



# Intra-abdominal Hypertension and Abdominal Compartment Syndrome

## A Comprehensive Overview

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Intra-abdominal hypertension has a prevalence of at least 50% in the critically ill population and has been identified as an independent risk factor for death. Yet, many of the members of the critical care team do not assess for intra-abdominal hypertension and are unaware of the consequences of untreated intra-abdominal hypertension. These consequences can be abdominal compartment syndrome, multisystem organ failure, and death. This article provides an overview of the pathophysiology of intra-abdominal hypertension and abdominal compartment syndrome. In addition, the evidence-based definitions, guidelines, and recommendations of the World Society of the Abdominal Compartment Syndrome are presented. (*Critical Care Nurse*. 2012;32[1]:19-32)

### CE Continuing Education

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Define intra-abdominal hypertension and abdominal compartment syndrome
2. Describe 2 methods of measuring intra-abdominal pressure
3. Discuss the management of intra-abdominal hypertension

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Collaborative management of acute and critically ill patients may result in complications associated with therapeutic measures. This article provides a comprehensive overview of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), the nurse's role in assessing and monitoring patients with these entities, and collaborative management of patients who have IAH and ACS.

The concept of IAH was proposed in the late 1800s, forgotten after World War I, and rediscovered near the end of the 20th century.<sup>1</sup> In 2004, a group of international physicians and surgeons formed the World Society of the Abdominal Compartment Syndrome (WSACS).<sup>2</sup> The goal of this new organization was to develop a cohesive approach to the management of IAH and ACS, foster education and research, and develop consensus statements and definitions. WSACS has developed evidence-based definitions, guidelines, and treatment algorithms and has identified evidence-based devices and methods to measure intra-abdominal pressure (IAP).

### Definitions

#### *Intra-abdominal Pressure*

IAP is the steady-state pressure within the abdominal cavity.<sup>2</sup> In

healthy persons, IAP is 0 to 5 mm Hg<sup>3</sup> and varies inversely with intrathoracic pressure during normal breathing. Various factors, such as coughing, sneezing, and loud singing, can cause IAP to increase drastically for short periods and then return easily to baseline. IAP is also increased in persons who are morbidly obese, have chronic ascites, or are pregnant. In these chronic forms, the increase develops slowly and the body adjusts to the change. Patients with chronically increased IAP do not experience the systemic effects of IAH. The mean IAP in critically ill adults is approximately 5 to 7 mm Hg.<sup>4</sup>

### *Intra-abdominal Hypertension*

IAH is a sustained or repeated pathological elevation of IAP of 12 mm Hg or greater.<sup>2</sup> WSACS has developed grades of IAH (Table 1).

### *Abdominal Perfusion Pressure*

Abdominal perfusion pressure (APP) is a measure of the relative

**Table 2** Primary and secondary conditions that cause intra-abdominal hypertension and abdominal compartment syndrome<sup>a</sup>

Primary	Secondary
Blunt/penetrating trauma	Severe intra-abdominal infection
Liver transplantation	Large-volume fluid replacement
Ruptured abdominal aortic aneurysm	Ascites
Postoperative bleeding	Pancreatitis
Retroperitoneal hemorrhage	Ileus
Mechanical intestinal obstruction	Sepsis
Postoperative closure of the abdomen under tension	Major burns
Bleeding pelvic fractures	Continuous ambulatory peritoneal dialysis
	Morbid obesity
	Pregnancy

<sup>a</sup> Based on information from Muckart et al<sup>3</sup> and Malbrain et al.<sup>4</sup>

adequacy of abdominal blood flow. APP is calculated by subtracting the IAP from the mean arterial pressure (MAP):  $MAP - IAP = APP$ . The APP in patients with IAH or ACS should be maintained at 60 mm Hg or higher.<sup>2-4</sup>

### *Abdominal Compartment Syndrome*

ACS is a sustained IAP greater than 20 mm Hg (with or without an APP <60 mm Hg) associated with new organ dysfunction or failure.<sup>2-4</sup>

### **Causes of ACS**

WSACS categorizes conditions that cause ACS as primary (surgical), secondary (medical), and recurrent<sup>4</sup> (Table 2). Primary conditions are ones that need surgical or interventional radiological treatment. Secondary conditions are due to medical causes that do not require surgery or

radiological intervention as an initial therapy. Recurrent conditions are ones in which ACS redevelops after surgical or medical treatment of primary or secondary causes of ACS.

### **Incidence and Prevalence**

Prevalence is a 1-day snapshot of a particular issue or concern. Malbrain et al<sup>5</sup> conducted a prevalence study in 13 intensive care units (ICUs) and assessed 97 patients. The overall prevalence of IAH was 58.8% (IAP >12 mm Hg). Prevalence was 65% in surgical patients and 54.4% in medical patients. However, the medical patients had a higher prevalence of an increased IAP (>15 mm Hg) than did the surgical patients (29.8% vs 27.5%). Also, the medical patients had a higher prevalence of ACS than did the surgical patients (10.5% vs 5%). Differences in IAH prevalence between the medical and surgical patients were not significant.

Incidence is the occurrence of a particular issue or concern over time. Vidal et al<sup>6</sup> studied the incidence of IAP in 83 critically ill patients in a single ICU. A total of 31% of the patients had IAH at the time of admission to

**Table 1** Grading system for intra-abdominal hypertension<sup>a</sup>

Grade	Intra-abdominal pressure, mm Hg
I	12 - 15
II	16 - 20
III	21 - 25
IV	≥25

<sup>a</sup> Based on information from the World Society of the Abdominal Compartment Syndrome.<sup>2</sup> Muckart et al,<sup>3</sup> and Malbrain et al.<sup>4</sup>

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the unit, and the condition developed in another 33% after admission. Compared with patients without IAH, patients with IAH were sicker and had a higher mortality rate (53% vs 27%;  $P = .02$ ). According to logistic regression, IAH was an independent predictor of mortality ( $P = .003$ ). ACS developed in 10 patients (12%), and 8 of the 10 (80%) died.

