Intensive Care Med (2007) 33:951–962
DOI 10.1007/s00134-007-0592-4

EXPERT PANEL

Michael L. Cheatham
Manu L. N. G. Malbrain
Andrew Kirkpatrick
Michael Sugrue
Michael Parr
Jan De Waele
Zsolt Balogh
Ari Leppäniemi
Claudia Olvera
Rao Ivatury
Scott D'Amours
Julia Wendon
Ken Hillman
Alexander Wilmer

Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations

Received: 9 April 2006
Accepted: 21 February 2007
Published online: 22 March 2007
© Springer-Verlag 2007

M. L. Cheatham
Orlando Regional Medical Center,
Department of Surgical Education,
86 West Underwood, Orlando 32806, FL,
USA

M. L. N. G. Malbrain (✉)
Campus Stuivenberg, Intensive Care Unit,
Ziekenhuis Netwerk Antwerpen,
Lange Beeldekensstraat 267, 2060
Antwerpen 6, Belgium
e-mail: manu.malbrain@skynet.be
Tel.: +32-3-2177399
Fax: +32-3-2177279

M. L. N. G. Malbrain · A. Wilmer
University Hospital Gasthuisberg, Intensive
Care Unit,
Herestraat 49, 3000 Leuven, Belgium

A. Kirkpatrick
Foothills Medical Centre, Departments of
Critical Care Medicine and Surgery,
29 St. NW, 1403 Calgary AB, T2N 2T9,
Canada

M. Sugrue · S. D'Amours
Liverpool Hospital, University of New
South Wales, Department of Trauma,
Sydney, NSW, Australia

M. Parr · K. Hillman
Liverpool Hospital, University of New
South Wales, Intensive Care Unit,
Sydney, NSW, Australia

J. De Waele
Universitair Ziekenhuis Gent, Surgical
Intensive Care Unit, Intensieve Zorgen
1K12-C,
De Pintelaan 185, 9000 Gent, Belgium

Z. Balogh
University of Newcastle, Department of
Trauma, Division of Surgery,
Newcastle, 2310, NSW, Australia

A. Leppäniemi
Meilahti Hospital, Department of
Emergency Surgery,
Haartmaninkatu, Finland

C. Olvera
American British Cowdray Medical Center,
Intensive Care Unit,
Mexico City, Mexico

R. Ivatury
Virginia Commonwealth University
Medical Center, Department of Surgery,
Trauma and Critical Care,
Richmond Va., USA

J. Wendon
King's College Hospital, Intensive Care
Unit,
London, UK

Abstract *Objective:* Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been increasingly recognized in the critically ill over the past decade. In the absence of consensus definitions and treatment guidelines the diagnosis and management of IAH and ACS remains variable from institution to institution. *Design:* An international consensus group of multidisciplinary critical care specialists convened at the second World Congress on Abdominal Compartment Syndrome to develop practice guidelines for the diagnosis, management, and prevention of IAH and ACS. *Methods:* Prior to the conference the authors developed a blueprint for consensus definitions and treatment guidelines which were refined both during and after the conference. The present article is the second installment of the final report from the 2004 International ACS Consensus Definitions Conference and is endorsed by the World Society of the Abdominal Compartment Syndrome. *Results:* The prevalence and etiological factors for IAH and ACS are reviewed. Evidence-based medicine treatment guidelines are

presented to facilitate the diagnosis and management of IAH and ACS. Recommendations to guide future studies are proposed. *Conclusions:* These definitions, guidelines, and recommendations, based upon current best evidence and expert opinion are proposed to assist clinicians in the management of IAH and ACS as well as serve as a reference for future clinical and basic science research.

Keywords Abdominal pressure · Abdominal hypertension · Abdominal compartment syndrome · Diagnosis · Management · Prevention · Guidelines

Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been increasingly recognized as causes of significant morbidity and mortality over the past decade [1–24]. Recognition of the widespread prevalence of elevated intra-abdominal pressure (IAP) in the critically ill, combined with advances in both the diagnosis and management of IAH and ACS, have resulted in significant improvements in patient survival from these evolving clinical syndromes during the same time period [1–4, 6, 8, 10, 13, 23–27].

IAP measurements are essential to the diagnosis and management of both IAH and ACS [1, 3, 14, 16, 17, 28–34]. Standardized techniques for IAP measurement as well as consensus definitions and treatment recommendations for IAH and ACS have been lacking, however. The World Society of the Abdominal Compartment Syndrome (WSACS, www.wsacs.org) has recently developed consensus definitions outlining standards for IAP measurement as well as diagnostic criteria for IAH and ACS based upon both the best available clinical evidence and expert opinion (Table 1). These definitions, if adopted in future studies of IAH and ACS, will promote improved comparison among trials as well as communication among clinicians worldwide. This second installment from the 2004 International ACS Consensus Definitions Conference proposes treatment guidelines for the diagnosis, management, and prevention of IAH and ACS as well as recommendations for future clinical investigation.

Table 1 Consensus definitions (*IAP* intra-abdominal pressure, *MAP* mean arterial pressure, *APP* abdominal perfusion pressure, *IAH* intra-abdominal hypertension, *ACS* abdominal compartment syndrome, *FG* filtration gradient, *GFP* glomerular filtration pressure, *PTP* proximal tubular pressure)

Definition 1	IAP is the 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 midaxillary line.
Definition 5	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml sterile saline.
Definition 6	Normal IAP is approx. 5–7 mmHg in critically ill adults.
Definition 7	IAH is defined by a sustained or repeated pathological elevation in IAP $\geq$ 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.

Methods

The rationale for and process by which the WSACS consensus definitions and recommendations were created has been described in detail in Part I [1]. During the preparation of Part II of this document, the writing committee has continually reviewed the most current literature and incorporated these data into the recommendations. These guidelines, in conjunction with the consensus definitions from Part I, serve as the final report of the 2004 International ACS Consensus Definitions Conference and are endorsed by the WSACS.

Evidence-based clinical practice guidelines are now commonplace in medicine. Such guidelines should assess not only the strength of the evidence but also the magnitude of net benefit vs. harm. Further, users need to know how much confidence they can place in the recommendations that expert guidelines offer. Numerous, sometimes confusing approaches for grading scientific evidence have been reported. To simplify the clinical application of these guidelines the WSACS has adopted a modification of the approach developed by the international GRADE group [35, 36]. This grading scheme classifies recommendations as either strong recommendations (grade 1) or weak suggestions (grade 2), according to the balance between the associated benefits and risks. The quality of evidence is further classified as high (grade A), moderate (grade B), or low (grade C)

according to the study design, consistency of results, and directness of the evidence (the similarity of the study patients to the population of interest). As a result a strong recommendation based upon high-quality evidence receives a grade of "1A" while a weak suggestion based upon low-quality evidence receives a grade of "2C". It is important to recognize that prospective, randomized clinical trials on IAH and ACS, the reference standard for evidence-based guideline development, are difficult to perform given the severity and acuity of these diseases and the need for informed consent. As a result the following recommendations are based largely upon nonrandomized prospective observational trials, retrospective database reviews, and even case reports, emphasizing the need for more rigorous clinical trials to be performed in the future. Where sufficient clinical evidence is available, evidentiary tables addressing pertinent management questions are provided.

