Abdominal Compartment Syndrome

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Definitions: abdominal compartment syndrome (ACS)—organ dysfunction due to acute and sustained elevation of intraabdominal pressure (IAP); timely operative decompression usually required (depending on severity); functional impairment of organs or systems usually resolves after decompression; increased IAP not synonymous with ACS (obesity, ascites, pregnancy, or tumor may increase IAP, but these do not necessitate immediate surgical decompression); IAP — defined as steady-state pressure concealed within abdominal cavity; elevated IAP common in intensive care unit (ICU); IAP increases and decreases with respiratory movement (may be affected by mechanical ventilation); IAP also affected by volume of hollow viscus organs, space-occupying lesions, and conditions that limit expansion of abdominal wall (eg, severe burns to anterior abdominal wall or trunk)

Etiologies: intraperitoneal — bleeding; bowel obstruction; massive ileus; acute gastric dilatation (patients may present with hypotension and abdominal distention; may appear to be bleeding, but hypotension resolves after decompression); pneumoperitoneum (associated with laparoscopy); abdominal packing; abscess peritonitis; visceral edema common factor; retroperitoneal — pancreatitis; bleeding; aortic surgery; ruptured abdominal aortic aneurysm; abscess; visceral edema common factor here as well; abdominal wall — burns; repair of defects in abdominal wall (eg, large abdominal hernias); tight closure of laparotomy; abdominal wall edema common factor; abdominal perfusion pressure — influences prognosis; analogous to cerebral perfusion pressure; better predictor of organ failure and outcome than arterial pH, lactate level, or base deficit

Pathophysiology
Cardiovascular: increased IAP decreases venous return and cardiac output (CO); systemic vascular resistance increases; cardiovascular system may collapse; hypovolemia (common in surgical patients) worsens symptoms, do does ventilation that requires high positive end-expiratory pressure (PEEP); wedge and central venous pressure may be falsely elevated due to effect of IAP on inferior vena cava (IVC); cardiovascular disturbance may eventually mimic congestive heart failure; cardiovascular symptoms should improve with fluid resuscitation in early stages of ACS

Pulmonary: high IAP limits compliance by causing elevation of diaphragm; suspect ACS if peak inspiratory pressure increases and remains elevated; case — patient appeared too edematous to allow closure of incision after trauma and exploratory laparotomy; although abdomen left open, packing placed around liver caused ACS
Renal: glomerular filtration rate (GFR) decreases with CO; urine output drops; fluid resuscitation may cause CO and blood pressure to normalize transiently; however, oliguria does not resolve if IAP severe; decompression of abdomen usually required to protect kidneys; compression of renal vein or kidney may contribute to renal failure; urine output and IAP inversely correlated; majority of patients develop oliguria if IAP 15 to 20 mm Hg; anuria occurs when IAP 20 to 30 mm Hg (volume, dopamine, and diuretics ineffective); decreased CO and GFR require immediate decompression
Gastrointestinal: hepatic arterial and microcirculatory compromise may occur, and portal flow decreases if IAP 10 mm Hg; mesenteric and mucosal blood flow impaired at IAP 20 mm Hg; intestinal ischemia occurs at IAP of 40 mm Hg (at microcirculatory level); IAP and splanchnic perfusion inversely correlated; metabolism of intestinal mucosa changes with diminished blood flow; generation of free radicals, translocation of bacteria, multiple organ failure, and sepsis possible; decreased perfusion due to increased IAP possible cause of ischemic gastritis, acalculous cholecystitis, postoperative pancreatitis, and ischemic colitis; effects on abdominal wall may include dehiscence, wound infection, and necrotizing fasciitis

Increased intracerebral pressure (ICP): often caused by increased IAP; University of Maryland has demonstrated that patients in ICU with severe traumatic brain injury, increased ICP, and no response to standard therapy experience decrease in ICP after abdominal decompression

