Abdominal compartment syndrome is a potentially lethal condition caused by any event that produces intra-abdominal hypertension; the most common cause is blunt abdominal trauma. Increasing intra-abdominal pressure causes progressive hypoperfusion and ischemia of the intestines and other peritoneal and retroperitoneal structures. Pathophysiological effects include release of cytokines, formation of oxygen free radicals, and decreased cellular production of adenosine triphosphate. These processes may lead to translocation of bacteria from the gut and intestinal edema, predisposing patients to multiorgan dysfunction syndrome. The consequences of abdominal compartment syndrome are profound and affect many vital body systems. Hemodynamic, respiratory, renal, and neurological abnormalities are hallmarks of abdominal compartment syndrome. Medical management consists of urgent decompressive laparotomy. Nursing care involves vigilant monitoring for early detection, including serial measurements of intra-abdominal pressure. (American Journal of Critical Care. 2003; 12:367-373)

A 32-year-old woman, P.L., was stabbed in the liver. She arrived in the emergency department hypotensive and unresponsive to pain. After she had a massive transfusion and an emergent laparotomy, she was admitted to the intensive care unit. During the next 6 hours, mechanical ventilation became progressively more difficult. Although P.L.’s central venous pressure remained slightly elevated, her cardiac output decreased markedly.

J.T., a 19-year-old, was the driver in a high-speed, single-vehicle rollover that caused blunt head trauma. He was wearing a seat belt and a shoulder harness at the time of the rollover. After his condition was stabilized at a small community hospital, he was transferred by air to a level I trauma center. When he arrived at the center 4 hours after the injury, he was intubated and still profoundly unconscious. Computed tomography of the head showed diffuse axonal injuries, and an intracranial pressure monitor was inserted. Despite aggressive management, J.T.’s intracranial pressure continued to increase and his hemoglobin level decreased.

E.K., an 87-year-old nursing home resident, had severe abdominal pain. His care providers reported that he had recently experienced several episodes of protracted vomiting progressing to hypotension, tachycardia, and a decreased level of consciousness. Transfer for tests to determine if he had sepsis was arranged, and E.K. was admitted to the critical care unit.
Despite administration of multiple boluses of fluid, he remained hypotensive and anuric. Continuous nasogastric suctioning produced a large amount of green-brown fluid, and an abdominal computed tomography scan revealed a complete obstruction of the small bowel.

Despite the varied histories and clinical features, abdominal compartment syndrome (ACS) developed in each of these patients.

**Abdominal Compartment Syndrome**

Compartment syndrome occurs whenever increasing pressure within a confined anatomic space undermines the normal cellular functions of the tissues contained within that space. Most commonly, compartment syndrome involves the extremities; tissue edema below the fascial layer causes ischemia and eventual muscle necrosis.

The peritoneal cavity is another confined anatomic space. Although the notion is not new, trauma clinicians are becoming more aware that increased pressure within the abdomen impairs organ function. This condition is known as intra-abdominal hypertension and can lead to the development of ACS, with potentially devastating consequences.

**Etiology and Pathophysiology**

Any abnormality that elevates the pressure within the abdominal cavity can induce intra-abdominal hypertension. In some situations, such as acute pancreatitis or ruptured abdominal aortic aneurysm, retroperitoneal processes are potential causes. However, mechanical obstruction of the bowel, and abdominal growths can all be precipitating events. However, blunt abdominal trauma with intra-abdominal bleeding from splenic, hepatic, and mesenteric injuries is the most common cause of intra-abdominal hypertension. Surgical placement of abdominal packing to control hemorrhage may also increase pressure within the peritoneal space. Finally, bowel distention, as a consequence of hypovolemic shock and massive volume replacement, is an important cause of intra-abdominal hypertension, and subsequent ACS, in trauma patients.

**Abdominal injury and disease, bowel distention, and generalized shock states can all lead to increased abdominal pressure.**

In shock states, vasoconstriction mediated by the sympathetic nervous system shunts blood away from the skin, muscles, kidneys, and gastrointestinal tract in favor of the heart and brain. Redistribution of blood from the gut produces cellular hypoxia in the tissues of the intestines. This hypoxia is associated with 3 sequelae crucial to the development of the positive feedback role of increased intra-abdominal pressure leading to intra-abdominal hypertension and the development of abdominal compartment syndrome.
feedback loop (see Figure) that characterizes the pathogenesis of intra-abdominal hypertension and its progression to ACS:

1. release of cytokines,
2. formation of oxygen free radicals, and
3. decreased cellular production of adenosine triphosphate.