Recommendations

Diagnosis

Risk factors for and surveillance of IAH/ACS

Originally thought to be a disease of the traumatically injured, IAH and ACS have now been recognized to occur in a wide variety of patient populations [3, 6, 11, 12, 21, 27–31]. The reported incidence and prevalence of IAH and ACS have varied significantly, however, largely due to the historical lack of consensus definitions and a common nomenclature. Table 2 depicts the current literature describing the incidence and/or prevalence of IAH and ACS. These data confirm the widespread and frequent development of both IAH and ACS among the critically ill with a significant associated risk of organ failure and increased mortality [2–4, 8, 10, 11, 13, 19–23, 26, 28, 29, 37–47].

Numerous risk factors for the development of IAH and/or ACS have been suggested. Three large-scale prospective trials have identified independent risk factors for the development of IAH/ACS. Malbrain et al. [3] identified that abdominal surgery, high-volume fluid resuscitation (> 3500 ml/24 h), ileus, and pulmonary, renal, or liver dysfunction predict IAH in a mixed medical-surgical population. Ivatury et al. [4, 6] identified that the severity of abdominal trauma, lactate level, and use of a temporary abdominal closure are predictors of survival among penetrating trauma patients with ACS. Balogh et al. [10] identified hypothermia, acidosis, anemia, oliguria, high crystalloid resuscitation volume, and high gastric regional minus end-tidal carbon dioxide tension as predictors of ACS in blunt thoracoabdominal trauma patients.

Given the broad range of potential etiological factors and the significant associated morbidity and mortality of IAH/ACS, a high index of suspicion and low threshold for IAP measurement appears appropriate in the patient possessing any of these risk factors. Fig. 1 depicts an algorithm for the initial evaluation of patients at risk for IAH. Based upon the above data we recommend that patients should be screened for IAH/ACS risk factors upon ICU admission and in the presence of new or progressive organ failure (grade 1B).

Intra-abdominal pressure measurement

The sensitivity of physical examination in detecting elevated IAP has been demonstrated in two separate prospective studies to be too low (40–60%) to be useful as a diagnostic tool [34, 47]. The diagnosis of IAH/ACS is therefore dependent upon the accurate and frequent measurement of IAP. IAP monitoring is a cost-effective,

Table 2 Should patients be routinely screened for IAH and ACS?

Studies	Design	Quality	Consistency	Directness	*n*	Findings
4 burn [19, 26, 37, 38]	3 prospective, 1 retrospective	No serious limitations	Some inconsistency [a]	Direct	1,067	IAH incidence 36–70%, ACS incidence 1–20%
1 liver transplant [29]	Prospective	No serious limitations	Only one study	Direct	108	IAH incidence 32%
3 abdominal surgery [13, 20, 39]	Prospective	No serious limitations	No important inconsistency	Direct	424	IAH incidence 33–41%
11 trauma [4, 8, 10, 11, 21–23, 34, 40–42]	6 prospective, 5 retrospective	No serious limitations	Some inconsistency [b]	Direct	3,070	IAH incidence 2–50%, ACS incidence 0.5–36%
2 medical/surgical [2, 3]	Prospective	No serious limitations	No important inconsistency	Direct	322	IAH prevalence 32–54%, ACS prevalence 4–8%
3 pancreatitis [28, 43, 44]	1 prospective, 2 retrospective	No serious limitations	Some inconsistency [a]	Direct	361	IAH incidence 30–78%, ACS incidence 36%
2 pediatric [45, 46]	Prospective	No serious limitations	No important inconsistency	Direct	2,814	ACS incidence 0.6–0.9%

[a] IAH/ACS definition variable
[b] intervention tresholds and management variable

