Rescue Mechanical Ventilatory Strategies In Abdominal Compartment Syndrome (ACS)
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Compartment Syndrome

A condition in which increased pressure within a confined anatomic space adversely affects function and validity of tissues contained within.
Intra abdominal Hypertension (IAH)
Sustained or repeated pathologic elevation of intra abdominal pressure (IAP) ≥ 12 mm or Hg*

Abdominal Compartment Syndrome (ACS)
Sustained IAP ≥ 20 mm of Hg (with or without an APP < 60 mm of Hg) that is associated with new organ dysfunction / failure*

Abdominal Compartment Syndrome (ACS) :

Historical evolution

• ACS was first used by Fietsam et al \(^1\) in late 1980s to describe the pathophysiologic alterations resulting from IAH resulting from Aortic aneurysm surgery

• World society on ACS (WSACS – www.wsacs.org) was founded in 2004 to serve as a peer-reviewed forum and educational resource for all clinicians with an interest in understanding IAH and ACS \(^2\)

• First consensus definitions were in 2006 \(^3\) and 2007 \(^4\) – they were updated in 2013 \(^5\)

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**Consensus definitions 2006**

**Definition 1**
IAP is the steady-state pressure concealed within the abdominal cavity.

**Definition 2**
APP = MAP – IAP

**Definition 3**
FG = GFP – PTP = MAP – 2 * IAP

**Definition 4**
IAP should be expressed in mm Hg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line.

**Definition 5**
The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline.

**Definition 6**
Normal IAP is approximately 5–7 mm Hg in critically ill adults.

**Definition 7**
IAH is defined by a sustained or repeated pathologic elevation of IAP ≥ 12 mmHg.

**Definition 8**
IAH is graded as follows:
- Grade I: IAP 12–15 mm Hg
- Grade II: IAP 16–20 mm Hg
- Grade III: IAP 21–25 mm Hg
- Grade IV: IAP > 25 mm Hg

**Definition 9**
ACS is defined as a sustained IAP > 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction / failure.

**Definition 10**
Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention.

**Definition 11**
Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region.

**Definition 12**
Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

**Abbreviations:** ACS, abdominal compartment syndrome; APP, abdominal perfusion pressure; FG, filtration gradient; GFP, glomerular filtration pressure; IAH, intra-abdominal hypotension; IAP, intra-abdominal pressure; MAP, mean arterial pressure; PTP, proximal tubular pressure.
2013 Updated definitions of IAH and ACS

1. IAP is the steady-state pressure concealed within the abdominal cavity
2. APP = MAP - IAP
3. IAP should be expressed in mm Hg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line
4. The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline
5. IAP is approximately 5–7 mm Hg and around 10 mm Hg in critically ill adults
6. IAH is defined by a sustained or repeated pathologic elevation of IAP > 12 mm Hg
7. IAH is graded as follows:
   Grade I: IAP 12–15 mm Hg
   Grade II: IAP 16–20 mm Hg
   Grade III: IAP 21–25 mm Hg
   Grade IV: IAP > 25 mm Hg
8. ACS is defined as a sustained IAP ≥ 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction/failure
9. Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention
10. Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region
11. Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

Effect of IAH on Respiratory System Physiologic Implication *

- Intra thoracic Pressure ↑
  - Pleural Pressure ↑
    - FRC↓
  - All Lung Volumes ↓
    - (~ Restrictive disease)
  - Auto PEEP↑
    - PAP↑
  - Plateau airway pressure↑
  - Dynamic Compliance ↓
- Static Respiratory System Compliance↓
  - Static Chest wall compliance↓
  - Static Lung Compliance =

Effect of IAH on Respiratory System
Physiologic Implication * (continued)

- Hypercarbia $\uparrow$
- $\text{PaO}_2 \downarrow$ and $\text{PaO}_2/\text{FiO}_2 \downarrow$
- Dead Space Ventilation $\uparrow$
- Intrapulmonary Shunt $\uparrow$
- Lower Inflection Point $\downarrow$
- Upper Inflection Point $\uparrow$
- Extra vascular Lung Water $\equiv$$\uparrow$
  - Alveolar edema $\uparrow$
  - Compression Atalectasis $\uparrow$

### The Two Compartments *

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Progression of IAH
Physiological effects of IAH
Progression of IAH (IAP 12-15 mm of Hg)

