# Intra-Abdominal Hypertension and Abdominal Compartment Syndrome: An Underappreciated Cause of Acute Kidney Injury

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**Intra-abdominal hypertension (IAH) and abdominal compartment syndrome are increasingly recognized in both medical and surgical critically ill patients and are predictive of death and the development of acute kidney injury. Although there are many risk factors for the development of IAH, in the era of goal-directed therapy for shock, brisk volume resuscitation and volume overload are the most common contributors. Abdominal examination is an unreliable predictor of intra-abdominal pressure (IAP), but IAP can be easily measured in a reproducible and reliable manner by a number of simple bedside techniques. Prompt recognition and intervention to decrease IAP and improve vital organ perfusion are essential to minimize the negative effects of IAH on somatic and visceral organ functions.**



**Key Words:** Intra-abdominal hypertension, Abdominal compartment syndrome, Abdominal perfusion pressure, Acute kidney injury (AKI)

## INTRODUCTION

Elevated pressure in a closed space has long been accepted as a cause of profound morbidity often leading to death in humans and animals. Tension pneumothorax, pericardial tamponade, and obstructive hydrocephalus or cerebral edema leading to cardiopulmonary or central nervous system collapse serve as potent examples of the lethal effects that arise from elevated pressures in closed spaces. Although it has been known for over 100 years that significant elevations of intra-abdominal pressure (IAP) has marked negative effect on organ function, historically, this was felt to be primarily a consequence of abdominal surgery or abdominal/visceral trauma. However, over the last 20 years, there has been a growing recognition that elevations of IAP occur with striking frequency in the general critically ill patient, and that even minor elevations in abdominal pressures may have wide-ranging physiologic effects on multiple organs.[1,2]

This article will review the definitions of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), discuss the prevalence of these findings among the critically ill, and explore the pathophysiology of IAH/ACS and its role as a cause of acute kidney injury (AKI), techniques for measuring IAP, and treatment strategies with a particular focus on preventing and ameliorating AKI. Finally, we will propose future directions and opportunities for clinical research.

## DEFINITIONS

The World Society of Abdominal Compartment Syndrome (now called "WSACS—the Abdominal Compartment Society" [WSACS])—an international multispecialty consortium of critical care specialists from internal medicine, surgery/trauma, and anesthesiology—published a consensus definition for IAH and ACS in 2006[3] and most recently revised in 2013.[4] IAP is the "steady state pressure concealed within the abdominal cavity."[3,4] The normal value ranges from 0 to 5 mm Hg in healthy adults and transiently rises with activities that cause an increase in intra-thoracic pressure such as coughing or sneezing. Increasing IAP also develops slowly in patients who are pregnant, obese, or with chronic ascites (cardiac or cirrhosis). However, as these processes tend to develop slowly over time, it has been postulated that the body adapts accordingly, and the individual may experience no adverse effects.[1] However, the pathophysiologic effects of these slow rises in IAP remain incompletely understood, and there are increasing concerns that elevated IAP may indeed contribute to end-organ dysfunction as seen in the hepatorenal or cardiorenal syndromes.[5-8]

WSACS defines IAH as a sustained or repeated pathologic elevation of IAP $\geq$ 12 mm Hg and ACS as a sustained elevation of IAP of >20 mm Hg with new organ dysfunction.[2,4] WSACS further divides IAH into 4 grades (Table 1),[4] but the clinical utility of these grades is less certain. In addition, abdominal perfusion pressure (APP) is the net arterial perfusion pressure in the abdominal vasculature after accounting for the impact of IAP. APP is equal to mean arterial pressure (MAP) − IAP. APP, compared to IAP alone, often informs a more complete picture of visceral organ perfusion.

## PREVALENCE IAH AND ACS IN CRITICAL ILLNESS

Over the last 10 to 15 years, it has become clear that IAH and/or ACS have a high prevalence in intensive care unit

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(ICU) patients across the spectrum of critical illness (emergent/elective admission, medical/surgical/trauma, and so forth). Using the aforementioned IAP cutoffs for IAH and ACS, in 2004, Malbrain and colleagues published a multicenter 1-day point prevalence study across 13 different medical and surgical ICUs in 6 countries, which found that 58.8% of patients had IAH, and 8.2% of them met criteria for ACS.[9] Over the subsequent 10 years, multiple studies have demonstrated similar findings—namely that the prevalence of IAH ranges from 30% to 85% and ACS 5% to 50%[10-18] (Table 2). It is important to note that highest rate of ACS usually occurs in settings of abdominal trauma, surgery, or other abdominal pathology (ie, severe acute pancreatitis). However, an IAH prevalence as high as 80% has been seen in medical ICUs in patients with septic shock although these patients usually lack clear abdominal pathology. In addition, approximately 30% to 50% of patients after coronary artery bypass grafting have been shown to have IAH.[19] Outside those patients with very high risk (such as severe acute pancreatitis, trauma, and so forth), frank ACS appears to occur in 5% to 25% of patients. Recognizing this is a large range, the true prevalence of ACS is likely closer to the lower end of this range as those studies enrolling a higher number of patients tend to show frank ACS prevalence in the single digits.

