CHEST

Intraabdominal Hypertension, Abdominal Compartment Syndrome, and the Open Abdomen

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Abdominal compartment syndrome (ACS) is the end point of a process whereby massive interstitial swelling in the abdomen or rapid development of a space-filling lesion in the abdomen (such as ascites or a hematoma) leads to pathologically increased pressure. This results in so-called intraabdominal hypertension (IAH), causing decreased perfusion of the kidneys and abdominal viscera and possible difficulties with ventilation and maintenance of cardiac output. These effects contribute to a cascade of ischemia and multiple organ dysfunction with high mortality. A few primary disease processes traditionally requiring large-volume crystalloid resuscitation account for most cases of IAH and ACS. Once IAH is recognized, nonsurgical steps to decrease intraabdominal pressure (IAP) can be undertaken (diuresis/dialysis, evacuation of intraluminal bowel contents, and sedation), although the clinical benefit of such therapies remains largely conjectural. Surgical decompression with midline laparotomy is the standard ultimate treatment once ACS with organ dysfunction is established. There is minimal primary literature on the pathophysiological underpinnings of IAH and ACS and few prospective randomized trials evaluating their treatment or prevention; this concise review therefore provides only brief summaries of these topics. Many modern studies nominally dealing with IAH or ACS are simply epidemiologic surveys on their incidence, so this paper summarizes the incidence of IAH and ACS in a variety of disease states. Especially emphasized is the fact that modern critical care paradigms emphasize rational limitations to fluid resuscitation, which may have contributed to an apparent decrease in ACS among critically ill patients.

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It is only within the past 25 years that abdominal compartment syndrome (ACS) has been broadly recognized as a distinct phenomenon, and only within the last 10 to 15 years have there been formal attempts to standardize and define terms and recommended treatments for the disease. The majority of medical literature on this process originates from a few dedicated clinicians collaborating through the research/advocacy group the Abdominal Compartment Society (WSACS;

ABBREVIATIONS: ACS = abdominal compartment syndrome; APP = abdominal perfusion pressure; IAH = intraabdominal hypertension; IAP = intraabdominal pressure; IVC = inferior vena cava; MAP = mean arterial pressure; PPV = pulse pressure variation; WSACS = the Abdominal Compartment Society

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http://www.wsacs.org).[1] Through their exceptional activism, but also because of paradigm changes in critical care medicine that favor restrictive fluid resuscitation for a variety of disease processes, the incidence of ACS may be decreasing. Nonetheless, the concept of IAH (and ACS) as a pathophysiological process requiring specific attention remains poorly understood and underrecognized.

Definitions and Pathophysiological Characteristics

The abdomen is a closed anatomic space with some partially compliant borders, like the diaphragm and abdominal musculature. For simplicity's sake, the abdominal contents can be considered to behave according to the principles of static fluid mechanics. Specifically, a pressure applied to any given part of the abdominal cavity is likely to be transmitted undiminished throughout the entirety of that anatomic space. Thus, a pressure measured at one point in the abdomen can be assumed to represent the pressure throughout the entire abdominal space; it is described as the intraabdominal pressure (IAP).

Pressure in the abdomen increases as intraabdominal *volume* increases (whether from air, tissue edema, liquid such as ascites or blood, or solids such as a tumor or gravid uterus). The mathematical relationship between pressure and volume (ie, how much IAP changes for a given change in volume) is the abdominal compliance (Fig 1), and it is largely determined by the elastic recoil of the abdominal wall and diaphragm. Decreased compliance (as from burn eschars or intraabdominal adhesions) restricts some of the volume accommodation that might otherwise occur and can contribute to a steep rise in pressure.[2] Massive IV fluid resuscitation with capillary leak, a positive fluid balance, and thus presumably a rapid increase in visceral edema and intraabdominal volume, was clearly associated with the development of high IAP in a meta-analysis and systematic review,[3] and abdominal compliance has been shown to be an important factor in predicting organ failure.[4]

Irrespective of the cause, elevated IAP can threaten the perfusion and thus viability of tissue in the abdominal compartment.[5] This and other end-organ effects of IAH are summarized in Figure 2.[6]

The terms IAH and ACS are used to represent pathologic points on a spectrum of pressures that can affect intraabdominal tissue viability and organ function.

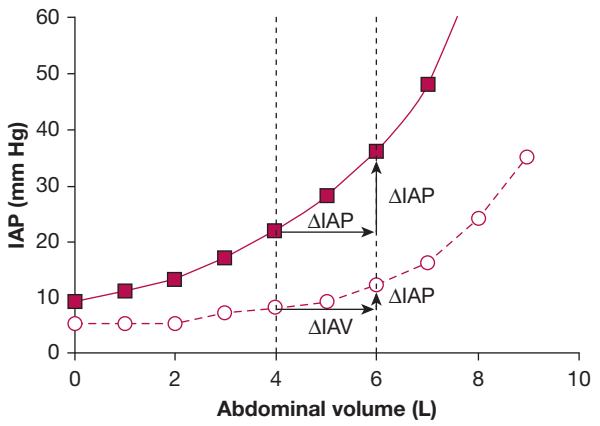

Figure 1 – *Abdominal compliance calculated as a change in volume over change in pressure. Because the abdominal compartment is a closed space with rigid (spine and pelvis) and semirigid (abdominal wall and diaphragm) borders, pressure in the abdomen increases as volume in the abdomen increases. Once a critical intraabdominal volume is reached, IAP increases exponentially with further increases in volume or as abdominal compliance decreases. The upper pressure-volume curve with closed squares represents a patient with poor abdominal compliance. IAH = intraabdominal hypertension; IAP = intraabdominal pressure; IAV = intraabdominal volume. (Reproduced with permission from Malbrain et al.[2])*

Throughout the remainder of this review, nomenclature will follow the terminology established by the WSACS (Table 1), although such terminology has only recently been standardized and many early studies of IAH and ACS used discordant definitions or cutoff pressure values.

