

# Abdominal compartment syndrome

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## Purpose of review

Intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS), the pathophysiologic implications of elevated intraabdominal pressure (IAP), have detrimental effects on all organ systems and are associated with significant morbidity and mortality. Within the past few years, the diagnosis and management of these syndromes have evolved tremendously.

## Recent findings

Consensus definitions and recommendations for the diagnosis and management of IAH/ACS have been proposed. Risk factors for IAH/ACS have been clearly defined. The timing and techniques for IAP measurement have been further described. A comprehensive evidence-based medical and surgical approach to the treatment of IAH/ACS has been developed.

## Summary

Liberal IAP measurement in the presence of known risk factors combined with implementation of an evolving and comprehensive resuscitation strategy have resulted in significant improvements in both short and long-term outcome for patients who develop IAH/ACS. All clinicians should be aware of the risk factors that predict development of IAH/ACS, the appropriate measurement of IAP, and the current resuscitation options for managing these highly morbid syndromes.

## Keywords

abdominal compartment syndrome, intraabdominal hypertension, intraabdominal pressure

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## Introduction

Intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS), the pathophysiologic manifestations of elevated intraabdominal pressure (IAP), represent significant causes of morbidity and mortality [1–9,10<sup>•</sup>–22<sup>•</sup>]. Implementation of bedside IAP monitoring, creation of consensus definitions and recommendations, and application of an evolving management strategy have resulted in significant improvements in patient survival [23,24<sup>••</sup>,25<sup>•</sup>,26<sup>•</sup>]. The incidence of IAH/ACS, principles and techniques for IAP monitoring, and current fluid resuscitation guidelines were recently reviewed in this journal [27<sup>•</sup>]. The following complementary article addresses state-of-the-art management of patients with IAH/ACS and identifies recent cutting-edge improvements.

## Current management of intraabdominal hypertension/abdominal compartment syndrome

The multiple disparate patient populations at risk for IAH/ACS, combined with numerous causative factors,

make a standardized therapeutic approach to these complex patients difficult. Several fundamental management principles, however, remain constant across all patient populations. Although surgical decompression is widely and erroneously considered the only treatment for IAH/ACS, nonoperative medical management strategies play a vital role in the prevention and treatment of IAP-induced organ dysfunction and failure [6–8,10<sup>•</sup>,27<sup>•</sup>,28,29<sup>•</sup>]. Appropriate management of IAH/ACS is based upon four general principles:

- (1) serial monitoring of IAP,
- (2) optimization of systemic perfusion and organ function,
- (3) institution of specific medical interventions to reduce IAP and the end-organ consequences of IAH/ACS,
- (4) prompt surgical decompression for refractory IAH.

Over 200 clinical trials, case reports/series, or review articles on IAH/ACS have been published within the past 2 years. The consensus definitions and recommendations recently published by the World Society of the Abdominal Compartment Syndrome (WSACS) represent a summation of our current understanding of IAH/ACS

**Table 1 World Society of the Abdominal Compartment Syndrome consensus definitions**

Definition 1	IAP is the pressure concealed within the abdominal cavity.
Definition 2	APP = MAP – IAP
Definition 3	Filtration gradient = 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 isotonic 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 at least 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 more than 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 abdominopelvic region that frequently requires early surgical or interventional radiological intervention.
Definition 11	Secondary ACS refers to conditions that do not originate from the abdominopelvic 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; GFP, glomerular filtration pressure; IAH, intraabdominal hypertension; IAP, intraabdominal pressure; MAP, mean arterial pressure; PTP, proximal tubular pressure.

(Tables 1 and 2). On the basis of these recommended standards, the WSACS has developed both a diagnostic and a therapeutic algorithm for patient management (Figs 1 and 2). These recommendations and algorithms will be briefly reviewed along with recently published developments.

#### Risk factors for and surveillance of intraabdominal hypertension/abdominal compartment syndrome

IAH and ACS have been witnessed to occur in virtually all patient populations [2,4–6,9,10\*,17\*,18\*,20\*,22\*,25\*,

