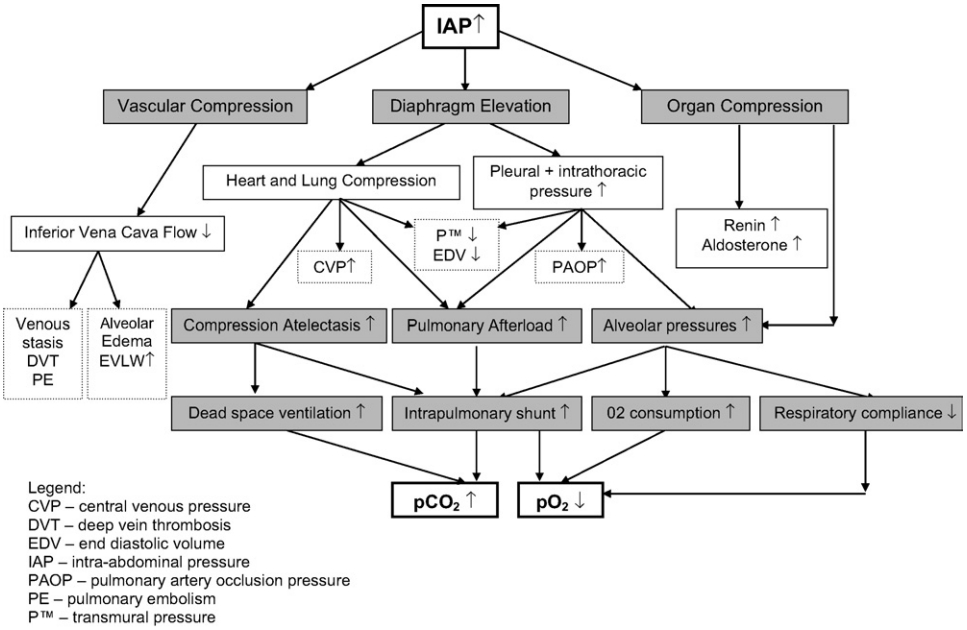

Figure 2 Respiratory effects of IAH. CVP: central venous pressure; DVT: deep vein thrombosis; EDV: end diastolic volume; IAP: intra-abdominal pressure; PAOP: pulmonary artery occlusion pressure; PE: pulmonary embolism; P™: transmural pressure.

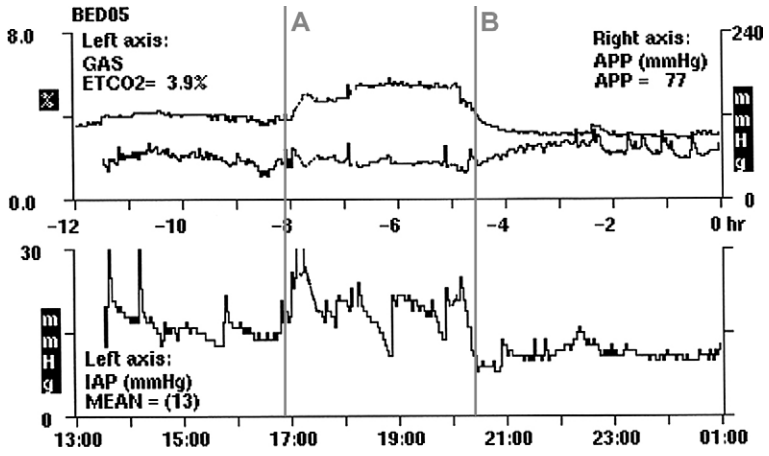

Figure 3 Upper panel: Trend tracing of endtidal CO_2 ($ETCO_2$) on the left axis and APP on the right axis in a mechanically ventilated patient. Lower panel: Trend tracing of IAP. Line A (16:50) marks the beginning of ventilator dyssynchrony due to fighting and abdominal muscle contractions with increased IAP up to 30 mmHg, increased $ETCO_2$ and decreased APP. Line B (20:40) marks the end of dyssynchrony with normalization of all parameters after the start of a continuous infusion with cisatracurium. This case nicely demonstrates the interactions between the abdominal and thoracic compartments.

