Abdominal Compartment Hypertension and Abdominal Compartment Syndrome

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KEYWORDS

• Abdominal compartment syndrome • Abdominal hypertension

KEY POINTS

- Abdominal compartment hypertension and syndrome should be expected following resuscitation in patients with an increase in the intraperitoneal contents (including tense ascites), and in those with decreased abdominal wall compliance.
- Measurement of bladder pressure is the standard of care by which intra-abdominal pressure should be measured in the intensive care unit in most instances.
- Abdominal hypertension is present when abdominal pressure exceeds 12 mm Hg and abdominal compartment syndrome is present with abdominal pressure exceeds 25 mm Hg.
- Although decompressive laparotomy is definitive treatment of all cases of abdominal compartment syndrome, some causes, such as tense ascites or hemoperitoneum, can be treated with paracentesis, and decreased abdominal wall compliance can be treated with pharmacologic paralysis and deep sedation.

INTRODUCTION

Abdominal compartment syndrome (ACS) is a rare but clinically significant outcome in critical illness. Although ACS is the most severe end point on a spectrum of disease related to increased intraperitoneal pressure, it is preceded by intra-abdominal hypertension (IAH), which is less likely to be associated with end-organ dysfunction.

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Although the exact incidence of either condition is poorly defined, the few available reports on the subject show that ACS occurs in between 10% and 35% of noninjured critically ill patients.[1] This rate also mirrors the incidence of ACS following major operation or trauma.[2,3] IAH, which has broader inclusion criteria than ACS, has a higher incidence than might be expected. Although also poorly defined, 30% to 70% of either subset of patients develop IAH. In both medical and surgical critical care patients, the presence of IAH or ACS is associated with a significant increase in mortality.

At the most basic level, IAH and ACS are physiologically similar to compartment syndromes in general and result in a derangement of tissue perfusion caused by increased pressure within the fixed volume of an anatomic compartment. The abdomen and pelvis collectively form one such compartment, bounded by the diaphragm, abdominal wall, back, and the peritoneal reflection at the bony pelvis. As with other compartment syndromes, increased pressures within the fixed abdominal compartment impair capillary and venous blood flow, thereby ultimately decreasing arteriole flow as well. The resultant cellular hypoxia leads to anaerobic respiration and lactic acidosis. This metabolic acidosis is often worsened by a respiratory acidosis arising from upward pressure on the diaphragm that prevents adequate ventilation and CO_2 exchange. Other common impairments seen in ACS include malperfusion of the intestines caused by decreased venous outflow from the splanchnic circulation, acute kidney injury caused by decreased glomerular blood flow, and decreased cardiac return as a result of compression of the inferior vena cava. Left unchecked, these events can lead to significant systemic acidosis and cardiovascular collapse.

DEFINITION AND CAUSES OF INTRA-ABDOMINAL HYPERTENSION/ABDOMINAL COMPARTMENT SYNDROME

The most commonly used definition of ACS was published by the World Society on Abdominal Compartment Syndrome (WSACS) in 2013.[4] This consensus document addresses clinical definitions and pressure measurement guidelines intended to assist clinicians and researchers in the diagnosis, treatment, and characterization of IAH/ACS. Intra-abdominal pressure (IAP) is defined as the end-expiratory abdominal pressure in the supine position in the setting of fully relaxed abdominal wall musculature. Measured IAP is used to calculate the abdominal perfusion pressure (APP) by subtracting IAP from the systemic mean arterial pressure (MAP); in this sense, APP can be thought of as the abdominal analog to cerebral perfusion pressure and can be used as a predictor of visceral perfusion. The WSACS statement defines IAH as a sustained IAP greater than 12 mm Hg, in contrast with normal IAP, which ranges from 2 to 7 mm Hg. IAH is further subdivided into grades I to IV (**Table 1**).

Table 1
Grading and treatment of IAH

Grade	IAP (mm Hg)	Treatments
I	12–15	Sedate patient, diurese, paracentesis, loosen abdominal closure device
II	16–20	Sedate patient, diurese, paracentesis, loosen abdominal closure device
III	21–25	Pharmacologically paralyze patient, loosen abdominal closure device, decompressive laparotomy
IV	>25	Decompressive laparotomy