Normal IAP in healthy individuals has been described as ranging between subatmospheric to 5 to 7 mm Hg, with higher levels found in obese individuals.[7] Pressures ≥ 12 mm Hg are considered to represent IAH, and pressures > 20 mm Hg in the context of new organ dysfunction is considered to be ACS (Table 1), although a variety of surveys found that there is likely an additional subset of nonobese noncritically ill individuals with chronically elevated IAP (eg, due to pregnancy or chronic ascites). Also, after uncomplicated abdominal surgery in noncritically ill patients, IAP may be transiently higher than levels usually defined as pathologic.[8]

An additional concept requiring definition is the "open abdomen." This is a surgical management strategy whereby the incisional defect in the abdominal wall is purposefully left temporarily unrepaired at the end of a procedure to relieve pressure so that the abdominal viscera are generally unprotected by a patient's own fascia, skin, or other soft tissue. To avoid evisceration and excessive heat or fluid losses from the abdominal defect, however, temporary closure with towels, sponges,

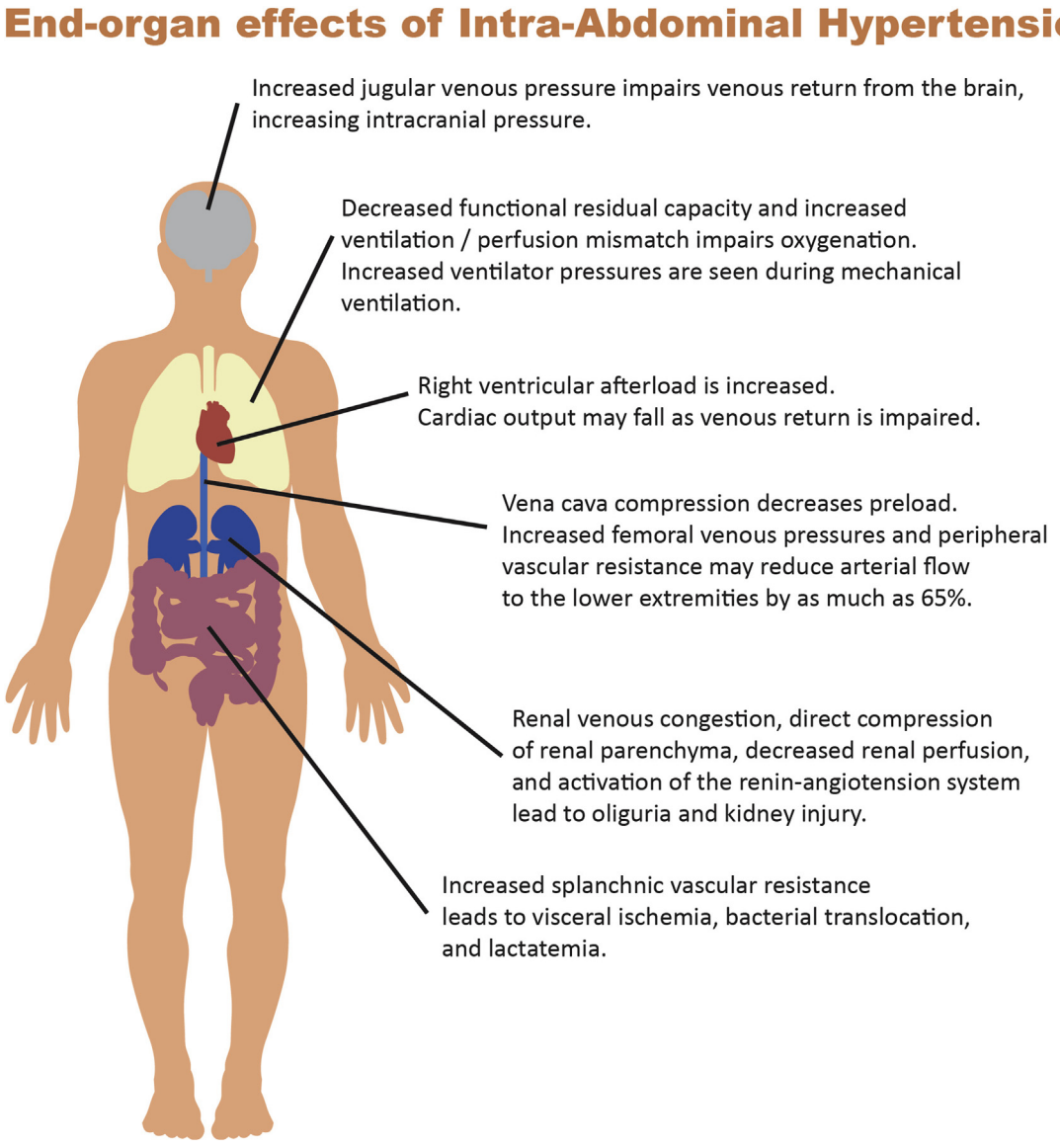

Figure 2 – *The adverse effects of IAH on other organ systems. As intraabdominal pressure increases, perfusion of viscera and other organs is adversely affected. Adapted from Balogh et al.*[6] *See Figure 1 legend for expansion of abbreviations.*

a prosthetic patch, or a translucent bag/cover, or a combination, is left in place.

