Abdominal compartment syndrome (ACS) is defined as sustained intra-abdominal pressure greater than 20 mm Hg (with or without abdominal perfusion pressure <60 mm Hg) associated with new organ failure or dysfunction. The syndrome is associated with 90% to 100% mortality if not recognized and treated in a timely manner. Nurses are responsible for accurately measuring intra-abdominal pressure in children with abdominal compartment syndrome and for alerting physicians about important changes. This article provides relevant definitions, outlines risk factors for abdominal compartment syndrome developing in children, and discusses an instructive case involving an adolescent with abdominal compartment syndrome. Techniques for measuring intra-abdominal pressure, normal ranges, and the importance of monitoring in the critical care setting for timely identification of intra-abdominal hypertension and abdominal compartment syndrome also are discussed. (Critical Care Nurse. 2012;32[6]:51-61)

Definitions
The World Society of Abdominal Compartment Syndrome (WSACS) recently developed definitions and diagnostic criteria for IAH and ACS and outlined standards for IAP measurement in adults. Currently, no standardized definitions specific for infants and children are available. Children have lower mean arterial pressures than adults do, so multiorgan failure may occur in children at lower IAP thresholds than those defined by WSACS. As a result, lower IAP cutoff values of 12 and 15 mm Hg have been used to define ACS in children. For an individual child, the actual IAP value may be less important than the impact of the pressure on organ function. Normal IAP is 7 (SD, 3) mm Hg in children, so ACS in a child may be more appropriately
defined as an IAP of greater than 10 mm Hg with evidence of new organ dysfunction or failure.4

Incidence, Risk Factors, and Indications for IAP Monitoring

IAH and ACS occur in a wide variety of patients, including infants, children, and adults who require medical or surgical treatment or treatment for burns, and is associated with a variety of diagnoses.7-12 The reported incidence of ACS varies from less than 1% to 60%, depending on the definitions used and the different populations of patients studied.7,12-13 Many health care providers think ACS may be underrecognized and thus underreported. According to WSACS,15 risk factors for IAH and ACS can be categorized into conditions associated with certain clinical characteristics (Figure 1). Examples of conditions in children include the following:

• Diminished abdominal wall compliance: gastroschisis, omphalocele, third-degree circumferential abdominal wall burns, and abdominal surgery with tight closure
• Increased intraluminal contents: constipation, Hirschsprung disease
• Increased abdominal contents: splenomegaly, hepatomegaly, intra-abdominal tumors (Wilms tumor), ascites, intraperitoneal or retroperitoneal bleeding
• Capillary leak/fluid replacement: systemic inflammatory response syndrome, sepsis

As a result of the favorable risk-benefit ratio of IAP monitoring and the marked associated morbidity and mortality of IAH and ACS, the WSACS recommends measuring IAP if a patient has 2 or more risk factors for IAH or ACS. If IAH is detected, serial IAP measurements should be performed (Figure 1).

Pathophysiology of ACS

ACS is due to persistently elevated pressure in the abdominal

### Table 1  Important definitions

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intra-abdominal pressure (IAP)a</td>
<td>Pressure concealed within the abdominal cavity1,2</td>
</tr>
<tr>
<td>Abdominal perfusion pressure (APP)b</td>
<td>The difference between the mean arterial pressure and IAPa</td>
</tr>
<tr>
<td>Intra-abdominal hypertension (IAH)c</td>
<td>Sustained or repeated elevation in IAP ≥12 mm Hg1,2</td>
</tr>
<tr>
<td>Grade I: IAP 12-15 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Grade II: IAP 16-20 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Grade III: IAP 21-25 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Grade IV: IAP &gt;25 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Abdominal compartment syndrome (ACS)d</td>
<td>Sustained IAP &gt;20 mm Hg (with or without APP &lt;60 mm Hg) associated with new organ dysfunction or failure1</td>
</tr>
<tr>
<td>Primary ACS</td>
<td>Condition associated with injury or disease in the abdominopelvic region1,2</td>
</tr>
<tr>
<td>Secondary ACS</td>
<td>Condition that does not originate from the abdominopelvic region1,2</td>
</tr>
<tr>
<td>Recurrent ACS</td>
<td>Condition in which ACS redevelops after previous surgical or medical treatment of primary or secondary ACS1,2</td>
</tr>
</tbody>
</table>

