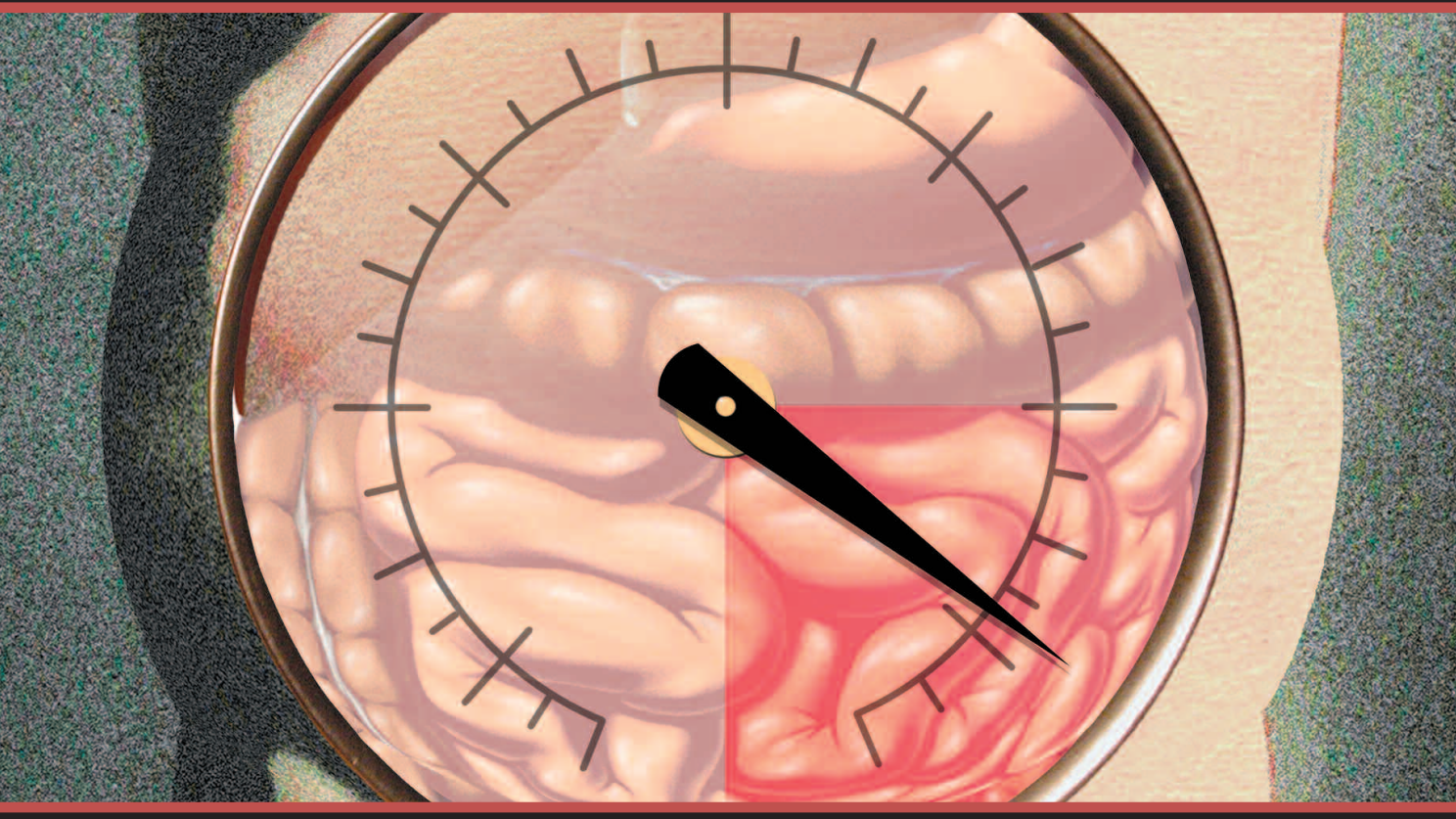
UNDER PRESSURE



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By Thomas T. Levins RN, CCRN, CFRN, BSN

Rapid identification and treatment of intra-abdominal hypertension and abdominal compartment syndrome may save lives.