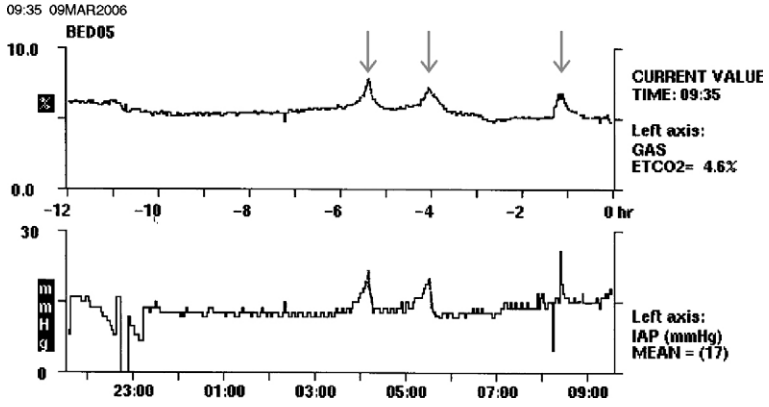

Figure 4 Another case demonstrating the interactions between the abdominal and thoracic compartments. Trend tracings of endtidal CO_2 ($ETCO_2$) on upper panel and IAP on lower panel. The arrows indicate administration of cisatracurium bolus during ventilator dyssynchrony due to fighting and abdominal muscle contractions with increased IAP up to 22 mmHg and increased $ETCO_2$.

lator dyssynchrony on endtidal CO_2 production and the effect of curarization in an individual patient. Table 4 schematically shows the pulmonary effects of IAH. Some key-issues to remember are:

- IAH decreases total respiratory system compliance by a decrease in chest wall compliance, while lung compliance remains unchanged [47,69].
- Best PEEP should be set to counteract IAP whilst in the same time avoiding over-inflation of already well-aerated lung regions:

$$\text{Best PEEP} = \text{IAP}$$

- The ARDS consensus definitions should take into account PEEP and IAP values.
- During lung protective ventilation, the plateau pressures should be limited to transmural plateau pressures Pplat™ below 35 cmH_2O:

$$\text{Pplat}^{\text{TM}} = \text{Pplat} - \text{IAP}/2$$

- The PAOP criterion in ARDS consensus definitions is futile in case of IAH and should be adapted (most patients with IAH and secondary ARDS will have a PAOP above 18 mmHg).
- IAH increases lung edema, within this concept monitoring of extravascular lung water index seems warranted [70].
- The combination of capillary leak, positive fluid balance and raised IAP poses the patient at an exponential danger for lung edema.

Table 4 Pulmonary effects related to IAP

Diaphragm elevation ↑
Intrathoracic pressure ↑
Pleural pressure ↑
Functional residual capacity ↓
All lung volumes (TLC, TV, …) ↓ (~restrictive disease)
Extrinsic compression lung parenchyma[a] ↑
Auto-PEEP ↑
Compression atelectasis ↑
Peak airway pressure ↑ (volume controlled MV)
Mean airway pressure ↑
Plateau airway pressure ↑
Pulmonary vascular resistance ↑
Alveolar barotrauma =↑
Alveolar volutrauma =↑
Dynamic compliance ↓
Static respiratory system compliance ↓
Static chest wall compliance ↓↓
Static lung compliance =
Upper inflection point on PV curve ↓
Lower inflection point on PV curve ↑
Hypercarbia – pCO_2 retention ↑
PaO2 ↓ and PaO2/FiO2 ↓
Alveolar oxygen tension ↓
Oxygen transport ↓
Dead-space ventilation ↑
Intrapulmonary shunt ↑
Ventilation perfusion mismatch ↑
Ventilation diffusion mismatch ↑↑
Oxygen consumption ↑
Metabolic cost and work of breathing ↑
Alveolar edema ↑
Extra vascular lung water (EVLW) =↗
Prolonged ventilation
Difficult weaning
Activated lung neutrophils ↑
Pulmonary inflammatory infiltration ↑
Pulmonary infection rate ↑

[a] Parenchymal compression is exacerbated in case of hemorrhagic shock or hypotension.

- Body position affects IAP:
  - Putting an obese patient in the upright position can cause ACS [71].
  - The abdomen should hang freely during prone positioning [30].
  - The anti-Trendelenburg position may improve respiratory mechanics, however it can decrease splanchnic perfusion [72].
- The use of curarization should be balanced against the beneficial effect on abdominal muscle tone resulting in decrease in IAP and improvement of APP, and the detrimental effect on lung mechanics resulting in atelectasis and sur-infection [73].
- The presence of IAH will lead to pulmonary hypertension via increased intrathoracic pressure with direct compression on lung parenchyma and vessels and via the diminished left and right ventricular compliance. In this case the administration of inhaled NO or ilomedine (prostacyclin) may be justified.
- The effect of IAP on parenchymal compression is exacerbated in case of hemorrhagic shock or hypotension.