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Figure 1 Intra-abdominal hypertension assessment algorithm.a

a Reprinted from World Society of the Abdominal Compartment Syndrome, 15 with permission.
The increased pressure can be due to increased volumes within the fixed space, a decrease in the volume of the compartment itself, or a combination of both. The underlying pathophysiology of all compartment syndromes is inadequate perfusion and oxygenation of the organ or tissues within the confined space due to an elevation in pressure. Most likely, cellular anoxia is the final pathway leading to death from compartment syndromes. Sustained elevation of IAP has multisystem adverse effects because of the size and central location of the abdominal compartment and the number of vital organs located within the compartment (Figure 2). Preexisting comorbid conditions such as renal failure or cardiomyopathy play an important role in aggravating the effects of IAH and may reduce the threshold of IAH that causes clinical manifestations of ACS. The underlying cause of a patient’s IAH is also of vital importance.

Effects of IAP

In normal physiological conditions, perfusion of an organ requires flow of oxygen and nutrient-rich blood to the organ along a pressure gradient from the heart to the capillaries in the organ. This blood flow is opposed by venous and interstitial pressures, which typically do not exceed the perfusion pressure to the organ, allowing blood to flow to the organ. The elevated pressure in the abdominal compartment is transmitted to the interstitial space and microvasculature, leading to diminished flow of oxygen and nutrient-rich blood to the intra-abdominal organs, resulting in ischemia, congestion, and swelling of those organs. The swelling further encroaches on the intra-abdominal space, worsening the IAP and setting up a vicious cycle of worsening compression of the vascular structures with worsening perfusion of the organs and subsequent progressive organ dysfunction. When IAP exceeds the perfusion pressure, blood flow to the organs is completely cut off, and cell death and necrosis occur.

The effect of sustained elevations in IAP also compromises respiratory, cardiovascular, renal, gastrointestinal, hepatic, and central nervous system homeostasis. Respiratory System. Elevated IAP causes increased pressure and upward displacement of the diaphragm, thereby diminishing the intrathoracic compartment and leading to increased intrathoracic pressure. Elevated intrathoracic pressure affects the lungs by decreasing lung compliance, causing atelectasis and ventilation-perfusion mismatch, all of which lead to hypoxia and hypercarbia. All these changes are manifested as increased oxygen requirements and the need for increased mechanical ventilation.

Cardiovascular System. Elevations in intrathoracic pressure affect the heart adversely by direct compression, causing transmission of elevated pressures across the atria. Consequently, higher filling pressures are needed to establish adequate preload to support cardiac output. This mechanical pressure also causes decreased end-diastolic
volume and decreased stroke volume, changes that lead to decreased cardiac output. The lower cardiac output results in lower arterial pressures, contributing to the vicious cycle of poor perfusion, fueling further organ swelling and worsening IAP.

Intra-abdominal Structures
Within the abdomen, elevated IAP causes direct compression of organs. This compression results in collapse of the inferior vena cava, portal vein, and mesenteric vessels, leading to decreased venous return and, subsequently, decreased cardiac output. The low cardiac output and renal plasma flow lead to secretion of catecholamines, angiotensin II, and aldosterone, with subsequent vasoconstriction, which results in increased systemic vascular resistance (afterload) and adversely affects cardiac output and organ perfusion. Ischemia of the intestines due to decreased perfusion may result in translocation of bacteria across the gut mucosa into the systemic circulation, leading to bacteremia, sepsis, and release of inflammatory mediators, changes that adversely affect patients’ overall hemodynamic status. Direct pressure on the kidneys results in decreased perfusion to the kidneys, thereby decreasing renal blood flow and glomerular filtration rate. The low cardiac output and renal plasma flow in turn cause increased secretion of catecholamines, angiotensin II, and aldosterone, with subsequent renal vasoconstriction. The vasoconstriction exacerbates the decrease in renal blood flow and glomerular filtration rate, alterations that are manifested clinically as decreased urine output and worsening fluid retention, compounding the swelling of intra- and extra-abdominal organs and further elevating the IAP.

Central Nervous System
Intracranial pressure is increased by elevation in IAP. The higher pressures in the superior vena cava and right atrium are transmitted via the elevated intrathoracic pressure, impeding venous return from the head.

Involvement of Multiple Organ Systems
The multisystem effects of IAP leads to ACS-related organ dysfunction. If left uncontrolled, the organ dysfunction may become irreversible and lead to death. However, ACS-related dysfunction can be difficult to separate from the organ damage caused by the underlying diagnosis.

