Positive pressure ventilation in cardiogenic shock: friend or foe?

ACCA Masterclass 2017

Josep Masip MD, PhD, FESC

Disclosures: Novartis advisor, ThermoFisher consultant, Philips and Orion speaker fees, Menarini travel-congress support
Respiratory disorders in cardiogenic shock

- Increase in dead-space (fall in pulmonary perfusion)
- Shunt effect (pulmonary edema - hypoxemia)
- Ventilation-Perfusion inequality (respiratory failure)
- Tissue hypoperfusion (Altered mental status)
  (Lactacidemia - metabolic acidosis – ↑ A-V difference- ↓ SVO2)
- Respiratory muscle dysfunction (Hypoventilation – Hypercapnia)
- Pulmonary inflammation (Cytokines release – SIRS)
- Tachypnea – Increasing work of breathing
MAIN GOALS OF MECHANICAL VENTILATION IN SHOCK

• Establish an adequate airway (CNS)
• Reduce VO2 (work of breathing)
• Improve oxygenation
• Reverse respiratory acidosis (hypercapnia)
• Decrease sympathetic tone
• Improve tissue perfusion and metabolic acidosis
Effects of MV in the thorax

Spontaneous breathing

Mechanical Ventilation

Positive pressure

Atmospheric pressure

Negative pressure

(+)

(–)
BENEFICIAL EFFECTS OF POSITIVE INTRATHORACIC PRESSURE

**RESPIRATORY**

- Recruitment of collapsed alveolar units
- Increase of FRC
- Maintenance of continuously opened alveoli
- Gas exchange during the whole respiratory cycle
- Intra-alveolar pressure against edema

**HEMODYNAMIC**

- Decrease in pulmonary shunt

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www.escardio.org/ACCA
OTHER HEMODYNAMIC CHANGES WITH POSITIVE INTRATHORACIC PRESSURE

- Decrease Preload RV - LV
- Systemic hypotension
- Reduction CO
- Fluid retention
- Increase RV Afterload
- Ventricular interdependence
- Venous return decreased
- Pulmonary vascular resistance increased
- Juxtacardiac pressure increased
- Left ventricle
- Right ventricle
- Alveolus

In AHF it may increase cardiac output

Martin J Tobin. NEJM 2001
Let's have a look at the real world
Table 1. Pathophysiology of Cardiogenic Shock

- **Acute myocardial infarction**
  - Loss of critical left ventricular myocardium
  - Right ventricular pump failure

- **Mechanical complications**
  - Acute mitral regurgitation due to papillary-muscle dysfunction or rupture
  - Ventricular septal rupture
  - Free-wall rupture
  - Left ventricular aneurysm

- **Miscellaneous conditions**
  - End-stage cardiomyopathy
  - Myocardial contusion
  - Myocarditis
  - Left ventricular outflow tract obstruction
    - Aortic stenosis
    - Hypertrophic obstructive cardiomyopathy
  - Left ventricular inflow tract obstruction
    - Mitral stenosis
    - Left atrial myxoma
  - Sequela of cardiopulmonary bypass