Measuring IAP

Comprehensive reviews of methods for measuring IAP are available,[9,10] but in clinical practice, IAP is almost always measured indirectly by bladder pressure through a Foley catheter. This has been shown to correlate well with directly measured IAP.[11] Figure 3 demonstrates a system for intermittent bladder pressure measurement using equipment readily available in most ICUs, and a stepwise algorithm for obtaining reliable IAP measurements is presented in Table 2.[12,13]

Although specialty products for continuous bladder pressure measurements are available and recommended by some authors,[14] measurement of IAP every 4 to 6 hours is probably adequate in critically ill patients deemed at risk of the development of IAH or ACS. Serial measurement of IAP by this method does not appear to lead to increased rates of catheter-associated urinary tract infections.[15]

Improper or absent measurement of IAP has been purported to contribute paradoxically to excessive fluid resuscitation. For example, assessment of hemodynamic parameters such as pulse pressure variation (PPV) on an arterial waveform or assessment of inferior vena cava

TABLE 1] Definitions and Diagnostic Criteria for IAH/ACS

IAH is defined by a sustained or repeated pathologic elevation of IAP ≥ 12 mm Hg
ACS is defined as a sustained IAP > 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction/failure
Primary IAH or ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention
Secondary IAH or ACS refers to conditions that do not originate from the abdominopelvic region
Recurrent IAH or ACS refers to the condition in which IAH or ACS redevelops following previous surgical or medical treatment of primary or secondary IAH or ACS
APP = MAP − IAP

The WSACS also recommends grading IAH by degree of elevation in IAP for research purposes, but since such distinctions are probably irrelevant for clinical purposes, given that specific pressure thresholds do not reliably predict organ dysfunction, they are not listed in this review. ACS = abdominal compartment syndrome; APP = abdominal perfusion pressure; IAH = intraabdominal hypertension; IAP = intraabdominal pressure; MAP = mean arterial pressure. (Adapted with permission from Kirkpatrick et al.[47])

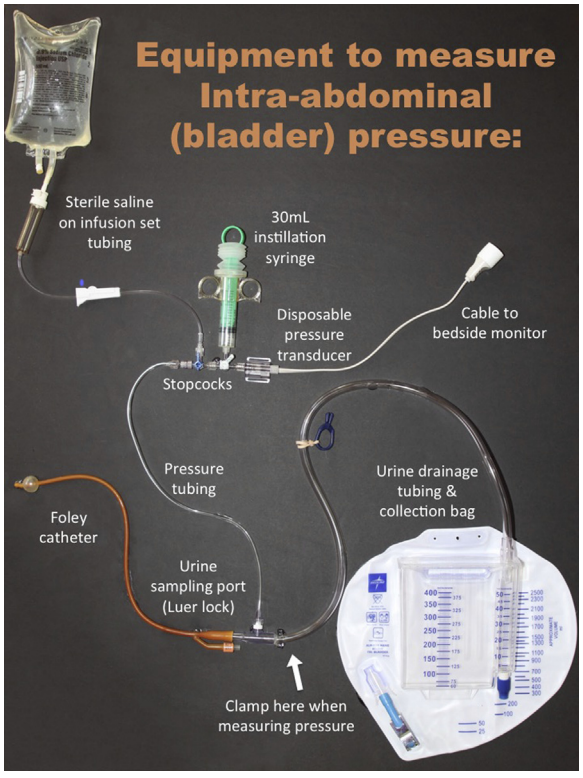

Figure 3 – *Equipment to measure IAP. An example of a closed system to facilitate intermittent measurement of vesicular (abdominal) pressures constructed with readily available standard ICU equipment. A standard IV infusion set is connected to 500 mL of normal saline, a ramp with three-way stopcocks, a Luer lock syringe, and a short segment of pressure tubing that can connect between a standard Foley catheter and urinary drainage tubing. Further instructions are given in Table 2. For an outstanding review of systems to measure IAP see Sugrue et al.*[10]

(IVC) diameter and distensibility by transabdominal ultrasonography are both promoted as reliable means to guide fluid resuscitation.[16,17] However, IAH can abolish or increase threshold values for PPV to predict fluid responsiveness (decreased intrathoracic compliance may cause dramatic increases in PPV with ventilation),[18] and IAH can cause a flat compressed IVC that mimics hypovolemia. A failure to recognize IAH could therefore cause a clinician to inappropriately administer fluids in an attempt at volume resuscitation, exacerbating capillary leakage and tissue edema and pushing a patient further toward ACS.

TABLE 2] Measuring IAP

1. Connect sterile saline infusion set, instillation syringe, and disposable pressure transducer using stopcocks and a segment of arterial pressure tubing to Foley catheter and urinary drainage tubing (Fig 3)
2. Place patient in supine position • *Head-up positioning may falsely elevate IAP measurement*[27,28]
3. Flush tubing all the way to the Foley catheter with sterile saline and "zero" transducer to atmospheric pressure at the iliac crest in the midaxillary line
4. Use syringe to instill a priming volume of < 25 mL sterile saline through Foley catheter into bladder; clamp urine drainage tubing immediately distal to the pressure sampling line • *Just enough volume to create a continuous fluid column and remove air is necessary, whereas excessively large instillation volumes may lead to falsely elevated IAP measurements*[12]
5. Wait 30-60 s after installation; ensure that stopcocks are "off" to the instillation syringe and IV tubing but "open" to the patient and transducer • *Allow time for bladder detrusor muscle relaxation, as instillation of priming fluid at room air temperature may cause muscle contraction*[13]
6. Measure pressure in the absence of active abdominal muscle contractions and at end-expiration • *Sedation or pharmacologic paralysis may be necessary in a dyspneic agitated patient to ensure adequate muscle relaxation and avoid falsely elevated IAP measurements* • *Reporting of IAP in mm Hg (1 mm Hg = 1.36 cm H_2O) is recommended for standardization and to facilitate calculation of abdominal perfusion pressure (APP = MAP − IAP)*
7. Remove clamp from urine drainage tubing so that the patient's bladder is allowed to drain
8. Obtain measurements every 4-6 h. Monitoring of IAP can cease when IAP is < 12 mm Hg for several h and the patient is clinically improving