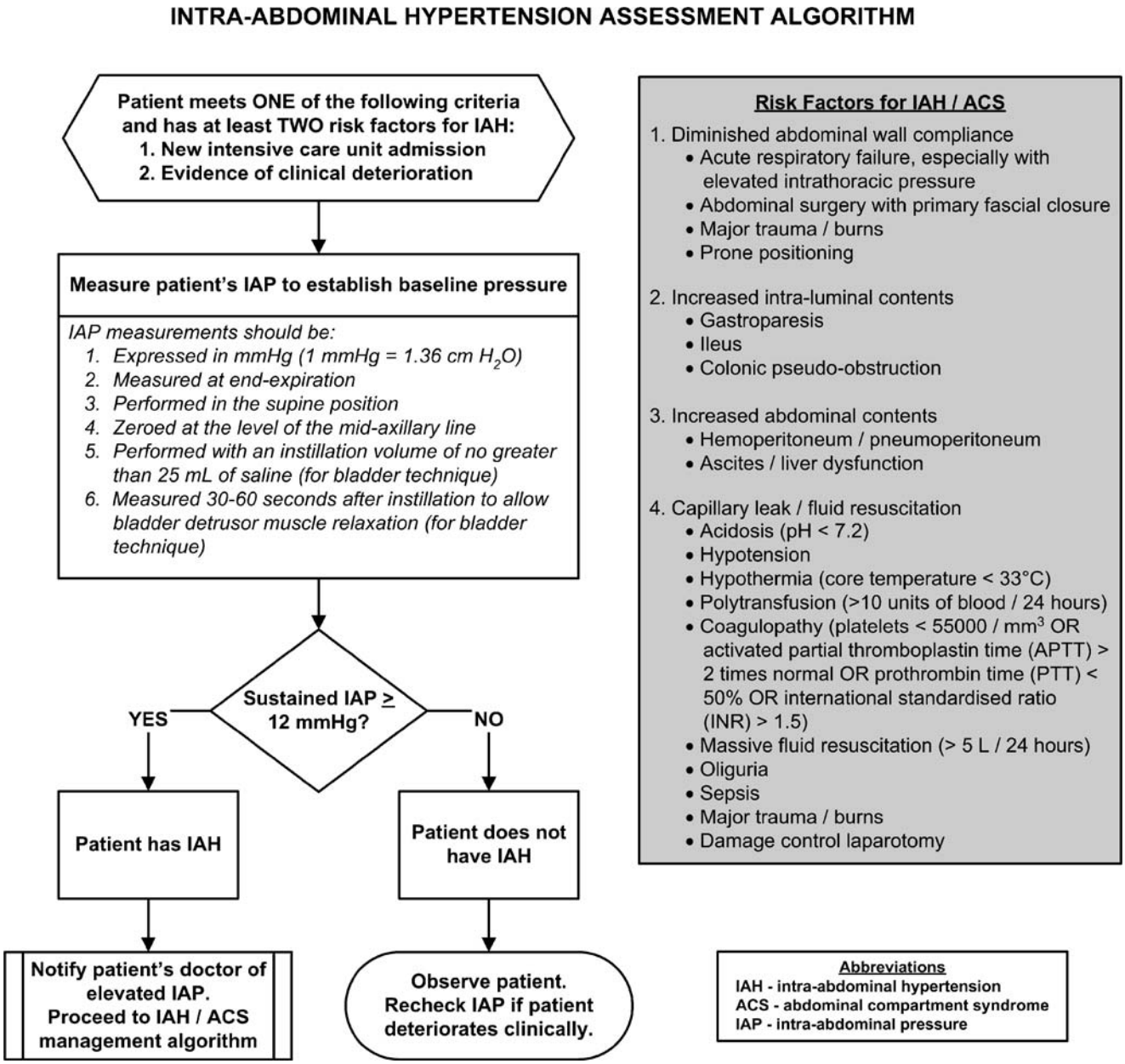

Fig. 1 Intra-abdominal hypertension assessment algorithm

safe, and accurate tool for identifying the presence of IAH and guiding resuscitative therapy for ACS [6, 14, 17, 48]. Given the favorable risk-benefit profile of IAP monitoring and the significant associated morbidity and mortality of IAH/ACS we recommend that (a) if two or more risk factors for IAH/ACS are present, a baseline IAP measurement should be obtained (grade 1B), and (b) if IAH is present, serial IAP measurements should be performed throughout the patient's critical illness (grade 1C).

The accuracy and reproducibility of IAP measurements are of paramount importance in the management of IAH/ACS and can directly impact upon both patient decision making and the interpretation of trials that include IAP as a clinical value. The optimal frequency of IAP monitoring, for example, has yet to be determined, as does the timing of measurements during the respiratory cycle. The intravesicular instillation volume, which varies widely from institution to institution, has been demonstrated to falsely elevate IAP when the high volumes originally advocated (50–100 ml) are used [33, 49, 50]. Patient head of bed elevation (addressed below) and the zero reference point utilized for IAP pressure transduction

(traditionally the symphysis pubis, but the midaxillary line at the iliac crest may be subject to less interpretation) also appear to significantly impact upon the variability in IAP measurements. Ongoing studies should shed significant light upon the optimal procedure for IAP measurements. To assist in ensuring the value and applicability of future clinical trials we suggest that studies should adopt the standardized IAP measurement method recommended by the consensus definitions OR provide sufficient detail of the technique utilized to allow accurate interpretation of the IAP data presented (grade 2C).

Management

The widely disparate patient populations that may develop IAH/ACS, in combination with a variety of underlying causal disease processes, make a standardized therapeutic approach to these diseases difficult. No one management strategy can be uniformly applied to every patient with IAH/ACS. Several fundamental management concepts, however, remain appropriate across all patient populations. While surgical decompression is commonly considered the only treatment for IAH/ACS, nonoperative medical management strategies are now recognized as playing a vital role in both the prevention and treatment of organ dysfunction and failure due to elevated IAP [7, 9, 10, 51].

Appropriate treatment and management of IAH and/or ACS is based upon four general principles: (a) serial monitoring of IAP, (b) optimization of systemic perfusion and organ function in the patient with elevated IAP, (c) institution of specific medical procedures to reduce IAP and the end-organ consequences of IAH/ACS, and (d) prompt surgical decompression for refractory IAH. An algorithm for the management of the patient with IAH/ACS is proposed in Fig. 2.

Abdominal perfusion pressure

The "critical IAP" that causes end-organ dysfunction varies from patient to patient as a result of differences in physiology and preexisting comorbidities. Thus a single threshold value of IAP cannot be globally applied to the decision making of all critically ill patients. Analogous to the widely accepted and utilized concept of cerebral perfusion pressure, calculation of the "abdominal perfusion pressure" (APP), defined as mean arterial pressure (MAP) minus IAP, assesses not only the severity of IAP present but also the relative adequacy of abdominal blood flow. APP has been studied as a resuscitation endpoint in four clinical trials [25, 27, 52]. Each of these studies demonstrated significant differences in APP between survivors and nonsurvivors with IAH/ACS. Cheatham et al. [25] in a retrospective trial of surgical/trauma patients with IAH (mean IAP 22 ± 8 mmHg) concluded that an APP value of ≥ 50 mmHg or higher optimized survival. APP was also found to be superior to arterial pH, base deficit, arterial lactate, and hourly urinary output in its ability to predict patient outcome. Malbrain et al. [27] and Cheatham et al. [52] in three subsequent trials in mixed medical-surgical patients (mean IAP 10 ± 4 mmHg) suggested that an APP value of ≥ 60 mmHg or higher represented an appropriate resuscitation goal. Persistence of IAH and failure to maintain APP at or above ≥ 60 mmHg by day 3 was found to discriminate between survivors and nonsurvivors. The apparent disparity in these APP endpoints may be related to differences in IAH severity and patient population between the trials.

APP as a resuscitation endpoint has yet to be subjected to a prospective, randomized clinical trial. Further, the therapeutic threshold above which raising MAP to achieve a particular APP becomes futile or even detrimental remains unknown. Indiscriminate fluid administration places the patient at risk for secondary ACS and should be avoided [7, 9, 10, 51, 53]. Target APP values may be achieved through a balance of judicious fluid resuscitation and application of vasoactive medications. Notwithstanding these concerns, maintaning APP at 50–60 mmHg appears to predict improved survival from IAH/ACS that is not identified by IAP alone. Given the significant benefit and limited risk, we recommend that APP should be maintained above 50–60 mmHg in patients with IAH/ACS (grade 1C).

Sedation and analgesia

Pain, agitation, ventilator dyssynchrony, and use of accessory muscles during work of breathing may all lead to increased thoracoabdominal muscle tone. This increased muscle activity can lead to increases in IAP [3, 6]. Patient sedation and analgesia can reduce muscle tone and potentially decrease IAP to less detrimental levels, although fentanyl has been reported to potentially increase abdominal muscle tone and IAP [54]. In addition to ensuring patient comfort, adequate sedation and analgesia would therefore appear to be prudent in the patient with evidence of IAH [55]. As no prospective trials have yet been performed evaluating the benefits and risks of sedation and analgesia in IAH/ACS, no recommendations can be made at this time.

