Intra-abdominal Hypertension and Abdominal Compartment Syndrome

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Increased intra-abdominal pressure (IAP), also referred to as intra-abdominal hypertension (IAH), affects organ function in critically ill patients and may lead to abdominal compartment syndrome (ACS). Although initially described in surgical patients, IAH and ACS also occur in medical patients without abdominal conditions. IAP can be measured easily and reliably in patients through the bladder using simple tools. The effects of increased IAP are multiple, but the kidney is especially vulnerable to increased IAP because of its anatomic position. Although the means by which kidney function is impaired in patients with ACS is incompletely elucidated, available evidence suggests that the most important factor involves alterations in renal blood flow. IAH should be considered as a potential cause of acute kidney injury in critically ill patients; its role in other conditions, such as hepatorenal syndrome, remains to be elucidated. Because several treatment options (both medical and surgical) are available, IAH and ACS should no longer be considered irrelevant epiphenomena of severe illness or critical care. An integrated approach targeting IAH may improve outcomes and decrease hospital costs, and IAP monitoring is a first step toward dedicated IAH management. IAH prevention, most importantly during abdominal surgery but also during fluid resuscitation, may avoid ACS altogether. However, when ACS occurs and medical treatment fails, decompressive laparotomy is the only option.

INDEX WORDS: Intra-abdominal pressure; abdominal compartment syndrome; intra-abdominal hypertension; acute kidney injury.

CASE PRESENTATION

A 37-year-old man was admitted to the intensive care unit (ICU) because of alcohol-induced severe acute pancreatitis. His abdominal pain had started 2 days before presentation. On admission, the patient was dyspneic and hypotensive and reported abdominal pain. He required massive fluid resuscitation for hemodynamic stabilization. APACHE II (Acute Physiology and Chronic Health Evaluation II) score on admission was 20, and Ranson score after 48 hours was 8. The patient required intubation 2 days after admission because of acute respiratory distress syndrome. Despite aggressive ventilatory strategies, his oxygenation remained tenuous, and urine output progressively decreased despite aggressive fluid therapy. Serum creatinine level at admission was 0.79 mg/dL (69.84 μmol/L) and had increased to 5.05 mg/dL (446.42 μmol/L) when intubation was required.

Transvesicular intra-abdominal pressure (IAP) monitoring (using a self-assembled set based on the Cheatham technique)1 was initiated shortly after admission. Initially, IAP was moderately increased (13 mm Hg) and increased steadily, reaching a maximum of 27 mm Hg 3 days after admission. In the setting of multigorgan failure, the diagnosis of abdominal compartment syndrome (ACS) was established.

Because of the inability to maintain oxygenation, hemodynamic instability, and decreased kidney function, formal surgical abdominal decompression was performed 70 hours after admission. IAP immediately decreased to 14 mm Hg, and within minutes, respiratory and hemodynamic function improved, with urine output increasing during the next few hours from 5 to 40 mL/h. Despite this improved diuresis, renal replacement therapy was required and continued for a total of 26 days.

The patient’s condition improved initially, but ongoing inflammation necessitated pancreatic necrosectomy 16 days after the initial decompressive laparotomy. Postoperative continuous lavage using abdominal drains and 2 additional surgical interventions for intra-abdominal infection were required before he could be discharged from the ICU on postoperative day 29. By this point, kidney function had completely recovered.

INTRODUCTION

For several decades, increased IAP has been increasingly recognized as both cause and consequence of many adverse events in critically ill patients. Increased IAP within the closed anatomic volume of the abdominal cavity can lead to decreased perfusion and ischemia of intra-abdominal organs. In addition, increased IAP also leads to physiologic changes and organ dysfunction beyond the abdominal cavity because of the close anatomic relationships with contiguous cavities. Depending on the severity of increased IAP and organ function, the condition is defined as intra-abdominal hypertension (IAH) or ACS (Box 1).

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In 1876, Wendt reported that an increase in IAP was associated with a decrease in urine output, and in 1947, Bradley and Bradley published a comprehensive experimental article describing the effect of IAP on kidney perfusion and function. Several investigators have since noted similar effects in animal models and clinical studies in the critically ill. Presumably because measurement of IAP was cumbersome and clinicians were unaware of the dangers, clinical effects of IAP were not reported again until the early 1980s. It was not until the landmark report by Kron et al, which reported that IAP could be monitored objectively and relatively easily through an indwelling intravesical catheter, that more clinical evidence was rapidly forthcoming concerning the deleterious effects of increased IAP on different organ systems.

