The Abdominal Compartment Syndrome
Definition

- A syndrome of intra-abdominal hypertension resulting in organ dysfunction which may be reversed by abdominal decompression
History

- 1863: Etienne-Jules Marey wrote that ‘the effects that respiration produces on the thorax are the inverse of those present in the abdomen’
- 1873: EC Wendt of Germany measured IAP through the rectum, noting that elevated pressures corresponded with diminished excretion of urine
- 1890: Heinricius of Germany found that IAPs between 27 and 46 cmH₂O were fatal to animals owing to prevention of respiration
History

- 1911: Haven Emerson publishes his treatise, 'intra-abdominal pressures'
- contraction of the diaphragm identified as chief factor in the rise of IAP during inspiration
- excessive IAP can cause death from cardiac failure even before terminal asphyxia develops
- Observed that cardiovascular collapse associated with 'distention of the abdomen with gas or fluid, as in typhoid fever, ascites, or peritonitis' are caused by 'overloading the resistance in the splanchnic area' and that 'relief of the laboring heart is constantly seen after removal of ascitic fluid.'

History

- **1940: Sir William Heneage Ogilvie**
  - In a letter to Lancet described ‘a dodge that has twice helped me out’, a technique for avoiding closing a ‘burst abdomen’
  - Sutured vaseline impregnated canvas to wound edges to cover abdominal contents

- **1984: Kron et al**
  - Published landmark case series on IAH
  - 11 patients with elevated IAP after aortic repair (>30 mmHg)
  - 7 patients decompressed with immediate diuresis
  - The other 4 patients died

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1 Ogilvie WH. The late complications of abdominal war wounds. Lancet 1940;2:253-256
Pathophysiology

- Causes of intra-abdominal hypertension
  - Primary: due to intra-abdominal process
    - Trauma: Intra-abdominal bleeding, MAST, damage control surgery
    - Retroperitoneal: Pancreatitis, ruptured AAA, abscess
    - Intraperitoneal: Gastric dilatation, bowel obstruction, visceral edema, tension pneumoperitoneum
    - Abdominal wall: Burn eschar, reduction of large hernias
  - Secondary: due to massive fluid administration for extra-abdominal process
    - Capillary leak
    - Ischemia-reperfusion: release of inflammatory mediators, free radicals

# Pathophysiology

## Clinical Effects of Increased Abdominal Pressure

<table>
<thead>
<tr>
<th>System</th>
<th>Clinical Effects</th>
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</thead>
<tbody>
<tr>
<td>Hemodynamics</td>
<td>Decreased cardiac output&lt;br&gt;Decreased preload&lt;br&gt;Increased afterload&lt;br&gt;Increased CVP and PCWP</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Increased peak inspiratory pressures&lt;br&gt;Increased airway pressures&lt;br&gt;Decreased PaO₂&lt;br&gt;Increased PaCO₂&lt;br&gt;Decreased dynamic compliance</td>
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<td>Renal</td>
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Pathophysiology

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Elevation of diaphragm transmits pressure to heart and great vessels.

CVP and PCWP are “spuriously” elevated – not a reflection of volume status.

Pathophysiology

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- Increases in pleural pressures evident at IAP of 15 mmHg or greater
- Exacerbated by PEEP
- Normalizes after surgical decompression

Pathophysiology

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- IAP of 15-20 mmHg coincides with oliguria; over 30 mmHg causes anuria
- Compression of renal vasculature, parenchyma
- Stimulation of juxtaglomerular apparatus

### Pathophysiology

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</tr>
<tr>
<td></td>
<td>Decreased pHo</td>
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- IAH found to decrease perfusion of every intra-abdominal viscus (except adrenals)
- Effect persists even when cardiac output is corrected

## Pathophysiology

### Clinical Effects of Increased Abdominal Pressure

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- Increase in IAP
- Increase in ITP
- Increase in CVP
- Decrease in CPP

Statistics

- Review of 13,817 consecutive trauma admissions revealed incidence of 15% among patients undergoing staged laparotomy with packing¹
- Of 145 acutely injured patients with ISS ≥ 15, twenty-one (14%) developed ACS²
- Review of 70 patients with life-threatening penetrating injuries revealed an incidence of 33%³
- In a prospective study of 706 consecutive patients admitted to a trauma ICU incidence of ICH was 2% and ACS 1%⁴

Diagnosis

- High index of suspicion
- Clinical signs:
  - Abdominal distention, tension
  - Decreased urine output
  - Elevated filling pressures
  - Elevated ICP
  - Worsening acidosis
  - Elevated peak airway pressures
- Confirmation

Balogh Z, McKinley BA, Holcomb JB, Miller CC, Cocanour CS, Kozar RA, Valdivia A, Ware DN, Moore FA. Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma 2003 May;54(5):848-59
Diagnosis

- Direct monitoring of IAP
  - Intraperitoneal catheter connected to water manometer or pressure transducer
  - Most accurate
  - Preferred in experimental studies
  - Clinical use limited by risk of peritoneal contamination, bowel perforation
Diagnosis

- Indirect monitoring of IAP
  - Measuring pressure within abdominal organs
  - Less invasive
  - Less reliable
  - Transfemoral caval catheter
  - Gastric tube
  - Rectal tube
  - Intravesical pressure monitoring
Diagnosis