Medical and surgical interventions can be used to interrupt the cascade of events related to ACS, but once ACS is established, mortality remains as high as 30% to 60% even with intervention. Thus, the best course of treatment remains early recognition and management of elevated IAP, before ACS develops.

Diagnosis of ACS
The clinical manifestations of ACS are as variable as the multiple organ systems affected and the many underlying diagnoses associated with the syndrome. This characteristic makes ACS difficult to recognize on the basis of a constellation of signs and symptoms alone. ACS can be diagnosed at the bedside, without the need for elaborate laboratory or radiological tests, by simply measuring IAP. All patients at risk for IAH should have routine IAP measurements until the IAP becomes normal and risk factors for IAH resolve. Because IAP monitoring is simple, a high index of suspicion and broad indications for IAP monitoring may help clinicians diagnose IAH and ACS early, with few drawbacks.

IAP Measurements
Methods
IAP can be measured directly or indirectly. With the direct method, a catheter or needle is placed into the peritoneal space, and IAP is determined by using a fluid column or pressure transducer system (Figure 3). With the indirect method, IAP is measured indirectly via the pressure transmitted to the lumen of an intra-abdominal structure or organ (Figure 4). Indirect measurements include those determined by using intragastric, intrarectal, venacaval, intraureteral, and intravesical methods. The gold standard for IAP measurement is the direct method, but this method is invasive and can be associated with complications such as bowel perforation and peritonitis. The most accepted standard for indirectly measuring IAP is the bladder method. The pressure transducer is leveled at the cross-section between the iliac crest and the midaxillary line. Appropriate volumes of saline are instilled into the bladder via a urethral catheter, and the pressure is measured after allowing time for equilibration of bladder pressures. In adults, the current recommended volume of saline instilled varies from 10 to 25 mL. In children, the minimum volume recommended is 3 mL; however, 1 mL/kg up to a maximum of 25 mL can be used to obtain an accurate IAP.

Factors That Affect Measurements
IAP increases with inspiration and decreases with expiration.

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because of diaphragmatic contraction and relaxation. Higher body mass index is correlated with higher IAP in adults, but not in children. Body position also affects IAP. Patients tend to have higher IAP in prone and semirecumbent positions than in the supine position.

Other factors that affect IAP measurements obtained via the intravesical method include tense abdominal muscles, volume of fluid instilled into the bladder for measurements, temperature of the fluid instilled, presence of air bubbles in the fluid column, and position of the transducer.

Recommendations

Before dissemination of the WSACS guidelines, the recommended position for the transducer was at the level of the symphysis pubis. IAP measurements obtained at this level are lower than those obtained with the transducer at the midaxillary position, the current recommended position. According to the WSACS, IAP should be expressed in millimeters of mercury. Measurements should be taken with patients in the supine position and at end expiration. The transducer should be zeroed at the level of the iliac crest in the midaxillary line. The instillation volume should be 3 mL of physiological saline for children who weigh less than 50 kg or 1 mL/kg up to a maximum of 25 mL. IAP should be measured 30 to 60 seconds after instillation to allow relaxation of the detrusor muscle and in the absence of abdominal muscle contractions (Figure 1).

Prognosis of ACS

Mortality associated with ACS is 40% to 90%, depending on the population of patients studied, and can be as high as 80% to 90% if the syndrome is unrecognized and untreated. Most deaths from ACS are attributed to sepsis and multi-system organ failure. Elevated IAP is an independent predictor of mortality among critically ill patients. Mortality remains high even when decompression of the abdomen is performed, a finding that validates the importance of detecting and treating IAH before end-organ damage occurs.

Medical Management

Optimal management of ACS is contingent on early recognition of IAH and prevention of ACS. Initially,
nonsurgical options should be tried in an attempt to control IAH. The goal of medical management is to decrease the IAP by using one of the following interventions:

- Evacuate the intraluminal contents of the intestines by using gastric suctioning, rectal enemas, and gastroprokinetic and coloprokinetic agents.44

- Evacuate the intra-abdominal space-occupying abnormalities to treat extraluminal pathological changes such as ascites, hemoperitoneum, or pneumoperitoneum, which may require procedures such as paracentesis.22,44

- Optimize fluid administration by using goal-directed therapies. Use of diuretics and continuous renal replacement therapy may be helpful in reducing fluid overload and organ, tissue, and abdominal wall edema contributing to IAH.21,44,45 These strategies could also be helpful in controlling capillary leak syndrome.