Since then, the clinical importance of IAH and ACS essentially has been rediscovered, largely by physicians and surgeons taking care of the sickest of the sick in the ICU. This rediscovery also reflects in part an increasing incidence of IAH and ACS in critically ill patients who were treated more aggressively than ever before, both peri- and intraoperatively and in the ICU. However, changes in organ function in patients undergoing laparoscopic surgery have shown that even at lower pressures in the range of 12-20 mm Hg, IAH is relevant and affects organ function.

In this article, we review the definitions and epidemiologic and pathophysiologic characteristics of IAH and ACS and discuss the available medical and surgical treatment options if ACS cannot be prevented. Finally, the effects of IAH on the kidney are addressed comprehensively.

**DEFINITIONS AND ETIOLOGY**

IAH is defined as sustained or repeated IAP ≥12 mm Hg and is divided into 4 grades (Box 1). The clinical picture involving sustained IAP ≥20 mm Hg with the development of new organ dysfunction or failure constitutes ACS. ACS can be categorized as primary ACS (referring to an intra-abdominal cause), secondary ACS (extra-abdominal cause), and recurrent ACS (recurrence despite previous treatment).

Normal IAP is ~5-7 mm Hg, with baseline levels in morbidly obese individuals often ranging from 9-14 mm Hg. Although this degree of IAH may affect organ function in other patients, it often appears to be tolerated in obese individuals. Normal IAP usually is lower in children. In general, an individual patient’s physiologic state must be taken into account when interpreting IAP measurements. IAP typically is expressed in millimeters of mercury and conversion from centimeters of water may be necessary (1 mm Hg = 1.36 cm H2O).

IAH usually is associated with situations in which either increased abdominal volume or decreased abdominal compliance may predominate, and often a combination of the 2 is to blame. The World Society of the Abdominal Compartment Syndrome (WSACS) recently listed conditions associated with these situations (Box 2).
IAP MEASUREMENT

Surveys of clinicians have shown that many continue to rely on physical examination to diagnose ACS.12,13 However, studies have shown that clinical assessment of IAP based on either abdominal diameter or palpation of the abdomen is unreliable; accordingly, more objective measures are recommended to screen for and diagnose IAH/ACS.14,15 Reliable IAP measurement is the first step in the appropriate clinical management of patients with IAH/ACS. Therefore, various methods for IAP measurement have been developed,16 which stem from the concept that the abdominal cavity is a closed system.2 It follows that the pressure found at any point within the abdominal cavity indicates the pressure throughout the cavity, in accordance with Pascal’s law,17 which states that pressure on liquid in a container will disperse equally to all parts of the container. Accordingly, IAP can be measured from any space within the abdomen. To be accurate in humans, pressure within the abdominal cavity also is influenced by gravity and dependent on the density of the fluid within, resulting in a pressure gradient from anterior to posterior when the patient is in the supine position.

IAP can be measured either directly (through needle puncture of the abdomen during peritoneal dialysis treatment or laparoscopy) or indirectly (using intravesicular pressure as measured through a bladder catheter or gastric pressure through a balloon catheter). Transvesicular measurement of IAP currently is the most popular technique, and several systems with or without the need for electronic equipment are available that allow IAP measurement in a non-ICU environment.18 All transvesicular IAP measurement techniques are based on the same principle; namely, that a fluid column in the bladder catheter and tubing to the collector serve as a pressure transducing medium. The pressure in this closed system can be measured either by means of an electronic system using a pressure transducer or by measuring the height of the fluid column in the tubing. More information regarding the different methods to measure IAP, including the pros and cons of each technique, can be found in a number of recent articles2,16,19 or on the WSACS website (www.wsacs.org).