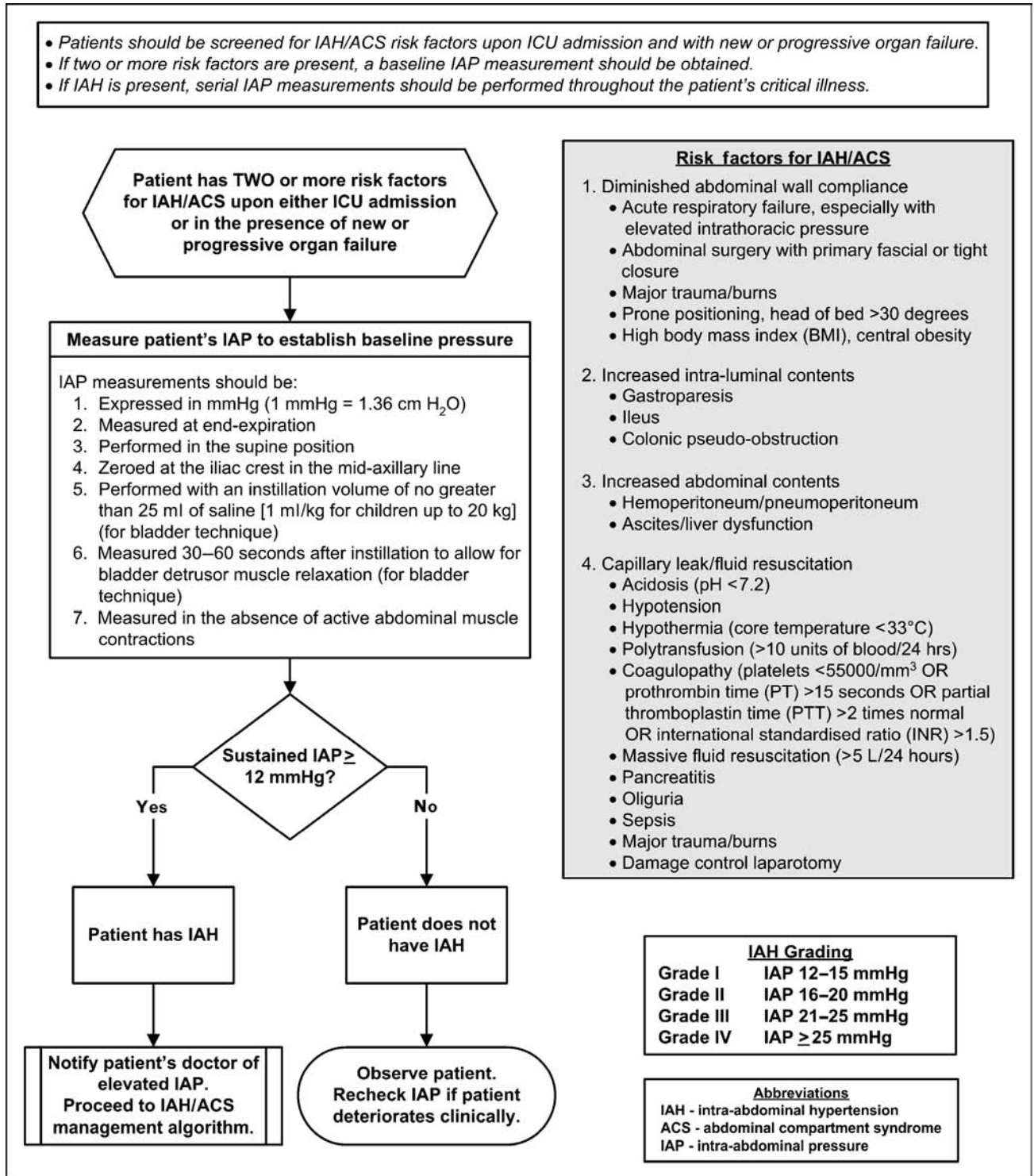
26\*,29\*,30\*,31,32\*]. Although numerous risk factors have been identified (Fig. 1), independent predictors of IAH/ACS include hypothermia, acidosis (elevated base deficit, elevated lactate, or high gastric regional devoid of end-tidal carbon dioxide tension), anemia, oliguria, abdominal surgery, abdominal trauma, high-volume fluid resuscitation (>3500 ml/24 h), ileus, and pulmonary, renal, or liver dysfunction [2,4,7,12\*]. Recently, the development of IAH has been identified as an independent predictor of the need for reexploration for intraabdominal catastrophe (abscess, intestinal ischemia, and perforation)