A 53-year-old male is admitted to the intensive care unit (ICU) from the emergency department (ED) with a diagnosis of pneumonia and septic shock. In the ED he was hemodynamically unstable and received a total of 9 liters of intravenous (I.V.) fluids to raise his central venous pressure (CVP) to 12 mm Hg, and was started on a norepinephrine infusion titrated to maintain a mean arterial pressure (MAP) of greater than 60 mm Hg. On arrival in the ICU he remains unstable with MAP's in the low 60s with a CVP of 18 mm Hg, poor urinary output, and increasing peak inspiratory pressures (PIP) (greater than 35 cm H_2O). What do you suspect?

Illustration by Craig Kiefer

Pathophysiology

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are both complications of critical illness and injury. Traditionally, ACS was considered to be a complication of abdominal trauma. Recently, ACS has been noted to be a problem in critically ill patients without trauma, especially those with the systemic inflammatory response syndrome.[1]

The primary pathophysiologic event causing IAH and ACS is interstitial edema of the bowel and mesentery secondary to capillary endothelial damage. The capillary damage is due to the ischemia related to the original physiologic insult (sepsis, hemorrhage, or trauma) and the release of proinflammatory cytokines secondary to the original insult. This may result in significant edema. As fluid accumulates, the abdominal wall and fascia are slowly stretched, compliance is decreased, and increased intra-abdominal pressure (IAP) results. Increased IAP results in many physiologic effects secondary to diminished organ perfusion. It also causes immune and inflammatory responses that can result in progressive organ failure. The most common time frame for developing IAH is within 8 to 16 hours following initial tissue insult, also the period of maximal immune responsiveness.[2]

Intra-abdominal pressure is normally 0 to 5 mm Hg in healthy adults and may be commonly elevated as high as 5 to 7 mm Hg in many ICU patients. Intra-abdominal hypertension is defined as IAP greater than 12 mm Hg, and ACS may result with pressures greater than 20 to 25 mm Hg. The reported incidence ranges from a few percent to over 40%.

As of 2004, only one study has systematically measured IAP in patients in multiple ICUs to determine the incidence of IAH and ACS. The results are impressive, with over 50% of patients studied exhibiting IAH (IAP greater than 12 mm Hg), over 28% exhibiting severe IAH (IAP greater than 15 mm Hg), and over 8% exhibiting ACS (IAP greater than 20 mm Hg).[3] Predicting patients who are at risk for IAH /ACS has proven to be difficult, as no specific type of disease process or patient has been identified to be at risk for IAH/ACS. The authors of this study recommend the routine measurement of IAP. The World Society of Abdominal Compartment Syndrome defines IAH as "The pathologic elevation of intra-abdominal pressure," and divides intra-abdominal hypertension into four grades of severity. (See "Grading IAH.")

Specific manifestations of IAH

How does IAH affect end-organ function and certain body systems? Consider the following:

Grading IAH

Sustained pressure at 12 mm Hg or higher indicates IAH. The grades and other patient criteria help determine treatment.

Grade	IAP
I	12 to 15 mm Hg
II	16 to 20 mm Hg
III	21 to 25 mm Hg
IV	Greater than 25 mm Hg

Source: World Society of the Abdominal Compartment Syndrome.