To facilitate understanding in communication and research, standardized methods and definitions are crucial. Therefore, per consensus, IAP is measured (in millimeters of mercury) at end-expiration; the patient should be supine and refrain from spontaneous muscle contractions. The midaxillary line is used as the zero reference level for IAP measurement. Methods for continuous IAP measurement also are available, but are not yet widely used.19 It is advised that IAP monitoring be based on a (site-specific) protocol, known risk factors, the monitoring equipment available, and nursing staff experience and should be linked directly to a local treatment protocol.15 It is recommended that patients with any of the conditions associated with IAH (Box 2) are monitored using a transvesicular technique at least every 4 hours until IAP remains <12 mm Hg for at least 24 hours in the absence of organ dysfunction.

EPIDEMIOLOGY

The “rediscovery” of IAH and ACS in the early 1980s was related largely to techniques used in patients after emergency abdominal surgery and trauma.20 In the setting of massive resuscitation with crystalloid fluids, the clinical picture of IAH and ACS may be dramatic, with severe and often irreversible organ damage. In subsequent years, it has increasingly been recognized that many other patients in the ICU, both adult and pediatric, are affected by IAH/ACS, with scenarios including elective surgical procedures, liver transplant,21 massive fluid resuscitation for extra-abdominal trauma,22 septic shock,23 severe acute pancreatitis,24 and severe burns,25 although it is likely that almost any critical illness may be a precipitant. The reported incidence rates of IAH and ACS vary considerably based on the patient population, and selection bias may affect these reports. Table 1 lists reported incidence rates. In intensive care populations, IAH is associated with adverse outcomes, including organ dysfunction and mortality,34-36 with studies that differentiate between primary and secondary IAH describing worse outcomes for patients with secondary ACS than with primary ACS.

As the use of preventive strategies in the surgical and trauma setting for ACS increases, epidemiologic characteristics may change significantly. This may be especially relevant in trauma and abdominal surgery, in which prophylactic laparostomy (or open abdomen

| Table 1. Reported Incidence of IAH and ACS in Contemporary Literature |
|---|---|
| IAH (%) | ACS (%) |
| Major abdominal surgery26-28 | NA | 33-41 |
| Liver transplant29 | NA | 31 |
| Major trauma30-33 | 50 | 13-36 |
| ICU35-37 | 30-54 | 5-12 |
| Septic shock23,38 | 51-76 | 33 |
| Severe acute pancreatitis24 | 59-84 | 25-56 |

Note: Values shown are incidence or range (both in percentage). Different definitions have been used to describe both IAH and ACS in most of the studies.

Abbreviations: ACS, abdominal compartment syndrome; IAH, intra-abdominal hypertension; ICU, intensive care unit; NA, not available.
treatment) has become the standard of care in patients at risk of ACS. Therefore, primary ACS probably will occur less frequently in our ICUs, and secondary ACS, a condition largely associated with massive fluid resuscitation, may become more prominent. Whether the present evolution away from massive crystalloid resuscitation fluids influences this prediction remains to be seen.

**IAH AS A RISK FACTOR FOR ACUTE KIDNEY INJURY**

The presence of IAH as a risk factor for acute kidney injury (AKI) has been shown in many clinical settings. After emergency abdominal surgery, IAH occurs in 33%-41% of patients and is associated with AKI and mortality. Similarly, in patients undergoing orthotopic liver transplant, Biancofiore et al reported not only an incidence of IAH (defined as IAP >25 mm Hg) of 32%, but also a linear relationship between IAH and severity of decreased kidney function. These somewhat dated studies used definitions with low sensitivity for both IAH and AKI. In recent years, several investigators have shown similar relationships between IAH and AKI in the ICU population using newer grading systems. Dalfino et al studied 123 consecutive patients (almost half were medical patients) admitted to a general ICU for at least 24 hours, finding that 30% of the study population developed IAH. IAP of 12 mm Hg was the most optimal cutoff for AKI defined using the RIFLE (risk, injury, failure, loss, end-stage disease) classification (sensitivity, 91%; specificity, 67%; area under the receiver operating characteristic curve, 0.85). In this cohort of unselected ICU patients, IAH also was an independent risk factor for the development of AKI (odds ratio, 2.44).

More recently, IAH also has been reported to complicate kidney transplant. Pertek et al reported 4 patients with increased IAP who developed early transplant dysfunction and subsequently were treated successfully using abdominal decompression. Similarly, Ball et al described retroperitoneal compartment syndrome, a subtype of secondary ACS, in 11 transplant patients. Presumably because of early recognition, all patients were treated successfully using decompression.

