

Abdominal Compartment Syndrome – Pressure is on

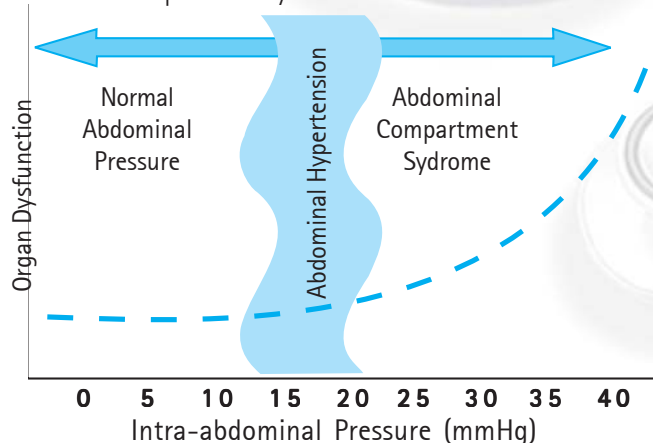
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Background

Compartment syndrome occurs when a fixed compartment, defined by myofascial elements or bone, becomes subject to increased pressure, leading to ischemia and organ dysfunction. The abdominal compartment syndrome (ACS) is the development of physiologic dysfunction in intra-abdominal and extra abdominal organs as the result of increased intra-abdominal pressure (IAP). The elevated IAP is a function of the rate of fluid accumulation within the abdominal cavity and the compliance of the abdomen. The pressure-volume curve for the abdominal cavity is non-linear. Due to the decreasing compliance of the abdomen, as fluid within the peritoneal cavity progressively accumulates, a greater increase in IAP results. The ACS may occur in patients with a variety of conditions where there is increased IAP.

Clinical settings which have been associated with the syndrome include ruptured abdominal aortic aneurysm, ascites and intraperitoneal hemorrhage. Organ dysfunction caused by intra-abdominal hypertension (IAH) is considered to be abdominal compartment syndrome. The dysfunction may be respiratory insufficiency secondary to compromised tidal volumes, decreased urine output caused by falling renal perfusion or any organ dysfunction caused by increased abdominal compartment pressure.

Abdominal compartment syndrome was recognized clinically in the 19th century when Marey and Burt observed its association with worsening respiratory function. In the early 20th century, Emerson's animal experiments demonstrated mortality associated with abdominal compartment syndrome.



Categories

Primary or acute ACS: This occurs when intra-abdominal pathology is directly and proximally responsible for the compartment syndrome.

Secondary ACS: This occurs when no visible intra-abdominal injury is present but injuries outside the abdomen cause fluid accumulation.

Chronic ACS: This occurs in the presence of cirrhosis and ascites, often in the later stages of the disease.

Recurrent ACS: in which the patient has recovered from the ACS once but because of secondary insults the cycle begins again. This variety is associated with very high mortality rate.

Systemic Manifestations of ACS

Increased IAP results in dysfunction of the respiratory, cardiovascular, renal and neurological systems.

Respiratory system: The hemi-diaphragms are elevated due to the increased IAP. A decrease in thoracic volume and compliance results. Peak inspiratory pressure and pulmonary vascular resistance increases and ventilation perfusion abnormalities occur. Increasing PEEP is required to oxygenate the patient.

Cardiovascular system: As the IAP increases, central venous pressure (CVP), pulmonary artery wedge pressure (PAWP) and systemic vascular resistance increases, due to an increased pleural pressure secondary to the increased IAP. Cardiac output (CO) decreases progressively as the IAP increases.

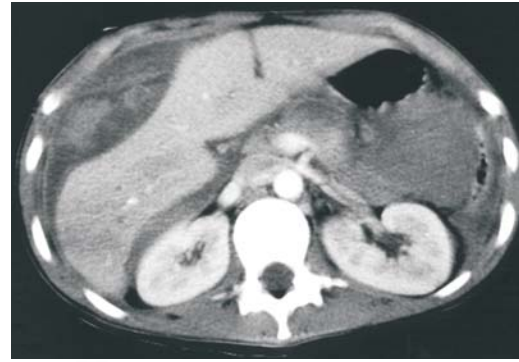
Intravenous volume expansion will increase the cardiac output and central filling pressures in ACS, but will not correct the other manifestations of ACS, including depressed renal function and splanchnic bloodflow. An actual depression of myocardial function occurs with ACS due to marked increase in afterload, as well as impairment of venous return.