Neuromuscular blockade

Diminished abdominal wall compliance due to pain, tight abdominal closures, and third-space fluid can increase IAP to potentially detrimental levels [3, 56]. NMB has been suggested in several reports to be an effective method for reducing IAP [55, 57, 58]. While NMB may well reverse the negative effects of mild to moderate IAH, it is unlikely

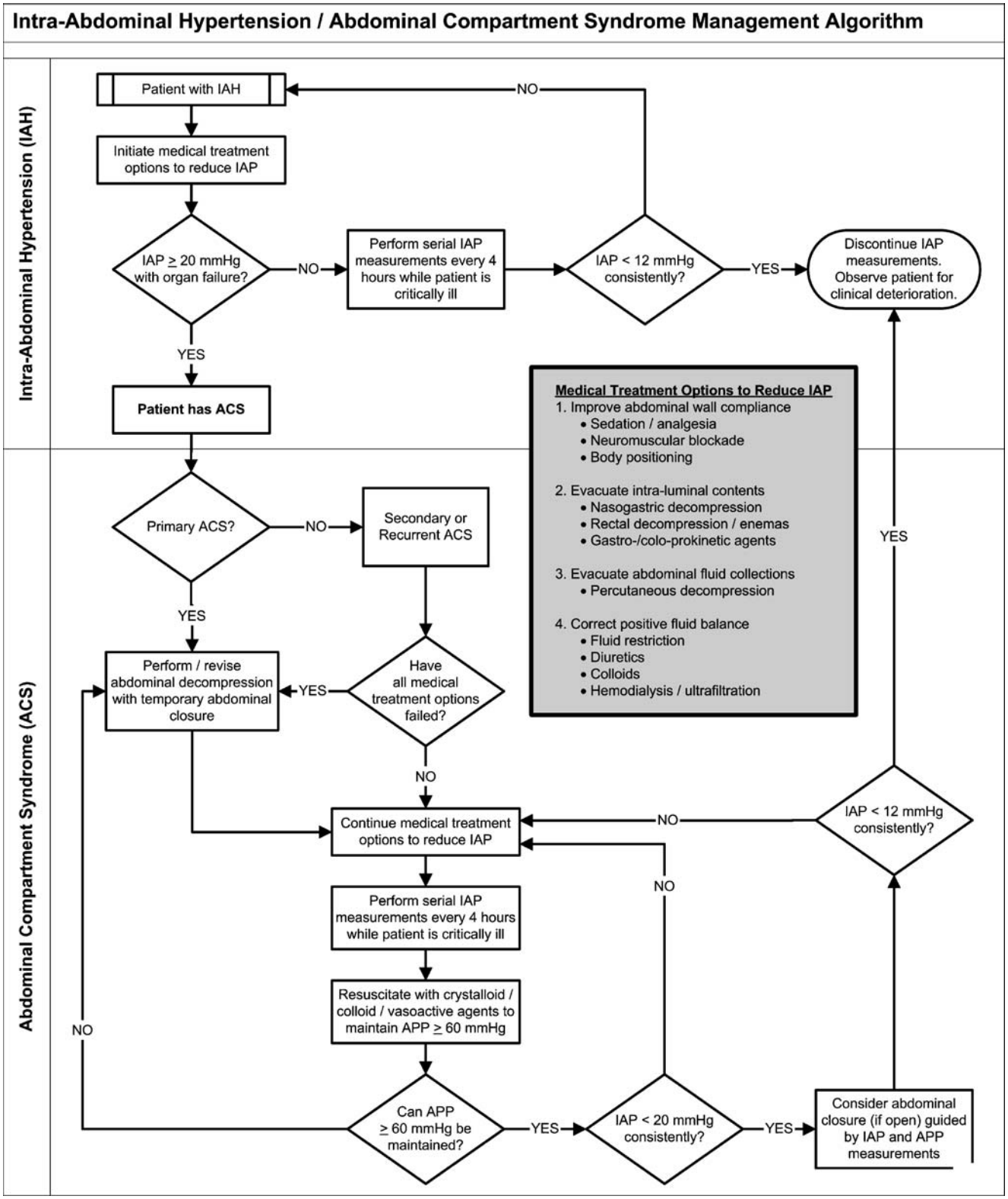

Fig. 2 Intra-abdominal hypertension/intra-abdominal compartment syndrome management algorithm

to be an effective therapy for patients with severe IAH or those who have progressed to ACS [59]. De Waele et al. [57] recently performed the first prospective trial of NMB in the management of IAH, demonstrating significant reductions in IAP in nine of ten patients following a single bolus dose of cisatracurium. The single patient who did not respond had a baseline IAP of 25 mmHg (grade IV IAH). The potential beneficial effects of NMB in reducing abdominal muscle tone, however, must be balanced against the risks of prolonged paralysis. Although such data are preliminary, we suggest that a brief trial of neuromuscular blockade may be considered in selected patients with mild to moderate IAH while other interventions are performed to reduce IAP (grade 2C).

Body positioning

IAP monitoring is traditionally performed in the supine position. Recently elevation of the patient's head has been emphasized to reduce the incidence of aspiration pneumonia [60, 61]. Such a change in body position, however, can increase measured IAP. Two as yet unpublished, prospective studies, including a large scale multicenter trial sponsored by the WSACS, have evaluated the impact of body positioning on IAP measurements, arriving at similar conclusions. Head of bed elevation significantly increases IAP compared to supine positioning, especially at higher levels of IAH. Such increases in IAP become clinically significant (increase ≥ 2 mmHg) when the patient's head of bed exceeds 20° elevation, well below that currently practiced in many intensive care units. As a result supine IAP measurements may underestimate the true IAP if the patient's head of bed is being elevated between measurements. Prone positioning for acute lung injury has also been demonstrated to significantly increase IAP [62, 63, 64]. Further research is necessary to fully characterize the impact of body position on IAP. In the interim, we suggest that the potential contribution of body position in elevating IAP should be considered in patients with moderate to severe IAH or ACS (grade 2C).

Nasogastric/colonic decompression, prokinetic motility agents

Gastrointestinal ileus is common among patients who have had abdominal surgery, peritonitis, major trauma, significant fluid resuscitation, or electrolyte abnormalities, many of which are independent risk factors for IAH/ACS [3, 4, 10]. Both air and fluid within the hollow viscera can raise IAP and lead to IAH/ACS. Nasogastric and/or rectal drainage, enemas, and even endoscopic decompression would appear to be simple and relatively noninvasive methods for reducing IAP and treating mild to moderate IAH [54]. The administration of prokinetic motility agents such as erythromycin, metoclopromide, or neostigmine would also appear to hold promise in evacuating the intraluminal contents and decreasing the size of the viscera [17, 65–69]. To date, however, no prospective trials have been performed to confirm the benefit of such therapies and no recommendations can be made at this time.

Fluid resuscitation

Fluid resuscitation to correct hypovolemia and avoid organ failure remains a cornerstone of critical care management. The concept of "early goal-directed therapy", originally described for the treatment of severe sepsis, also applies to the patient with IAH/ACS. Adequate intravascular volume is especially important in IAH/ACS as the combination of hypovolemia and intrathoracic pressure due to mechanical ventilation appears to aggravate the pathophysiological effects of elevated IAP [58, 70, 71]. Excessive fluid resuscitation, an independent predictor of both IAH and ACS, should be avoided, however, and represents a major etiology for secondary ACS, where fluid resuscitation actually worsens survival [8–10, 53].