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Califf R. NEJM 1994

CARDSHOCK STUDY

N = 220
Causes of Cardiogenic shock

- Acute Coronary Syndrome: 80%
- Other causes: 20%

N=220

Harjola V-P. Eur J Heart Fail 2015
220 patients with CS

ACS 81%

STEMI 68%
NSTEMI 13%

Mechanical complications 9%

Severe low-output failure 10%

Ischemic CMP
Dilated CMP

Other 9%

Valvular cause 5%
Takotsubo 2%
Myocarditis 2%

Harjola V-P. Eur J Heart Fail 2015
## Causes of Mechanical Ventilation in ACS

### AHF

<table>
<thead>
<tr>
<th></th>
<th>n=27 (26%)</th>
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<td>Age</td>
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<td>69</td>
<td>58</td>
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<td>Diabetes</td>
<td>59</td>
<td>43</td>
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<td>HTA</td>
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<td>86</td>
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<tr>
<td>Smoker</td>
<td>26</td>
<td>21</td>
<td>83</td>
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<tr>
<td>In hospital ETI (%)</td>
<td>63</td>
<td>54</td>
<td>16</td>
</tr>
<tr>
<td>NIV</td>
<td>9</td>
<td>-</td>
<td>3</td>
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<tr>
<td>Swan Ganz (%)</td>
<td>37</td>
<td>36</td>
<td>14</td>
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<tr>
<td>IABP (%)</td>
<td>56</td>
<td>50</td>
<td>15</td>
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<tr>
<td>Renal RT (%)</td>
<td>15</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>Major bleeding (%)</td>
<td>11</td>
<td>29</td>
<td>3</td>
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<tr>
<td>Transfusions (%)</td>
<td>26</td>
<td>36</td>
<td>11</td>
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<tr>
<td>In H mortality (%)</td>
<td>22</td>
<td>43</td>
<td>33</td>
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<tr>
<td>Mortality (%)</td>
<td>41</td>
<td>43</td>
<td>33</td>
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<tr>
<td>Non-card. Mortality</td>
<td>36</td>
<td>17</td>
<td>52</td>
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### Shock

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</tr>
<tr>
<td>Non-card. Mortality</td>
<td>36</td>
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### Cardiac Arrest

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<td>Diabetes</td>
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<td></td>
<td></td>
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<tr>
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<td></td>
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<tr>
<td>NIV</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>In H mortality (%)</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality (%)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Non-card. Mortality</td>
<td>36</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Lazzeri Ch. Cardiol J 2013

Ariza A. Eur Heart J Acute Card Care 2013
USA National Inpatient Sample (NIS) from 2002 to 2013: 1,867,114 STEMI, 72,220 IMV (3.9%) and 7,030 NIV (0.4%)
SHOCK Trial

Mechanical ventilation (88 %)

Revascularization (n=152) 83%

Medical therapy (n=150)

Mechanical ventilation (78 %)

Hochman J et al. NEJM 1999
Oxygen Therapy in Card-Shock Study

Hongisto M. International J Cardiol 2017
<table>
<thead>
<tr>
<th></th>
<th>MV (n = 137)</th>
<th>NIV (n = 26)</th>
<th>Oxygen (n = 56)</th>
<th>p</th>
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</thead>
<tbody>
<tr>
<td>Hemoglobin (g/L)</td>
<td>130</td>
<td>125</td>
<td>124</td>
<td>0.3</td>
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<tr>
<td>Arterial lactate (mmol/L)</td>
<td>3.7</td>
<td>1.7</td>
<td>2.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Hs-TroponinT (ng/L)</td>
<td>1597</td>
<td>3631</td>
<td>2427</td>
<td>0.06</td>
</tr>
<tr>
<td>NT-proBNP (pg/mL)</td>
<td>2367</td>
<td>7375</td>
<td>1860</td>
<td>0.04</td>
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<tr>
<td>Creatinine (mmol/L)</td>
<td>110</td>
<td>100</td>
<td>107</td>
<td>0.1</td>
</tr>
<tr>
<td>eGFR (mL/min/1.73 m2)</td>
<td>64</td>
<td>67</td>
<td>59</td>
<td>0.6</td>
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<tr>
<td>CRP (g/L)</td>
<td>15</td>
<td>37</td>
<td>15</td>
<td>0.2</td>
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Hongisto M. International J Cardiol 2017
### Baseline arterial blood gases