See Table 1 legend for expansion of abbreviations. (An alternative method for measuring IAP via foley catheter, and more complete checklist, is available from Sugrue et al.[10])

When bladder pressures are not immediately available—or if bladder hematoma, severe pelvic fracture, or peritoneal adhesions are present that may affect bladder pressure measurements—some of the other simple or uncommon methods of screening for IAH include manometry from Jackson-Pratt abdominal drains,[19] intragastric pressure measurements through a nasogastric tube,[20,21] or measuring pressure from a central venous catheter placed through the femoral vein into the IVC.[22,23] In general, these methods should not be considered reliable ways to accurately assess IAP. Even less reliable, however, are attempts to estimate IAP or diagnose IAH based on changes in abdominal circumference or clinical examination. Even in the hands of experienced surgeons, clinical examination exhibits phenomenally poor sensitivity and accuracy for identifying elevated IAP.[24,25]

Finally, it is worth noting that studies consistently document how patient positioning can have a significant effect on measured IAP. The reason for this is somewhat unclear. One possible explanation is that any apparent changes in IAP as measured by bladder pressure are artefactual (gravitational compression of the bladder could result in altered intravesicular pressure that does not reflect the pressure exerted on abdominal viscera). Many authors propose instead that flexion can affect how visceral contents are compressed between the rigid thorax and the pelvis and thus that apparent IAP changes with position changes reflect true differences. Based on this assumption, flexion and a head-of-the-bed angle $> 30°$ almost certainly contribute to clinically relevant increases in IAP,[26-28] whereas prone positioning (as for acute lung injury) appears to lead to slight increases in IAP, which are of unclear clinical significance.[29] These increases with prone positioning may be influenced by whether or not the abdomen is suspended vs resting directly on a mattress.[30] All that said, the fact that reverse Trendelenberg positioning without flexion also seems to adversely affect IAP[2] suggests that mechanical compression of the abdomen cannot completely explain the documented differences in measured IAP with positioning changes. In addition, the presence of positive end-expiratory pressure on a ventilator has not been found to affect measured IAP to a clinically relevant degree.[27]

Incidence/Epidemiology and Diagnosis-Specific Management

Many of the most-cited studies on the incidence of IAH and ACS are from a generation ago, when aggressive IV fluid administration was still emphasized in surgical, medical, and burn resuscitation.[31-33] This pattern of aggressive fluid resuscitation led to an alarmingly high incidence of severe IAH and ACS in a variety of disease processes (Table 3).[34-42] It can be difficult to compare rates of IAH and ACS across eras, as it was not until the mid-2000s that standardized definitions of IAH and ACS were commonly used. Nonetheless, as the paradigms for management of trauma and critical illness have changed over the past 10 to 15 years, with an emphasis on rationally limiting volume resuscitation,[43] the incidence of IAH and ACS *does* appear to be decreasing.[44,45]

Although some studies associate the development of IAH with increased mortality, the clinical significance of a finding of IAH in the absence of organ dysfunction is unclear. It may therefore be best to simply think of IAH as a potential harbinger of decompensation.[46] Patients who have undergone high-volume fluid resuscitation or those with high-risk disease processes, such as abdominal trauma, massive burns, ruptured aortic aneurysms, and severe pancreatitis, should likely be monitored prophylactically for worsening IAH with serial assessments of IAP.[47]

Trauma

Modern management of catastrophic abdominal trauma frequently adheres to the principles of "damage control" surgery,[48,49] whereby definitive surgical correction of a pathologic condition is not meant to be achieved in one emergent trip to the operating room. Rather, there is an initial focus on efficient control of hemorrhage and contamination followed by maintenance of an open abdomen, as described previously, and transport to an ICU. In the ICU, resuscitation continues with a goal of correcting derangements such as dilutional coagulopathy, hypothermia, and acidosis before eventual return to the operating room for definitive surgery. This strategy appears to have dramatically decreased the incidence of ACS in trauma patients in recent years (Table 3).

According to a systematic review primarily evaluating data from the late 1990s[50] and a more recent expert appropriateness rating study,[51] factors that should lead to consideration of an abbreviated initial surgery and an abdomen left open after trauma include a pH lower than 7.2, core temperature lower than 34°C, estimated blood loss > 4 L, a transfusion requirement of > 10 units of packed red blood cells, systolic blood pressure $<$ 70 mm Hg, lactate levels > 5 mmol/L, base deficit > -6 in patients older than 55 years or > -15 in patients younger than 55 years, or an international normalized