- Intravesical monitoring
  - Most closely reflects direct monitoring\(^1,\(^2\)
  - Foley clamped distal to aspiration port
  - 50 to 100 cc saline injected into bladder
  - 16-guage needle connected to pressure transducer, inserted into aspiration port

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Treatment: An Ounce of Prevention

- Identify patients at risk
  - Major trauma, damage control surgery
  - Laparotomy for major bleeding
  - Edematous and/or ischemic bowel
  - Abdominal vascular procedures
  - Mechanically difficult closure
  - High-volume resuscitation

- Avoid primary fascial closure

Treatment: An Ounce of Prevention
Treatment: Surgical decompression

- Timing of intervention
  - IAH ≠ ACS
  - Recommendations differ
    - Modest IAH + organ dysfunction\(^1\)
    - Marked IAH\(^2\)
  - No absolute evidence-based guidelines

Treatment: Surgical Decompression

- Proposed ACS grading system:

<table>
<thead>
<tr>
<th>Grade</th>
<th>IAP (mmHg)</th>
<th>Associated signs</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>10–15</td>
<td>No signs of ACS</td>
<td>Maintain normovolemia</td>
</tr>
<tr>
<td>II</td>
<td>16–25</td>
<td>May have increased PAWP and oliguria</td>
<td>Hypervolemic resuscitation may be employed but could have drawbacks</td>
</tr>
<tr>
<td>III</td>
<td>26–35</td>
<td>Anuria, decreased cardiac output, raised PAWP</td>
<td>Consider abdominal decompression</td>
</tr>
<tr>
<td>IV</td>
<td>&gt;35</td>
<td>Anuria, decreased cardiac output, raised PAWP</td>
<td>Abdominal decompression and re-exploration</td>
</tr>
</tbody>
</table>

**TABLE III**

<table>
<thead>
<tr>
<th>Grade</th>
<th>UO &lt; 0.5 mL/kg/hr</th>
<th>PAP &gt; 45</th>
<th>SVR &gt; 1000</th>
<th>DO₂l &lt; 600</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>II</td>
<td>0%</td>
<td>40%</td>
<td>20%</td>
<td>20%</td>
</tr>
<tr>
<td>III</td>
<td>85%</td>
<td>78%</td>
<td>65%</td>
<td>57%</td>
</tr>
<tr>
<td>IV</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

PAP = peak arterial pressure (cm H2O); DO₂l = oxygen delivery index (mL O₂/min/m²); SVR = systemic vascular resistance (dynes/cm² * s); UO = urine output (mL/min).

Treatment: Hazards of Laparostomy

- “Reperfusion injury”
  - Morris et al\(^1\) reported fatal cardiac arrest in 4 of 16 patients undergoing decompressive laparostomy
  - Prevention
- Abrupt shift in vent requirements
- Sudden fluid shifts
- Loss of tamponade
- Complications of open abdomen
  - Large surface for fluid loss
  - Exposes viscera to trauma, desiccation
  - Route for infection

Treatment: Nonoperative management

- Progression of IAH to ACS may be arrested by nonoperative maneuvers
  - Paralysis
  - Diuresis or fluid resuscitation

- Attempts at management of ACS with percutaneous decompression have been almost universally catastrophic
  - Patients with ACS secondary to abdominal burns may represent an exception
  - Alain and Sherman (2001):
    - Case series in which ACS in burn patients was managed successfully by percutaneous intraperitoneal drainage catheter

Outcome

- Intervention successful vis-à-vis early endpoints
  - Airway pressures
  - Cardiac output
  - Urine output

- High mortality rate (10.6-68%)
  - Most commonly succumb to MOF, sepsis
  - Paucity of data on short-term and long-term morbidity

ACS and the General Surgeon

- Preponderance of data on ACS based on trauma patients
- Retrospective review by McNelis et al of nontrauma SICU admissions developing ACS:
  - Study population:
    - Eighteen patients
    - M:F ratio 1:2
    - 8 AAA repairs
    - 6 laparotomies
    - 3 cases of pancreatitis
    - 1 cerebral aneurysm
  - Appropriate response to decompression (↑UO, ↓PIP, ↑CO)
  - Mortality 61.1%

ACS and Acute Pancreatitis

- Current paradigm for acute pancreatitis:
  - Delayed operation
  - Operation for infected necrosis
- Retrospective review of 23 patients with pancreatitis and ACS:

<table>
<thead>
<tr>
<th>Severe Acute Pancreatitis complicated with ACS³</th>
<th>Total</th>
<th>Laparostomy performed</th>
<th>No laparostomy performed</th>
<th>SIRS stage</th>
<th>Infected stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>23</td>
<td>18</td>
<td>5</td>
<td>17</td>
<td>6</td>
</tr>
<tr>
<td>Mortality (%)</td>
<td>7(30.4)</td>
<td>3(16.7)</td>
<td>4(80%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Conclusions

- Abdominal compartment syndrome is a potentially fatal constellation of symptoms with many disparate etiologies
- A high index of suspicion and astute decision-making are required for successful management
- Further data would help guide treatment of this syndrome in both the injured and the general surgical population