Gastrointestinal: Edema within the gastrointestinal cavity leads to distention of the abdomen and increased IAP.
Cardiovascular: Elevated IAP compresses the inferior vena cava, reducing venous return to the right side of the heart and decreasing preload and cardiac output.
Pulmonary: Elevated IAP displaces the diaphragm cephalad, reducing intrathoracic volume and increasing intrathoracic pressure. To maintain adequate tidal volumes, the PIPs increase, causing barotrauma, hypercapnia, and hypoxemia. The inflammatory mediators released also result in pulmonary capillary damage and interstitial edema. The elevated pulmonary pressures also reduce venous return to the right side of the heart and falsely elevate CVP and pulmonary artery occlusion pressure (PAOP) readings.
Renal: Compression of the retroperitoneal and renal parenchyma by IAH leads to poor renal perfusion and congestion of the kidney. The reduced cardiac output also contributes to reduced renal perfusion. This mechanism decreases the glomerular filtration rate, decreases urine output, and can lead to renal insufficiency, and eventually renal failure.
Neurologic: Elevated IAP reduces venous return from the brain secondary to increased pressures within the superior vena cava. This may result in increased intracranial pressure and decreased cerebral perfusion pressure. (See "Pathophysiologic implications of IAH on end-organ function.")

Hemodynamic parameters

Numerous studies have shown that critically ill and injured patients who have their cardiopulmonary function optimized experience reduced organ failure, reduced mortality, and better outcomes.[4] However, elevated IAP has significant effects on measured hemody-

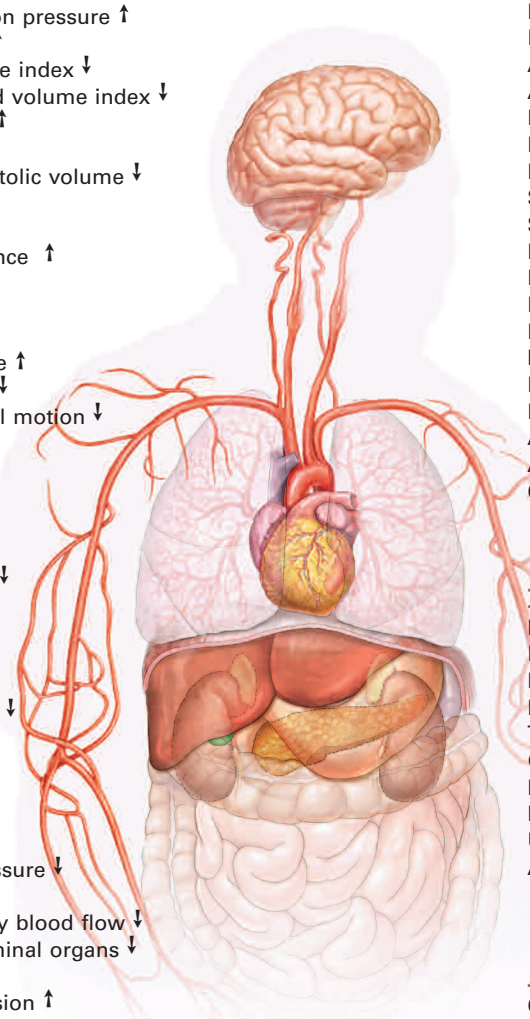
Pathophysiologic implications of IAH on end-organ function

Central nervous system
Intracranial pressure ↑
Cerebral perfusion pressure ↓

Cardiovascular system*
Difficult preload assessment
Pulmonary artery occlusion pressure ↑
Central venous pressure ↑
Intrathoracic blood volume index ↓
Global end diastolic blood volume index ↓
Extravascular lung water ↑
Stroke volume ↓
Right ventricular end diastolic volume ↓
Cardiac output ↓
Venous return ↓
Systemic vascular resistance ↑
Venous thrombosis ↑
Heart rate ↑
Mean arterial pressure ↓
Pulmonary artery pressure ↑
Left ventricle compliance ↓
Left ventricle regional wall motion ↓

Hepatic system
Hepatic arterial flow ↓
Portal venous blood flow ↓
Portocollateral flow ↑
Lactate clearance ↓
Glucose metabolism ↓
Mitochondrial function ↓
Cytochrome P450 function ↓