Reintam et al<sup>7</sup> performed a study to identify the differences in incidence, course, and outcome of primary and secondary IAH and to determine if IAH is an independent risk factor for death. A total of 257 patients receiving mechanical ventilation were enrolled in the study. IAP was measured repeatedly. IAH developed in 95 of the patients; 60 had primary IAH and 35 had secondary IAH. During the first 3 days, mean IAP decreased in the patients with primary IAH and increased significantly ( $P = .05$ ) in those with secondary IAH. The patients with IAH had a significantly higher mortality than did patients without IAH. ICU mortality was 37.9% for patients with IAH and 19.1% for patients without IAH ( $P = .001$ ). The 28-day mortality was 48.4% vs 27.8% ( $P = .001$ ), and the 90-day mortality was 53.7% vs 35.8% ( $P = .004$ ). Patients with secondary IAH had a significantly higher mortality than did patients with primary IAH ( $P = .03$ ). Reintam et al<sup>7</sup> concluded that development of IAH is an independent risk factor for death. They further concluded that compared with primary IAH, secondary IAH does not occur as often, has a different development course, and has worse outcomes.

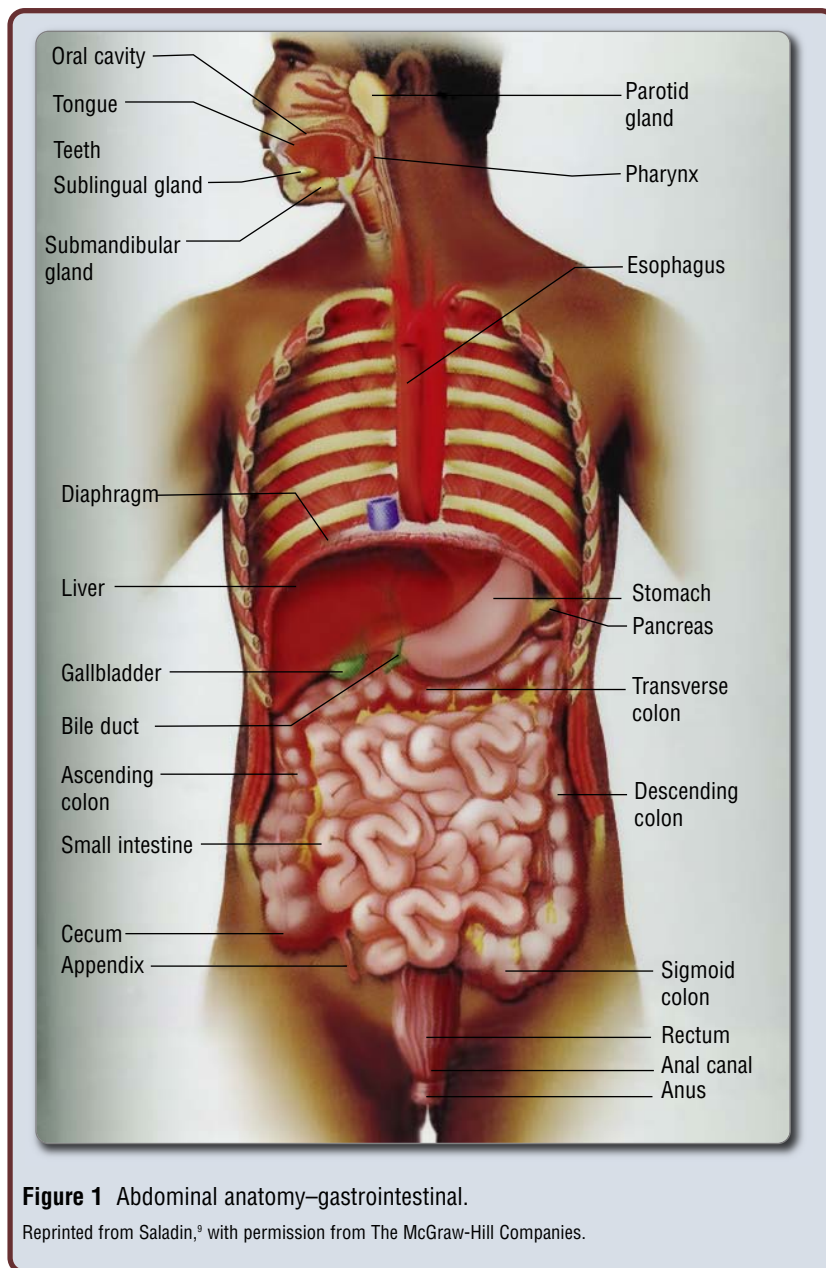
These 3 studies<sup>5-7</sup> indicate that IAH occurs frequently and may worsen patients' outcome. Health

care practitioners tend to be aware of the development of IAH in surgical patients and in patients with obviously distended abdomens. However, the team must also be aware of the implications of IAH in medical patients.

### Pathophysiological Effects of IAH and ACS

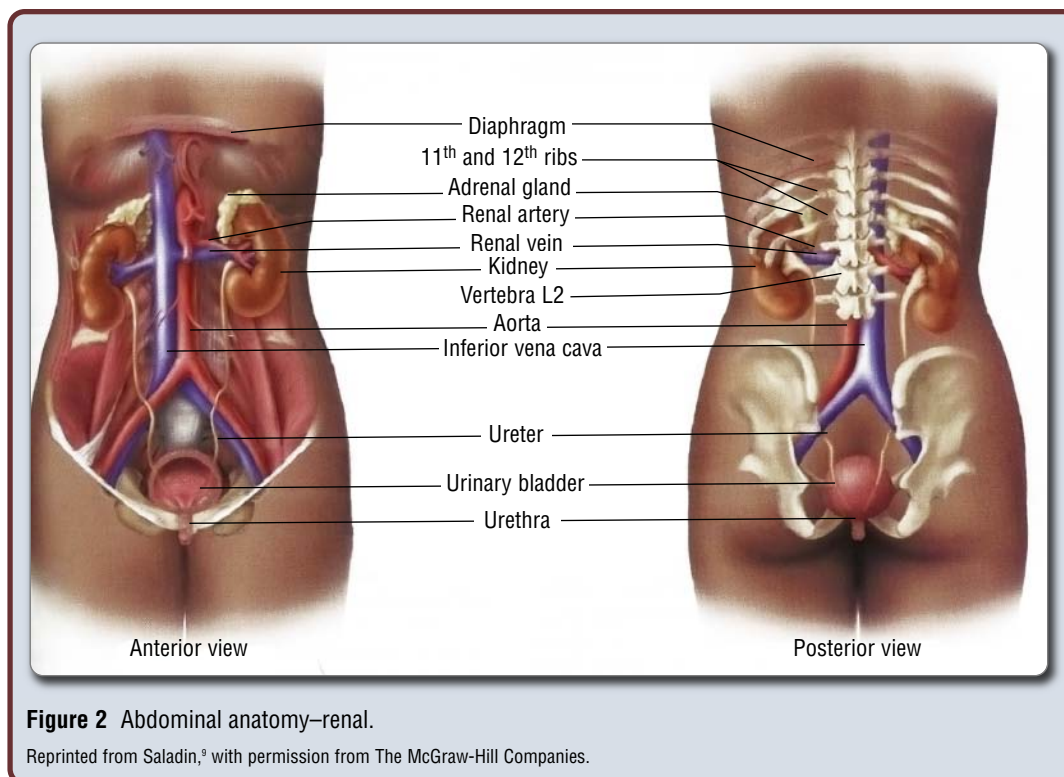
The abdomen can be considered a compartment with the spine, pelvis,