Renal system: Oliguria develops despite measured normal or mildly elevated CVP and PAWP. The renal vein and inferior vena cava are compressed. In addition, renal vascular resistance increases several fold in ACS. Direct compression of the renal parenchyma also contributes to the renal dysfunction. Oliguria is often the earliest sign of ACS and anuria follows if the IAP is not reduced.

Abdominal and visceral effects: Clinically, the abdominal girth increases and the abdomen becomes more tense as the IAP increases. Splanchnic blood flow decreases as ACS develops. These studies have identified physiologic derangements that occur with increased IAP which may play a role in the development of SIRS in patients with ACS.

Central nervous system: Elevated IAP resulted in increased intracranial pressure (ICP) and decreased cerebral perfusion pressure (CPP). The proposed mechanism is functional obstruction of jugular venous drainage due to the elevated pleural pressures and CVP.

Eyes: Increased IAP has been associated with the rupture of retinal capillaries, resulting in the sudden onset of decreased central vision (Valsalva retinopathy).



A positive "round belly" sign with a ratio of anteroposterior-to-transverse diameter superior to 1:0.80. CT scan at level at which left renal vein crosses aorta shows hemoperitoneum. At this time, ratio of anteroposterior-to-transverse diameter was equal to 1:0.64.

INTRA-ABDOMINAL HYPERTENSION (IAH) ASSESSMENT ALGORITHM

- Patients should be screened for IAH/ ACS risk factors upon ICU admission and with new or progressive organ failure.
- If two or more risk factors are present a baseline IAP measurement should be obtained.
- If IAH is present serial IAP measurements should be performed throughout the patient's critical illness.

Patient has PvVO or more risk factors for IAH/ACS upon either ICU admission or in the presence of new or progressive organ failure

Measure patient's IAP to establish baseline pressure

AP measurements should be:

1. Expressed in mmHg (1 mmHg=1.36 cm H₂O)
2. Measured at end-expiration
3. Performed in the supine position
4. Zeroed at the iliac crest in the mid-axillary line
5. Performed with an instillation volume of no greater than 25 ml of saline [1 mL/kg for children up to 20 kg]
6. Measured 30-60 seconds after instillation to allow for bladder detrusor muscle relaxation (for bladder technique)
7. Measured in the absence of active abdominal muscle contractions

Sustained IAP ≥ 12 mmHg?

YES

NO

Patient has IAH

Patient does not have IAH

Notify patient's doctor of elevated IAP. Proceed to IAH/ACS management algorithm.

Observe patient. Recheck IAP if patient deteriorates clinically.

Risk Factors for IAH / ACS

1. Diminished abdominal wall compliance
 - Acute respiratory failure especially with elevated intrathoracic pressure
 - Abdominal surgery with primary fascial or tight closure
 - Major trauma / burns
 - Prone positioning, head A bed > 30 degrees
 - High body mass index (BMI) central obesity
2. Increased intra-luminal contents
 - Gastroparesis
 - Ileus
 - Colonic pseudo-obstruction
3. Increased abdominal contents
 - Hemoperitoneum / pneumoperitoneum
 - Ascites / liver dysfunction
4. Capillary leak / fluid resuscitation
 - Acidosis (pH<7.2)
 - Hypotension
 - hypothermic (core temperature <33°C)
 - Polytransfusion (>10 units of blood / 24hrs)
 - Coagulopathy(platelets < 55000/mm³ OR partial thromboplastin time (PTT) > 2 times normal OR prothrombin time (PTT) <50% OR international standardized ratio (INR) > 1.5)
 - Massive fluid resuscitation (> 5 L /24 hours)
 - Pancreatitis
 - Oliguria
 - Sepsis
 - Major trauma /burns
 - Damage control laparotomy

IAH Grading	
Grade I	IAP 12-15 mmHg
Grade II	IAP 16-20 mmHg
Grade III	IAP 21-25 mmHg
Grade IV	IAP ≥ 25 mmHg