Clinical management

The management of patients with IAH is based on three principles [74,75]:

- specific procedures to reduce IAP and the consequences of ACS;
- general support (intensive care) of the critically ill patient;
- optimization after surgical decompression to perhaps counteract some of the specific adverse effects associated with decompression.

Medical treatment

Before surgical decompression is considered less invasive medical treatment options should be optimized. Different medical treatment procedures have been suggested to decrease IAP [19]. These are based on five different mechanisms:

- improvement of abdominal wall compliance;
- evacuation of intraluminal contents;
- evacuation of abdominal fluid collections;
- correction of capillary leak and positive fluid balance;
- specific treatments.

Table 5 gives an overview of the different medical treatment options.

Surgical decompression

Although decompression remains the only definite management for ACS, the timing of this procedure still remains controversial. During the intervention specific anesthetic challenges need to be solved and after decompression the patient is at risk for ischemia reperfusion injury, venous stasis and fatal pulmonary embolism [76]. Maintaining adequate preload and APP are the key to success [18,21,60]. Open abdomen treatment (or laparostomy) was initially intended for patients with diffuse intra-abdominal infections, and often used in combination with a planned relaparotomy approach. Due to the increased awareness of the deleterious effects of IAH, open abdomen treatment, either prophylactic or therapeutic, is more common in the intensive care unit [38,77]. Several methods for temporary abdominal closure are have been described (we refer the reader to more specific textbooks for further information [77]):

- **Moist gauze** used to be the preferred method of covering the abdomen, but this is no longer used.
- **"Bogota bag":** a plastic sheet is cut from a sterile 3L irrigation bag, and sewn to the skin or fascia.

Table 5 Medical treatment options for IAH and ACS

Improvement of abdominal wall compliance
Sedation
Pain relief (not fentanyl!) [84]
Neuromuscular blockade [20,73,85-87]
Body positioning [29,30,72]
Negative fluid balance
Skin pressure decreasing interfaces [29]
Weight loss [88]
Percutaneous abdominal wall component separation [89,90]
Evacuation of intraluminal contents
Gastric tube and suctioning [91-94]
Gastroprokinetics (erythromycin, cisapride, metoclopramide) [95-97]
Rectal tube and enemas [91-94]
Colonoprokinetics (neostygmine, prostygmine bolus or infusion) [98-100]
Endoscopic decompression of large bowel
Colostomy
Ileostomy
Evacuation of peri-intestinal and abdominal fluids
Ascites evacuation in cirrhosis [101-106]
CT- or US-guided aspiration of abscess
CT- or US-guided aspiration of hematoma
Percutaneous drainage of (blood) collections
Ascites evacuation in cirrhosis [90,107,108]
Correction of capillary leak and positive fluid balance
Albumin in combination with diuretics (furosemide) [60,109,110]
Correction of capillary leak (antibiotics, source control, …)
Colloids instead of cristalloids [111,112]
Dobutamine (not dopamine!) [113]
Dialysis or CVVH with ultrafiltration [114-116]
Ascorbinic acid in burn patients [117,118]
Specific therapeutic interventions
Continuous negative abdominal pressure (CNAP) [119,120]
Negative external abdominal pressure (NEXAP) [121-123]
Targeted APP [18,19,51]
(Experimental: Octreotide and melatonin in secondary ACS) [124,125]

- **Towel clip closure** is often used as an initial method of temporary abdominal closure after damage control surgery.
- **Removable prosthetic material**. Examples are the zippers, Wittman patch (which uses a Velcro closure system), etc.
- **Vacuum assisted fascial closure systems**.

Conclusions

First suggested in 1863 by Marey, ACS is a constellation of the physiologic sequelae of increased IAP, termed IAH. Recent observations suggest an increasing frequency of this complication in all types of patients. The presence of IAH and ACS are significant causes of organ failure, increased resource utilization, decreased economic productivity, and increased mortality among a wide variety of patient populations [23,78].

Despite its obvious clinical implications, attention is not paid to IAP, IAH and ACS. Only a few medical and surgical intensivists believe in the concept and actively attempt its prevention and treatment [5].