and costal arch as its inflexible edges and the diaphragm and abdominal wall as its more pliable edges.<sup>8</sup> The internal contents of this compartment consist of the stomach, large and small intestine, omentum, liver, spleen, pancreas, gall bladder, kidneys, adrenal glands, ureters, bladder, and, in females, the uterus (Figures 1 and 2). Major blood vessels also course through this compartment. The abdominal aorta,



**Figure 1** Abdominal anatomy—gastrointestinal.

Reprinted from Saladin,<sup>9</sup> with permission from The McGraw-Hill Companies.



**Figure 2** Abdominal anatomy—renal.

Reprinted from Saladin,<sup>9</sup> with permission from The McGraw-Hill Companies.

40%, and pressures up to 40 mm Hg can decrease mesenteric perfusion by 70%.<sup>12</sup> The correction of IAH can lead to ischemia-reperfusion injury and send inflammatory cytokines to other organs, setting the ground work for multisystem organ failure.<sup>12</sup>

IAH affects the abdominal wall by impos-

ing pressure directly on the vessels feeding the wall. The pressure leads to edema and ischemia of abdominal wall tissues. Distention of the abdominal wall by IAH leads to decreased compliance of the wall, further compounding the IAH. The decreased blood flow leads to poor healing and possible dehiscence of abdominal surgical wounds.<sup>11</sup>

with its branches of the celiac axis and the superior and inferior mesenteric arteries, perfuses the gut and the accessory organs. All the venous blood from the gut drains into the portal vein to the liver and leaves the liver via the hepatic vein to be drained into the inferior vena cava. The abdominal compartment contains solid organs, hollow organs, fluid, gas, solids, and adipose tissue. When a condition arises that persistently increases pressure in the abdominal cavity to 12 mm Hg or greater, not only the gut but all major body systems can be affected, and the effects can lead to multisystem organ failure and death.

### *Gastrointestinal System*

The effect of IAH on the splanchnic organs leads to diminished gut perfusion. The consequences of this change lead to ischemia, acidosis of the mucosal bed, capillary leak,

intestinal edema, and translocation of gut bacteria.<sup>10</sup> As IAP increases, pressure is placed on the arteries, capillaries, and veins in the abdominal cavity. This increased pressure causes diminished arterial blood flow to the organs and resistance to drainage into the veins.

The diminished oxygenation to the gut leads to intramucosal acidosis.<sup>10,11</sup> The ischemic intestine loses its protective mucosal barrier and becomes more permeable to the intestinal contents. Edema develops in the intestinal wall and further increases the IAP. The increased permeability allows the intestinal flora to translocate via the villi into the lymph and vascular systems. This microbial translocation sets the stage for the development of sepsis.<sup>12</sup> Patients with IAH are also at high risk for stress ulcers because of the loss of the mucosal barrier.<sup>8</sup>

An IAP of up to 20 mm Hg can decrease mesenteric perfusion by

The vascular liver is extremely susceptible to IAH. Persistent pressures as low as 10 mm Hg can decrease hepatic perfusion and impair liver function.<sup>10,12</sup> If varices are present, this same pressure can lead to increased variceal stress and possible rupture.<sup>13</sup> With increased IAP, blood flow decreases in both the hepatic artery and the portal vein. This change in blood flow leads to decreased glucose metabolism, mitochondrial malfunction, and decreased lactate clearance by the liver.<sup>12</sup> Diminished lactate clearance leads to lactic acidosis.

### *Renal System*

IAH leads to impaired renal function.<sup>14</sup> Persistent pressures of 15 mm Hg or greater have been independently associated with renal impairment.<sup>14</sup> The acute kidney injury that accompanies IAH is multifactorial. The primary factor is decreased renal perfusion. Other factors are decreased APP (<60 mm Hg), decreased cardiac output, and increased systemic vascular resistance.<sup>14</sup> The renal impairment, as indicated by an elevated serum level of creatinine, may not appear until 2 to 3 days after the incident of IAH.<sup>14</sup>

### *Pulmonary System*

As the abdomen distends with intestinal gas, fluid, or edematous organs, the diaphragm is pushed upward, impinging on the thoracic cavity. Approximately 50% of the IAP is dispersed across the diaphragm and affects respiration and ventilation.<sup>11</sup> Pulmonary dysfunction may be one of the earliest signs of ACS.<sup>15</sup> Because the lungs cannot expand fully, respiratory excursion is limited, thereby reducing inhaled tidal volume, leading to hypoxemia. Conversely, carbon dioxide is retained, causing hypercarbia and respiratory acidosis. Compression atelectasis adds to the ventilation-perfusion mismatch and decreases the ratio of PaO<sub>2</sub> to fraction of inspired oxygen. Hospital- or ventilator-acquired pneumonia associated with the compression atelectasis may develop.

IAH can cause increases in peak airway and plateau pressures in patients treated with mechanical ventilation. The increases can

be suggestive of acute lung injury and lead to a shift to lung protective strategies and low tidal volumes. When acute lung injury is not present, but ACS is, low tidal volumes will not improve the respiratory picture.<sup>16</sup> However, in patients with ACS, acute lung injury or acute respiratory distress syndrome can be a sequela.<sup>15</sup>

### *Cardiovascular System*

One conundrum of IAH is the effect of the abnormality on the cardiovascular system. The increased intrathoracic pressure compresses the heart and major vessels, causing a tamponade-like picture, especially with the higher grades of IAH. Central venous pressures (CVPs) and pulmonary artery wedge pressures (PAWPs) are fictitiously elevated because of the effects of IAH.<sup>17</sup> These elevations may lead clinicians to surmise that a patient is volume loaded or overloaded.