Gastrointestinal system
Abdominal perfusion pressure ↓
Celiac blood flow ↓
Superior mesenteric artery blood flow ↓
Blood flow to intra-abdominal organs ↓
Mucosal blood flow ↓
Mesenteric vein compression ↑
Intramucosal pH ↓
Regional CO_2 ↑
Successful enteral feeding ↓
Intestinal permeability ↑
Bacterial translocation ↑
Multiple organ failure ↑
Gastrointestinal ulcer (re)bleeding ↑
Variceal wall distress ↑
Variceal (re)bleeding ↑



Respiratory system
Intrathoracic pressure ↑
Pleural pressure ↑
Functional residual capacity ↓
All lung volumes ↓
Auto-PEEP ↑
Peak airway pressure ↑
Plateau airway pressure ↑
Dynamic compliance ↓
Static respiratory system compliance ↓
Static chest wall compliance ↓
Hypercarbia ↑
PaO_2 ↓ and PaO_2/FiO_2 ↓
Dead-space ventilation ↑
Intrapulmonary shunt ↑
Extravascular lung water ↑
Prolonged ventilation
Difficult weaning
Activated lung neutrophils ↑
Alveolar edema ↑
Compression atelectasis ↑

Renal system
Renal perfusion pressure ↓
Filtration gradient ↓
Renal blood flow ↓
Diuresis ↓
Tubular dysfunction ↑
Glomerular filtration rate ↓
Renal vascular resistance ↑
Renal vascular compression ↑
Ureteral compression ↑
Antidiuretic hormone ↑

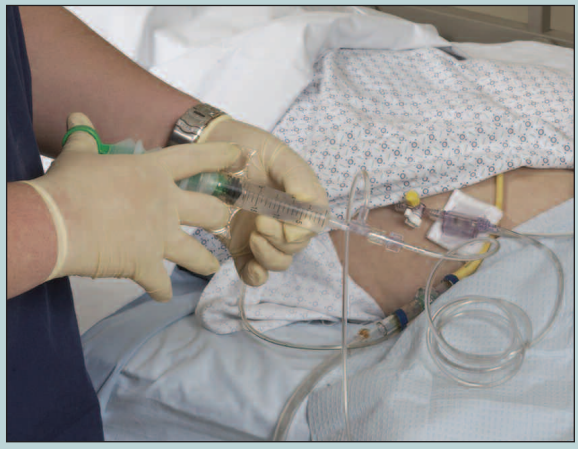
Abdominal wall
Compliance ↓
Rectus sheath blood flow ↓
Wound complications ↑
Incisional hernia ↑

Endocrine system
Release pro-inflammatory cytokines ↑ (IL-1b-TNF-a, IL-6)

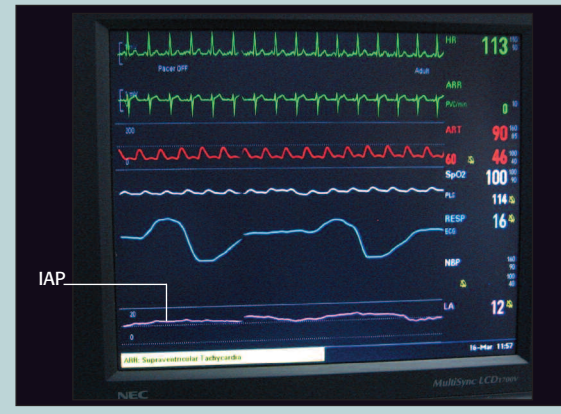
*Cardiovascular effects are exacerbated in case of hypovolemia, hemorrhage, ischemia, and high PEEP ventilation.

Adapted from Malbrain M. Is it wise not to think about intraabdominal hypertension in the ICU? *Curr Opin Crit Care.* 2004;10:132-145.

IAP monitoring technique



1. Draw 20 mL of saline into the syringe and inject it into the patient's bladder with enough force to actuate the valve (not too slowly, but not aggressively). The pressure reading will appear on the monitor for 60 to 90 seconds.