In response to hypoxic tissue injury, proinflammatory cytokines are released. These molecules promote vasodilatation and increase capillary permeability, leading to the development of edema. After cellular reperfusion, oxygen free radicals are generated. These agents have a toxic effect on cell membranes that is further aggravated by the presence of cytokines, which stimulate the release of even more free radicals. Additionally, insufficient oxygen delivery to the tissues limits production of adenosine triphosphate and this decreased availability of adenosine triphosphate impairs energy-dependent cellular activities. Particularly affected is the sodium-potassium pump. Efficient functioning of the pump is essential for regulation of intracellular electrolytes. When the pump fails, sodium leaks into the cells, pulling water along with it. As cells swell, the membranes lose their integrity, spilling intracellular contents into the extracellular environment and further promoting inflammation. Inflammation quickly leads to the formation of edema, as a result of capillary leakage, and the increasingly swollen tissues of the gut cause intra-abdominal pressures to soar. As pressure mounts, intestinal perfusion is impaired, and the cycle of cellular hypoxia, cell death, inflammation, and edema continues unabated.

Clinical Manifestations

The effects of intra abdominal hypertension on the peritoneal organs are well known. In studies in swine, increasing intra-abdominal pressure to 20 mm Hg markedly decreased blood flow in the mesenteric arteries and decreased gastric mucosal pH. Perfusion of the hepatic microvasculature and the hepatic artery was significantly reduced at an intra-abdominal pressure of only 10 mm Hg. In intra-abdominal hypertension, gut hypoxia has been implicated in the release of cytokines from the Kupffer cells of the liver and bacterial translocation from the gut to the mesenteric lymph nodes.

Intestinal ischemia plays an important role in the “2 hit” hypothesis of the development of multiple organ dysfunction syndrome. According to this theory, the initial traumatic injury is the inciting event or “first hit,” which leads to the development of a mild systemic inflammatory response syndrome. This condition is considered beneficial and is the normal physiological reaction to stress. However, any “second hit” results in an exaggerated systemic inflammatory response that is maladaptive and can rapidly progress to multiple organ dysfunction syndrome. In animal studies of ACS, bacteria translocated from the lumen of the gut to the lymphatic system, as a consequence of bowel ischemia, and such translocation may provide the second hit. These bacteria can incite the release of proinflammatory cytokines and perpetuate this destructive cycle.

ACS is distinguished by its immediate impact on a variety of organ systems. Patients have a complex constellation of sometimes subtle signs and symptoms that, taken together, indicate the development of the syndrome. Consequently, careful ongoing vigilance is essential for early detection of this potentially devastating condition. The cardiovascular system is affected by decreased venous return to the heart due to compression of the inferior vena cava by high pressure in the peritoneal space. In addition, this increased pressure also increases systemic vascular resistance as a whole, thus impairing left ventricular ejection. The combination of decreased venous return and increased afterload leads to a precipitous decrease in cardiac output, adding to the ischemia.

Intra-abdominal hypertension also impairs functioning of the pulmonary system. Elevated pressure in the abdomen impedes diaphragmatic excursion, reducing functional residual capacity and further exacerbating the increasing oxygen debt. Clinical signs of elevated intra-abdominal pressure in patients receiving mechanical ventilation include increases in peak airway pressures during volume ventilation and decreases in tidal volumes when pressure modes are used.

A dwindling urine output progressing to oliguria is the renal manifestation of ACS, even when blood pressure is normal. Compression of the renal veins and collecting systems by high intraperitoneal pressure causes this phenomenon. Kidney ischemia also promotes activation of the renin-angiotensin-aldosterone system. If prolonged, these responses progress to acute tubular necrosis and renal failure.

Finally, ACS has been implicated in elevating intracranial pressure in patients with multiple trauma.
that includes a brain injury. Increases in intrathoracic pressure cause obstruction in cerebral venous outflow, leading to vascular congestion. In addition, the ACS-induced decrease in cardiac output can combine with increased intracranial pressure to significantly decrease cerebral perfusion pressure. These events are poorly tolerated in patients who already have impaired cerebral autoregulation.

Management

As ACS evolves, a positive feedback loop is created whereby increases in intra-abdominal pressure promote further elevations in intra-abdominal pressure. Because the underlying problem is that the abdominal compartment has become too small, the immediate solution is to enlarge the size of the compartment. Consequently, current treatment of patients with ACS is urgent decompressive laparotomy, either in the operating room or at the bedside. This procedure provides rapid relief of intra-abdominal hypertension. The peritoneal cavity is usually left open postoperatively, and the exposed contents are covered with a sterile dressing such as an iodine-impregnated plastic adhesive drape. Closure of the incision is attempted when the swelling subsides. Unfortunately, once ACS is well advanced, surgical decompression may have serious adverse effects resulting in massive washout of anaerobic products, profound hypotension, and asystolic arrest.