The components of cardiac output (ie, preload, afterload, and contractility) are all adversely affected by increased IAH.<sup>17</sup> Compression of the inferior vena cava causes a decrease in venous return to the heart, affecting preload and causing a decrease in cardiac output. Contractility is affected mainly by changes in right ventricular mechanics.<sup>17</sup> The elevated intrathoracic pressure caused by IAH also increases pulmonary vascular resistance and right ventricular afterload. The right ventricle is a thin-walled chamber that normally acts as a passive conduit in pumping blood to the left side of the heart. In adapting to the increased pulmonary vascular resistance, the right ventricle dilates and pushes the intraventricular septum into the left ventricle. This situation

increases the workload of the right ventricle and decreases the ability of the left ventricle to fill. The increased workload increases myocardial oxygen demand. Through the compensatory measures of the sympathetic nervous system to maintain arterial pressure, systemic vascular resistance increases. In addition, the direct compression of the abdominal aorta, due to IAH, further increases systemic vascular resistance and the workload on the left ventricle.<sup>11,17</sup>

IAH also causes pressure on the femoral veins. This pressure increases venous stasis and the development of deep vein thrombosis. When the IAH is resolved, the risk of pulmonary emboli increases.<sup>11</sup>

### *Central Nervous System*

Studies in animals and humans have correlated the effects of IAH on intracranial pressure.<sup>18,19</sup> The Munro-Kellie hypothesis states that the cranium is a nondistensible vault filled with brain tissue, cerebrospinal fluid, and blood.<sup>20</sup> If 1 of the vault's substances increases in size, the other 2 substances adjust their volume to compensate and maintain equilibrium. Cerebrospinal fluid and the brain's venous drainage both leave the brain via the jugular vein.<sup>20</sup> Elevations in IAP are transferred into the thoracic compartment. Increased intrathoracic pressure puts back pressure on the jugular veins and decreases drainage of cerebrospinal fluid and blood, leading to increased intracranial pressure.<sup>18,20</sup> In patients with increased intracranial pressure, the effects of IAH can cause a further increase in the pressure in the cranium and decrease cerebral perfusion pressure.

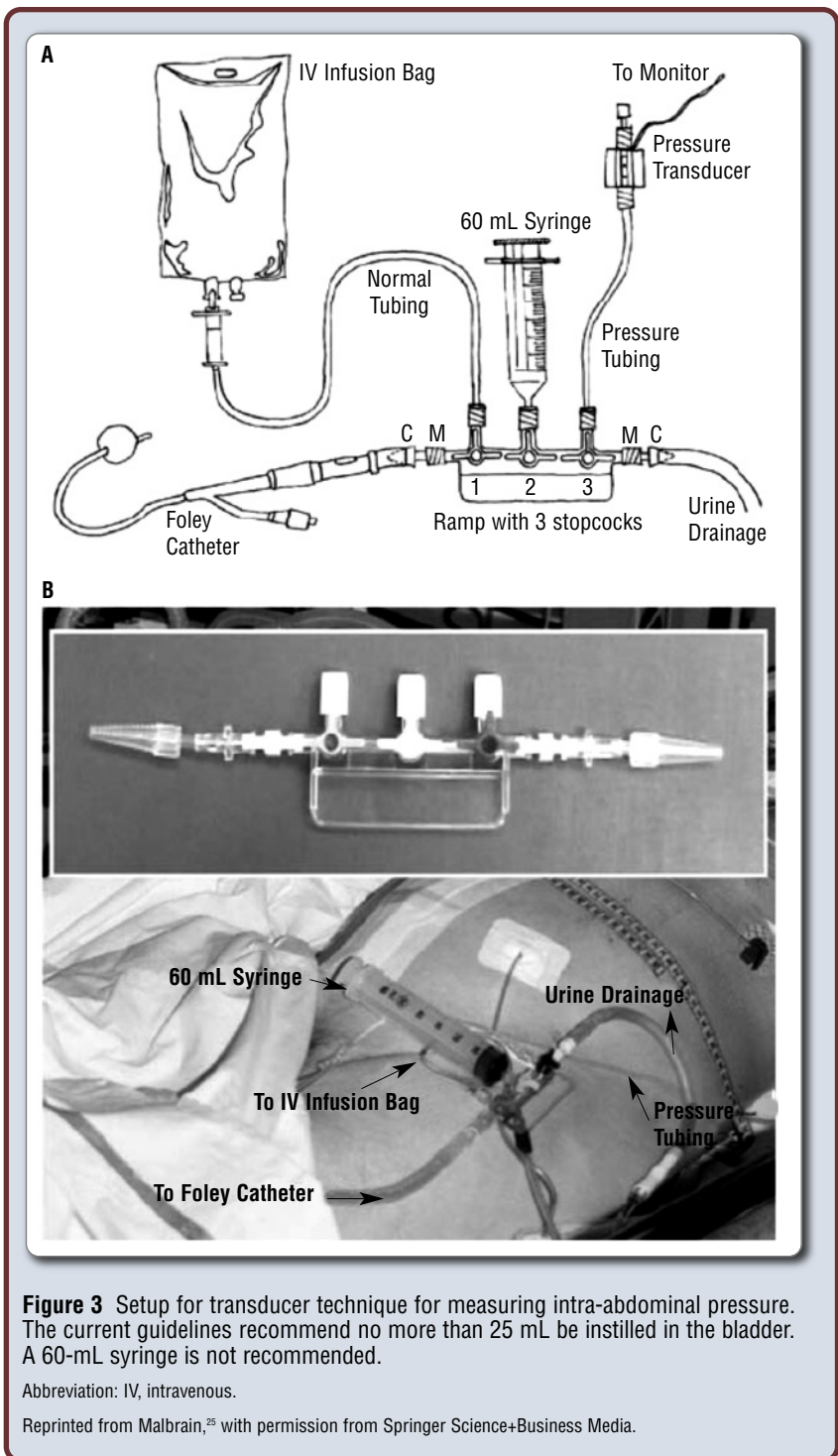
## Summary

In summary, IAH can affect almost all body systems. IAH has been proposed as the initial fall of the dominoes on the pathway of multisystem organ failure.<sup>11,21</sup> Manu Malbrain,<sup>22</sup> a past president of WSACS from Belgium, states that just as researchers and clinicians have focused on acute kidney injury and acute kidney failure and on acute lung injury and acute respiratory distress syndrome, they need to raise the concepts of IAH and ACS to “acute bowel injury and acute intestinal distress syndrome.”