**Table 2 World Society of the Abdominal Compartment Syndrome consensus recommendations**

Risk factors for IAH/ACS	Patients should be screened for IAH/ACS risk factors upon ICU admission and in the presence of new or progressive organ failure (Grade 1B).
IAP Measurement	If two or more risk factors for IAH/ACS are present, a baseline IAP measurement should be obtained (Grade 1B). If IAH is present, serial IAP measurements should be performed throughout the patient's critical illness (Grade 1C). 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).
APP	APP should be maintained above 50–60 mmHg in patients with IAH/ACS (Grade 1C).
Sedation and analgesia	Insufficient data exist to make recommendations at this time.
NMB	A brief trial of NMB may be considered in selected patients with mild-to-moderate IAH, while other interventions are performed to reduce IAP (Grade 2C).
Body positioning	The potential contribution of body position in elevating IAP should be considered in patients with moderate-to-severe IAH or ACS (Grade 2C).
Gastric/colonic decompression	Insufficient data exist to make recommendations at this time.
Fluid resuscitation	Fluid resuscitation volume should be carefully monitored to avoid overresuscitation in patients at risk for IAH/ACS (Grade 1B). Hypertonic crystalloid and colloid-based resuscitation should be considered in patients with IAH to decrease the progression to secondary ACS (Grade 1C).
Diuretics/hemofiltration	Insufficient data exist to make recommendations at this time.
Percutaneous decompression	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 decompression should be performed in patients with ACS that is refractory to other treatment options (Grade 1B). 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	Insufficient data exist to make recommendations at this time.

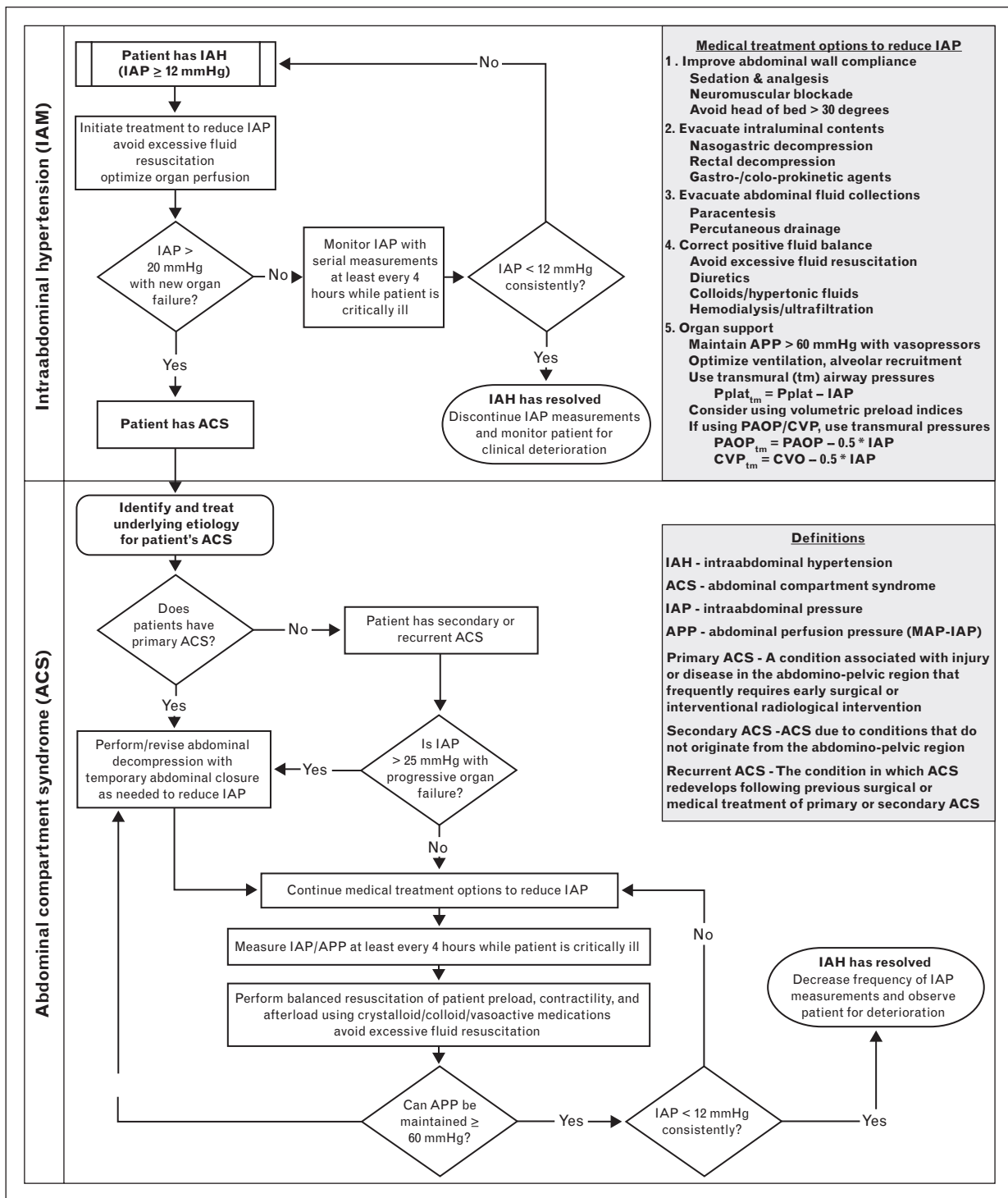
The Grade scheme ([www.gradeworkinggroup.org](http://www.gradeworkinggroup.org)) classifies recommendations as either strong recommendations (Grade 1) or weak suggestions (Grade 2) according to the balance between the associated benefits and risks. The quality of evidence is classified as high (Grade 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 will receive a Grade of '1A', whereas a weak suggestion based upon low-quality evidence will receive a Grade of '2C'. ACS, abdominal compartment syndrome; APP, abdominal perfusion pressure; IAH, intraabdominal hypertension; IAP, intraabdominal pressure; NMB, neuromuscular blockade.