**SIGNS and SYMPTOMS**

**Very subtle clinical findings**

- Occult ischaemia is occurring with little clinical evidence beyond IAP level
- Clinicians Can not feel abdomen or measure its circumference and gain any meaningful insight into the patients IAP level
  - May be difficult to mobilise excess fluid
  - May be difficult to wean from the ventilator
Progression of IAH
Physiological effects of IAH

Occult Organ Ischemia
IAP 16 – 20 mmHg

- Increased ICP, Decreased CPP
- Increased lung dysfunction
- Rising IAP pushes diaphragm further into chest
- Increased bowel edema and ischemia

- Increased CVP, Increased Wedge pressure (falsely elevated)
- Further decrease in Cardiac Output
- Worsening vena caval compression
- Decreased perfusion, oliguria, difficulty mobilizing fluids
- Increased acidosis
Progression of IAH (IAP 16-20 mm of Hg)

SIGNS and SYMPTOMS
All of the previous slide plus

- Unexplained acidosis
- CVP and wedge pressure are often falsely elevated (due to IAP transmission into CVP catheter)
  - Cardiac output decreasing
  - Urine output decreased
  - Peak and plateau pressures increasing on the ventilator
    - Hypoxemia, Hypercarbia and Atelectasis
- Abdominal distension might be visible but not reliable in obese patients
Progression of IAH
Physiological effects of IAH

Onset of Multiple Organ Dysfunction Syndrome (MODS) IAP > 20 mmHg

- Brain swelling and ischemia
- Increased peak pressure, difficult ventilation and oxygenation; VILI/ARDS
- Increased gut ischemia, Impending necrosis
- Cardiovascular instability
- Vena caval flattening
- Anuria/Acute Renal Failure (ARF)
- Further worsening of acidosis
Progression of IAH (IAP > 20 mm of Hg)

**SIGNS and SYMPTOMS**
All of the previous slide plus

- Worsening Acidosis
- Abdomen tense (exam not reliable in today’s obese population
  - Renal insufficiency /failure
- Pulmonary failure with significant difficulty ventilating
  - Cardio vascular instability
- Rising intra cranial pressure
How does mechanical ventilation influence intra abdominal pressure in critically ill without other risk factors for IAH *

• The study was conducted on 50 patients on MV and 50 patients without MV
  • The mean IAP in patients on MV was 6.7 ± 4.1 mm of Hg
  • The mean IAP in patients without MV was 3.6 ± 2.4 mm of Hg

How does IAH affect VILI?

- VILI is caused by strain on Lung structures

- IAH results in poor alveolar recruitment and increased shear stress with opening and closing of alveoli and thus increasing the incidence of VILI

- In Primary ACS, distant failure and secondary ARDS are caused by circulating cytokines

How does IAH affect Lung edema formation?

• In porcine model, it has been found that application of an IAP of 15 mm of Hg after Oleic Acid induced lung injury resulted in a greater than two fold increase in pulmonary edema as measured by extra vascular lung water (EVLW) *

Therapeutic consideration in IAH : Patient on MV

- Peep should be set to counteract IAP (also ITP) while at same time avoiding over inflation of already well ventilated regions of lungs*

- The fact that PEEP is expressed in cm of $H_2O$ (with conversion factor of 1.36 to mm of Hg) takes into account the average abdomino thoracic index of transmission of around 50%. Best PEEP (CM $H_2O$)=IAP(mm of Hg)

- During lung protective ventilation, plateau pressures should be limited to transmural or trans pulmonary plateau pressure ($P_{plat_{tm}}$) below 30 to 35 cm $H_2O$, otherwise end tidal $CO_2$ will increase**

$$P_{plat_{tm}} = P_{plat} - ITP = P_{plat} - IAP/2 < 35 \text{ cm } H_2O$$


Therapeutic considerations in IAH in Patients on MV (continued)

- IAH increases Lung Edema, therefore monitoring of Extra Vascular Lung Water Index (EVLWI) seems warranted *
- The combination of capillary leak, positive fluid volume and raised IAP put the patient for exponential risk for lung edema

**Body Position effects IAP**

- Putting an obese patient in upright position can cause ACS
- The abdomen should hang freely during prone positioning
- The reverse Trendelenburg position may improve respiratory mechanics, however it can decrease splanchnic perfusion

**Therapeutic consideration in IAH : Patient on MV**

- Consideration of neuromuscular blockade should balance the potentially beneficial effects on abdominal muscle tone resulting in decreased IAP and improved APP against the potentially detrimental effect on lung mechanics resulting in atelectasis and super infection*