These studies suggest a clear link between IAH and the development of AKI. In addition, evidence regarding the impact of IAH comes from studying the effect of IAP-lowering interventions. In a review of 10 studies reporting IAP before and after decompressive laparotomy, IAP decreased from 35 to 16 mm Hg. Although these studies showed an inconsistent effect on kidney function, postdecompression urinary output increased in most. Although the timing of decompression, now considered to be an important element, was not always reported, the very high IAP before decompression suggests that the injurious process may have been progressing for longer periods. Accordingly, acute tubular necrosis may have developed, potentially explaining the variable results. Beneficial effects of nonsurgical treatment options have been described, including improvement after abdominal paracentesis in patients with hepatorenal syndrome and after the administration of neuromuscular blockers. Similar to recent consensus for a classification system for AKI, IAH now also has a more systematic definition and grading system. Until 5 years ago, the impact of less severe IAH on AKI was virtually unrecognized. We are hopeful that the systematic application of both IAH severity classes, as well as AKI stages (using RIFLE or AKIN [Acute Kidney Injury Network] criteria), may increase knowledge and awareness of the impact of earlier phases of IAH and less severe IAH on kidney function, as well as lead to more early and efficacious interventions.

**PATHOPHYSIOLOGY**

IAH can negatively affect the function of organs both inside and outside the abdominal cavity. In this review, we focus on the pathologic process that has direct implications on the clinical care of the critically ill or injured in this section, with the specific effects of IAH on the kidney extensively reviewed in the next section.

Several factors account for the effects of IAH on the cardiovascular system, all of which ultimately decrease cardiac output, even if systemic blood pressure is not obviously affected. First, reflecting cranial displacement of the diaphragm during IAH, intrathoracic pressure is increased. Both animal experiments and studies of humans show that 20%-80% of IAP is transferred to the thorax, resulting in compression of the heart and a decrease in end-diastolic volume. Moreover, cardiac preload also is reduced because of a decrease in venous return from the abdomen (and possibly also the lower limbs). Third, because of direct compression of vascular beds, systemic afterload initially is increased. Finally, generalized vasoconstriction occurs, likely reflecting activation of the sympathetic nervous system and renin-angiotensin-aldosterone system, the latter presumably caused by decreased blood flow to the kidney associated with IAH. Although mean arterial blood pressure (MAP) may increase at first because of blood moving out of the abdominal cavity, it soon stabilizes or decreases. In critically ill and ventilated patients, these cardiovascular manifestations are wors-
duced by hypovolemia and use of positive end-expiratory pressure. The increase in intrathoracic pressure also effectively increases all manometric measurements obtained in the thorax, including central venous pressure, pulmonary artery occlusion pressure, and pulmonary artery pressure. Thus, the measured values may have different interpretations than they would in an individual without IAH. This finding has important implications if not considered. The Surviving Sepsis Campaign guidelines recommend that resuscitation target a central venous pressure of 8-12 mm Hg; based on the physiologic process discussed, higher targets should be used in patients with IAH. The respiratory system also is affected by the transmission of IAP to the thorax because IAH decreases total respiratory system compliance (by a decrease in chest wall compliance) at the same time that lung compliance is essentially constant. This leads to increased inspiratory pressures or decreased tidal volumes, depending on the mode of mechanical ventilation, such that increased positive end-expiratory pressure may be required to adequately oxygenate patients with IAH and ACS. Moreover, both animal studies and studies of humans have suggested a direct relationship between IAP and intracranial pressure. A number of previous publications have argued that the increase in intracranial pressure arising from IAH is explained by increased intrathoracic pressure, which results in increased central venous pressure and decreased venous return from the brain and subsequent venous congestion and brain edema. Refractory intracranial hypertension has been treated successfully using abdominal decompression or neuromuscular blockers.

Because pressures are well transmitted throughout the thoracoabdominal cavities, intra-abdominal organs beyond the kidney also are affected: IAH causes diminished perfusion of all intra-abdominal organs, including the gut, liver, and pancreas, and causes mucosal acidosis. ACS leads to splanchnic hypoperfusion that may occur without hypotension or decreased cardiac output. As shown in animal experiments, this may lead to increased mucosal permeability and bacterial translocation, particularly when it occurs with ischemia-reperfusion injury.