<table>
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<tr>
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<th>MV (n = 137)</th>
<th>NIV (n = 26)</th>
<th>Oxygen (n = 56)</th>
<th>p</th>
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<tbody>
<tr>
<td>pH</td>
<td>7.27</td>
<td>7.39</td>
<td>7.38</td>
<td>&lt;0.001</td>
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<tr>
<td>PaO2 (mm Hg)</td>
<td>96.7</td>
<td>84</td>
<td>105.1</td>
<td>0.2</td>
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<tr>
<td>PaCO2 (mm Hg)</td>
<td>41.2</td>
<td>33.8</td>
<td>36.8</td>
<td>0.01</td>
</tr>
<tr>
<td>HCO3 mmol/L</td>
<td>19.6</td>
<td>22</td>
<td>21.9</td>
<td>0.001</td>
</tr>
<tr>
<td>FiO2 (%)</td>
<td>76</td>
<td>60</td>
<td>32</td>
<td>0.001</td>
</tr>
<tr>
<td>PaO2/FiO2 (mm Hg)</td>
<td>141</td>
<td>167</td>
<td>311</td>
<td>0.3</td>
</tr>
<tr>
<td>200–300 n (%)</td>
<td>35</td>
<td>7</td>
<td>7</td>
<td>0.9</td>
</tr>
<tr>
<td>100–200 n (%)</td>
<td>54</td>
<td>14</td>
<td>7</td>
<td>0.2</td>
</tr>
<tr>
<td>&lt;100 n (%)</td>
<td>40</td>
<td>4</td>
<td>0</td>
<td>0.1</td>
</tr>
</tbody>
</table>

*Hongisto M. International J Cardiol 2017*
# Characteristics of the Patients with CS According to the Type of Oxygen Therapy

## Devices and Outcomes

<table>
<thead>
<tr>
<th></th>
<th>MV (n = 137)</th>
<th>NIV (n = 26)</th>
<th>Oxygen (n = 56)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary angiogram</td>
<td>114 (83)</td>
<td>23 (89)</td>
<td>45 (80)</td>
<td>0.8</td>
</tr>
<tr>
<td>PCI</td>
<td>90 (66)</td>
<td>19 (73)</td>
<td>40 (71)</td>
<td>0.5</td>
</tr>
<tr>
<td>CABG</td>
<td>5 (4)</td>
<td>3 (12)</td>
<td>1 (2)</td>
<td>0.1</td>
</tr>
<tr>
<td>IABP</td>
<td>85 (62)</td>
<td>16 (62)</td>
<td>21 (38)</td>
<td>1.0</td>
</tr>
<tr>
<td>In-hospital mortality</td>
<td>62 (45)</td>
<td>5 (19)</td>
<td>13 (23)</td>
<td>0.01</td>
</tr>
<tr>
<td>90-day mortality</td>
<td>67 (49)</td>
<td>7 (27)</td>
<td>15 (27)</td>
<td>0.03</td>
</tr>
<tr>
<td>ICU/CCU (days)</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>0.2</td>
</tr>
<tr>
<td>In-hospital (days)</td>
<td>17</td>
<td>12</td>
<td>8</td>
<td>0.2</td>
</tr>
</tbody>
</table>

*Hongisto M. International J Cardiol 2017*
Clinical Findings

<table>
<thead>
<tr>
<th></th>
<th>MV (n = 137)</th>
<th>NIV (n = 26)</th>
<th>Oxygen (n = 56)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (mmHg)</td>
<td>78</td>
<td>83</td>
<td>75</td>
<td>0.03</td>
</tr>
<tr>
<td>Heart rate (b/m)</td>
<td>91</td>
<td>87</td>
<td>89</td>
<td>0.2</td>
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<tr>
<td>LVEF (%)</td>
<td>32</td>
<td>33</td>
<td>36</td>
<td>0.7</td>
</tr>
<tr>
<td>Confusion n (%)</td>
<td>113</td>
<td>8</td>
<td>26</td>
<td>0.001</td>
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</tbody>
</table>

Hongisto M. International J Cardiol 2017
Disadvantages of mechanical ventilation
Disadvantages of mechanical ventilation

- Artificial airway (intubation-tracheostomy)
- Need for Sedation
- Initial hypotension
- Atrophy (ciliar)
- Ventilator lung injury
- Diaphragmatic dysfunction
  - Ventilator associated pneumonia
- Increased RV afterload → Acute Cor Pulmonale
Inconvenients of Tracheal Intubation

At the time of Intubation
- Gastric aspiration
- Barotrauma
- Hypotension and arrhythmias
- Sedation
- Local trauma (dental, pharynge, larynge or trachea)

Related to Extubation
- Dysphagia, odinophagia or dysphonia
- Hemoptysis
- Obstruction (chordae dysfunction/edema)
- Tracheal stenosis