Figure 1 World Society of the Abdominal Compartment Syndrome intraabdominal hypertension assessment algorithm



Adapted from [23].

**Figure 2 World Society of the Abdominal Compartment Syndrome intraabdominal hypertension/abdominal compartment syndrome management algorithm**



Adapted from [24\*\*].

[33<sup>•</sup>]. Given the associated significant morbidity and mortality, patients should be routinely screened for IAH/ACS risk factors in the presence of new or progressive critical illness.

### **Intraabdominal pressure measurement**

Physical examination is insensitive (40–60%) for detecting elevated IAP [1]. Serial measurements of IAP are essential and have been demonstrated to be cost-effective, safe, and accurate in identifying the presence of IAH and guiding resuscitative therapy for ACS [23,27<sup>•</sup>,31,32<sup>•</sup>,34,35,36<sup>•</sup>,37<sup>•</sup>]. When IAH/ACS risk factors are present, a baseline IAP measurement should be obtained and serial IAP measurements performed throughout the patient's critical illness.

IAP is most commonly determined using the intravesicular or 'bladder' technique, which has been well described [23,24<sup>••</sup>,27<sup>•</sup>,34,38]. The recent literature, however, has addressed several important issues, which bear consideration. Although an isotonic saline instillation volume of no more than 25 ml is widely recognized as being optimal, volumes as low as 2–3 ml have been demonstrated to be accurate and volumes more than 25 ml potentially misleading by overestimating IAP [39<sup>•</sup>]. The optimal frequency of IAP monitoring has yet to be determined, although measurements every 4–6 h are now commonplace in many ICUs. IAP clearly varies with respiration, should be measured at end-expiration, and may be useful as a marker of abdominal wall compliance [40<sup>•</sup>]. IAP also varies with BMI such that obese patients have a higher baseline IAP [41<sup>•</sup>]. As a result, obesity-induced increases in IAP should be considered when evaluating patients for IAH/ACS. The temperature of the isotonic saline instilled is also important; room temperature (RT) isotonic saline significantly increases IAP, presumably due to contraction of the bladder detrusor muscle, potentially leading to a falsely elevated IAP [42<sup>•</sup>]. Waiting 30–60 s after isotonic saline instillation to allow warming of the fluid and relaxation of the detrusor muscle has thus been recommended [23,24<sup>••</sup>].

A recent multicenter international trial evaluated three different zero reference transducer positions for IAP measurement (pubic symphysis, phlebostatic axis, and mid-axillary). The authors found that there can be considerable variation in determining the exact location of both the phlebostatic axis and the symphysis pubis clinically resulting in differing IAP values [43<sup>•</sup>]. They concluded that IAP transducers should be zeroed at the mid-axillary line to ensure consistency from measurement to measurement.

IAP monitoring has traditionally been performed in the supine position. Several recent studies [44<sup>•</sup>,45–50] have

evaluated the impact of head of bed elevation (to prevent ventilator-associated pneumonia) upon IAP measurements. These studies have routinely found that head of bed elevation significantly increases IAP compared with supine measurements. Such increases in IAP become clinically significant (increase >2 mmHg) when the patient's head of bed exceeds 20° elevation. Consequently, traditional supine IAP measurements may underestimate the patient's true IAP if the head of bed is being elevated between measurements. Prone positioning for acute lung injury has also been demonstrated to significantly increase IAP. Although some authors believe such increases are due to visceral bladder compression as a result of gravity, intragastric pressures are also increased by head of bed elevation, suggesting that this is a true physiologic and clinically important effect [47]. As clinical decision thresholds for nonsupine IAP measurements have not yet been defined, until further research is available, IAP measurements should be performed in the supine position and the potential contribution of body position considered when interpreting IAP measurements [24<sup>••</sup>,27<sup>•</sup>].