EFFECTS OF IAH AND ACS ON THE KIDNEY

Because of its location deep within the posterior retroperitoneal space, the kidney is especially vulnerable to the deleterious effects of increased IAP resulting in AKI (IAH-AKI).

Hemodynamic Effects

Blood flow both to and from the kidney is impaired in patients with IAH because IAH decreases cardiac output (to a variable extent), leading to decreased arterial blood flow to the kidney. However, decreased cardiac output likely is not the primary cause of IAH-AKI because animal experiments have shown that correction of cardiac output by volume loading does not prevent IAH-AKI. However, the role of volume status remains unclear because one study showed a beneficial effect on kidney function after volume expansion. Possibly this observed benefit may occur in only extreme volume depletion.

In addition to decreased cardiac output and systemic perfusion, local arterial inflow to the kidney may be impaired by IAH. Highlighting the importance of IAP to kidney perfusion, kidney perfusion pressure can be defined as MAP minus IAP. Therefore, in theory, decreased kidney function can be prevented by either decreasing IAP or increasing MAP, either of which will increase kidney perfusion pressure. Ulyatt suggested that filtration gradient (FG) is a more appropriate parameter to explain AKI associated with IAH. FG reflects the balance between hydrostatic forces in glomerular capillaries that promote fluid transfer into Bowman space and oncotic forces that promote transfer into glomerular capillaries. In normal physiologic states, hydrostatic pressure in Bowman space (and therefore in the proximal tubules) is negligible, promoting glomerular filtration; accordingly, glomerular filtration pressure can be approximated as equal to kidney perfusion pressure and thus equal to MAP – IAP. However, in the presence of IAH, Bowman space and proximal tubular pressure will be close to IAP; therefore, FG can be approximated as FG = MAP – (2 × IAP), assuming that autoregulation of glomerular perfusion is not present. Although mechanistically attractive, no clinical evidence presently supports this hypothesis for the development of IAH-AKI.

Several studies also have focused on the influence of IAH on venous kidney blood flow. In animal models, IAH leads to increased venous pressure and decreased venous blood flow, which also leads to decreased renal arterial blood flow and decreased perfusion of the renal cortex. Direct Effects on the Kidney and Ureter

Another study by Doty et al of the influence of increased parenchymal pressure in the kidney found that direct parenchymal compression had no effect on cardiac output, MAP, renal blood flow, or inulin clearance. Interestingly, Stone and Fulenwider subjected animal kidneys to ischemia by aortic cross-clamping and decapsulating 1 kidney. After 8-20 days,
the capsulated kidney had significantly less kidney function than the decapsulated contralateral kidney. This led to the term renal compartment syndrome and suggests that parenchymal compression may be an important contributor to AKI when it is applied in an injured kidney that is subjected to ischemia-reperfusion injury (which may better reflect clinical IAH). Finally, IAH is not believed to lead to postrenal AKI through ureteral compression because placement of ureteral stents has not resolved IAH-AKI.80

Unfortunately, the kidney vasculature is not readily accessible for monitoring in a clinical setting, which has been a major obstacle for both clinical research and bedside monitoring. Bedside ultrasound measurements of renal artery resistance index have been suggested as a monitoring tool.43,81

**IAH IN CLINICAL NEPHROLOGY PRACTICE: SUMMARY POINTS**

**AKI in Critically Ill Patients**

Given the frequency of IAH in critically ill patients, the dose-dependent effect of IAH on kidney function, and the identification of IAH as an independent risk factor for AKI,37 IAH should be considered in every patient with AKI in the ICU. When IAH is present in an oliguric patient, fluid resuscitation can be continued, but IAP should be monitored carefully and crystalloid use should be avoided or limited. Specific medical treatment options to decrease IAP should be considered (discussed later).