- Improve abdominal wall compliance by using adequate analgesia, sedation, and neuromuscular blockade. A maneuver as simple as body positioning (ie, having a patient lie more supine) can decrease IAP.22,35,44

If these nonoperative measures are not successful, the specific surgical treatment for ACS is abdominal decompression by laparotomy with postoperative open-abdomen management.20,27,44,46 The vacuum pack technique, also known as negative-pressure dressing for temporary abdominal closure, has been associated with good outcomes in adults and children.25,47 In a retrospective study in neonates,47 use of the vacuum pack technique resulted in 85.7% successful primary fascial closure in a median of 4 days. Surgical decompression and temporary abdominal closure release the IAP by creating a larger abdominal compartment; the goal is to reestablish intra-abdominal organ perfusion. Successful early management of ACS may reverse organ dysfunction, as evidenced by improved urine output and more stable hemodynamic status and ventilatory parameters, enabling clinicians to start weaning the child from cardiac and respiratory support. Although it is a lifesaving strategy, the open abdomen has been associated with morbidity in some cases. Abdominal abscesses, fistulas, and major herniations of the abdominal wall have all been reported.43 Patients with planned ventral hernias after open-abdomen management require major reconstructive procedures several months after the initial precipitating factor.43 The complications of ACS can decrease when a comprehensive evidence-based management strategy is used that includes early use of open-abdomen management in patients at risk. In a recent study25 in adults, overall survival improved from 50% to 72% between 2002 and 2007, the ability to achieve successful primary fascial closure increased from 59% to 81%, and the number of patients with complications

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**Table 2 Factors that affect measurements of intra-abdominal pressure**

<table>
<thead>
<tr>
<th>Factors to consider</th>
<th>Effect on measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higher body mass index</td>
<td>Increases in adults</td>
</tr>
<tr>
<td>Elevation of the head of the bed above 0˚</td>
<td>Increases with inspiration</td>
</tr>
<tr>
<td>Respiration</td>
<td>Decreases with expiration</td>
</tr>
<tr>
<td>Contraction of abdominal muscles (agitation, valsalva maneuver, coughing)</td>
<td>Increases</td>
</tr>
<tr>
<td>Transducer below the reference point§</td>
<td>Increases</td>
</tr>
<tr>
<td>Transducer above the reference point§</td>
<td>Decreases</td>
</tr>
<tr>
<td>Midaxillary line as reference point for placement of transducer compared with the symphysis pubis</td>
<td>Increases</td>
</tr>
<tr>
<td>Presence of air bubbles in the fluid column</td>
<td>Is unreliable</td>
</tr>
<tr>
<td>Larger instilled volume (&gt;50 mL) in the bladder for measurement</td>
<td>Increases</td>
</tr>
<tr>
<td>Temperature of fluid instilled into the bladder for measurement</td>
<td>Very cold water may increase</td>
</tr>
</tbody>
</table>

§ Reference point refers to the cross-section at the level of the iliac crest and the midaxillary line.
CASE STUDY

A 13-year-old boy was brought to the emergency department via ambulance after a horse kicked him in the left flank and left upper abdominal quadrant. Vital signs were heart rate 69/min, blood pressure 117/72 mm Hg, respirations 16/min, and oxygen saturation 100%. Abdominal computed tomography revealed no free fluid and a mild splenic contusion. The boy was admitted by the pediatric surgery team for observation and serial abdominal examinations. Within 24 hours, he began having increased pain, nausea, and vomiting. Repeat abdominal computed tomography showed free intraperitoneal fluid. The patient was taken to the operating room for an exploratory laparotomy. A perforation in the jejunum was found and repaired, and primary closure of the fascia was achieved. However, within 24 hours of surgery, the boy had altered mentation, tachycardia to 160/min, and hypotension (blood pressure as low as 33/24 mm Hg). He was emergently given 4 L of physiological saline, and inotropic support was started during transfer to the pediatric intensive care unit.

Vital signs upon admission to the unit were heart rate 165/min, respirations 60/min, blood pressure 90/25 mm Hg. He had worsening abdominal distention, decreased urine output, and decreased perfusion to his lower extremities. His IAP was measured by using the indirect intravesical method with appropriate instillation volumes for his weight: 25 mL for a weight of 69 kg. The initial IAP was 30 mm Hg. This value with the new development of organ failure was indicative of ACS. Because the underlying cause of ACS was within the abdomen, the syndrome was defined as primary ACS.