Fluid resuscitation remains a highly controversial subject and a complete discussion is beyond the scope of this document [72]. The current literature addressing fluid resuscitation in patients at risk for IAH/ACS will be briefly discussed (Table 3). Balogh et al. [8] retrospectively evaluated two trauma resuscitation strategies in which patients were fluid resuscitated to achieve an oxygen delivery index of either 500 or 600 ml min^{-1} m^{-2}. Patients resuscitated using the "supranormal" endpoint required significantly more fluid and were twice as likely to demonstrate IAH, ACS, organ failure, and death than those under the more restrictive fluid resuscitation strategy. McNelis et al. [12] performed a retrospective matched case-control study of nontrauma surgical patients with and without ACS. In multivariate analysis 24-h fluid balance was an independent predictor for development of ACS. Oda et al. [73] retrospectively reviewed burn patients (> 40% total body surface area) who received either isotonic or hypertonic crystalloid resuscitation. Hypertonic resuscitation was associated with a significantly decreased fluid requirement and lower peak inspiratory pressures, and significantly higher APP levels. Further, isotonic resuscitation was associated with a 3.5-fold increased risk for developing IAH (defined as IAP > 30 cmH_2O). Oda et al. concluded that the lower fluid load afforded by hypertonic crystalloid resuscitation could reduce the risk of secondary ACS. O'Mara et al. [31] performed the only prospective, randomized controlled trial evaluating fluid resuscitation in patients with IAH/ACS. Thirty-one burn patients were randomized to receive either crystalloid or colloid-based resuscitation. The crystalloid patients required significantly greater volumes of fluid to maintain urinary output

Table 3 Should resuscitation fluid volume be limited in patients at risk for IAH/ACS? (*OR* odds ratio associated with aggressive fluid resuscitation)

Studies	Design	Quality	Consistency	Directness	*n*	Findings
1 medical/surgical [3]	Prospective	No serious limitations	Only one study	Direct	265	IAH OR 1.9
1 trauma [8]	Retrospective	No serious limitations	Only one study	Direct	156	IAH OR 1.8, ACS OR 1.7, MOF OR 2.0, mortality OR 2.1
1 surgical [12]	Retrospective	No serious limitations	Only one study	Direct	44	24-h fluid balance, independently predicts ACS
1 burn [73]	Retrospective	Some limitations [a]	Only one study	Direct	36	Mortality OR 3.5 for isotonic vs. hypertonic resuscitation
1 burn [31]	Prospective, randomized	No serious limitations	Only one study	Direct	31	IAP significantly lower with colloid resuscitation; no difference in survival

[a] Probable selection bias

(0.56 ± 0.16 vs. 0.36 ± 0.17 l/kg) and developed significantly higher peak IAP (33 ± 10 vs. 16 ± 8 mmHg). Based upon these studies we recommend that (a) fluid resuscitation volume should be carefully monitored to avoid overresuscitation in patients at risk for IAH/ACS (grade 1B), and (b) hypertonic crystalloid and colloid-based resuscitation should be considered in patients with IAH to decrease the progression to secondary ACS (grade 1C).

Diuretics and continuous venovenous hemofiltration/ultrafiltration

For IAH patients who develop oliguria or anuria despite resuscitation early institution of renal replacement therapy has been reported with fluid removal by intermittent dialysis or continuous hemofiltration/ultrafiltration [54, 74, 75]. This may be an appropriate intervention rather than continuing to volume load and increase the likelihood of secondary ACS. Diuretic therapy, in combination with colloid, may also be considered to mobilize the third-space edema once the patient is hemodynamically stable. These therapies have not been subjected to clinical study in IAH/ACS patients, however, and no recommendations can be made regarding their use.

Percutaneous catheter decompression

Given the morbidity of open abdominal decompression, less invasive means of reducing IAP would certainly be appealing. Percutaneous catheter decompression represents a less invasive method for treating IAH or secondary ACS due to free intra-abdominal fluid, air, abscess, or blood. As illustrated in Table 4, this technique appears to be effective in reducing IAP and potentially correcting IAH-induced organ dysfunction [38, 76–83]. Commonly performed under ultrasound or computed tomography guidance, percutaneous decompression, in appropriate patients, appears to be effective in resolving IAH/ACS and avoiding the need for surgical decompression. Latenser et al. evaluated this technique in burn patients ($> 40\%$ total body surface area) with five of nine patients who developed IAH (defined as IAP ≥ 25 mmHg) being successfully treated with percutaneous decompression while four progressed to ACS requiring decompressive laparotomy [38]. Inhalational injury and burn area of more than 80% total body surface area appeared to predict failure of percutaneous decompression. Gotlieb et al. [78] performed percutaneous drainage of symptomatic ovarian ascites in women with grade III or IV IAH. IAP was significantly reduced in 33 of 35 patients with improvements in both

Table 4 Should percutaneous catheter decompression be performed for IAH/ACS?

Studies	Design	Quality	Consistency	Directness	*n*	Findings
1 pediatric trauma [79]	Retrospective	No serious limitations	Only one study	Direct	2	Successful treatment of ACS
3 medical [77, 82, 83]	Retrospective	No serious limitations	No important inconsistency	Direct	3	Successful treatment of ACS
3 burn [38, 76, 80]	Retrospective	No serious limitations	No important inconsistency	Direct	12	Successful treatment of ACS in 60% of patients
1 trauma [81]	Retrospective	No serious limitations	Only one study	Direct	1	Successful treatment of ACS
1 oncology [78]	Prospective	No serious limitations	Only one study	Direct	35	Significant decrease in IAP in 33

hemodynamic and pulmonary function. In light of the potential benefits of avoiding abdominal decompression we suggest that percutaneous catheter decompression should be considered in patients with intraperitoneal fluid, abscess, or blood who demonstrate symptomatic IAH or ACS (grade 2C).

Abdominal decompression

Surgical abdominal decompression has long been the standard treatment for the patient who develops ACS. It represents a life-saving intervention when a patient's IAH has become refractory to medical treatment options and organ dysfunction and/or failure is evident [4, 23–25, 84]. Delays in surgical decompression and disregard for high IAP levels is associated with significant increases in patient mortality [25]. Presumptive decompression or "leaving the abdomen open" in surgical patients at risk who are undergoing laparotomy has been demonstrated to significantly reduce the subsequent development of IAH/ACS and improve survival [25]. While seemingly aggressive and disabling, Cheatham et al. [24] have demonstrated excellent long-term physical and mental health function as well as ability to resume gainful employment among patients who require surgical decompression.

Surgical decompression results in an "open abdomen", which must be covered by some form of protective dressing or "temporary abdominal closure" (TAC). A variety of equivalent TAC techniques have been described, including towel clips, "vacuum-pack closure", "Bogota bag", Wittmann patch, and vacuum-assisted closure [5, 6, 85–90]. None of these techniques has been subjected to a comparative prospective evaluation to prove efficacy over the others [84]. It is essential to recognize that recurrent ACS is possible with any of the TAC techniques, especially if they are applied in a fashion that does not allow continued visceral expansion during resuscitation [5, 18]. If recurrent ACS develops, the TAC should immediately be removed and reapplied so as to reduce IAP to an acceptable level.

Based upon the above literature and the significant morbidity and mortality of untreated ACS we recommend that (a) surgical decompression should be performed in patients with ACS that is refractory to other treatment options (grade 1B), and (b) presumptive decompression should be considered at the time of laparotomy in patients who demonstrate multiple risk factors for IAH/ACS (grade 1C).