## Measuring Intra-abdominal Pressure

To manage patients with IAH appropriately, nurses must be cognizant of the proper procedure for IAP measurement. Hands-on assessment of the abdomen and serial measurements of abdominal girth are not sensitive or specific enough to detect IAH.<sup>23,24</sup> A distended abdomen that has increased slowly over time, as in chronic ascites or pregnancy, will not necessarily have an elevated IAP.<sup>23</sup> Conversely, clinically important IAH can occur in the absence of a distended abdomen with the onset of an acute condition.<sup>23</sup>

Various methods are used for direct and indirect measurements of IAP.<sup>22</sup> The gold standard of indirect measurement is measurement via a urinary bladder catheter.<sup>2,11,23</sup> Either a transducer technique or a manometer technique can be used. The tools to measure IAP are readily available in any ICU,<sup>19,21</sup> and specialized kits can be purchased. The techniques and tools described here have been deemed acceptable by the WSACS.<sup>4</sup> The society does not



promote one device over another. The qualities a clinician should look for in a device for measuring IAP are reproducibility of results, patient and staff safety, effectiveness, ease of use, and cost. According to Michael Cheatham, past

president of the WSACS, “The best technique is the one that you and your nursing staff will use.”<sup>23</sup>

### Transducer Technique

Figure 3 is an illustration of the transducer technique,<sup>25</sup> and Table 3

**Table 3** Procedure for transducer setup with a manifold

**Equipment needed**

Indwelling urinary catheter with drainage bag  
A manifold ramp with 3 stopcocks or 3 stopcocks connected to one another  
Conical connectors for each end of the manifold  
Sterile scissors  
Disinfectant (povidone-iodine solution, chlorhexidine, or alcohol)  
Sterile gloves  
Sterile drape  
Standard intravenous setup with a 500-mL container of normal saline  
20- to 25-mL syringe  
Transducer and pressure tubing

**Setup**

Using aseptic technique, set up the manifold system, container of normal saline, the 20-mL to 25-mL syringe, and the pressure tubing and transducer as shown in Figure 3.

A pressure bag connected to the pressure tubing is not required.

Open the intravenous fluid and flush the system.

Don sterile gloves and place the sterile drape so it is centered under the area where the catheter connects to the drainage tubing.

Cleanse the drainage tubing with an appropriate disinfectant from the connection to the catheter to about 60 cm down the drainage tubing.

With the sterile scissors, cut the drainage tubing about 40 cm after the culture aspiration port and insert the stopcock ramp into the drainage tubing as shown in Figure 3.

To work the stopcocks to obtain a measurement, turn the first stopcock off to the patient and on to the intravenous bag, and open the second stopcock to the syringe.

Aspirate 20 to 25 mL of the normal saline.

Turn the first stopcock off to the intravenous bag and on to the patient.

Turn the third stopcock on to the patient and transducer and off to the drainage tubing; instill the 20 mL of saline into the bladder.

After the measurement is obtained, ensure that stopcocks are turned so that urine can flow from the patient to the urinary drainage bag.

provides a list of the equipment needed and the procedure for setup. (Of note, Figure 3 was published before the recommendation was made to use only 25 mL at the most for instillation.) Figure 4 and Table 4 provide information about an alternative setup method. The advantages of the transducer technique include no need for specialized equipment, cost-effectiveness, and safety. Once the initial setup is completed, the system remains

closed, with minimal risk for urinary tract infection.<sup>26</sup> Each measurement requires less than 2 minutes of nursing time, and the setup can be used for repetitive measurements for 2 to 3 weeks.<sup>13,23,25</sup> One drawback is that the stopcock system can be confusing, and if stopcock 3 is left in the off-to-drainage position after a measurement is obtained, urinary drainage will be obstructed. A patient safety concern with the specimen-port setup is that nurses must remember

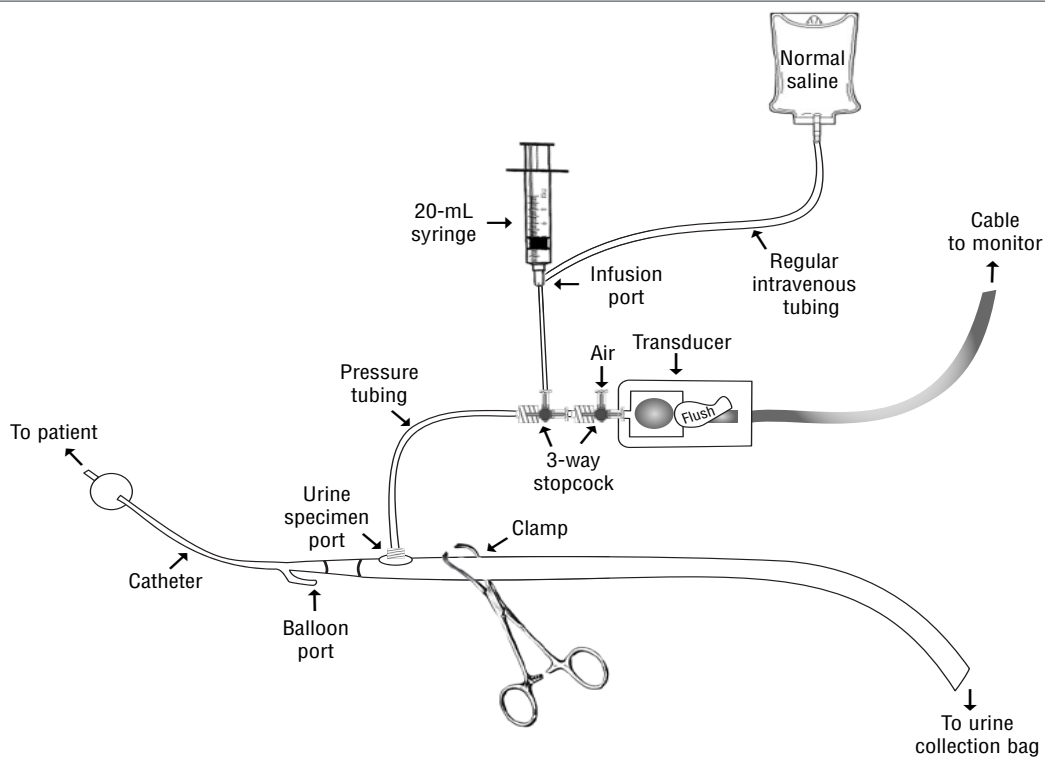
to remove the clamp after the measurement to ensure flow of urine.