Diagnosis: requires high index of suspicion; suspect development of ACS (or increased IAP) in patients with tense or distended abdomen, or any evidence of organ dysfunction during surgery or in early postoperative period; measurement of IAP — consider as additional vital sign; measurement of central venous pressure via catheter in iliac veins or IVC possible surrogate for IAP, as gastric pressure measured with nasogastric tube; however, measurement of pressure in bladder most sensitive and accurate indirect method of measuring IAP

Measurement of intrabladder pressure: evidence of organ dysfunction or physical abdominal distention adequate indication for objective measurement of IAP in postoperative patients; technique — place Foley catheter into bladder; empty, then inject 50 to 100 mL of sterile saline into bladder; place...
needle into aspiration port of drainage tube and connect to pres-  
...sure transducer monitor; normal IAP — 0 to 5 mm Hg; (5–7 mm Hg acceptable in ICU); 10 to 15 mm Hg acceptable after laparotomy (septic shock and acute abdomen increase IAP); IAP > 15 mm Hg can cause significant end-organ dysfunction; if higher cutoff used or additional testing performed to con-  
firm organ dysfunction, window of opportunity during which intervention effective may be missed; data show devastating outcomes associated with failure to intervene when IAP > 25 mm Hg; grading — see Table 1; indications — consider routine measurements in patients with severe injuries or who required aggressive fluid resuscitation after surgery or procedure; pit-  
falls — conditions that may cause inaccurate measurements include neurogenic or small bladder, history of bladder surgery, intra- 
peritoneal adhesions, asites, hepatic failure, and cirrhosis  

Summary: common signs of ACS include unexplained meta-  
bo lic acidosis, increasing abdominal distention, oliguria, elevated peak-airway pressures, hyperventilation refractory to  
increased ventilation, hypoxemia, and elevated ICP; IAP fol-  

ows continuum from normal, to abdominal hypertension without organ dysfunction, to development of ACS if elevated  

pressure remains untreated

Secondary ACS: causes — include sepsis, capillary leak syn- 
drome, circumferential burns, and massive resuscitation; bowel edema and ascites induced by massive fluid resuscita- 
tion; studies — administration of 20 L crystalloid or 30 U of  

blood associated with 67% mortality; 16 U of crystalloid or 13  
U of packed cells associated with mortality of 38% in trauma  

patients and 100% in nontrauma patients  

Management: be aware of patients at increased risk; monitor IAP serially; optimize systemic perfusion; institute medical interventions; perform surgical decompression in patients with poor response; decompressive laparotomy — speaker prefers performing in operating room rather than ICU; timely inter- 
vention may result in reversal of organ dysfunction in < 93% of patients; grading and organ dysfunction — oliguria of 65% and 100% associated with ACS grades III and IV; cardiovas- 
cular disturbance increases with grade (100% at grade IV); treatment — depends on grade of ACS (see Table 1); reperfu- 
sion syndrome — common after decompression; patients may worsen metabolically before improving; treat with continued fluid, bicarbonate, mannitol, and vasopressors

TABLE 1. GRADING AND TREATMENT OF ACS

<table>
<thead>
<tr>
<th>Grade</th>
<th>Bladder Pressure (mm Hg)</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>I</td>
<td>10 to 15</td>
<td>Maintain normovolemia</td>
</tr>
<tr>
<td>II</td>
<td>16 to 25</td>
<td>Hypervolemic resuscitation</td>
</tr>
<tr>
<td>III</td>
<td>26 to 35</td>
<td>Decompression</td>
</tr>
<tr>
<td>IV</td>
<td>&gt;35</td>
<td>Decompression and reexploration</td>
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Abdominal closure: towel clamps — do not prevent increase of IAP; no longer used for temporary closure; sterile plastic (Bogota) bag — stitched over wound; allows sterile bowel evisceration; wound gradually closed over time; does not apply negative pressure, so skin graft may be needed to close abdominal wall (results in large hernia); mesh — does not apply negative pressure; covers bowel, but also requires skin graft; speaker’s method — plastic, large towel, and surgical sponges used to cover wound; Jackson-Pratt (JP) drains placed and exteriorized through separate stab wounds; wound covered with antimicrobial incise drape (Ioban); JP drains attached to negative pressure, which maintains minimal distance between edges of abdominal wall; success rate for complete closure in 7 and 10 days higher than with closure that does not apply negative pressure