TABLE 3] Selected Studies Evaluating the Incidence of Severe IAH/ACS

Study/Year	Population	Findings
	Trauma	
Ivatury et al[35]/1998	70 patients with "severe abdominal trauma"	32% incidence of ACS
Balogh et al[34]/2003	188 consecutive patients with major torso trauma requiring shock resuscitation	14% incidence of ACS
Balogh et al[45]/2011	81 consecutive shock/trauma patients admitted to an ICU	0% incidence of ACS; 75% incidence of IAP > 12 mm Hg
	Burn	
Ivy et al[36]/2000	10 severely burned patients	20% incidence of ACS requiring surgical decompression; 70% incidence of peak IAP > 25 mm Hg
Strang et al[55]/2014	Systematic review of 50 publications, reporting 1,616 severely burned patients	4%-17% prevalence of ACS; 65%-75% prevalence of IAP > 12 mm Hg
	Ruptured abdominal aortic aneurysm	
Karkos et al[37]/2014	Meta-analysis of 1,134 patients in 39 studies undergoing endovascular repair of ruptured abdominal aortic aneurysms	8%-17% incidence of ACS
Adkar et al[38]/2017	1,241 patients undergoing endovascular repair of ruptured abdominal aortic aneurysms	7% incidence of need for concomitant laparotomy[a]
	Pancreatitis	
Al-Bahrani[65]/2008	18 patients with severe acute pancreatitis	56% incidence of ACS
Aitken et al[46]/2014	218 patients admitted to a medical ICU with acute pancreatitis	1% incidence of ACS; 14% incidence of IAP > 12 mm Hg on admission
	Mixed populations	
Malbrain et al[39]/2004	One day snapshot prevalence study of all 97 patients in 13 general and specialized ICUs across 6 countries	8% prevalence of ACS; 59% prevalence of IAP > 12 mm Hg
Malbrain et al[40]/2005	265 consecutive patients admitted to 14 general and specialized ICUs across 6 countries during a 4 week period	4% incidence of ACS; 32% incidence of IAP > 12 mm Hg on admission; 13% prevalence of ACS in patients with IAH
Daugherty et al[41]/2007	40 patients admitted to a medical ICU with a minimum net positive fluid balance of 5 L within the preceding 24 h	25% incidence of ACS; 33% incidence of peak IAP > 20 mm Hg
	Other disease processes	
Biancofiore et al[42]/2003	108 consecutive liver transplant recipients on arrival in ICU	Incidence of ACS not evaluated; 32% incidence of IAP > 25 mm Hg
Mazzeffi et al[71]/2016	Patients undergoing cardiac surgery with cardiopulmonary bypass	0% incidence of ACS; 83% incidence of IAP > 12 mm Hg

Although some degree of IAH remains a relatively common finding among critically ill patients, the incidence of severe IAH and ACS appears to be decreasing in a variety of primary disease processes. The definition of IAH was not consistent among studies. ACS defined variously as IAP > 20 or 25 mm Hg associated with new-onset organ failure. See Table 1 legend for expansion of abbreviations.

[a]Need for emergent postoperative laparotomy was presumed to be a surrogate marker for ACS.

ratio > 1.6.[50] International consensus guideline documents further recommend empirically maintaining an open abdomen after patients undergo a damage control laparotomy and have extreme visceral or retroperitoneal swelling or elevated bladder pressure noted at the time of laparotomy.[52,53]

Burns

Patients with burn injuries encompassing > 60% of total body surface area or with concurrent inhalational or intraabdominal injuries appear to be at high risk for the development of ACS.[54] Large volume fluid resuscitation is a clear risk factor for the development of ACS in burn

patients.[55,56] The type of fluid used during burn resuscitation has been purported, but not proved, to be associated with the risk of ACS.[55,57]

Mortality in burn patients with ACS may be as high as 75%,[55] and an improvement in overall outcomes with decompressive laparotomy has been difficult to demonstrate. An open abdomen can complicate the management of burn dressings and conversely, since the normal protective skin barrier is already compromised, management of protective dressings for an open abdomen can be more difficult. Decompressive laparotomy has been suggested to reduce mortality from ACS in burn patients in small retrospective studies,[58,59] although other studies have documented only an improvement in hemodynamic parameters after laparotomy without improvement in rates of acute lung injury or other organ dysfunction.[60] Some centers have reported moderate success with the use of less invasive procedural or medical therapies, such as paracentesis or percutaneous peritoneal lavage catheters and protocolized evacuation of gastric or rectal contents, as a way to prevent progression of IAH to ACS or even to treat established ACS and avoid the need for decompressive laparotomy.[56,61]

Emergent Aortic Repair

ACS is recognized as a complication after both open and endovascular repair of ruptured abdominal aortic aneurysms, although the increase in IAP may not necessarily be due to bleeding, and surgical decompression may not improve overall outcomes despite improving hemodynamics.[62] In patients treated initially with an open operation, massive fluid resuscitation for shock, hypothermia, and insensible fluid losses clearly contributes to the development of IAH and ACS postoperatively.[63] Conversely, patients undergoing endovascular repair of ruptured abdominal aortic aneurysms may have large space-filling retained hematomas that contribute to high IAP.

Although small studies have suggested that factors such as the use of balloon occlusion of the aorta during endovascular repair may be associated with the development of ACS, larger reviews and meta-analyses have failed to find consistent factors besides fluid resuscitation that clearly increase the risk of ACS.[37]

A Swedish center has reported some success with percutaneous drain-based management of ACS after ruptured abdominal aortic aneurysms. Using CT-guided placement of a drain into large retroperitoneal hematomas, tissue plasminogen activator is injected to facilitate evacuation of the coagulated hematomas and decrease abdominal pressure.[64]

Acute Pancreatitis

Pancreatitis is an inflammatory process associated with capillary permeability and hypoalbuminemia. With or without aggressive volume resuscitation, significant intraperitoneal or retroperitoneal and visceral edema can develop,[46] which in turn contribute to IAH and the potential for ACS.