Two commercial kits are available for the transducer method. The AbViser AutoValve (Wolfe Tory Medical, Inc, Salt Lake, Utah; Figure 5) has a valve that automatically opens 1 to 3 minutes after the saline has been instilled, adding a measure of safety to this device. Another IAP transducer kit is the Bard intra-abdominal pressure monitoring device (Bard Medical Division, Covington, Georgia). Regardless of the transducer setup used, the way in which an IAP measurement is obtained should remain the same. WSACS recommends the procedure<sup>2,25,27</sup> given in Table 5 and Figure 6.

*Manometer Technique*

The manometer technique is similar to the method of measuring CVP with a fluid column. The patient should have a urinary catheter in place; the only equipment needed is a centimeter ruler. This technique is called the U-tube technique (Figure 7) and is a modification of a technique first developed by an emergency department nurse.<sup>23,25,28,29</sup> Table 6 gives the steps for using the technique. The clinical validation with the U-tube technique is poor, and the method is recommended primarily for screening.

Holtech Medical (Charlottenlund, Denmark) has developed a urinary manometry tool (Figures 8 and 9) that is simple to use and is marked in millimeters of mercury instead of centimeters of water, so no conversion is needed. Studies<sup>23,25</sup> with the Holtech device indicate that it can provide reproducible, consistent, and accurate measurements.



**Figure 4** Transducer setup with a specimen port.

Sketch by Teri Weiser, RN.

**Table 4** Procedure for transducer setup with a specimen port

**Equipment needed**

- Indwelling urinary catheter with drainage bag and needleless specimen port
- Disinfectant (povidone-iodine solution, chlorhexidine, or alcohol)
- Standard intravenous setup with 500 mL of normal saline
- 20- to 25-mL syringe
- Transducer and pressure tubing
- Clamp for urinary drainage tubing

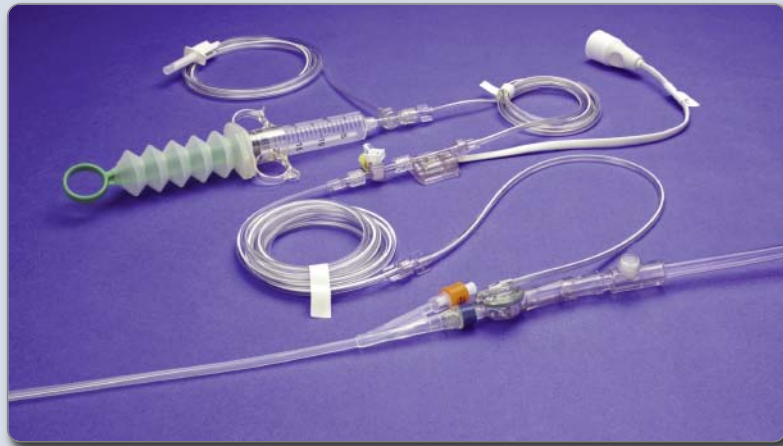
**Setup**

- Using aseptic technique, set up the manifold system, intravenous container of normal saline, the 20- to 25-mL syringe, and the pressure tubing and transducer as shown in Figure 4.
- A pressure bag connected to the pressure tubing is not required.
- Open the intravenous fluid and flush the system.
- Connect the pressure system setup to the needleless specimen port.
- To obtain a measurement, clamp the urinary drainage tubing distal to the specimen port.
- Aspirate 20 to 25 mL of the normal saline.
- Instill 20 to 25 mL of normal saline.
- After the measurement, ensure that the clamp is removed from the tubing.

The advantage of the manometry method is that it can be used outside the ICU. One concern is that reinstallation of urine and saline into the bladder can cause a urinary tract infection.<sup>30</sup>

The preceding techniques are used for intermittent measurements. A validated continuous technique with a 3-way urinary catheter has been deemed acceptable by WSACS.<sup>23,25,31,32</sup> Although the measurements with this third technique may be continuous, they would not be considered accurate unless the patient were supine with the head of the bed flat, and the abdominal muscles relaxed. In essence, with this continuous method, measurements could only be obtained “intermittently,” because patients do need to be turned, repositioned, and so on.





**Figure 5** AbViser Autovalve.

Image courtesy Wolfe Tory Medical Inc, Salt Lake City, Utah.

**Table 5** Procedure for measuring intra-abdominal pressure with the transducer method

Position the patient supine with the head of the bed flat.

The patient should be quiet, not agitated, restless, coughing, or talking; ensure that abdominal muscle contractions are absent.

Level transducer at the iliac crest in line with the midaxillary line and zero the transducer. The transducer can be secured to the patient's thigh or on a pole with a transducer holder.

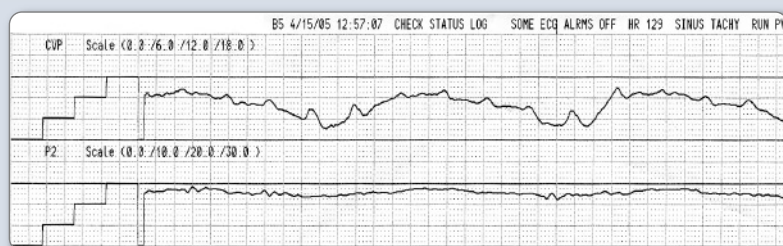
Draw up 20 to 25 mL of sterile normal saline via the intra-abdominal pressure monitoring system, and instill it into the bladder. Instill for 10-15 seconds.

Wait 30 seconds before obtaining a measurement so that the bladder detrusor muscle can relax and the monitor can equilibrate.