In a recent survey, Mayberry et al assessed the knowledge level, experience, and current ACS management techniques of expert trauma surgeons in the United States. The majority of the respondents could correctly recognize the signs and symptoms of ACS and had experience in its treatment. Nonetheless, prophylactically leaving the abdomen open after major trauma surgery was not a common practice.

Urgent decompressive laparotomy provides rapid relief of intra-abdominal pressure.

Nursing Care

Patients at high risk for ACS are those who have either undergone abdominal procedures or experienced any event that predisposes them to increased intra-abdominal pressure. Classically, this group includes patients who have had marked abdominal trauma with concomitant shock. However, patients with pancreatitis, bowel obstructions, abdominal aortic aneurysm rupture, pregnancy, large tumors, circumferential full-thickness burns of the abdomen, and those who have received massive volume resuscitation for whatever reason are also at risk for ACS.

Nurses caring for any of these patients must remain vigilant to prevent ACS by watching closely for the hallmark renal, pulmonary, cardiovascular, and neurological signs that indicate the development of this syndrome. Patients who have low urinary output and hypotensive shock unresponsive to fluid resuscitation or who have increased peak airway or intracranial pressures should be considered at risk for ACS.

The definitive assessment finding for ACS, however, is not a constellation of ambiguous clinical signs and symptoms but rather the presence of elevated intra-abdominal pressure. Animal studies in which a catheter was placed directly into the peritoneal cavity indicated that normal intra-abdominal pressure is atmospheric or even subatmospheric ($\leq 0$ mm Hg). Elevations in intra-abdominal pressure are classified as mild (10-20 mm Hg), moderate (20-40 mm Hg), and severe (>40 mm Hg). The exact level at which intra-abdominal hypertension requires intervention is not well established. However, most experts recommend abdominal decompression in symptomatic patients whose intra-abdominal pressure reaches 30 mm Hg.

In clinical practice, direct measurement of intra-abdominal pressure is impractical because it would require placement of an invasive line into the peritoneal space solely for the purpose of measuring the pressure. Fortunately, a reliable, indirect method of assessing intra-abdominal pressure exists. The urinary bladder is an intra-abdominal structure with a compliant wall. When the bladder is partially filled, the pressure in it accurately reflects intra-abdominal pressure.

Two well-recognized techniques are used to determine bladder pressure. In the first method, an indwelling urinary (Foley) catheter is inserted into the bladder and the bladder is emptied. The drainage bag is then clamped off, and 50 to 100 mL of sterile isotonic sodium chloride solution is instilled through the catheter tubing. When the volume in the bladder is in this range, the organ acts as a passive diaphragm. With the patient supine and the symphysis pubis as a zero reference point, a pressure transducer is connected to

Urinary bladder pressure, which accurately reflects intra-abdominal pressure, should be monitored frequently.
the sampling port of the Foley catheter and the pressure (in millimeters of mercury) is displayed on the bedside monitor.2,4,5

Alternatively, instead of connecting the patient’s Foley catheter to a pressure transducer after clamping of the catheter tubing and instillation of sterile iso-tonic sodium chloride solution, the catheter tubing is simply raised vertically above the symphysis pubis at a 90° angle to the patient’s pelvis. Then, the tubing is unclamped and the distance (in centimeters) between the symphysis pubis zero point and the maximal height of the fluid column is recorded6 (1.36 cm H2O = 1 mm Hg).2 Similar in concept to using a water manometer to measure central venous pressure, this simple technique requires little effort or equipment.1 Ease of use has made it the preferred procedure at our institution. It provides a quick and accurate assessment of whether the intra-abdominal pressure is elevated and can be performed by emergency or critical care nurses without a specific medical order or sophisticated invasive monitoring equipment. A recent study21 indicated that although both the transducer technique and the catheter tubing method accurately reflected intra-abdominal pressure, the catheter method had a slightly stronger correlation ($r^2 = 0.98$ vs $r^2 = 0.93$) between bladder pressure and intra-abdominal pressure.21

**Summary**

ACS is a potentially lethal condition caused by any event that produces intra-abdominal hypertension and causes ischemia of the peritoneal organs. Pathophysiological effects are wide-ranging and predispose patients to multiorgan dysfunction syndrome. Hemodynamic, respiratory, renal, and neurological abnormalities are classic findings. Urgent decompressive laparotomy can decrease morbidity and mortality. Trauma nurses play an essential role in the early detection of this syndrome.

**Commentary by Mary Jo Grap (see shaded boxes).**

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Pathophysiology and Management of Abdominal Compartment Syndrome
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Am J Crit Care 2003;12 367-371
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