Prospective studies have documented an incidence of IAH as high as 61% in patients with severe acute pancreatitis.[65] The authors of one study noted that although IAH developed concurrently with other organ failure in most patients, IAH appeared to precede other organ dysfunction in some patients.[46] They therefore proposed that IAH might be a useful screening tool to identify high-risk patients with pancreatitis. Another paper suggested that the mean admission IAP value in patients with pancreatitis did not differ significantly from the maximum pressure measured in the first 5 days, so the authors suggested that IAH could be used as a reliable marker of severe disease.[66] This is in contradistinction to papers that have documented that in nonsurvivors of acute pancreatitis, IAP may continue to increase throughout the first week of illness, especially if left untreated, whereas in survivors, it plateaus or eventually decreases after about 4 or 5 days.[46,67]

Although modern management of uncomplicated pancreatitis generally emphasizes avoidance of surgical intervention, a few small uncontrolled case series suggest a potential benefit to surgical decompression on mortality and outcomes like respiratory and renal failure in patients with acute pancreatitis and ACS.[65,68]

Other Disease Processes

Case reports have suggested that IAH can be a complication of severe ileus.[69] Elective surgical procedures such as abdominal wall reconstruction in patients with massive ventral hernias[44] have been associated with IAH or ACS, as have cardiac procedures.[70-73] Animal studies have suggested that the degree of hemodilution that develops after initiation of cardiopulmonary bypass may affect IAP and mesenteric circulation.[74]

Pregnancy may be associated with a state of chronic IAH, and a variety of case reports have confirmed that complications of pregnancy, such as severe

pre-eclampsia and HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome, or abdominal ascites as a complication of increased capillary permeability seen with ovarian hyperstimulation syndrome, may convert a compensated state of IAH to uncompensated ACS.[75] In a study of 100 pregnant women at term undergoing scheduled cesarean delivery in the absence of labor, IAP was measured through a bladder catheter in the supine position with leftward tilt immediately after the placement of spinal anesthesia. The median preoperative IAP measurement was 22 mm Hg (range, 15-29 mm Hg), and the median postoperative IAP measurement immediately after cesarean delivery was 16 mm Hg.[76] Although there are questions about the methods used to measure IAP in this study,[75] and other studies have not documented as significant IAH in healthy term parturients,[77,78] it would appear that even after delivery of a gravid uterus some degree of IAH may persist. This could be exacerbated by volume resuscitation during management of severe postpartum hemorrhage.

General Treatment Principles

Nonsurgical Therapies

Targeting an abdominal perfusion pressure (APP), defined as the difference between mean arterial pressure (MAP) and IAP, > 60 mm Hg has sometimes been proposed as a resuscitation end point more predictive of outcome than IAP,[79] but this is not universally accepted, and treatment strategies should probably focus on mitigating IAH rather than driving up MAP. A consensus algorithm identifying a variety of interventions purported to help circumvent or treat ACS is given in Figure 4, and a recent comprehensive review of many of these therapies is available.[80] As a general principle, anything that may help improve abdominal wall compliance or correct a positive fluid balance can be tried to help negate worsening IAH, but once a diagnosis of ACS is suspected or definitively made, there should probably be rapid progression to surgical decompression.

The use of deep sedation and analgesia, or even neuromuscular blockade, may transiently improve abdominal wall compliance and reduce IAP[81] while more durable treatments are being pursued. Removal of restrictive bandages or surgical release of restrictive burn eschars or scar tissue may help. Paracentesis and large-volume (> 1 L) removal of ascites or hematoma has been documented to significantly decrease IAP in a variety of disease processes.[82] Evacuation of other intraabdominal contents—as with nasogastric tube suction of air and fluid, bowel decompression with enemas and prokinetic agents such as metoclopramide or neostigmine, or delivery of a gravid uterus—is presumed (but not proved) to provide some benefit. Since head-of-bed elevation and patient flexion significantly increase IAP, as documented earlier, temporary repositioning of a patient to a supine position may provide some transient benefit in decreasing IAH.

Pharmacologic diuresis or removal of fluid, or both, with continuous renal replacement therapies resulting in net ultrafiltration has been suggested to have a significant impact on IAP. For example, a review of 13 published case series suggested that aggressive fluid removal could result in a significant and clinically relevant decrease in IAP.[83] An average total body fluid removal of 4.9 L resulted in a drop in IAP from 19.3 ± 9.1 mm Hg to 11.5 ± 3.9 mm Hg in this review, but it was not reported over what time course these results were obtained, and effects on major patient outcomes were not reported. Individual papers included in the review only reported improvements in minor outcomes such as an apparent decrease in serum levels of inflammatory markers for disease processes such as severe acute pancreatitis[84,85] and septic shock.[86] Despite these encouraging case reports, the benefits of active fluid removal after resuscitation are less clearly defined than the benefits of a restrictive fluid administration strategy during active resuscitation in critical illness.[87]

Surgical Decompression

In the face of a failure of nonsurgical methods to decrease IAP, surgical abdominal decompression and temporary maintenance of an open abdomen is considered the standard of care. Delays in progression to surgical decompression after development of ACS can dramatically increase the risk of mortality in both patients who have undergone trauma and nonsurgical patients.[79,88] Surgical decompression both improves visceral perfusion and, when combined with negative pressure peritoneal therapy (as described in Fig 5), has been proposed to reduce transmission of inflammatory mediators to the bloodstream, thereby potentially mitigating a septic spiral that can otherwise contribute to progressive organ dysfunction.[89-92]

Because IAH and ACS are frequently encountered in the context of emergent surgical disease processes, acute care surgeons may be more attuned to the risks and