- The presence of IAH will lead to pulmonary hypertension via ↑ IAP with direct compression on lung parenchyma and vessels and via the diminished left and right ventricular compliance

MV can precipitate IAH and ACS by reducing abdominal wall compliance: How? *

- USE of PEEP or presence of Auto PEEP
- Fighting with ventilator and use of accessory muscles

* Reference: De Laet IE et al. Med Intensiva; 31(2) : 88-99
Effect of IAH on cardio vascular system relevant to patient on MV *

• A very important concept is the abdomino thoracic transmission, which means that the intra thoracic pressure increases during IAH due to cephaloid movement of diaphragm. About 20-80% of IAP is transmitted to the thorax.

• As such traditional filling pressures, PAOP are always falsely elevated in presence of IAH and do not reflect true cardiac filling.

For hemodynamic monitoring, CVP & PAOP should be corrected for ITP

- This means that transmural CVP(CVPTM) is equal to CVP minus ITP and PAOPTM = PAOP – ITP
- Since abdomino thoracic transmission amounts to 20-80%, ITP can be assumed to be IAP/2 and transmural filling pressure can be estimated as:
  \[ \text{CVP}^{\text{TM}} = \text{CVP (observed)} - \text{IAP}/2 \]
  \[ \text{PAOP}^{\text{TM}} = \text{PAOP(observed)} - \text{IAP}/2 \]
- The SSC guide lines targeting initial and ongoing resuscitation towards a CVP of 8 to 12 mmHg * and other studies targeting a MAP of 65 mmHg ** should be interpreted and adjusted according to these findings

Impact of IAH on ARDS definition and management *

- ARDS consensus definition should take into account PEEP and IAP values
- The PAOP criterion in the ARDS consensus definition is inappropriate in the case of IAH as a result of inaccuracy of PAOP measurements
- So it must be adjusted upward because most patients with IAH and secondary ARDS will have a PAOP above 18 mm of Hg as a result of transmission of IAP to the intrathoracic compartments and intracardiac filling pressure measurements

What is an optimum ventilatory strategy: Optimal Tidal Volume in ARDS

- A protective ventilation strategy with a low TV (6 ml/kg ideal body weight) and airway plateau pressure below 30 cm H₂O improves survival in ARDS

- On the other hand, low TV may lead to alveolar derecruitment in presence of ARDS associated with IAH.

- The effect of TV in IAH depends on the degree inspiratory trans pulmonary pressure that is usually reduced because of the cephalic shift of diaphragm.

- One study showed that in extra pulmonary ARDS with IAH, high TV (10ml/Kg ideal body weight) decreased atelectasis and inflammatory response of the lung tissue and improved oxygenation by increasing the end expiratory trans pulmonary pressure.

What is optimum ventilatory strategy: Recruitment maneuvers

- In the presence of IAH and increased chest wall elastance, the inspiratory pressure of the respiratory system applied during recruitment manoeuvres should be higher than usually applied.

- This will achieve an inspiratory trans pulmonary pressure that is large enough to reopen collapsed alveoli.
What is an optimum ventilatory strategy: Open Abdomen and Prone positioning

• One should know that higher PEEP in presence of IAH markedly increases pleural pressure, negatively affecting cardiovascular function *

• It is likely that in case in which higher PEEP levels are required, the open abdomen might be considered according to patients clinical condition **

• In patients with severe ARDS and IAH the prone positioning could be used to reduce the cranial diaphragmatic load, even if prone positioning may induce a mild elevation of IAP or ameliorate or exacerbate IAH and may be difficult to apply inpatients with transiently open abdomen treatment ***

Recommendations can be made in terms of ventilation strategy for patients with IAH

- *Best PEEP should be set to counteract IAP whist in the same time avoiding over inflation of already well aerated lung regions*
  
  $\text{Best PEEP} = \text{IAP}$

- During lung protective ventilation, the plateau pressures should be limited to transmural plateau pressures, which should be below 35 cm of $\text{H}_2\text{O}$
  
  $\text{Pplat}^{\text{TM}} = \text{Pplat} - \text{IAP}/2$

- **Monitoring of extra vascular lung water index (EVLWI) seems warranted in risk patents since IAH is associated with increased risk of pulmonary edema**