Definitive abdominal closure

Following surgical decompression and resolution of the patient's ACS the next therapeutic goal should be definitive closure of the patient's abdomen. Most patients, if decompressed early prior to development of significant organ failure, tolerate primary fascial closure within 5–7 days. Most patients who remain critically ill past this time period with significant loss of abdominal domain require either split-thickness skin grafting of the exposed viscera, with subsequent fascial closure 9–12 months later, or cutaneous advancement flap ("skin-only") closure, which allows earlier fascial closure [25, 84]. Various observational studies have been performed reporting the success of various techniques for long-term management of the open abdomen [84]. Prospective trials to identify the optimal method for such management, however, have yet to be performed. As a result, recommendations for definitive abdominal closure cannot be made at this time.

Future Investigation

The consensus definitions proposed in Part I and the evidence-based guidelines advocated in Part II are presented as a means to establish a foundation upon which future clinical trials may be performed. The use of such definitions and guidelines should facilitate the interpretation and comparison of one study against another so that clinically useful decisions may be made regarding the optimal treatment strategy for the patient with IAH/ACS. While multiple areas for future research have been identified in the above discussion, several additional suggestions are appropriate.

As illustrated in Table 2, the reported incidence and prevalence of IAH and ACS have varied widely from one study to the next, largely due to the prior lack of consensus definitions. To facilitate determination of accurate incidence and prevalence estimates and assist in the comparison of future trials, we recommend that incidence and prevalence estimates of IAH/ACS should be based upon the consensus definitions (grade 1C).

The frequency of IAP monitoring may affect mean and maximal daily IAP levels as well as the incidence and prevalence of IAH when different thresholds are utilized. Further, the time spent above a critical IAP threshold during each 24-h period may be of greater clinical significance than isolated maximal IAP values. To facilitate communication of the severity of IAH in future trials, we suggest that mean, median, and maximal IAP values should be provided both on admission and during the study period (grade 2C).

Summary

Significant progress has been made over the past decade towards understanding the etiology and pathophysiology surrounding IAH and ACS. The absence of consensus definitions and treatment guidelines, however, has led to confusion over both the prevalence of IAH and ACS as

well as the most effective treatment strategies for such patients. The 2004 International ACS Consensus Definitions Conference committee has proposed the consensus definitions outlined in Part I and the evidence-based management guidelines in Part II to serve as a basis for future consensus. Both of these documents highlight the significant need for well-designed, prospective clinical trials to clarify the many questions and issues that remain unanswered with respect to IAH and ACS. The authors anticipate that these definitions and recommendations will be dynamic and will change as new research is published.

References

1. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppaniemi A, Olvera C, Ivatury R, D'Amours S, Wendon J, Hillman K, Johansson K, Kolkman K, Wilmer A (2006) Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med 32:1722–1732
2. Malbrain ML, Chiumello D, Pelosi P, Wilmer A, Brienza N, Malcangi V, Bihari D, Innes R, Cohen J, Singer P, Japiassu A, Kurtop E, De Keulenaer BL, Daelemans R, Del Turco M, Cosimini P, Ranieri M, Jacquet L, Laterre PF, Gattinoni L (2004) Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. Intensive Care Med 30:822–829
3. Malbrain ML, Chiumello D, Pelosi P, Bihari D, Innes R, Ranieri VM, Del Turco M, Wilmer A, Brienza N, Malcangi V, Cohen J, Japiassu A, De Keulenaer BL, Daelemans R, Jacquet L, Laterre PF, Frank G, de Souza P, Cesana B, Gattinoni L (2005) Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. Crit Care Med 33:315–322
4. Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM (1998) Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma 44:1016–1021
5. Cheatham ML (1999) Intra-abdominal hypertension and abdominal compartment syndrome. New Horiz 7:96–115
6. Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (2006) Abdominal compartment syndrome. Landes Biosciences, Georgetown
7. Biffl WL, Moore EE, Burch JM, Offner PJ, Franciose RJ, Johnson JL (2001) Secondary abdominal compartment syndrome is a highly lethal event. Am J Surg 182:645–648
8. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Valdivia A, Sailors RM, Moore FA (2003) Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Arch Surg 138:637–642
9. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Holcomb JB, Ware DN, Moore FA (2002) Secondary abdominal compartment syndrome is an elusive early complication of traumatic shock resuscitation. Am J Surg 184:538–543
10. Balogh Z, McKinley BA, Holcomb JB, Miller CC, Cocanour CS, Kozar RA, Valdivia A, Ware DN, Moore FA (2003) Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma 54:848–859
11. Ertel W, Oberholzer A, Platz A, Stocker R, Trentz O (2000) Incidence and clinical pattern of the abdominal compartment syndrome after "damage-control" laparotomy in 311 patients with severe abdominal and/or pelvic trauma. Crit Care Med 28:1747–1753
12. McNelis J, Marini CP, Jurkiewicz A, Fields S, Caplin D, Stein D, Ritter G, Nathan I, Simms HH (2002) Predictive factors associated with the development of abdominal compartment syndrome in the surgical intensive care unit. Arch Surg 137:133–136
13. Sugrue M, Buist MD, Hourihan F, Deane S, Bauman A, Hillman K (1995) Prospective study of intra-abdominal hypertension and renal function after laparotomy. Br J Surg 82:235–238
14. Malbrain ML (1999) Abdominal pressure in the critically ill: measurement and clinical relevance. Intensive Care Med 25:1453–1458
15. Ivatury RR, Sugerman HJ (2000) Abdominal compartment syndrome: a century later, isn't it time to pay attention? Crit Care Med 28:2137–2138
16. Sugrue M (2002) Intra-abdominal pressure: time for clinical practice guidelines? Intensive Care Med 28:389–391
17. Malbrain ML (2000) Abdominal pressure in the critically ill. Curr Opin Crit Care 6:17–29
18. Gracias VH, Braslow B, Johnson J, Pryor J, Gupta R, Reilly P, Schwab CW (2002) Abdominal compartment syndrome in the open abdomen. Arch Surg 137:1298–1300
19. Ivy ME, Possenti PP, Kepros J, Atweh NA, D'Aiuto M, Palmer J, Pineau M, Burns GA, Caushaj PF (1999) Abdominal compartment syndrome in patients with burns. J Burn Care Rehabil 20:351–353
20. Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K (1999) Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Arch Surg 134:1082–1085
21. Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, Burch JM (1997) Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 174:667–672
22. Maxwell RA, Fabian TC, Croce MA, Davis KA (1999) Secondary abdominal compartment syndrome: an underappreciated manifestation of severe hemorrhagic shock. J Trauma 47:995–999
23. Hong JJ, Cohn SM, Perez JM, Dolich MO, Brown M, McKenney MG (2002) Prospective study of the incidence and outcome of intra-abdominal hypertension and the abdominal compartment syndrome. Br J Surg 89:591–596
24. Cheatham ML, Safcsak K, Llerena LE, Morrow CE, Block EFJ (2004) Long-term physical, mental, and functional consequences of abdominal decompression. J Trauma 56:237–242
25. Cheatham ML, White MW, Sagraves SG, Johnson JL, Block EF (2000) Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. J Trauma 49:621–626
26. Hobson KG, Young KM, Ciraulo A, Palmieri TL, Greenhalgh DG (2002) Release of abdominal compartment syndrome improves survival in patients with burn injury. J Trauma 53:1129–1133