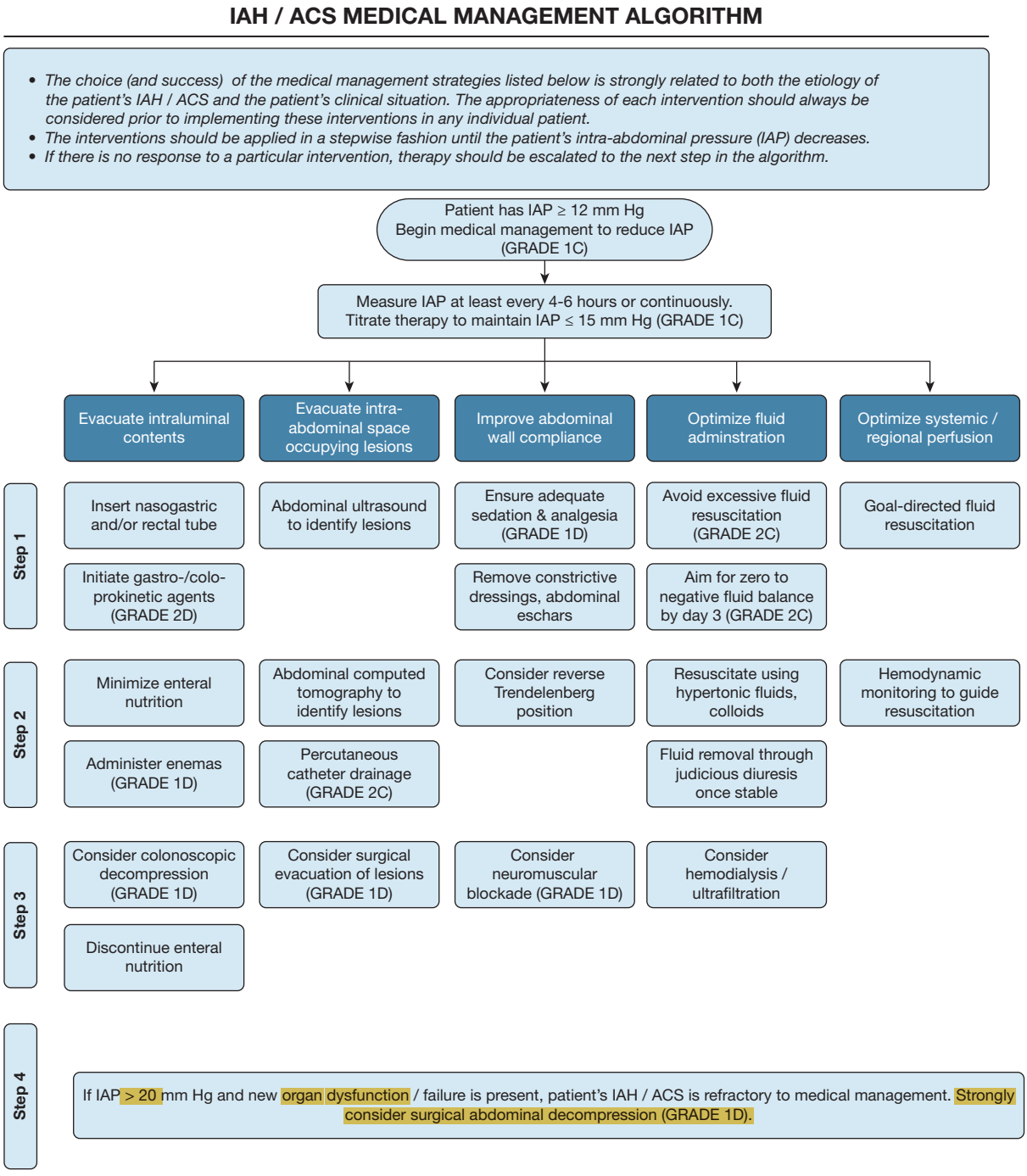

Figure 4 – *IAH/ACS management algorithm. Quality of evidence for each recommendation and strength of recommendation is rated along a four-point ordinal scale in accordance with Grading of Recommendations Assessment, Development and Evaluation guidelines (http://www.gradeworkinggroup.org), in which each evidence grade is symbolized by a letter from D to A: very low (D), low (C), moderate (B), and high (A) and strength of recommendation is given by a number: strong (1) and weak (2). ©Copyright by WSACS, the Abdominal Compartment Society (http://www.wsacs.org). ACS = abdominal compartment syndrome. See Figure 1 legend for expansion of other abbreviations. (Adapted with permission from Kirkpatrick et al.[47])*

more likely to screen for this process. In one study, the time required to diagnose ACS in patients without trauma was twice that required in patients with injuries, and there were significantly longer times to decompression and a higher incidence of multiorgan failure in medical patients.[93] It has been anecdotally suggested that acute care general surgeons may be more likely to pursue surgical decompression and an open abdomen than clinicians from other surgical specialties.[94]