**Kidney Transplant**

As discussed, it has been reported that IAH also may complicate kidney transplant, but may be treated successfully using decompression.39,40 Mesh abdominal closure or other techniques have been reported to treat or prevent this complication successfully.82-84 Although clinical diagnosis may be difficult, Doppler ultrasound is an invaluable tool to detect this complication. Reversed diastolic blood flow in interlobar and segmental renal arteries plus minimized venous flow have been proposed as objective criteria indicative of retroperitoneal compartment syndrome.40

**Hepatorenal Syndrome and Severe Ascites**

Hepatorenal syndrome likely reflects prerenal decreased kidney function, occurs in patients with liver failure and ascites, and does not respond to volume loading. The pathophysiologic mechanism leading to hepatorenal syndrome is believed to be splanchnic and systemic vasodilatation leading to kidney hypoperfusion. Paracentesis has been performed as treatment for hepatorenal syndrome, but generally was not advocated because of fears of inducing hypovolemia and further exacerbating kidney impairment. However, several investigators have hypothesized that IAH may be an important contributing factor in the pathogenesis of hepatorenal syndrome, with observations in small studies that paracentesis and parenteral administration of albumin may lead to improved kidney function in critically ill patients with cirrhosis admitted with variceal bleeding, as well as in stable patients with hepatorenal syndrome.42,85 These findings warrant further study.

**MANAGEMENT OF IAH AND ACS**

Tremendous progress has been made in the management of IAH and ACS. For a long time, surgical decompression was considered the only option for patients who developed overt ACS and that IAH was an irrelevant epiphenomenon of critical care. However, in a prospective study, Cheatham and Safcsak86 found that an integrated and diligent approach to comprehensively manage IAH/ACS led to decreased mortality, earlier and higher abdominal closure rates, and decreased costs. Whereas the earlier focus often was on the end stage of IAH, namely full-blown ACS, this study was the first to show that aggressively managing IAH in patients at highest risk can improve outcomes. Accordingly, contemporary management of patients with IAH/ACS is based on 4 elements (Fig 1): IAP measurement, prevention, medical management, and surgical management.

**IAH Monitoring**

An essential first step in management is early recognition of IAH.87 The only way to accomplish this is through awareness of IAH as an important clinical condition, and then by monitoring IAP in patients who are at risk. The WSACS has listed a number of conditions associated with IAH (Box 2) and recommends screening for these risk factors at ICU admission and when organ dysfunction occurs during an ICU stay.2,88 In at-risk patients, IAP should be assessed at baseline, and if there is IAH, IAP
should be monitored at least every 4-6 hours throughout the course of critical illness. 

Prevention of IAH and ACS

Prevention of IAH and ACS has been studied most extensively in patients with primary ACS. In patients with trauma and patients after ruptured abdominal aortic aneurysms, prophylactic use of open-abdomen strategies has proved to be beneficial, although it sometimes can be a complicated undertaking. Decreased postinterventional IAH is another benefit of nonsurgical interventional techniques for repair of ruptured aneurysms. As discussed, fluid resuscitation is an important contributor to IAH in critically ill patients, and as discussed in the next section, there is evidence in burn patients that colloid and hypertonic lactated saline lead to lower IAP incidence than crystalloid-based resuscitation. Finally, because ACS typically is the final stage of prolonged exposure to IAH, it may be prevented or ameliorated through treating IAH using medical management options.

Medical Management

Medical interventions aimed at decreasing IAP target the 3 important contributors to IAH: (1) solid-organ and hollow-viscera volume; (2) space occupying lesions, such as ascites, blood, fluid, or tumors; and (3) conditions that limit expansion of the abdominal wall. When using medical management options to decrease IAP, it is important to always consider individualized pathophysiologic mechanisms leading to IAH because these may differ considerably from one patient to another. Critically, in patients with IAH, small changes in intra-abdominal volume may have a pronounced effect on IAP.

Ileus is a common finding in critically ill patients, especially those with abdominal conditions such as pancreatitis, peritonitis, and abdominal trauma, and postoperative patients. Nasogastric drainage can be a simple first step to decrease IAP in these patients. When colonic ileus is most pronounced, insertion of a rectal cannula can produce similar effects. Administration of prokinetic agents, such as metoclopramide or erythromycin, often is used to overcome abdominal distention and ileus and thus is a treatment option for IAH. When such pharmacologic measures are unsuccessful in decreasing intraluminal volume, endoscopic decompression can be considered.