Other techniques for IAP determination are gaining acceptance. Intragastric balloon catheters, direct intraabdominal catheters, and noninvasive IAP estimation using near-infrared spectroscopy have all been described and validated against the traditional intravesicular method [51–53]. Continuous IAP measurement has been demonstrated to be an accurate and clinically useful monitoring technology, although superiority over intermittent measurements has not been established [51,54<sup>•</sup>,55,56].

Ejike *et al.* [57<sup>•</sup>], in a landmark paper, addressed an important question: what is the normal range of IAP among critically ill infants and children? They confirmed that 3 ml of isotonic saline represents the minimal instillation volume necessary to measure IAP in children below 20 kg body weight. The WSACS currently recommends an instillation volume of 1 ml/kg up to 20 kg, which is within the ranges supported by the Ejike *et al.* study [58].

### **Abdominal perfusion pressure**

The 'critical IAP' that causes end-organ dysfunction or failure varies from patient to patient as a result of differences in physiology and preexisting comorbidities. It may be inappropriate, therefore, to assume that a single threshold IAP can be applied to the clinical decision-making of all patients. 'Abdominal perfusion pressure' (APP), defined as mean arterial pressure (MAP) devoid of IAP, assesses not only the severity of IAP present but also the relative adequacy of abdominal blood flow [59]. APP has been demonstrated in multiple studies to be superior to IAP measurements alone [30<sup>•</sup>,59]. Failure to maintain an APP of at least 60 mmHg has been found to discriminate

between survivors and nonsurvivors and appears to be a useful resuscitation endpoint [59]. Maintenance of an APP of at least 60 mmHg, achieved through a balance of judicious fluid resuscitation and application of vasoactive medications, has been demonstrated to reduce the incidence of acute renal failure in two recent studies [30,60]. The WSACS is currently subjecting APP-directed resuscitation to a prospective, randomized, multicenter trial.

### **Sedation and analgesia**

Pain, agitation, ventilator dyssynchrony, and use of accessory muscles during work of breathing may all lead to increased thoracoabdominal muscle tone and decreased abdominal wall compliance, resulting in increases in IAP [2,28,61,62]. Patient sedation and analgesia can reduce muscle tone and potentially decrease IAP to less detrimental levels. In addition to ensuring patient comfort, adequate sedation and analgesia thus appear to play a useful therapeutic role in the patient with IAH [28,63]. Hakobyan and Mkhoyan [61] recently reported a prospective, blinded trial of epidural vs. intravenous postoperative opioid therapy as treatment of moderate IAH. Both therapies were equivalent in reducing IAP to less detrimental levels and increasing APP to ensure adequate systemic perfusion.

### **Neuromuscular blockade**

Diminished abdominal wall compliance due to pain, tight abdominal closures, and third-space fluid can increase IAP to potentially detrimental levels [2,40,61]. Neuromuscular blockade (NMB) has been reported anecdotally to be an effective method for reducing IAP in mild-to-moderate IAH [28,62]. De Laet *et al.* [64] recently performed the first prospective trial of NMB in the management of IAH, demonstrating significant reductions in IAP in nine of 10 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), suggesting that NMB is not efficacious in the presence of severe IAH or ACS. NMB is not without risk, however, and the potential benefits of such therapy must be balanced against the risks of prolonged paralysis. The WSACS suggests that a brief trial of NMB may be considered in patients with mild-to-moderate IAH in conjunction with other interventions to reduce IAP.

### **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 [2,4]. Excessive air and fluid within the hollow viscera can raise IAP and lead to organ dysfunction and failure. Nasogastric, rectal drainage, or both, enemas, and

even endoscopic decompression are simple and relatively noninvasive methods for reducing IAP and treating mild-to-moderate IAH in patients with visceral distention [28]. The administration of prokinetic motility agents such as erythromycin, metoclopramide, or neostigmine is also of use in evacuating the intraluminal contents and decreasing visceral volume [31].

