Intra-abdominal Compartment Syndrome

Abdominal compartment syndrome is defined by intra-abdominal hypertension associated with multi-organ dysfunction; having an adverse impact on pulmonary, cardiovascular, renal, splanchnic, musculoskeletal, integumentary, and CNS physiology, resulting in significant morbidity and mortality and requiring high index of suspicion, prompt recognition, and timely intervention

ANESTHETIC CONSIDERATIONS:

- Syndrome of intra-abdominal hypertension leading to dysfunction of multiple end organs including respiratory, cardiovascular and renal systems:
  - Decreased cardiac function and end organ blood flow
  - Hypoxia secondary to restrictive ventilation (decreased pulmonary compliance)
  - Associated with ARDS (sepsis, transfusion of more than 15 units of pRBCs in 24 hours, pulmonary contusion, and long bone fractures)
  - Full Stomach
  - ARF (oliguria)
  - Hypovolemia, coagulopathy
- Risk of systemic acid and potassium loading upon abdominal decompression leading to potential hypotension or fatal arrhythmia

ANESTHETIC GOALS:

- Hemodynamic Goals :
  - Maintain high preload (fluid will rescue esp. when fluid removed)
  - Maintain afterload
  - Increase contractility (inotropic support if necessary)
  - NSR
  - High rate (as preload is compromised and therefore CO is HR dependent)

HISTORY

- High index of suspicion is important
- Setting is usually one of a ventilated patient in the ICU with risk factors
- Review of chart will reveal considerations of AMPLE history, looking for events thus far in the patient's care → past history, diagnoses, and surgery thus far, degree of fluid resuscitation etc.
- Review current and past monitored vitals for signs of worsening of airway pressures, minute ventilation, hypercarbia, hypoxia, acidosis (both respiratory and metabolic), lactate, renal function and urine output
- Review vitals, filling pressures, CO and SVR (if available)
- Ask about monitoring of IAP thus far if available
- On evaluation at the bedside, ensure a functioning airway and review the status of oxygenation and ventilation

PHYSICAL

- **VITALS**
- **CVS** – signs of right and left heart failure, further, evaluate the cardiovascular status for signs of shock and hypoperfusion
- **RESP** – examine to rule out other causes of restricted ventilation / high airway pressure (if present) including a partially obstructed ETT, bronchospasm, and pneumothorax (high airway pressures with low tidal volumes indicate poor compliance and restrictive lung pattern)
- **GI** – examine for a tense (and perhaps tender, depending on LOC) abdomen
- **GU** – U/O

INVESTIGATIONS

- **Labs**
  - CBC (Hb), lytes, BUN / Cr, lactate
  - ABG (hypercarbia, hypoxia, acidosis)
  - Ensure a crossmatch done
- **Imaging**
  - Evaluate CXR and ECG
- **Measuring IAP**
  - Can use direct intraabdominal pressure monometer (as in laparoscopic surgery), inferior vena cava line, gastric or most commonly bladder pressures
  - Bladder pressure procedure
    - Infuse 50-100 mL via Foley, clamp distal to aspiration port
    - Attach pressure transducer to aspiration port
    - Zero reference is the top of the symphysis pubis in supine position

Grades and Management of Abdominal Compartment Syndrome

Grade	Bladder Pressure (mmHg)	Clinical signs of ↑ Intraabdominal Pressure	Recommended Intervention
I	10-15	None	Maintain normovolemia
II	16-25	Oliguria, Mild ↑ Peak A/W Pressure	Hypervolemic Resuscitation
III	26-35	Anuric, ↓ CO, ↑ Peak A/W Pressure	Decompression
IV	>35	Anuric, ↓ CO, ↑ Peak A/W Pressure	Decompression and Exploration

OPTIMIZATION

- Surgeons unlikely to decompress abdomen based solely on elevation in bladder pressure in absence of clinical deterioration; unfortunately, once the elevation in intra-abdominal pressure is clinically overt, the damaging effects are likely to have already occurred
  - Earlier abdominal decompression of patients at high risk, especially those with higher peak airway pressures, may be the only available means of reducing morbidity and mortality
- Type of resuscitation fluid:

- Colloids and hypertonic saline / dextran may not improve early resuscitation hemodynamics, but may reduce edema and ascites in the first 24 hours post-injury
    - DCS and ACS are virtually unknown entities in Europe and the United Kingdom, where field resuscitation includes colloids
- Additionally, the use of antioxidants and other anti-inflammatory agents may reduce endothelial cell injury and the resultant tissue edema in the early post-injury period

- Perhaps consider the judicious administration of loop diuretics in the ICU
    - This may be facilitated by the availability of blood substitutes to optimize plasma volume without the penalty of interstitial volume expansion
- Aggressively correct coagulation deficits, hypothermia, acidosis and hypovolemia prior to decompression
- Reperfusion syndrome = severe hypotension, acidosis, hyperkalemia, coagulopathy +/- asystole with decompression
- Frequency of death is correlated with duration of abdominal compartment syndrome and degree of ARF

ANESTHETIC OPTIONS

- GETA

ANESTHETIC SETUP

- **Drugs**
    - Dopamine, epinephrine
- **Equipment**
    - Standard CAS monitors
    - Position in reverse Trendelenburg
    - Level 1, fluid warmer, a. line, CVP
    - NG, Foley
    - Pneumatic compression stockings