Obtain the measurement at end expiration (which corresponds to the trough on the ventilator patient's respiration waveform, and the peak in a nonventilator patient's respiration waveform) and record.

When measurement is completed, reposition patient to appropriate position for the patient's condition.

Deduct the amount of instilled saline from the patient's urinary output.



**Figure 6** Intra-abdominal pressure measurement by using the transducer method. The top waveform is the central venous pressure (CVP) waveform of a patient undergoing mechanical ventilation. End expiration would be at the end of the CVP trough and is approximately 5 mm Hg. The intra-abdominal pressure is labeled as P2, and is approximately 22 to 24 mm Hg.

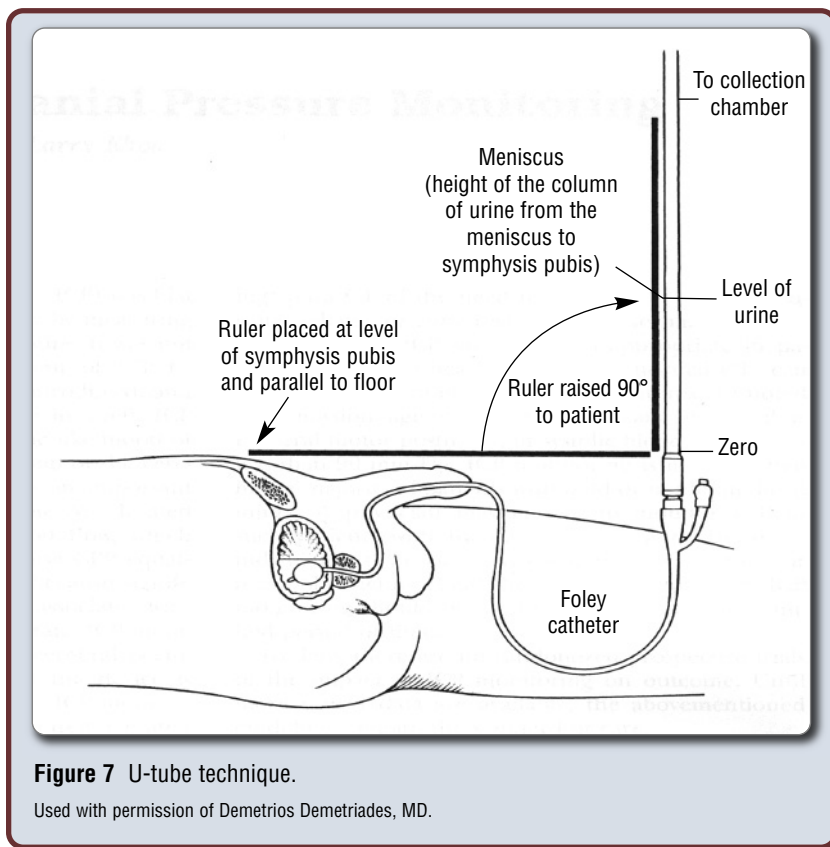
Clinicians can check the WSACS Web site (<http://www.wsacs.org>) for new evidence on acceptable methods and devices for measuring IAP. Trying out the do-it-yourself pressure-measuring devices and manufacturers' ready-made kits is also recommended, so a consensus among the collaborative team can be made on which device to use for consistency of practice.

Several caveats are associated with use of IAP monitoring via a urinary catheter. This method may be contraindicated in patients who have bladder surgery or trauma and may not be reliable in patients who have neurogenic bladder.<sup>25</sup> In patients with anuria, bladder pressure monitoring can still be done if at least 20 to 25 mL of saline is in the bladder.<sup>25</sup> The difficulties of inserting a urinary catheter into a patient with anuria must be weighed against the benefits of information that may be needed to prevent ACS.<sup>25</sup>

*Screening and Monitoring*

Once an IAP measuring device has been decided on and put into practice, nurses must know how to interpret the measurements. The critical IAP can vary from patient to patient; an IAP of 15 mm Hg may be tipping one patient over the edge, whereas another patient may tolerate a persistent IAP of 20 mm Hg. The APP is a better marker of abdominal organ perfusion than is IAP alone.<sup>4,33</sup> Calculation of APP is similar to that for cerebral perfusion pressure:  $MAP - IAP = APP$ . WSACS<sup>2</sup> recommends that the APP be maintained at greater than 60 mm Hg in patients with IAH or ACS.

WSACS has developed guidelines for screening and monitoring



**Figure 7** U-tube technique.

Used with permission of Demetrios Demetriades, MD.

**Table 6** U tube method for the manometer technique

- Obtain a centimeter ruler.
- Position the patient supine with the head of the bed flat.
- The patient should be quiet, not agitated, restless, coughing, or talking. Ensure that abdominal muscle contractions are absent.
- Raise the urinary catheter above the patient, allowing a U-shaped loop to develop.
- Level the connection site (the zero level) where the catheter meets the drainage tubing in line with the symphysis pubis.
- The urine in the tubing should fluctuate with respirations.
- Allow the fluid column to settle, and using the centimeter ruler, measure from the "zero" connection site to the meniscus of the fluid column.
- Convert from centimeters of water to millimeters of mercury (1.00 cm H<sub>2</sub>O = 0.74 mm Hg).
- When measurement is completed, reposition patient to appropriate position for patient's condition.

patients for IAH (Figure 10—available online only). In summary, any patient admitted to a critical care unit or in whom new organ failure develops should be screened for risk factors for IAH and ACS. If a patient has at least 2 of the risk factors, a baseline

IAP measurement should be obtained. In the study by Vidal et al,<sup>6</sup> almost one-third of patients had IAH at the time of admission to the ICU, and the abnormality developed in another one-third after admission. Perhaps, initially the U-tube method

could be used as a screening tool, and then once the IAP is greater than 12 mm Hg for 2 consecutive measurements, a more reliable measuring device should be used. Patients at risk for IAH or ACS should have IAP measured at least every 4 to 6 hours.<sup>11</sup> Patients with unstable hemodynamic status and patients with rapidly deteriorating organ dysfunction should have IAP measured hourly.<sup>11</sup> IAP measurements can be discontinued once the condition causing IAH has resolved and the IAP has remained at 10 to 12 mm Hg or less for 24 to 48 hours.<sup>11</sup>

A few of the risk factors identified by WSACS need to be studied more closely. The Institute for Healthcare Improvement<sup>34</sup> has recommended the use of a sepsis bundle. Sepsis is already a risk factor for IAH. The goal-directed fluid therapy that is a part of this bundle adds another risk factor (ie, >5 L of intravenous fluid in 24 hours). Large blood transfusions (>10 units of packed red blood cells in 24 hours) required by patients with trauma, ruptured abdominal aortic aneurysm, or gastrointestinal bleeding are another risk factor. Burn patients, especially those with large areas of abdominal eschar, are at risk for IAH because of the restriction of the abdominal wall by the eschar and the large volumes of fluid needed for fluid replacement. Therapeutic hypothermia for comatose survivors of cardiac arrest also puts patients at risk for IAH; both the hypothermia and the fluid replacement add to the possibility of IAH. Adding measurement of IAH as a part of protocols specifically for these comatose patients should be considered.