Photos by Gary Donnelly

2. Let the system equilibrate and take the reading at end expiration, when IAP is the lowest. After 60 to 90 seconds, the valve will automatically open and drain and the pressure will return to zero. Using the S hook, hang the syringe in a convenient location until you need it for the next pressure reading.

namic parameters, making patient management challenging. As mentioned earlier, elevated IAP causes reduced intrathoracic volume and increased intrathoracic pressure, the latter of which falsely elevates the CVP and PAOP. This false elevation doesn't accurately reflect volume and may lead to under fluid resuscitation.[5]

Elevated IAP also causes compression of the inferior vena cava, which results in reduced venous return to the right side of the heart, leading to decreased preload, reduced stroke volume, and reduced cardiac output. One attempt at improving measured hemodynamic parameters (CVP, PAOP) in patients with IAH has been advocated by Malbrain and Cheatham.[6] The equation is as follows:

CVP corrected = CVP measured – IAP/2

PAOP corrected = PAOP measured – IAP/2

While the effect of IAP on CVP and PAOP is complicated and can't be accounted for by any formula, these formulas may allow more accurate assessment of CVP/ PAOP in patients with IAH. However, in some patients, echocardiography or right ventricular volumetric monitoring may give a more accurate picture of volume status.

Case study

On arrival in the ICU, the patient is found to be hemodynamically unstable with a MAP in the 50s and a CVP of 20 mm Hg. His pulse oximetry reveals an SpO_2 of 90% with PIPs in the 40s. His abdomen is firm and distended. He's restless but responsive to verbal stimuli. What should you do?

Measuring IAP

The detection of increased IAP by physical examination alone was studied in 2000; experts found there was less than 50% correlation in detecting IAH by physical examination alone. They recommended that routine measurements of bladder pressure in at-risk patients should be performed.[7] How does one go about this? The current standard is IAP monitoring through the indwelling urinary catheter. The bladder acts as a passive reservoir and accurately reflects IAP at volumes of 100 mL or less. This monitoring has been shown to be comparable to direct intra-peritoneal measurement of IAP and is less invasive. There are two commonly used techniques to accomplish this. (See "IAP monitoring technique.")

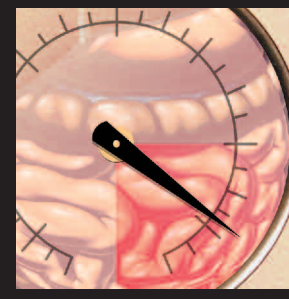
1. Homemade technique: This technique uses a pressure transducer primed with normal saline, stopcocks, 60 mL luer lock syringe, and a needle or Angiocath. The patient should be placed in the supine position. For adults, the bladder is filled with 50 mL of normal saline utilizing sterile technique.[8] For pediatric patients, 1 mL/kg is used.[9] After instillation, the indwelling urinary catheter is clamped. The transducer is zero'd and calibrated at the symphysis pubis. The

IAP is measured at end expiration.

Problems encountered with this technique include the lack of standardization, as setup may vary from clinician to clinician. It's done infrequently and it's time consuming. There's a risk of a needle stick while using the needle or Angiocath to penetrate the system. There's also the risk of infection due to the recurrent penetration of the system.

2. AbViser IAP pressure monitoring kit: This is a closed system that's placed inline with the indwelling urinary catheter. Once it's attached, it's left in place. The patient is placed supine. The bladder is filled with 20 mL for adults and 1 mL/kg up to 20 mL for pediatric patients. The transducer is placed at the symphysis pubis and zero'd. The IAP is measured at end expiration.

One advantage of this system is its standardization; everything is contained in one kit. It's also a closed system which requires no needles for access. Additionally, it's quicker to perform as the system remains in line and once set up, requires no additional set up.



Once optimal volume status is achieved, abdominal blood flow should be optimized through the use of inotropic agents.