Abbreviations
IAH - intra-abdominal hypertension
ACS - abdominal compartment syndrome
IAP - intra-abdominal pressure

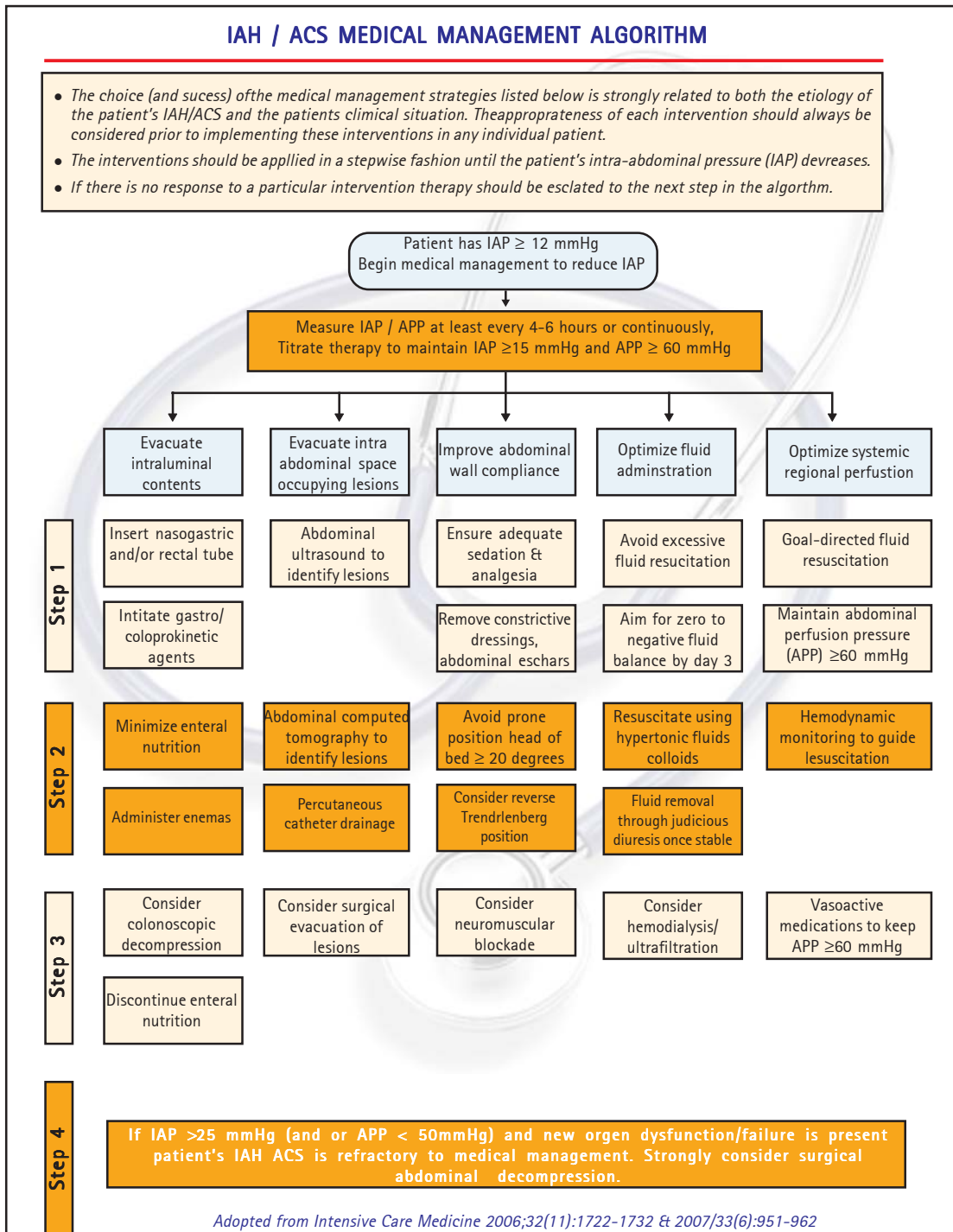
Diagnosis

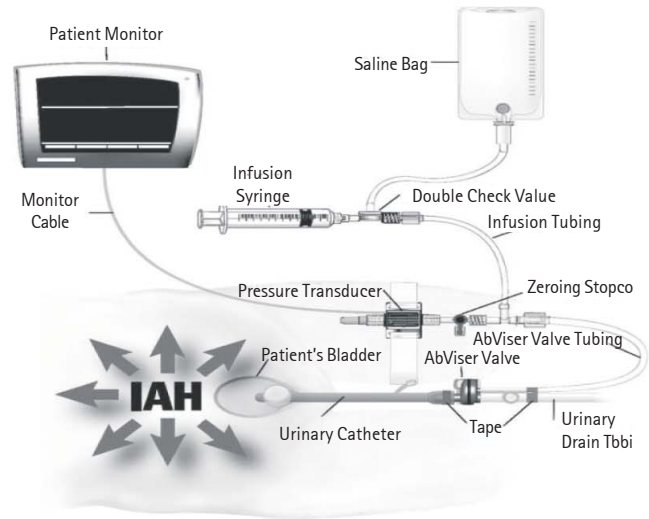
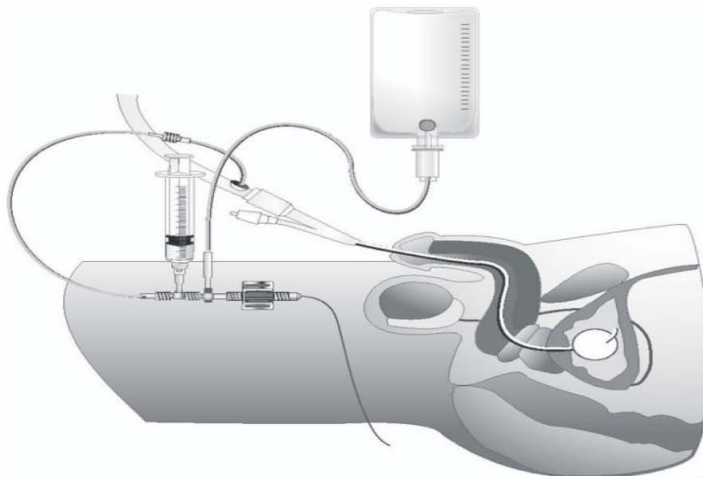
To diagnose and intervene early in the course of ACS, a high index of suspicion must be maintained. Clinically, the syndrome consists of the association of abdominal distension with increasing peak inspiratory pressures, increased central venous pressure (if the patient is euvoletic), oliguria and hypercarbia. Often, a diagnosis of ACS should be made on the basis of clinical suspicion and

decompressive laparotomy performed without attempts at measuring IAP. In the early phases of ACS, when oliguria may be the only sign, measurement of IAP is useful.

Measurement of IAP:

Measurement of bladder pressure, measurement of the gastric pressure or measurement of the IAP using a long femoral venous catheter placed in the inferior vena cava. The most accurate and





simple way to determine the IAP is indirectly by measurement of the bladder pressure using a Foley catheter. The bladder pressure is essentially equivalent to the IAP.

The AbViser system integrates directly with the patient's Foley catheter, transducer and monitoring system. Its unique valves allow clinicians to easily infuse saline into the bladder and rapidly measure Intra-Abdominal Pressure.

Surgical Interventions

If these medical treatments fail, decompressive laparotomy has been shown to effectively reduce IAP and improve organ function in critically ill patients. Open abdomen treatment or laparostomy was initially intended for patients with diffuse intra-abdominal infections, but open abdomen treatment—either prophylactic or the therapeutic—is becoming more common in the ICU. Several techniques to cover the open abdomen are available, but the Bogota bag, prosthetic material and vacuum systems are most often used. The abdomen may be opened in the intensive care unit, however, the operating room is preferable. If the abdomen is opened in the ICU, the operating room must be prepared to accept the patient if surgically correctable bleeding is identified at the time of decompressive laparotomy. A forced fascial closure of the abdomen should be avoided, such as in patients with massive retroperitoneal hematoma, visceral edema or intra-abdominal packs.

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 medical management and/or decompressive laparotomy can decrease morbidity and mortality.

References

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