- The presence of IAH will lead to pulmonary hypertension via increased ITP with direct compression on lung parenchyma and vessels via diminished left & right ventricular compliance. In this case inhaled NO or prostacycline may be justified

* Reference: De Laet IE et al. Med Intensiva; 31(2) : 88-99
Additional recommendation: How do you perform Alveolar Recruitment in your patient with IAH *

• Higher opening pressures are needed as a result of the compression of the pulmonary parenchyma and decrease in trans pulmonary pressures

• An appropriate recruitment manoeuvre would consist of serially increasing recruitment pressures up to a maximum of 40+ IAP/2 cm of H$_2$O for 40 seconds guided by patients response and improvement of oxygenation

Rescue Mechanical Ventilation needs management of IAH
Rescue Mechanical Ventilation needs management of IAH
IAP 12-15 mm of Hg

- Sedation
- Pain Control
- Avoid prone position
- Reposition bed: Reverse trendelenburg without flexion of hip
  - Remove all constricting bandages

**Carefully assess fluid administration**
- Do not over resuscitate – use goal directed volumes and reassess
  - Avoid unneeded fluid bolus & concentrate all drips
  - Aim for neutral to negative fluid balance by day 3

- Naso gastric and rectal tubes
  - Bowel pro kinetic agents
Rescue Mechanical Ventilation needs management of IAH

Occult Organ Ischemia
IAP 16 – 20 mmHg

- Increased ICP, Decreased CPP
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- Increased CVP, Increased Wedge pressure (falsely elevated)
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- Decreased perfusion, oliguria, difficulty mobilizing fluids
- Increased acidosis
Rescue Mechanical Ventilation needs management of IAH
IAP 16—20 mm of Hg

Prior slide plus

- Enteral nutrition at trophic levels only
  - Colloids plus diuretics
  - Hemo filtration/Dialysis to remove excess fluids
- USG or CT abdomen to identify free fluid. Space occupying lesions amenable to drainage
  - Paracentasis catheter to drain any free fluid
- CT or Ultra sound guided drainage of abscess/hematomas
Rescue Mechanical Ventilation needs management of IAH
Rescue Mechanical Ventilation needs management of IAH
IAH \(> 20\) mm of Hg

All prior two slides plus

- Neuro muscular blockade and infusion
- Colonoscopy to decompress distended colon
- Stop enteral nutrition
- Surgical evacuation of any tumors or masses
- Surgical consultation to plan decompressive laparotomy if above intervention fails, IAP \(> 25\) mm of Hg or organ failure ensues
**Case Report** *

- A 62 year old woman presents to a hospital with obvious septic shock and hypoxemia (SaO$_2$ 82% on FiO$_2$ 0.21)

- She was orally intubated and was placed on ACV. She was febrile to 101.7°F and had tachycardia (Ht rate 114/min). She was placed on crystalloid resuscitation.

- Abdomen was distended and tender. A CT scan of abdomen showed perforated diverticulitis with free fluid and air.

- She remained in Shock and was placed on Norepinephrine and Vasopressin whilst she was prepared for surgery.

• Despite achieving a MAP greater than 65 mm Hg, she remained hypoxemic (SaO2 90 % on FiO₂ 1.0)

• Her ventilation settings were as follows – AC 14/ Vₜ 650/ 100 %=+5, Q = 65 L/min, P_{aw peak} 34, P_{aw mean} 9; decelerating wave form

  • Her initial ABG : 7.28/32/ 62, Lactate = 5.2 mmol/L

• Her Chest X-Ray indicated low lung volumes and a right lower lobe opacity consistent with early aspiration pneumonia

• Her PaO₂/FiO₂ was 62/1.0 and there was a large A-a gradient (611; age expected normal A-a gradient = 18)
Case Report (continued)

- At this point, she underwent sigmoid colectomy and peritoneal debridement. A temporary abdominal closure was placed.
- Now she appeared hypoxic in surgical ICU with the previous ventilator settings.
- Now her chest X-Ray demonstrated additional loss of lung volumes and patchy infiltrates.
- The ventilator indicated high Paw\text{peak} (38 cm H\textsubscript{2}O pressure). The high airway pressure indicated burgeoning ARDS with loss of compliance.
- Thus a ventilator strategy that established low tidal volume was thought of.
- However, with her decreasing lung volumes and therefore worsened derecruitment, further decreasing her lung volumes would increase hypoxemic pulmonary vasoconstriction, decreasing RVEF, impede venous return and diminish cardiac performance. In addition, patient was on vasopressor for MAP management.
Case Report (continued)