Treatment options

True abdominal compartment syndrome is a surgical emergency requiring emergent decompressive laparotomy. On the other hand, IAH, if detected early, may be treated without surgical intervention. If these medical interventions are instituted early, before IAH progresses, ACS may be prevented. Current evidence suggests that IAP measurements should be performed every 1 to 2 hours initially until a trend is established.

When IAH is established, the initial intervention is optimal fluid resuscitation. Once optimal volume status is achieved, abdominal blood flow should be optimized through the use of inotropic agents. If the abdominal perfusion pressure (APP) is less than 60 mm Hg, other interventions may be necessary. The APP is calculated by subtracting the IAP from the MAP. If, despite optimal volume loading and inotropic support, IAP remains elevated, reduction of intra-abdominal contents is appropriate. This is accomplished through the placement of a nasal or oral gastric tube to decompress the stomach, in addition to the use of cathartics, enemas, or rectal tubes to reduce the content of the large bowel. Adequate analgesia and sedation will also assist in reducing IAP in any patient with pain or anxiety. The use of neuromuscular blocking agents may also be necessary to further relax the abdominal wall.

In patients with excess peritoneal fluid (ascites) or retroperitoneal fluid collections, percutaneous drainage may reduce IAP.[10] However, most cases if IAH aren't secondary to fluid collection, they're due to interstitial edema. In investigating the effect of cytokines on IAH, experts found that continuous hemofiltration reduced serum cytokine levels and the overall volume of interstitial fluid.[11]

What's the status?

The patient's current hemodynamic status is: MAP 64 mm Hg, CVP 22 mm Hg, SpO_2 89%, PIP on mechanical ventilation 44, urine output less than 10 mL last hour, and he remains restless and agitated.

The patient is immediately sedated with I.V. midazolam and I.V. fentanyl. Intravenous vecuronium is also administered. While the patient is no longer restless and agitated, his hemodynamic parameters do not change significantly. Bladder pressure monitoring is instituted via the indwelling urinary catheter and reveals Grade III IAH with a measured bladder pressure of 22 mm Hg. His APP is 42 mm Hg. Since his measured CVP is 22 mm Hg, he is felt to be adequately volume loaded. A dobutamine infusion is started and his nasogastric tube is placed to continuous low suction and drains approximately 400 mL of bilious material. His urine output continues to be less than 10 mL/hr. His respiratory profile remains unchanged. Repeat bladder pressure measurements reveal a bladder pressure of 30 mm Hg; IAH has now progressed to ACS, a surgical emergency. He's immediately taken to the operating room (OR) for decompressive laparotomy.

Your patient returns from the OR after decompressive laparotomy. Temporary abdominal closure is achieved with a silastic (plastic) closure, leaving the abdominal incision open. He remains sedated and paralyzed postoperatively. His current hemodynamic status is: MAP 84 mm Hg, CVP 14 mm Hg, SpO_2 98%, PIP on vent 24, urine output 35 mL the past hour.

After completing your initial postop assessment, you measure his bladder pressure. You find his IAP to be 14

mm Hg, which is Grade 1 IAH. His APP is 70 mm Hg. Since his overall hemodynamic profile is markedly improved after decompressive laparotomy, he requires no other intervention at this time other than frequent monitoring of his IAP via the indwelling urinary catheter. You continue to monitor his IAP every 2 hours until a trend is established. He does not develop significant IAH/ACS again. His abdomen is closed on postoperative day 2. He's successfully extubated and the remainder of his hospital course is uneventful.

Early identification

The physical findings of IAH and ACS are easily noted on assessment. The hemodynamic findings of hypotension, elevated central venous pressures, elevated inspiratory pressures, and poor urinary output are classic for this syndrome. The key is to suspect IAH and ACS as the cause of the physical assessment and hemodynamic changes, measure IAP, and institute rapid treatment. Rapid identification and treatment of IAH may prevent the development of ACS and prevent the need for surgical intervention. Rapid identification and treatment of ACS may be lifesaving. M

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The author has disclosed that he has no significant relationship with or financial interest in any commercial companies that pertain to this educational activity.

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