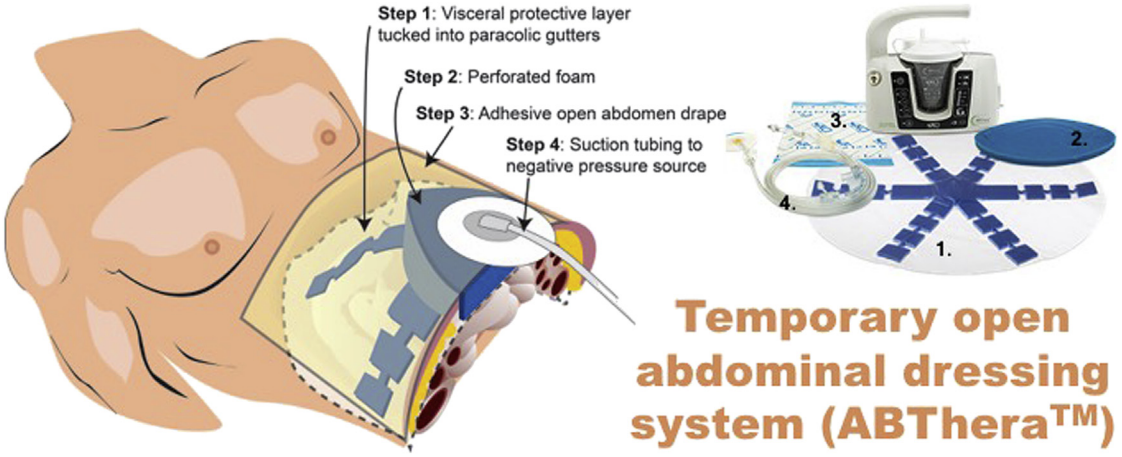

Figure 5 – *A negative pressure vacuum system to temporarily manage an open abdomen. Dedicated systems, such as the ABThera Active Abdominal Therapy (KCI, San Antonio, TX), are commercially available to facilitate management of an open abdomen after surgical decompression for ACS. A large midline incision through all abdominal wall layers releases tension. After confirmation of adequate hemostasis and cleanliness of the abdominal wound, a fenestrated visceral protective layer is placed over the open abdominal cavity (Step 1), covering intestines, omentum, and other organs. This layer is tucked into the paracolic gutters and under the abdominopelvic wall in all directions. A thick perforated foam wedge is then placed into the wound cavity over the visceral protective layer (Step 2). This foam layer both helps indirectly transfer negative pressure to the visceral protective layer to promote active fluid removal, and, as the foam collapses with negative pressure, can provide medial tension to the wound edges of the abdominal wall. This helps maintain the fascial domain (but can also inadvertently contribute to persistent intraabdominal hypertension/abdominal compartment syndrome if there is inadequate relief of tension and decompression of the abdominal contents). An occlusive drape is placed over the foam and intact skin (Step 3) and then a hole is pierced in this drape and an interface pad applied (Step 4) that allows application of negative pressure service. Similar systems such as the so-called Barker's vacuum pack can be created with readily available material (eg, a sterile polyurethane sheet perforated several times with a scalpel is used in place of the fenestrated visceral protective layer; moistened surgical towels are placed over the polyurethane sheet in place of foam; two closed-suction 10F flat silicone Jackson-Pratt drains are draped over the moistened surgical towels and attached to 20 mm Hg of wall suction; and a large adhesive film dressing covers the drains, towels, and skin). (Adapted with permission from www.abthera.com/product-information, ©copyright 2013 KCI Licensing, Inc.)*

Nonetheless, it must be remembered that surgical decompression and an open abdomen, while potentially lifesaving, can be associated with significant morbidity.[94] Complications from an open abdomen can include stimulation of a hypercatabolic state and protein loss through removal of peritoneal fluid,[95] enterocutaneous or other intestinal fistulas,[96-98] retraction of the abdominal wall and development of large ventral hernias, and potentially even lethal hemorrhagic complications including exsanguination and reperfusion syndrome.[99] The risk of fistulas and ventral hernias increases the longer an open abdomen is maintained.[97,100] Bacterial colonization of wounds is common and increases with the length of time the abdomen is left open; this can lead to long-term infectious complications in patients who underwent decompressive surgery for ACS after aortic repair (and so have a synthetic aortic graft in place). Surgical decompression may not even be durably effective in treating ACS[62,101]; recurrent ACS from persistent bleeding, sepsis, or tissue edema has been suggested to occur in up to 20% of patients who undergo decompression.[94] In some cases, it may also be that temporary abdominal closure methods do not adequately increase the abdominal volume to the degree necessary to prevent an increase in IAP. Continued manometric monitoring for ACS is therefore necessary even after surgical decompression.

Common techniques to help manage an open abdomen and provide temporary covering of the abdominal defect before definitive closure can occur include (1) a negative pressure vacuum system or (2) a patch closure (whereby a prosthetic material is suture-interposed between edges of fascia and slowly brought together as intraabdominal edema declines). A systematic review suggested that these techniques also had low rates of complications and higher rates of successful eventual fascial closure compared with other techniques.[102] The negative pressure vacuum system involves placing a perforated plastic sheet over the viscera and a sponge or moistened towels between the fascial edges; the wound and sponge/towels are then covered by an airtight seal pierced by a suction drain connected to negative pressure that collects excess abdominal fluid and helps resolve edema while maintaining tension on fascial edges (Fig 4). The patch closure system often uses VELCRO hook-and-loop sheets (Textol Systems) sutured to fascial edges over a protective plastic sheet, allowing for stepwise reapproximation of fascial edges.

In general, an open abdomen should be closed as early as possible, with most surgeons planning staged attempts at fascial closure approximately every 48 hours. The abdomen may be left open for more than a week with "relook" surgeries intermittently performed.

Conclusions

ACS is a highly morbid disease process caused by sustained acute elevations in IAP > 20 mm Hg associated with new organ dysfunction. Direct injury to abdominal organs, massive fluid resuscitation, or any other process that leads to interstitial edema in the abdominopelvic region can be an inciting factor for IAH and so should be seen as a trigger to institute routine screening for IAH. IAP is routinely measured indirectly through intrabladder pressure. Trauma, burn, aortic rupture, and pancreatitis are disease processes that are especially prone to concurrent development of IAH and ACS. Once IAH is recognized, nonsurgical steps to decrease IAP can be undertaken, such as diuresis, paracentesis, evacuation of intraluminal bowel contents, and sedation, although the clinical benefit of such therapies remains largely conjectural. Urgent surgical evaluation for abdominal decompression is necessary once ACS is diagnosed, although this in itself can be a highly morbid procedure.

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