- In this setting, a pressure limited ventilation strategy was thought to be ideal
  - Her ventilation set up was changed to PCV. HFOV was not considered as patient was thought to need to go to OR
  - Her set up was switched to AC 10/PCV 25, T,25S/100 %/+10;
  - $\text{Paw}_{\text{peak}}$ 35, $\text{Paw}_{\text{mean}}$ 14, decelerating; $V_T$ resultant 720 cc, $V_E$ 7.2L/min with a 30 min post change ABG of 7.34/46/186
  - Permissive Hypercapnia was established and pH was buffered to offset acidosis (both metabolic and respiratory)
Case Report (continued)

- Her CVP was noted to be 3 and additional plasma volume expansion (PVE) was initiated as she remained on high dose of vasopressors (Norepinephrine and vasopressin) and empirical steroid.

- Despite PVE her urine output began to fall and once again her airway pressures began to rise and her resultant TV began to fall by about 100 cc from their baseline.

- Her SaO₂ declined to 93% on an FiO₂ of 0.6.

- At this point, she was net positive 18L of isotonic fluid, 4 units of packed RBC, and 8 units of FFP. Her CVP now was 10 mm Hg.
A repeat X-Ray chest did not demonstrate new infiltrates, pneumothorax, ETT mal position or full pulmonary hila but did identify decreased lung volumes consistent with decreased compliance and decreased $V_T$.

So attention was diverted to abdomen as the observation above were consistent with ACS. Her measured IAP (intra vesical pressure) was 24 mmHg whereas concomitant MAP was 52 mmHg. Her APP was calculated to be 28 mmHg (Normal = 50 mm Hg).

ACS can recur in patients with an already opened abdomen with temporary abdominal closure.

Anticipating visceral edema and presence of ascites, relaparotomy was done. Post relaparotomy IAP was 12 mmHg and her MAP was 70 mm Hg. Similarly her ventilator returned to their previous baseline and her hypoxemia resolved.
Case Report (continued)

- 12 hours later with a well preserved urine output and an IAP of 16, her \( V_{\text{Tresulant}} \) again fell and was accompanied by recurrent hypoxemia necessitating an increase to \( \text{FiO}_2 \) of 100 % and there was also a rise of \( \text{PCO}_2 \).

- She was still on pressor. MAP was 65 mmHg. X-Ray chest showed worsening infiltrates and there was worsening of lung volumes. This condition was consistent with ARDS secondary to ischaemia and reperfusion at the site remote from the lungs and it was also consistent with burgeoning pneumonia.

- In this setting APRV was chosen as the ventilator strategy as APRV was transportable and could be used in the OR. It was also known to be associated with improvements in cardiac performance, renal and intestinal blood flow.
Case Report (continued)

- Initial APRV settings were $P_{\text{high}}$ 30 cm H$_2$O, $P_{\text{low}}$ 2 cm H$_2$O, $T_{\text{high}}$ 6.0 seconds, $T_{\text{low}}$ 0.8 seconds and $FiO_2$ 100 %. These settings generated a $V_{\text{trelease}}$ of 650cc (initially) and rapidly resolved her hypoxaemia allowing one to decrease her $FiO_2$ to 0.5
  - Her one hour post change ABG was 7.36/46/212 leading to further decrease of $FiO_2$ to 0.4 .
    - By 4 hours $V_{\text{trelease}}$ was 820 cc and her ABG was 7.48/34/186
  - Her chest X-Ray demonstrated improved lung volumes and a relatively narrow mediastinum, and her CVP fell from 14 cm to 8 cm
    - This patient gradually recovered
Conclusion: Take home message

- ACS poses a complex scenario affecting respiratory, cardio vascular and other systems
  - Increased IAP adversely affects respiratory and cardio vascular system, so ventilator parameters often needs adjustment for better interpretation
  - Mechanical ventilation using PEEP and Inverse ratio ventilation often cause IAP to rise
  - Successful rescue ventilation depends on reducing IAP and paying attention to co-morbidities and adverse effects on cardio vascular and other systems
    - ACS involving severely increased IAP requires abdominal decompression/decompressive laparotomy for rescue ventilatory strategy to work
- The SSC guidelines targeting CVP of 8-12 mm Hg and studies targeting MAP of 65 mm Hg should be interpreted using CVP and PaOP adjusted according to IAP
Thank you

Questions?
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