### **Fluid resuscitation**

Strategies for appropriate fluid resuscitation and assessment of intravesicular volume status in patients with IAH/ACS have previously been described [27]. In brief, hypovolemia aggravates the pathophysiologic effects of elevated IAP, whereas hypervolemia (i.e. excessive volume resuscitation) is an independent predictor for subsequent development of ACS [7,12,22,62,65,66]. Fluid resuscitation of patients at risk for IAH/ACS should be carefully monitored to avoid overresuscitation. Hypertonic crystalloid and colloid-based resuscitation have recently been demonstrated to reduce IAP and decrease the risk of subsequent secondary ACS [67,68].

### **Diuretics and continuous venovenous hemofiltration/ultrafiltration**

Early intermittent hemodialysis or continuous hemofiltration/ultrafiltration may be more appropriate than continuing to volume load and increase the likelihood of secondary ACS with its attendant morbidity and mortality [69]. Diuretic therapy, in combination with colloid, may also be considered to mobilize third-space edema once the patient is hemodynamically stable [67].

### **Percutaneous decompression**

Percutaneous catheter drainage of free intraabdominal fluid, air, abscess, or blood is an effective technique for reducing IAP and potentially correcting IAH-induced organ dysfunction [67,70]. Performed under ultrasound or computed tomography guidance, percutaneous decompression can significantly reduce IAP and decrease the need for morbidity of surgical decompression. This minimally invasive approach to IAH/ACS management is most effective in patients with secondary ACS due to excessive resuscitation, burns, acute pancreatitis, or ascites. Patients with IAH/ACS refractory to percutaneous catheter decompression should undergo abdominal decompression.

### **Abdominal decompression**

Surgical decompression of the abdomen has long been the standard treatment for IAH/ACS and can be life-saving when a patient's organ dysfunction, failure, or both are refractory to medical treatment [10,71]. Delayed abdominal decompression and disregard of high IAP levels are associated with significant increases in patient mortality [18,59,72]. 'Prophylactic' decompression and creation of a 'temporary abdominal closure' in surgical

patients at risk for IAH/ACS significantly reduces the subsequent development of IAH/ACS and improves survival [25<sup>•</sup>]. Although seemingly aggressive and potentially disabling, patients at risk for IAH/ACS who are treated with prophylactic decompression demonstrate identical long-term physical and mental health function as well as resumption of gainful employment compared with similar patients who undergo primary fascial closure [73<sup>•</sup>].

A variety of different methods ['vacuum-pack closure', 'Bogota bag' or silo, Velcro burr, absorbable mesh, and vacuum-assisted closure (VAC)] have been described for managing the 'open abdomen' [71,74<sup>•</sup>–77<sup>•</sup>]. No one technique demonstrates superiority in all clinical situations and a surgeon must be familiar with the relative benefits of indications for each method. It is essential to recognize that recurrent ACS is possible with any of these techniques, especially if applied in a fashion that does not allow continued visceral expansion during resuscitation [9,33<sup>•</sup>,71]. If recurrent ACS develops, the dressing should be immediately removed and reapplied so as to reduce IAP to an acceptable level [77<sup>•</sup>].

Leppäniemi *et al.* [78] have recently described subcutaneous release of the linea alba as a less invasive technique for abdominal decompression in patients with acute pancreatitis. This technique reduces IAP and restores organ function while maintaining the skin and peritoneum intact for visceral protection. This technique has recently been shown to be an effective alternative to open decompression in the traumatically injured as well [79<sup>•</sup>].

### 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, will tolerate primary fascial closure within 5–7 days. Cheatham and Safcsak [25<sup>•</sup>] recently reported an 80% same admission primary fascial closure rate among patients decompressed for IAH/ACS. Those patients who remain critically ill past this time period with significant loss of abdominal domain will likely 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 fascial closure after only 3–6 months [25<sup>•</sup>,59,71].

### Conclusion

The diagnosis and management of IAH/ACS is evolving at a rapid rate. Liberal IAP measurement in the presence of known risk factors, implementation of comprehensive

medical interventions to reduce elevated IAP, earlier surgical decompression, and aggressive attempts to achieve primary fascial closure following resolution of a patient's critical illness have resulted in significant improvements in both short and long-term outcome for patients who develop IAH/ACS. All clinicians should be aware of the risk factors that predict development of IAH/ACS, the appropriate measurement of IAP, and the current resuscitation options for managing these highly morbid syndromes.

### Acknowledgement

The author is President-elect of the World Society of the Abdominal Compartment Syndrome (WSACS).

### References and recommended reading

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- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 177–178).

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