MANAGEMENT OF ANESTHESIA

- **Induction**
    - Nothing special, often intubated already, coming from ICU or postoperative
- **Maintenance**
    - PPV and careful with PEEP
- **Emergence**
    - Often returning to ICU

DISPOSITION & MONITORING

- ICU

COMPLICATIONS

- Cardiovascular collapse after reperfusion

PATHOPHYSIOLOGY

- Abdominal compartment syndrome (ACS) is defined by intra-abdominal hypertension (IAH) associated with organ dysfunction
    - Definition:
        - Intraabdominal pressure (> 20 mmHg or 27 cmH_20) (normal = 0-5 mmHg) + one of:
            - Decrease renal function (< 0.5 cc/kg/h or Cr >150 mmol/L) or
            - Peak Inspiratory Pressure > 50 cmH_2O
- Mortality range 25-71%
- High intra-abdominal pressure (IAP) has an adverse impact on pulmonary, cardiovascular, renal, splanchnic, musculoskeletal, integumentary, and CNS physiology, resulting in significant morbidity and mortality and requiring high index of suspicion, prompt recognition, and timely intervention
- **At risk patients** include:
    - Severe blunt and penetrating abdominal trauma
    - Ruptured abdominal aortic aneurysms
    - Retroperitoneal hemorrhage
    - Pneumoperitoneum
    - Neoplasm
    - Pancreatitis
    - Massive ascites
    - Liver transplantation
    - Massive fluid resuscitation, accumulation of blood and clot, bowel edema, and forced closure of a non-compliant abdominal wall are other common factors
    - External abdominal compression such as a circumferential burn eschar can also cause rise in IAP leading to the syndrome
- **Pulmonary dysfunction** is progressive with IAP rising above 15 mmHg - ensuing pressure effects on the thoracic cavity reduce pulmonary compliance, TLC, FRC and RV and increase atelectasis, shunt and PVR (the latter from reduced alveolar O_2 tension and increased intrathoracic pressures)
    - This leads to hypoxia, hypercapnia (from reduced minute ventilation), and ultimately respiratory failure – this is promptly reversible with abdominal decompression
- **Cardiovascular compromise** manifests as a reduced cardiac output with IAP rising above 20 mmHg, resulting from both a decreased venous return and an increased SVR
    - The preload reduction results from direct compression of the IVC and the portal vein and also from high intrathoracic pressure effects on both IVC / SVC flow and on diastolic filling by directly compressing the LV (when using PPV with high airway pressures)
    - The afterload effects are a combination of arteriolar vasoconstriction and elevated IAP
    - Increase in HR and contractility are only partly compensatory and the fall in cardiac output may further be compromised by concomitant pathophysiology (hypovolemia, acidosis, etc.)
    - Remember that CVP and PCWP measurements can be very misleading in the face of high intrathoracic pressures (although trending may still help)
- **Renal dysfunction** is reflected by reductions in renal plasma flow and GFR incrementally with rising IAP

- Oliguria develops at IAP of 15-20 mmHg and progresses to anuria above 30
- Mechanisms are pre-renal and renal
  - Pre-renal effect is related to cardiac output and therefore renal insufficiency partly improves with CO
  - Renal parenchymal compression elevates renal vascular resistance by compressing arterioles and veins (IAPs of 20 and 40 mmHg increase the resistance by 500 and 1500% respectively)
  - Ultimately renal plasma flow and GFR decrease and circulating renin, ADH and aldosterone increase (further increasing SVR) and acute renal failure ensues

- Splanchnic blood flow abnormalities are similarly affected by incremental changes in IAP with resultant organ dysfunction (**gut, liver**)
  - The effect on intestinal mucosa has the added problem of breakdown in the normal gut mucosal barrier allowing bacterial translocation and predisposition to septic complications
- **ICP** also rises in the setting of high IAP, although the mechanism is less clear (possibly related to impaired central venous outflow)
  - This relationship becomes more important in the setting of concomitant head injury and the CPP / ICP relationship

Laparoscopic surgery: Summary of potential physiologic changes	
• **TRENDELENBURG POSITION** ○ Circulation ○ Heart rate ○ Stroke volume ○ Respiration ○ Minute volume ○ Work of breathing ○ Lung volumes ○ Gas exchange	• **EXOGENOUS CO_2** ○ Circulation ○ Dysrhythmias ○ Contractility ○ Venous gas embolization ○ Respiration ○ Ventilation (dead space) ○ CO_2 hemostasis
• **PNEUMOPERITONEUM** ○ Circulation ○ Venous return (cardiac filling pressures) ○ Contractility (neural / humoral) ○ Afterload ○ Respiration ○ Minute ventilation ○ Airway pressure ○ Lung volumes (functional residual capacity) ○ Gas exchange (hypoxemia / hypercapnia)	• **REVERSE TRENDELENBURG** ○ Circulation ○ Venous return ○ Afterload ○ Respiration ○ Lung volumes ○ Work of breathing ○ Minute ventilation ○ Gas exchange

REFERENCES

- Christopher D. Raeburn, Ernest E. Moore, M.D. , The abdominal compartment syndrome is a morbid complication of post-injury damage control surgery, American Journal of Surgery, Volume 182 • Number 6 • December 2001
- Zsolt Balogh, M.D. , Bruce A. McKinley, Ph.D., Secondary abdominal compartment syndrome is an elusive early complication of traumatic shock resuscitation American Journal of Surgery Volume 184 • Number 6 • December 2002
- Abdominal Compartment Syndrome, Critical Care 2000, 4:23-29