We must study and learn from the past and, at the same time, proactively "invent" the future. As aptly described by Dr. Ivatury and Sugerman [79], IAH/ACS is "…a clinical entity that had been ignored for far too long… …the mystery of IAH and ACS continues to unfold, transgressing the boundaries of acute and chronic illness and medical and surgical specialties." The future of IAH and ACS is in our hands and the results of recent multicenter studies confirm the importance of IAH and ACS on patient outcome [78,80,81]. It is time to pay attention, this was the title of a recent review [24] and the slogan of the Third World Congress on Abdominal Compartment Syndrome (WCACS2007) held in Antwerp, Belgium in 2007, March 22-24 (www.wcacs.org).

References

[1] Schein M. Abdominal compartment syndrome: historical background. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 1-7.

[2] Fietsam Jr R, Villalba M, Glover JL, Clark K. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg 1989;55:396-402.

[3] Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I Definitions Intensive Care Med 2006;32:1722-32.

[4] Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Abdominal compartment syndrome: it's time to pay attention! Intensive Care Med 2006;32:1912-4.

[5] Ivatury RR. Abdominal compartment syndrome: a century later, isn't it time to accept and promulgate? Crit Care Med 2006;34:2494-5.

[6] Kirkpatrick AW, Brenneman FD, McLean RF, et al. Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients? Can J Surg 2000;43:207-11.

[7] Sugrue M, Bauman A, Jones F, et al. Clinical examination is an inaccurate predictor of intraabdominal pressure. World J Surg 2002;26:1428-31.

[8] Malbrain ML. Different techniques to measure intra-abdominal pressure (IAP): time for a critical re-appraisal. Intensive Care Med 2004;30:357-71.

[9] Malbrain M, Jones F. Intra-abdominal pressure measurement techniques. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 19-68.

[10] Sanchez NC, Tenofsky PL, Dort JM, et al. What is normal intra-abdominal pressure? Am Surg 2001;67:243-8.

[11] Sugerman H, Windsor A, Bessos M, Wolfe L. Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med 1997;241:71-9.

[12] Sugerman HJ. Effects of increased intra-abdominal pressure in severe obesity. Surg Clin North Am 2001;81:1063-75 vi.

[13] Davis PJ, Koottayi S, Taylor A, Butt WW. Comparison of indirect methods of measuring intra-abdominal pressure in children. Intensive Care Med 2005;31:471-5.

[14] De Potter TJ, Dits H, Malbrain ML. Intra- and interobserver variability during in vitro validation of two novel methods for