Extremity Compartment Syndrome

George Velmahos, MD, PhD, Professor of Surgery, Harvard Medical School, and Chief of Trauma, Massachusetts General Hospital, Boston

Background: extremity compartment syndrome (ECS) commonly seen but diagnosis often missed; all surgeons should be knowledgeable about ECS; entire human body compilation of compartments confined within inelastic fascia or bone

Pathophysiology: edema or hemorrhage within compartment elevates pressure, which leads to compartment tamponade fol- 

lowed by ischemia of muscle; ischemia destroys tight cell junc- 
tions of vascular endothelium; resultant leakage of fluid into third space exacerbates edema and microhemorrhage; nerves (most sensitive structure) affected early (often irreversibly) in process; may cause muscle infarction, Altmann contractures, loss of limb, and (possibly) loss of life; missed or delayed diag- 

nosis and treatment of ECS can be life threatening; destruction of muscle releases myoglobin, which results in damage to kid-

neys and other organs; ischemia and reperfusion injury cause organ failure and, eventually, death

Compartment pressure in ECS: 0 to 10 mm Hg normal; 10  

to 20 mm Hg allowable in patients with critical illness and edema; 20 to 30 mm Hg considered “gray zone”; >30 mm Hg incompatible with survival of muscle and nerves (decompres- 

sion required); pressure ranges discovered during experiments in dogs; fluid injected into animal limbs increased pressures; when distention no longer possible (at 25–30 mm Hg), small increases in volume cause large increases in pressure; distal pulse — large vehicle blood vessels operate at higher pres- 

su res than smaller perfusion vessels (arterioles, venules, and capillary network), which close off at lower pressures; there- 

fore, distal pulse can remain intact in large vessels in full- 

blown ECS despite lack of oxygen or nutrient transport within compartment

Diagnosis: use “6 Ps” mnemonic; pain — out of proportion to injury, or persisting after appropriate treatment; pressure — requires palpation of tense compartment; edema noncompressible; paresthesia — early sign of ECS; nerves sensitive to pressure early in process; paralysis — permanent damage occurs when pressure on nerves persists; pulse — lost only after ECS advanced; pallor — occurs late in process (eg, foot remains perfused and pink during development of ECS in calf)

ECS of calf: location of 70% of cases; calf contains 4 compart- 
ments; anterior compartment located next to tibia; lat- 

eral compartment overlies fibula; superficial compartment located posteriorly; deep posterior compartment closer to bone; deep peroneal nerve runs through anterior compart- 

ment; superficial peroneal nerve runs through lateral compart- 

ment; sural nerve in superficial posterior; tibial nerve in deep posterior compartments; anterior and lateral compart- 
m ents most inelastic and develop ECS early in process; deep peroneal nerve compartment innervates web space between first and second toes (lack of sensation in web space provides early indication of ECS in anterior compartment); measurement of pressure — several tools available; measurement in all compartments crucial (20% of patients do not have involvement of all 4); measurements valuable but not always definitive (combine with physical examination [PE], symp-

toms, and risk factors when making diagnosis)

Treatment: leave windows in (or open) casts or dressings to allow for PE; mannitol and bicarbonate — may help balance meta- 

bo lic acidosis and scavenge 02 free radicals (evidence limited); decompression — anterior and lateral compartments acces- 

sible through lateral incision in calf; superficial and deep pos- 

terior compartments accessible through medial incision; create
transverse incision in fascia to locate intermuscular septum and differentiate between anterior and lateral compartments; speaker recommends use of large incisions for fasciotomies (particularly if inexperienced); subcutaneous fasciotomy — blind opening of fascia inside small skin incision; risks damage to nerves or inadequate decompression if surgeon inexperienced; single-incision fasciotomy — useful for experienced surgeons; make low single incision over fibula (instead of 2); allows access to all 4 compartments after elevation of skin