Ascites and blood are the most common components of space-occupying lesions, but abscesses and free air also can contribute to IAH. When located in the free intraperitoneal space, these collections may be easy targets for percutaneous drainage, which can be performed at the bedside in the ICU under ultrasound guidance. Limited abdominal wall compliance also may be an important contributor to IAH. Increased abdominal muscle tone, most often due to pain or agitation, can be relieved by adequate analgesia and sedation if necessary. Use of restrictive bandages should be avoided. Neuromuscular blockade repeatedly has effectively decreased IAP in patients with IAH. A trial with neuromuscular blocking agents could be considered when simpler measures are not sufficient or are ineffective, and continuous infusion of these agents could be considered when a clinically relevant effect is shown.

Fluid resuscitation also may contribute to the development of IAH, a risk that seems especially relevant in patients with capillary leakage, in which fluids accumulate in the bowel wall and mesentery, free peritoneal cavity, retroperitoneum, and abdominal wall. This mechanism was described first in trauma patients, but numerous studies of both general ICU and postoperative patients have confirmed that overall positive fluid accumulation is a risk factor for IAH. Furthermore, as mentioned, 2 randomized controlled trials in burn patients clearly showed that limiting fluid resuscitation by using colloids or hypertonic solutions results in a lower IAP incidence than standard resuscitation schemes based on crystalloids. Similarly, in patients with acute pancreatitis, low-volume resuscitation is associated with a decreased incidence of ACS.

This role of fluid accumulation in IAH suggests that all efforts should be made to remove excessive fluid from a volume-overloaded patient with overt ACS. In addition, when a patient’s acute illness has subsided and fluid accumulation is the cause of a more chronic form of IAH, a similar strategy may be warranted. Depending on the clinical situation, either ultrafiltration or diuretics can be used.

The WSACS recently proposed a medical treatment algorithm based largely on expert opinion that is aimed at both decreasing IAP and optimizing fluid resuscitation and systemic perfusion (Fig 2). The medical treatment options discussed may be applied in a stepwise fashion; critically, the present level of evidence supporting these and other elements of this algorithm is limited, and the separate elements are not supported by clinical outcome data. However, this algorithm was part of an integrated approach that Cheatham and Safcsak found to improve outcome and decrease hospital costs.

Surgical Management

If attempts to decrease IAP using medical treatment are not effective, formal decompressive laparotomy should be considered. Also in patients with rapidly progressive organ dysfunction caused by IAH, early surgical decompression may be indicated because,
although invasive, decompressive laparotomy is effective in decreasing IAP and improving organ function. However, if overt ACS has occurred, IAP often remains in the range of 12-20 mm Hg, and despite often dramatic improvements, true normalization of organ function rarely is observed.\textsuperscript{41} The timing of the intervention thus also is important; for example, Mentula et al\textsuperscript{101} described poor outcomes in patients with severe acute pancreatitis when decompression was performed more than 4 days after admission to the ICU.

A full midline laparotomy from the xiphoid down to the pubis is the technique most commonly used, but other less invasive modalities have been developed, with subcutaneous linea alba fasciotomy one of the most promising approaches.\textsuperscript{102} Obviously, decompression means that the patient is left with an open abdomen, which can result in serious fluid losses, infection, enterocutaneous fistulas, ventral hernia, and cosmetic concerns. Temporary abdominal closure techniques have improved significantly in recent years, leading to lower complication rates and earlier fascial closure rates. Several more detailed reviews of available surgical techniques have been published recently.\textsuperscript{103,104}

\section*{CONCLUSIONS}

IAH frequently occurs in critically ill patients, and multiple factors often contribute to the problem. It may affect all organ systems, but respiratory, cardiovascular, and kidney function are affected most often. IAP monitoring is a first and essential step in the diagnosis and treatment of IAH, can be performed easily in any ICU, and also is applicable in less intensive hospital settings.
Intra-abdominal Hypertension and AKI

IAH can be the sole cause of or contributing factor in AKI and other kidney-related problems, such as delayed transplant function after kidney transplant and hepatorenal syndrome. Therefore, it is important for every nephrologist and intensivist caring for patients with acute or chronic kidney disease to be aware of the existence of IAH, its pathologic implications, and available methods to decrease IAP. Several nonsurgical interventions are available and may avoid the need for surgery. If medical management fails and the patient progresses to ACS, surgical decompression should be performed without delay.

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