intra-abdominal pressure monitoring. Intensive Care Med 2005;31:747–51.
[15] De Waele J, Pletinckx P, Blot S, Hoste E. Saline volume in transvesical intra-abdominal pressure measurement: enough is enough. Intensive Care Med 2006;32:455–9.
[16] Malbrain ML, Deeren DH. Effect of bladder volume on measured intravesical pressure: a prospective cohort study. Crit Care 2006;10:R98.
[17] Ball CG, Kirkpatrick AW. 'Progression towards the minimum': the importance of standardizing the priming volume during the indirect measurement of intra-abdominal pressures. Crit Care 2006;10:153.
[18] Cheatham ML, White MW, Sagraves SG, et al. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. J Trauma 2000;49:621–6 discussion 626-7.
[19] Malbrain ML. Abdominal perfusion pressure as a prognostic marker in intra-abdominal hypertension. In: Vincent JL, editor. Yearbook of Intensive Care and Emergency Medicine. Berlin: Springer-Verlag; 2002. p. 792–814.
[20] Deeren D, Dits H, Malbrain MLNG. Correlation between intra-abdominal and intracranial pressure in nontraumatic brain injury. Intensive Care Med 2005;31:1577–81.
[21] Cheatham M, Malbrain M. Abdominal perfusion pressure. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 69–81.
[22] Saggi B, Ivatury R, Sugerman HJ. Surgical critical care issues: Abdominal compartment syndrome. In: Holzheimer RG, Mannick JA, editors. Surgical Treatment. Evidence-Based and Problem-Oriented. München: W. Zuckschwerdt Verlag München; 2001.
[23] Malbrain ML. Is it wise not to think about intraabdominal hypertension in the ICU? Curr Opin Crit Care 2004;10:132–45.
[24] Malbrain ML, Deeren D, De Potter TJ. Intra-abdominal hypertension in the critically ill: it is time to pay attention. Curr Opin Crit Care 2005;11:156–71.
[25] Diebel LN, Wilson RF, Dulchavsky SA, Saxe J. Effect of increased intra-abdominal pressure on hepatic arterial, portal venous, and hepatic microcirculatory blood flow. J Trauma 1992;33:279–82 discussion 282-3.
[26] Wendon J, Biancofiore G, Auzinger G. Intra-abdominal hypertension and the liver. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 138–43.
[27] Biancofiore G, Bindi ML, Boldrini A, et al. Intraabdominal pressure in liver transplant recipients: incidence and clinical significance. Transplant Proc 2004;36:547–9.
[28] Biancofiore G, Bindi ML, Romanelli AM, et al. Intra-abdominal pressure monitoring in liver transplant recipients: a prospective study. Intensive Care Med 2003;29:30–6.
[29] Michelet P, Roch A, Gainnier M, et al. Influence of support on intra-abdominal pressure, hepatic kinetics of indocyanine green and extravascular lung water during prone positioning in patients with ARDS: a randomized crossover study. Crit Care 2005;9:R251–R257.
[30] Hering R, Vorwerk R, Wrigge H, et al. Prone positioning, systemic hemodynamics, hepatic indocyanine green kinetics, and gastric intramucosal energy balance in patients with acute lung injury. Intensive Care Med 2002;28:53–8.
[31] Biancofiore G, Bindi ML, Romanelli AM, et al. Postoperative intra-abdominal pressure and renal function after liver transplantation. Arch Surg 2003;138:703–6.
[32] Sugrue M, Hallal A, D'Amours S. Intra-abdominal pressure hypertension and the kidney. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 119–28.
[33] Sugrue M, Buist MD, Hourihan F, et al. Prospective study of intra-abdominal hypertension and renal function after laparotomy. Br J Surg 1995;82:235–8.
[34] Sugrue M, Jones F, Deane SA, et al. Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Arch Surg 1999;134:1082–5.
[35] Ivatury R, Diebel L. Intra-abdominal hypertension and the splanchnic bed. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 129–37.
[36] Diebel LN, Dulchavsky SA, Brown WJ. Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. J Trauma 1997;43:852–5.
[37] Diebel LN, Dulchavsky SA, Wilson RF. Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 1992;33:45–8 discussion 48-9.
[38] Balogh Z, Moore FA. Postinjury secondary abdominal compartment syndrome. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 170–7.
[39] Raeburn CD, Moore EE. Abdominal compartment syndrome provokes multiple organ failure: Animal and human supporting evidence. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 157–69.
[40] Sugrue M, Jones F, Lee A, et al. Intraabdominal pressure and gastric intramucosal pH: is there an association? World J Surg 1996;20:988–91.
[41] Ivatury RR, Porter JM, Simon RJ, et al. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma 1998;44:1016–21 discussion 1021-3.
[42] Balogh Z, McKinley BA, Cocanour CS, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Arch Surg 2003;138:637–42 discussion 642-3.
[43] Balogh Z, McKinley BA, Cox Jr CS, et al. Abdominal compartment syndrome: the cause or effect of postinjury multiple organ failure. Shock 2003;20:483–92.
[44] Moore FA. The role of the gastrointestinal tract in postinjury multiple organ failure. Am J Surg 1999;178:449–53.
[45] Eleftheriadis E, Kotzampassi K, Papanotas K, et al. Gut ischemia, oxidative stress, and bacterial translocation in elevated abdominal pressure in rats. World J Surg 1996;20:11–6.
[46] Diebel L, Saxe J, Dulchavsky S. Effect of intra-abdominal pressure on abdominal wall blood flow. Am Surg 1992;58: 573–5.
[47] Mutoh T, Lamm WJ, Embree LJ, et al. Volume infusion produces abdominal distension, lung compression, and chest wall stiffening in pigs. J Appl Physiol 1992;72:575–82.
[48] Kashtan J, Green JF, Parsons EQ, Holcroft JW. Hemodynamic effect of increased abdominal pressure. J Surg Res 1981;30: 249–55.
[49] Ridings PC, Bloomfield GL, Blocher CR, Sugerman HJ. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. J Trauma 1995;39:1071–5.
[50] Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 1976;20:401–4.
[51] Malbrain ML, Cheatham ML. Cardiovascular effects and optimal preload markers in intra-abdominal hypertension. In: Vincent J-L, editor. Yearbook of Intensive Care and Emergency Medicine. Berlin: Springer-Verlag; 2004. p. 519–43.
[52] Cheatham M, Malbrain M. Cardiovascular implications of elevated intra-abdominal pressure. In: Ivatury R, Cheatham M,

Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 89–104.
[53] Dellinger RP, Carlet JM, Masur H, et al. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. Intensive Care Med 2004;30:536–55.
[54] Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345:1368–77.
[55] Cheatham ML, Block EF, Nelson LD, Safcsak K. Superior predictor of the hemodynamic response to fluid challenge in critically ill patients. Chest 1998;114:1226–7.
[56] Cheatham ML, Nelson LD, Chang MC, Safcsak K. Right ventricular end-diastolic volume index as a predictor of preload status in patients on positive end-expiratory pressure. Crit Care Med 1998;26:1801–6.
[57] Schachtrupp A, Graf J, Tons C, et al. Intravascular volume depletion in a 24-hour porcine model of intra-abdominal hypertension. J Trauma 2003;55:734–40.
[58] Michard F, Alaya S, Zarka V, et al. Global end-diastolic volume as an indicator of cardiac preload in patients with septic shock. Chest 2003;124:1900–8.
[59] Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002; 121:2000–8.
[60] Simon RJ, Friedlander MH, Ivatury RR, et al. Hemorrhage lowers the threshold for intra-abdominal hypertension-induced pulmonary dysfunction. J Trauma 1997;42:398–403 discussion 404-5.
[61] Burchard KW, Ciombor DM, McLeod MK, et al. Positive end expiratory pressure with increased intra-abdominal pressure. Surg Gynecol Obstet 1985;161:313–8.
[62] Pelosi P, Ravagnan I, Giurati G, et al. Positive end-expiratory pressure improves respiratory function in obese but not in normal subjects during anesthesia and paralysis. Anesthesiology 1999;91:1221–31.
[63] Sugrue M, D'Amours S. The problems with positive end expiratory pressure (PEEP) in association with abdominal compartment syndrome (ACS). J Trauma 2001;51:419–20.
[64] Sussman AM, Boyd CR, Williams JS, DiBenedetto RJ. Effect of positive end-expiratory pressure on intra-abdominal pressure. South Med J 1991;84:697–700.
[65] Bloomfield GL, Ridings PC, Blocher CR, et al. A proposed relationship between increased intra-abdominal, intrathoracic, and intracranial pressure. Crit Care Med 1997;25:496–503.
[66] Mertens zur Borg IR, Verbrugge SJ, Olvera C. Pathophysiology: Respiratory. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 105–18.
[67] Ranieri VM, Brienza N, Santostasi S, et al. Impairment of lung and chest wall mechanics in patients with acute respiratory distress syndrome: role of abdominal distension. Am J Respir Crit Care Med 1997;156:1082–91.
[68] Gattinoni L, Pelosi P, Suter PM, et al. Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes? Am J Respir Crit Care Med 1998;158:3–11.
[69] Mutoh T, Lamm WJ, Embree LJ, et al. Abdominal distension alters regional pleural pressures and chest wall mechanics in pigs in vivo. J Appl Physiol 1991;70:2611–8.
[70] Quintel M, Pelosi P, Caironi P, et al. An increase of abdominal pressure increases pulmonary edema in oleic acid-induced lung injury. Am J Respir Crit Care Med 2004;169:534–41.
[71] De Keulenaer BL, De Backer A, Schepens DR, et al. Abdominal compartment syndrome related to noninvasive ventilation. Intensive Care Med 2003;29:1177–81.