**Other common sites of ECS:** forearm — second most common; commonly considered compartments dorsal, ventral, and (possibly) mobile wad; 2 incisions adequate to reach compartments; dorsal incision straight; straight medial incision may extend over carpal tunnel to palm (“lazy S”-shaped incision also used); thigh — third most common; contains 3 compartments; use medial incision to decompress medial compartment; use lateral incision to decompress anterior and posterior compartments

**Uncommon sites:** include gluteal, foot, deltoid, and iliacus muscles; dark urine and elevated creatinine phosphokinase in ill patients should prompt examination to avoid missing ECS; gluteal compartment syndrome — often seen in elderly patients treated with warfarin (e.g., Coumadin, Jantoven, Marevan) after fall and development of large gluteal hematoma; if untreated, necrosis of muscle and compression of sciatic nerve may ensue

**Pinfalls in management:** diagnosis — failure to suspect ECS and follow closely (e.g., ignoring persistent pain; failing to recognize pain out of proportion to injury); use of pressure measurements only, without correlation with clinical signs; failure to examine under casts and bandages; treatment — inadequate subcutaneous fasciotomy (making incision too small); failure to decompress all compartments; failure to treat with mannitol and bicarbonate before decompression; inadequate skin incision

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**Suggested Reading**

1. All the following statements about abdominal compartment syndrome (ACS) are true, except:
   (A) Untreated ACS leads to organ dysfunction
   (B) All significant increases in intraabdominal pressure (IAP) represent ACS
   (C) ACS usually requires timely operative decompression
   (D) Functional impairment of organs or systems usually resolves after decompression

2. Which of the following is the common factor in intra- and retroperitoneal etiologies of ACS?
   (A) Bowel obstruction
   (B) Peritonitis
   (C) Visceral edema
   (D) Acute gastric dilatation

3. Increase in IAP can lead to increases in:
   1. Systemic vascular resistance
   2. Urine output
   3. Peak inspiratory pressure
   4. Portal blood flow
   5. Generation of free radicals
   (A) 1,2
   (B) 3,4
   (C) 1,3,5
   (D) 2,4,5

4. Elevation of IAP has no effect on organ systems outside the abdomen.
   (A) True
   (B) False

5. Choose the correct statement about measurement of IAP.
   (A) Should be considered an additional vital sign
   (B) Is inaccurate if measured via the urinary bladder
   (C) Is required only in patients who have had surgery or have an abdominal tumor
   (D) Intervention is not indicated until readings exceed 30 mm Hg

6. Patients with grade II ACS require _______, while patients with grade _______ ACS require decompression and reexploration.
   (A) Maintenance of normovolemia; III
   (B) Maintenance of normovolemia; IV
   (C) Hypervolemic resuscitation; III
   (D) Hypervolemic resuscitation; IV

7. All the following statements about extremity compartment syndrome (ECS) are true, except:
   (A) It is an extremely rare condition
   (B) Nerves are affected early on in the process
   (C) Leads to compartment tamponade, followed by ischemia
   (D) It can be life threatening

8. ECS can generally be ruled out if the distal pulse is intact.
   (A) True
   (B) False

9. Which of the following statement(s) about ECS is(are) true?
   (A) It is not necessary to measure all compartments in the calf
   (B) Low pressure measurements rule out ECS even if the patient has pain and the calf feels tense
   (C) A and B
   (D) Neither A nor B

10. Which of the following muscles is(are) possible site(s) of ECS?
    (A) Gluteal
    (B) Deltoid
    (C) Iliacus
    (D) All the above

Answers to Audio-Digest General Surgery Volume 60, Issue 09: 1-C, 2-B, 3-A, 4-A, 5-D, 6-C, 7-B, 8-A, 9-A, 10-D