**CLINICAL SUMMARY**

1. Intra-abdominal hypertension (IAH) and abdominal compartment syndrome are common complications of critical illness whose risk factors are common in the critically ill.
2. By decreasing abdominal perfusion pressure and increasing venous congestion, IAH is an underrecognized cause of acute kidney injury in the intensive care unit especially in patients dependent on vasopressors.
3. Intra-abdominal pressure should be measured in critically ill patients with risk factors for IAH, and physical examination has poor sensitivity for IAH and abdominal compartment syndrome.
4. Lowering intra-abdominal pressure and increasing abdominal perfusion pressure may ameliorate or prevent acute kidney injury, but more research is needed.

## IAH/ACS RISK FACTORS

IAH/ACS is divided into primary (ie, surgical or trauma) or secondary (medical) based on the underlying cause/pathology.[20] Abdominal trauma (including visceral injury), major abdominal surgery, and severe acute pancreatitis are frequent causes of primary IAH and ACS. Risk factors for (and causes of) secondary IAH/ACS are divided into 4 general categories on the basis of pathophysiology (Table 3): (1) diminished abdominal wall compliance; (2) increased intra-luminal visceral contents; (3) increased abdominal cavity contents; and (4) fluid resuscitation/capillary leak. Among the critically ill, the most common risk factors for IAH/ACS are large volume/fluid resuscitation, sepsis, and polytransfusion. In the era of goal-direct therapy of sepsis, large volume resuscitation appears to be the primary risk factor for IAH in the critically ill patient,[11,12,18] and there appears to be a linear correlation between volume expansion and increasing IAP.[19]

## DIAGNOSING INTRA-ABDOMINAL HYPERTENSION

Direct measurement of IAP via an intra-peritoneal catheter is impractical secondary to the attendant risk of visceral injury, bleeding, and introducing infection into the intra-peritoneal cavity. Transducing the pressure inside a hollow intra-abdominal organ is an indirect measure of IAP with intragastric and intravesicular techniques published. Measuring intravesicular (bladder) pressure in a standardized fashion is well validated and is currently the gold standard for IAP measurement. The WSACS consensus statement recommends IAP measurement using an intravesicular method in all critically ill or injured patients with known risk factors for IAH/ACS (grade 1C recommendation) and to continue intermittent monitoring every 4 hours at a minimum when IAP $\geq$ 12 mm Hg (grade 1C).[4] There are many published techniques for intermittent measurement of bladder pressure using either standard ICU equipment (ie, Foley catheter, pressure tubing, and so forth) or by using commercially available closed system products.[21] Lee[20] published an excellent review of various techniques for bladder pressure measurements as of 2012. To summarize, bladder pressure measurements involve the following steps:

1. Insert a Foley and fully drain the bladder (assuming no contraindications to Foley catheter insertion).
2. Position patient supine and flat. Ensure pain and agitation are adequately treated before obtaining a measurement as they can lead to falsely elevated readings.
3. Attach the Foley side port to a sterile pressure transducer system (ie, central venous pressure transducer).
4. Place the pressure transducer at iliac crest in the midaxillary line and zero the pressure transducer.
5. Instill 20 to 25 mL of fluid (0.9% NaCl most commonly used) into the bladder via Foley catheter side port and temporarily clamp the Foley to retain fluid in bladder.
6. Wait 20 to 30 seconds to allow for relaxation of the bladder detrusor muscle and transduce pressure at end-expiration.
7. After measurement, unclamp Foley and return the head of the bed to its original position.

Proper technique for bladder pressure measurement is important to accurately measure IAP with attention to transducer positioning (at the iliac crest in the midaxillary line), appropriate fluid volume in bladder (25 mL) and allowing sufficient time of approximately 30 seconds for relaxation of bladder detrusor muscle before measurement.[2,20] Instillation of too much fluid or failure to allow the bladder to completely relax will falsely elevate measurement. Body positioning does impact accurate measurement of IAP with at least 1 study demonstrating