[72] Hering R, Wrigge H, Vorwerk R, et al. The effects of prone positioning on intraabdominal pressure and cardiovascular and renal function in patients with acute lung injury. Anesth Analg 2001;92:1226–31.
[73] De Waele JJ, Benoit D, Hoste E, Colardyn F. A role for muscle relaxation in patients with abdominal compartment ysndrome? Intensive Care Med 2003;29:332.
[74] Mayberry JC. Prevention of abdominal compartment syndrome. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 221–9.
[75] Parr M, Olvera C. Medical management of abdominal compartment syndrome. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 230–7.
[76] Mertens zur Borg IR. Verbrugge SJ, Kolkman KA. Anesthetic considerations in abdominal compartment syndrome. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 252–63.
[77] Balogh Z, Moore FA, Goettler CE, et al. Management of abdominal compartment syndrome. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 264–94.
[78] Malbrain ML, Chiumello D, Pelosi P, et al. Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. Crit Care Med 2005;33:315–22.
[79] Ivatury RR, Sugerman HJ. Abdominal compartment syndrome: A century later, isn't it time to pay attention? Crit Care Med Crit Care Med 2000;28:2137–8.
[80] Malbrain ML, Critically Ill and Abdominal Hypertension (CIAH) Study Group. Incidence of intraabdominal hypertension in the intensive care unit. Crit Care Med 2005;33:2150–3.
[81] Malbrain ML, Chiumello D, Pelosi P, et al. Prevalence of intraabdominal hypertension in critically ill patients: a multicentre epidemiological study. Intensive Care Med 2004;30:822–9.
[82] Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. Surg Clin North Am 1996;76:833–42.
[83] Ivatury RR, Sugerman HJ, Peitzman AB. Abdominal compartment syndrome: recognition and management. Adv Surg 2001;35:251–69.
[84] Drummond GB, Duncan MK. Abdominal pressure during laparoscopy: effects of fentanyl. Br J Anaesth 2002;88:384–8.
[85] Macalino JU, Goldman RK, Mayberry JC. Medical management of abdominal compartment syndrome: Case report and a caution. Asian J Surg 2002;25:244–6.
[86] Kimball EJ, Mone M. Influence of neuromuscular blockade on intra-abdominal pressure. Crit Care Med 2005;33:A38.
[87] Kimball WR, Loring SH, Basta SJ, et al. Effects of paralysis with pancuronium on chest wall statics in awake humans. J Appl Physiol 1985;58:1638–45.
[88] Sugerman H, Windsor A, Bessos M, et al. Effects of surgically induced weight loss on urinary bladder pressure, sagittal abdominal diameter and obesity co-morbidity. Int J Obes Relat Metab Disord 1998;22:230–5.
[89] Voss M, Pinheiro J, Reynolds J, et al. Endoscopic components separation for abdominal compartment syndrome. Am J Surg 2003;186:158–63.
[90] Latenser BA, Kowal-Vern A, Kimball D, et al. A pilot study comparing percutaneous decompression with decompressive laparotomy for acute abdominal compartment syndrome in thermal injury. J Burn Care Rehabil 2002;23:190–5.
[91] Bauer JJ, Gelernt IM, Salky BA, Kreel I. Is routine postoperative nasogastric decompression really necessary? Ann Surg 1985;201:233–6.
[92] Cheatham ML, Chapman WC, Key SP, Sawyers JL. A meta-analysis of selective versus routine nasogastric decompression after elective laparotomy. Ann Surg 1995;221:469–76.