27. Malbrain ML (2002) Abdominal perfusion pressure as a prognostic marker in intra-abdominal hypertension. In: Vincent JL (ed) Yearbook of intensive care and emergency medicine. Springer, Berlin Heidelberg New York, pp 792–814
28. Pupelis G, Austrums E, Snippe K, Berzins M (2002) Clinical significance of increased intraabdominal pressure in severe acute pancreatitis. Acta Chir Belg 102:71–74
29. Biancofiore G, Bindi ML, Boldrini A, Consani G, Bisa M, Esposito M, Urbani L, Catalano G, Filipponi F, Mosca F (2003) Intra-abdominal pressure monitoring in liver transplant recipients: a prospective study. Intensive Care Med 29:30–36
30. Efstathiou E, Zaka M, Farmakis M (2005) Intra-abdominal pressure monitoring in septic patients. Intensive Care Med 31:S183
31. O'Mara MS, Slater H, Goldfarb IW, Caushaj PF (2005) A prospective, randomized evaluation of intra-abdominal pressures with crystalloid and colloid resuscitation in burn patients. J Trauma 58:1011–1018
32. Malbrain ML (2004) Different techniques to measure intra-abdominal pressure (IAP): time for a critical re-appraisal. Intensive Care Med 30:357–371
33. De Waele J, Pletinckx P, Blot S, Hoste E (2006) Saline volume in transvesical intra-abdominal pressure measurement: enough is enough. Intensive Care Med 32:455–459
34. Kirkpatrick AW, Brenneman FD, McLean RF, Rapanos T, Boulanger BR (2000) Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients? Can J Surg 43:207–211
35. Guyatt G, Gutterman D, Baumann MH, Addrizzo-Harris D, Hylek EM, Phillips B, Raskob G, Lewis SZ, Schunemann H (2006) Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians Task Force. Chest 129:174–181
36. Schunemann HJ, Jaeschke R, Cook DJ, Bria WF, El-Solh AA, Ernst A, Fahy BF, Gould MK, Horan KL, Krishnan JA, Manthous CA, Maurer JR, McNicholas WT, Oxman AD, Rubenfeld G, Turino GM, Guyatt G, ATS Documents Development, Implementation Committee (2006) An official ATS statement: grading the quality of evidence and strength of recommendations in ATS guidelines and recommendations. Am J Respir Crit Care Med 174:605–614
37. Greenhalgh DG, Warden GD (1994) The importance of intra-abdominal pressure measurements in burned children. J Trauma 36:685–690
38. Latenser BA, Kowal-Vern A, Kimball D, Chakrin A, Dujovny N (2002) A pilot study comparing percutaneous decompression with decompressive laparotomy for acute abdominal compartment syndrome in thermal injury. J Burn Care Rehabil 23:190–195
39. Sugrue M, Jones F, Lee A, Buist MD, Deane S, Bauman A, Hillman K (1996) Intraabdominal pressure and gastric intramucosal pH: is there an association? World J Surg 20:988–991
40. Morris JA Jr, Eddy VA, Blinman TA, Rutherford EJ, Sharp KW (1993) The staged celiotomy for trauma. Issues in unpacking and reconstruction. Ann Surg 217:576–584
41. Offner PJ, de Souza AL, Moore EE, Biffl WL, Franciose RJ, Johnson JL, Burch JM (2001) Avoidance of abdominal compartment syndrome in damage-control laparotomy after trauma. Arch Surg 136:676–681
42. Raeburn CD, Moore EE, Biffl WL, Johnson JL, Meldrum DR, Offner PJ, Franciose RJ, Burch JM (2001) The abdominal compartment syndrome is a morbid complication of postinjury damage control surgery. Am J Surg 182:542–546
43. De Waele JJ, Hoste E, Blot SI, Decruyenaere J, Colardyn F (2005) Intra-abdominal hypertension in patients with severe acute pancreatitis. Crit Care 9:R452–457
44. Tao HQ, Zhang JX, Zou SC (2004) Clinical characteristics and management of patients with early acute severe pancreatitis: experience from a medical center in China. World J Gastroenterol 10:919–921
45. Beck R, Halberthal M, Zonis Z, Shoshani G, Hayari L, Bar-Joseph G (2001) Abdominal compartment syndrome in children. Pediatr Crit Care Med 2:51–56
46. Diaz FJ, Fernandez SA, Gotay F (2006) Identification and management of abdominal compartment syndrome in the pediatric intensive care unit. P R Health Sci J 25:17–22
47. Sugrue M, Bauman A, Jones F, Bishop G, Flabouris A, Parr M, Stewart A, Hillman K, Deane SA (2002) Clinical examination is an inaccurate predictor of intraabdominal pressure. World J Surg 26:1428–1431
48. Malbrain MLNG, Jones F (2006) Intra-abdominal pressure measurement techniques. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (eds) Abdominal compartment syndrome. Landes Biomedical, Georgetown, pp 19–68
49. Fusco MA, Martin RS, Chang MC (2001) Estimation of intra-abdominal pressure by bladder pressure measurement: validity and methodology. J Trauma 50:297–302
50. Malbrain ML, Deeren DH (2006) Effect of bladder volume on measured intravesical pressure: a prospective cohort study. Crit Care 10:R98
51. Kirkpatrick AW, Balogh Z, Ball CG, Ahmed N, Chun R, McBeth P, Kirby A, Zygun DA (2006) The secondary abdominal compartment syndrome: iatrogenic or unavoidable? J Am Coll Surg 202:668–679
52. Cheatham ML, Malbrain MLNG (2006) Abdominal perfusion pressure. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (eds) Abdominal compartment syndrome. Landes Biomedical, Georgetown, pp 69–81
53. Balogh Z, Moore FA (2006) Postinjury secondary abdominal compartment syndrome. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (eds) Abdominal compartment syndrome. Landes Biomedical, Georgetown, pp 170–177
54. Drummond GB, Duncan MK (2002) Abdominal pressure during laparoscopy: effects of fentanyl. Br J Anaesth 88:384–388
55. Parr MJ, Olvera CI (2006) Medical management of abdominal compartment syndrome. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (eds) Abdominal compartment syndrome. Landes Biomedical, Georgetown, pp 232–239
56. Deeren D, Dits H, Malbrain MLNG (2005) Correlation between intra-abdominal and intracranial pressure in nontraumatic brain injury. Intensive Care Med 31:1577–1581
57. De Waele J, Delaet I, Hoste E, Verholen E, Blot S (2006) The effect of neuromuscular blockers on intraabdominal pressure. Crit Care Med 34:A70
58. Mertens zur Borg IR, Verbrugge SJ, Kolkman KA (2006) Anesthetic considerations in abdominal compartment syndrome. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (eds) Abdominal compartment syndrome. Landes Biomedical, Georgetown, pp 254–265
59. Mayberry JC (2006) Prevention of abdominal compartment syndrome. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (eds) Abdominal compartment syndrome. Landes Biomedical, Georgetown, pp 223–231
60. Kollef MH (1993) Ventilator-associated pnuemonia: a multivariate analysis. JAMA 270:1965–1970