The boy was taken back to the operating room for emergent abdominal decompression and reexploration. He returned to the intensive care unit with his abdomen open and a negative-pressure wound dressing in place to further manage the ACS. The IAP decreased to 19 mm Hg after this intervention. Serial IAP monitoring indicated ongoing IAH with increasing IAP. The patient was returned to the operating room once more for another decompressive laparotomy to treat recurrent ACS (IAP 32 mm Hg) that occurred despite leaving the abdomen open. The dressing was reexpanded to increase the volume of the abdominal compartment, thereby decreasing the IAP. The open abdomen was again managed with a negative-pressure wound dressing. The patient subsequently had 4 more trips to the operating room for irrigation, debridement, and dressing changes until postoperative day 28, when the abdominal fascia was closed with a soft-tissue graft. The abdominal wound was allowed to heal by secondary intention.

The multiorgan dysfunction caused by ACS gradually resolved. The patient had been treated with high-frequency oscillator ventilation for respiratory failure complicated by acute respiratory distress syndrome. The boy was successfully weaned from mechanical ventilation after 19 days. Continuous renal replacement therapy was used to support the patient’s renal failure for 3 days until return of optimal urine output. The patient was eventually discharged home on postoperative day 50; he was receiving parenteral nutrition and required wound dressing management. His subsequent course was complicated by development of a gastrocutaneous fistula, which was managed with parenteral nutrition, application of a negative-pressure wound dressing, and subsequent surgical excision of the fistula after 3 months. He eventually had reconstructive surgery of the abdominal wall and was functioning as a normal teenager, 5 years after the injury.

This case highlights the importance of IAP monitoring in a patient at risk for ACS. Perhaps, if the IAP had been monitored immediately after the initial exploratory laparotomy, evidence of increasing pressures might have prompted the physicians to explore the abdomen or decompress it before the IAP reached 30 mm Hg and the patient had multiorgan dysfunction. Other lessons learned from this patient include the need to continue monitoring IAP despite surgical intervention for ACS, because of the potential for recurrent ACS even when the abdomen is left open. IAP management can be used to determine timing for reexploration and decompression. Support of organ dysfunction during management of ACS is vital for survival. For example, in this case pulmonary function was supported by using high-frequency oscillator ventilation, cardiac function with inotropes, and renal function with continuous renal replacement therapy.

Complications of the open abdomen, such as the development of an enterocutaneous fistula, can be managed with good outcomes.
associated with an incisional hernia decreased from 41 to 19. In a retrospective study in 26 children undergoing decompressive laparotomy for ACS, all survivors eventually had closure of the abdominal wall. Time to definitive closure was 8.6 days (range, 1-61), and the mean number of separate operations before final closure was achieved was 3.2. Specific complications related to open-abdomen management included fascial dehiscence (n=1) and enterocutaneous fistula (n=1). Overall mortality was 58%, and mortality was higher in children who had higher preoperative serum lactate levels.

**Nursing Management**

Detection of ACS requires close surveillance of IAP in patients at risk for IAH. Nursing management should focus on risk assessment for IAH and routine IAP monitoring. Nurses should be proficient in obtaining accurate IAP measurements by the method used at their institution and should understand the clinical factors that affect IAP measurements. A clear understanding of the definitions of IAH and ACS can guide bedside nurses in the early detection of IAH and potentially lead to early medical interventions and prevention of ACS. Nurses should also be familiar with simple measures that can be taken to decrease IAP, such as aspiration of gastric contents or suctioning, rectal decompression, and use of adequate levels of sedation and neuromuscular blockade to discourage voluntary abdominal muscle contractions. These interventions ultimately would improve patients’ outcomes.

**Conclusion**

ACS is associated with a high mortality rate and can cause significant morbidity in survivors. Thus, ACS is a clinically important problem in critically ill patients that can be ameliorated by early recognition of IAH and appropriate medical or surgical intervention for IAH and impending ACS. Bedside critical care nurses are responsible for accurately measuring IAP and alerting physicians about important observed changes. Nurses’ knowledge of IAH and ACS, awareness of the patients at risk for IAH, and recognition of IAH and progression to ACS are important. A high index of suspicion and active IAP surveillance for at-risk patients are essential in early detection and management of ACS.

**References**


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