**Figure 8** Urinary manometry tool.

Used with permission of Holtech Medical, Charlottenlund, Denmark.



**Figure 9** Urinary manometry tool.

Used with permission of Holtech Medical, Charlottenlund, Denmark.

## Collaborative Management

Once IAH has been detected in a susceptible patient, the goal is to decrease the IAP to 15 mm Hg or less, maintain the APP at 60 mm Hg or greater, and prevent ACS.<sup>2,11,13</sup> The WSACS has developed a medical management algorithm<sup>2,35</sup> based on the causes of IAH and the condition of the patient. The algorithm is set up in an escalating step-wise approach (Figure 11—available online only). Many of the recommendations are specifically for physicians and advanced practice nurses, but many are within the domain of bedside nurses.

Recommendations for evacuating intraluminal contents include monitoring and recording daily bowel movements and implementing a bowel protocol before a patient becomes constipated. Patients who are paralyzed,

unconscious, or sedated should be checked daily for fecal impactions. Abdominal radiographs and computed tomography reports should be reviewed for evidence of impacted feces. Maintaining the patency of the nasogastric tube and the rectal tube, if used, is important. Patients' tolerance to enteral feedings should be assessed, and if residuals are greater than accepted levels, the amounts administered should be decreased or feedings should be discontinued. IAH should be reevaluated as a possible cause of increases in residual volumes. For patients who are able to eat, gas-producing foods should be minimized or eliminated. Recommendations for evacuating intra-abdominal space-occupying lesions are part of the purview of physicians or advanced practice nurses. Bedside nurses ensure that the diagnostic studies are safely carried out and assist with any bedside interventional procedures.

Positioning patients to achieve stability has been a mainstay of acute and critical care nurses' practice. Recommendations to improve abdominal wall compliance include avoiding the prone position and elevating the head of bed more than 20°. <sup>36,37</sup> Raising the head of the bed is a conflict with the recommendations of the ventilator bundle to prevent ventilator-associated pneumonia, which calls for elevating the head of the bed at least 30°. One way to compromise is to place patients in a reverse Trendelenberg position. However, when IAP is measured, patients must be supine with the head of the bed flat.

The recommendations to improve abdominal wall compliance are interdisciplinary. Debriding of abdominal

eschar and removal of constrictive abdominal dressings are advised. Patients who are in pain or are agitated should be given adequate doses of analgesics and sedatives and should be assessed for relief. As a last resort, patients may need to be intubated and given paralytic agents to decrease the effects of muscle contraction on IAP.

Fluid replacement is a known risk factor for IAH, especially if a patient has capillary leak. Monitoring and recording daily intake and output and assessing cumulative fluid balance are important nursing actions in managing these patients. The recommendations for optimizing fluid administration are to avoid excessive fluid administration and to aim for a goal of an equal or negative fluid balance by the third day in the ICU. If nurses have a fluid replacement protocol to follow and the protocol includes an option to use colloids or crystalloids; the colloids should be chosen. Members of the health care team should discuss possible use of hypertonic saline in patients who have bowel edema. Compared with crystalloids, colloids and hypertonic saline appear to provide a better response in patients with IAH and capillary leak.<sup>11,38-40</sup> Diuresis, continuous renal replacement therapy, and hemodialysis are stepwise escalating recommendations according to the medical management algorithm (Figure 11—available online only).<sup>2</sup>

## dotmore

To learn more about intra-abdominal pressure measurement, read "Revision of the Procedure for Monitoring Intra-abdominal Pressure" by Rhonda Anderson in *Critical Care Nurse*, October 2007;27:67-70. Available at [www.ccnonline.org](http://www.ccnonline.org).

The last category in the medical management algorithm is optimizing systemic and regional perfusion. Again, goal-directed fluid replacement is recommended. If the APP cannot be maintained at 60 mm Hg or greater with fluids, inotropes or vasopressors can be given. This category includes the need for hemodynamic monitoring to guide fluid replacement. IAH causes fictitious elevations in the CVP and PAWP. To negate this effect, the WSACS<sup>2</sup> recommends using the following correction formula:

$$\text{CVP}_{\text{corrected}} = \text{CVP}_{\text{measured}} - (\text{IAP}/2)$$

$$\text{PAWP}_{\text{corrected}} = \text{PAWP}_{\text{measured}} - (\text{IAP}/2)$$

An example is as follows:

A patient has a measured CVP of 15 mm Hg and an IAP of 20 mm Hg

$$\text{CVP}_{\text{corrected}} = 15 - (20/2) = 5 \text{ mm Hg}$$

A patient has a measured PAWP of 22 mm Hg and an IAP of 12 mm Hg

$$\text{PAWP}_{\text{corrected}} = 22 - (12/2) = 16 \text{ mm Hg}$$

Because of the inaccuracies associated with using these pressure values, the WSACS<sup>2,41</sup> recommends using volumetric indices such as stroke volume variation or pulse pressure variation.

If the escalating medical interventions do not decrease IAH or prevent ACS, a decompressive laparotomy is recommended<sup>2</sup> (Figure 12—available online only). Nurses need to prepare patients and patients' family members for this emergent procedure, which may need to take place at the bedside. After the surgery, patients often have an open abdomen until bowel edema subsides. IAP measurements should still be obtained.

## It Is Time

We are well into a new century. The members of WSACS will not let the concepts of IAP and ACS fade

away again. WSACS has been the impetus to coalesce the critical care community to identify and act on IAH and ACS. **CCN**

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