[93] Moss G, Friedman RC. Abdominal decompression: increased efficency by esophageal aspiration utilizing a new nasogastric tube. Am J Surg 1977;133:225–8.
[94] Savassi-Rocha PR, Conceicao SA, Ferreira JT, et al. Evaluation of the routine use of the nasogastric tube in digestive operation by a prospective controlled study. Surg Gynecol Obstet 1992;174:317–20.
[95] Wilmer A, Dits H, Malbrain ML, et al. Gastric emptying in the critically ill–the way forward. Intensive Care Med 1997;23: 928–9.
[96] Madl C, Druml W. Gastrointestinal disorders of the critically ill. Systemic consequences of ileus. BestPractResClinGastroenterol 2003;17:445–56.
[97] Malbrain ML. Abdominal pressure in the critically ill. Curr Opin Crit Care 2000;6:17–29.
[98] Ponec RJ, Saunders MD, Kimmey MB. Neostigmine for the treatment of acute colonic pseudo-obstruction. N Engl J Med 1999;341:137–41.
[99] Gorecki PJ, Kessler E, Schein M. Abdominal compartment syndrome from intractable constipation. J Am Coll Surg 2000;190:371.
[100] van der Spoel JI, Oudemans-van Straaten HM, Stoutenbeek CP, et al. Neostigmine resolves critical illness-related colonic ileus in intensive care patients with multiple organ failure–a prospective, double-blind, placebo-controlled trial. Intensive Care Med 2001;27:822–7.
[101] Sugrue M. Abdominal compartment syndrome. Curr Opin Crit Care 2005;11:333–8.
[102] Corcos AC, Sherman HF. Percutaneous treatment of secondary abdominal compartment syndrome. J Trauma 2001;51: 1062–4.
[103] Luca A, Feu F, Garcia-Pagan JC, et al. Favorable effects of total paracentesis on splanchnic hemodynamics in cirrhotic patients with tense ascites. Hepatology 1994;20:30–3.
[104] Cabrera J, Falcon L, Gorriz E, et al. Abdominal decompression plays a major role in early postparacentesis haemodynamic changes in cirrhotic patients with tense ascites. Gut 2001;48:384–9.
[105] Reckard JM, Chung MH, Varma MK, Zagorski SM. Management of intraabdominal hypertension by percutaneous catheter drainage. J Vasc Interv Radiol 2005;16:1019–21.
[106] Escorsell A, Gines A, Llach J, et al. Increasing intraabdominal pressure increases pressure, volume, and wall tension in esophageal varices. Hepatology 2002;36:936–40.
[107] Gotlieb WH, Feldman B, Feldman-Moran O, et al. Intraperitoneal pressures and clinical parameters of total paracentesis for palliation of symptomatic ascites in ovarian cancer. Gynecol Oncol 1998;71:381–5.
[108] Navarro-Rodriguez T, Hashimoto CL, Carrilho FJ, et al. Reduction of abdominal pressure in patients with ascites reduces gastroesophageal reflux. Dis Esophagus 2003;16:77–82.
[109] Friedlander MH, Simon RJ, Ivatury R, et al. Effect of hemorrhage on superior mesenteric artery flow during increased intra-abdominal pressures. J Trauma 1998;45:433–89.
[110] Gargiulo 3rd NJ, Simon RJ, Leon W, Machiedo GW. Hemorrhage exacerbates bacterial translocation at low levels of intra-abdominal pressure. Arch Surg 1998;133:1351–5.
[111] O'Mara MS, Slater H, Goldfarb IW, Caushaj PF. A prospective, randomized evaluation of intra-abdominal pressures with crystalloid and colloid resuscitation in burn patients. J Trauma 2005;58:1011–8.
[112] The SAFE Study Investigators. A Comparison of Albumin and Saline for Fluid Resuscitation in the Intensive Care Unit. N Engl J Med 2004;350:2247–56.
[113] Agusti M, Elizalde JI, Adalia R, et al. Dobutamine restores intestinal mucosal blood flow in a porcine model of intra-abdominal hyperpressure. Crit Care Med 2000;28:467–72.
[114] Oda S, Hirasawa H, Shiga H, et al. Management of intra-abdominal hypertension in patients with severe acute pancreatitis with continuous hemodiafiltration using a polymethyl methacrylate membrane hemofilter. Ther Apher Dial 2005;9:355–61.
[115] Kula R, Szturz P, Sklienka P, et al. A role for negative fluid balance in septic patients with abdominal compartment syndrome? Intensive Care Med 2004;30:2138–9.
[116] Vachharajani V, Scott LK, Grier L, Conrad S. Medical Management Of Severe Intra-abdominal Hypertension With Aggressive Diuresis And Continuous Ultra-filtration. The Internet Journal of Emergency and Intensive Care Medicine 2003;6.
[117] Matsuda T, Tanaka H, Williams S, et al. Reduced fluid volume requirement for resuscitation of third-degree burns with high-dose vitamin C. J Burn Care Rehabil 1991;12:525–32.
[118] Tanaka H, Matsuda T, Miyagantani Y, et al. Reduction of resuscitation fluid volumes in severely burned patients using ascorbic acid administration: a randomized, prospective study. Arch Surg 2000;135:326–31.
[119] Bloomfield G, Saggi B, Blocher C, Sugerman H. Physiologic effects of externally applied continuous negative abdominal pressure for intra-abdominal hypertension. J Trauma 1999; 46:1009–14 discussion 1014-6.
[120] Saggi BH, Bloomfield GL, Sugerman HJ, et al. Treatment of intracranial hypertension using nonsurgical abdominal decompression. J Trauma 1999;46:646–51.
[121] Valenza F, Irace M, Guglielmi M, et al. Effects of continuous negative extra-abdominal pressure on cardiorespiratory function during abdominal hypertension: an experimental study. Intensive Care Med 2005;31:105–11.
[122] Valenza F, Bottino N, Canavesi K, et al. Intra-abdominal pressure may be decreased non-invasively by continuous negative extra-abdominal pressure (NEXAP). Intensive Care Med 2003;29:2063–7.
[123] Valenza F, Gattinoni L. Continuous negative abdominal pressure. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience; 2006. p. 238–51.
[124] Kacmaz A, Polat A, User Y, et al. Octreotide improves reperfusion-induced oxidative injury in acute abdominal hypertension in rats. J Gastrointest Surg 2004;8:113–9.
[125] Sener G, Kacmaz A, User Y, et al. Melatonin ameliorates oxidative organ damage induced by acute intra-abdominal compartment syndrome in rats. J Pineal Res 2003;35:163–8.