61. Drakulovic MB, Torres A, Bauer TT, Nicolas JM, Nogue S, Ferrer M (1999) Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: a randomized trial. Lancet 354:1851–1858
62. Pelosi P, Tubiolo D, Mascheroni D, Vicardi P, Crotti S, Valenza F, Gattinoni L (1998) Effects of the prone position on respiratory mechanics and gas exchange during acute lung injury. Am J Respir Crit Care Med 157:387–93
63. Hering R, Wrigge H, Vorwerk R, Brensing KA, Schroder S, Zinserling J, Hoeft A, Spiegel TV, Putensen C (2001) The effects of prone positioning on intraabdominal pressure and cardiovascular and renal function in patients with acute lung injury. Anesth Analg 92:1226–1231
64. Hering R, Vorwerk R, Wrigge H, Zinserling J, Schroder S, von Spiegel T, Hoeft A, Putensen C (2002) Prone positioning, systemic hemodynamics, hepatic indocyanine green kinetics, and gastric intramucosal energy balance in patients with acute lung injury. Intensive Care Med 28:53–58
65. Madl C, Druml W (2003) Gastrointestinal disorders of the critically ill. Systemic consequences of ileus. Best Pract Res Clin Gastroenterol 17:445–456
66. Ponec RJ, Saunders MD, Kimmey MB (1999) Neostigmine for the treatment of acute colonic pseudo-obstruction. N Engl J Med 341:137–141
67. Wilmer A, Dits H, Malbrain ML, Frans E, Tack J (1997) Gastric emptying in the critically ill-The way forward. Intensive Care Med 23:928–929
68. Gorecki PJ, Kessler E, Schein M (2000) Abdominal compartment syndrome from intractable constipation. J Am Coll Surg 190:371
69. van der Spoel JI, Oudemans-van Straaten HM, Stoutenbeek CP, Bosman RJ, Zandstra DF (2001) 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 27:822–827
70. Sugrue M, D'Amours S (2001) The problems with positive end expiratory pressure (PEEP) in association with abdominal compartment syndrome (ACS). J Trauma 51:419–420
71. Sussman AM, Boyd CR, Williams JS, DiBenedetto RJ (1991) Effect of positive end-expiratory pressure on intra-abdominal pressure. South Med J 84:697–700
72. Pruitt BA Jr (2000) Protection from excessive resuscitation: "Pushing the pendulum back." J Trauma 49:567–568
73. Oda J, Ueyama M, Yamashita K, Inoue T, Noborio M, Ode Y, Aoki Y, Sugimoto H (2006) Hypertonic lactated saline resuscitation reduces the risk of abdominal compartment syndrome in severely burned patients. J Trauma 60:64–71
74. Vachharajani V, Scott LK, Grier L, Conrad S (2003) Medical management of severe intra-abdominal hypertension with aggressive diuresis and continuous ultra-filtration. Internet J Emerg Intensive Care Med 6(2)
75. Kula R, Szturz P, Sklienka P, Neiser J, Jahoda J (2004) A role for negative fluid balance in septic patients with abdominal compartment syndrome? Intensive Care Med 30:2138–2139
76. Corcos AC, Sherman HF (2001) Percutaneous treatment of secondary abdominal compartment syndrome. J Trauma 51:1062–1064
77. Reckard JM, Chung MH, Varma MK, Zagorski SM (2005) Management of intraabdominal hypertension by percutaneous catheter drainage. J Vasc Interv Radiol 16:1019–1021
78. Gotlieb WH, Feldman B, Feldman-Moran O, Zmira N, Kreizer D, Segal Y, Elran E, Ben-Baruch G (1998) Intraperitoneal pressures and clinical parameters of total paracentesis for palliation of symptomatic ascites in ovarian cancer. Gynecol Oncol 71:381–385
79. Sharpe RP, Pryor JP, Gandhi RR, Stafford PW, Nance ML (2002) Abdominal compartment syndrome in the pediatric blunt trauma patient treated with paracentesis: report of two cases. J Trauma 53:380–382
80. Parra MW, Al-Khayat H, Smith HG, Cheatham ML (2006) Paracentesis for resuscitation-induced abdominal compartment syndrome: an alternative to decompressive laparotomy in the burn patient. J Trauma 60:1119–1121
81. Kozar RA, Moore FA, Cothren CC, Moore EE, Sena M, Bulger EM, Miller CC, Eastridge B, Acheson E, Brundage SI, Tataria M, McCarthy M, Holcomb JB (2006) Risk factors for hepatic morbidity following non-operative management. Arch Surg 141:451–459
82. De Cleva R, Pineiro da Silva F, Zilberstein B, Machado DJB (2001) Acute renal failure due to abdominal compartment syndrome: report on four cases and literature review. Rev Hosp Clin Fac Med Sao Paulo 56:123–130
83. Etzion Y, Barski L, Almog Y (2004) Malignant ascites presenting as abdominal compartment syndrome. Am J Emerg Med 22:430–431
84. Balogh Z, Moore FA, Goettler CE, Rotondo MF, Schwab CW, Kaplan MJ (2006) Surgical management of abdominal compartment syndrome. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M (eds) Abdominal compartment syndrome. Landes Biomedical, Georgetown, pp 266–296
85. Brock WB, Barker DE, Burns RP (1995) Temporary closure of open abdominal wounds: the vacuum pack. Am Surg 61:30–35
86. Barker DE, Kaufman HJ, Smith LA, Ciraulo DL, Richart CL, Burns RP (2000) Vacuum pack technique of temporary abdominal closure: a 7-year experience with 112 patients. J Trauma 48:201–206
87. Smith LA, Barker DE, Chase CW, Somberg LB, Brock WB, Burns RP (1997) Vacuum pack technique of temporary abdominal closure: a four-year experience. Am Surg 63:1102–1107
88. Navsaria PH, Bunting M, Omoshoro-Jones J, Nicol AJ, Kahn D (2003) Temporary closure of open abdominal wounds by the modified sandwich-vacuum pack technique. Br J Surg 90:718–722
89. Wittmann DH, Aprahamian C, Bergstein JM, Edmiston CE, Frantzides CT, Quebbeman EJ, Condon RE (1993) A burr-like device to facilitate temporary abdominal closure in planned multiple laparotomies. Eur J Surg 159:75–79
90. Block EFJ, Cheatham ML (2001) Closing the abdomen that won't close. In: Cameron JL (ed) Current surgical therapy, 6th edn. Mosby, St. Louis, pp 1129–1135