Tracheostomy
- Hemorrhage
- Infection or obstruction
- False lumen
- Mediastinitis
- Lesions in trachea, esophagus and blood vessels
BP vs TIME

INTUBATION
ENDOTRACHEAL INTUBATION IN ACS

2001-2002: 458 patients
Germany (BEAT registry)

- In-hospital: 60%
- Pre-hospital: 40%
- Mortality: 48%

Kouraki K. Clin Res Cardiol 2011

2009-2012: 106 patients
Barcelona
Primary PCI 74%

- In-hospital: 34%
- Pre-hospital: 66%
- Mortality: 29%

Ariza et al. EHJ Acute Cardiovasc Care 2013
INTUBATION CARE BUNDLE MANAGEMENT
CDC surveillance paradigm (2013)

Incidence rates range:
10–15 events per 1,000 ventilator-days or
4 – 7 events per 100 episodes of MV

VAEs are approximately twice as likely to die, associated with more time on MV, longer ICU stays, and higher rates of antimicrobial use

*Klompas. Am J Resp Crit Care 2015*
Ventilator Associated Events (VAEs)

NEW PARADIGM

at least 2 days of stable or decreasing ventilator settings followed by at least 2 days of increased ventilator settings

PEEP : 3 cm H2O

(FIO2) of at least 20 points

<table>
<thead>
<tr>
<th>Date</th>
<th>PEEP (min)</th>
<th>FiO2 (min)</th>
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<td>Jan 1</td>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>Jan 2</td>
<td>5</td>
<td>50</td>
</tr>
<tr>
<td>Jan 3</td>
<td>5</td>
<td>40</td>
</tr>
<tr>
<td>Jan 4</td>
<td>5</td>
<td>40</td>
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<tr>
<td>Jan 5</td>
<td>8</td>
<td>60</td>
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<td>Jan 6</td>
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<td>Jan 7</td>
<td>8</td>
<td>40</td>
</tr>
<tr>
<td>Jan 8</td>
<td>5</td>
<td>40</td>
</tr>
<tr>
<td>Jan 9</td>
<td>5</td>
<td>40</td>
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Clinical Events Associated with Ventilator-associated Events

<table>
<thead>
<tr>
<th>Clinical Event</th>
<th>Klompas et al. (15)* (n = 44)</th>
<th>Hayashi et al. (17) (n = 153)</th>
<th>Klein Klouwenberg et al. (12)* (n = 81)</th>
<th>Boyer et al. (20) (n = 67)</th>
<th>All Studies Combined* (n = 345)</th>
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<tbody>
<tr>
<td>Pneumonia and/or aspiration</td>
<td>10 (23%)</td>
<td>66 (43%)</td>
<td>28 (35%)</td>
<td>21 (31%)</td>
<td>125 (36%)</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>5 (11%)</td>
<td>23 (15%)</td>
<td>12 (15%)</td>
<td>8 (12%)</td>
<td>48 (14%)</td>
</tr>
<tr>
<td>Acute respiratory distress syndrome</td>
<td>7 (16%)</td>
<td>10 (6.5%)</td>
<td>—</td>
<td>14 (21%)</td>
<td>48 (9.0%)</td>
</tr>
<tr>
<td>Mucous plugging</td>
<td>1 (2%)</td>
<td>—</td>
<td>9 (11%)</td>
<td>—</td>
<td>1 (0.3%)</td>
</tr>
<tr>
<td>Abdominal distension/compartment syndrome</td>
<td>1 (2%)</td>
<td>2 (1.3%)</td>
<td>—</td>
<td>—</td>
<td>12 (3.5%)</td>
</tr>
<tr>
<td>Pulmonary embolus</td>
<td>1 (2%)</td>
<td>3 (2.0%)</td>
<td>—</td>
<td>—</td>
<td>1 (1.2%)</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>—</td>
<td>—</td>
<td>2 (2.5%)</td>
<td>2 (3.0%)</td>
<td>6 (1.8%)</td>
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<tr>
<td>Radiation pneumonitis</td>
<td>1 (2%)</td>
<td>—</td>
<td>—</td>
<td>1 (0.3%)</td>
<td>1 (0.3%)</td>
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<tr>
<td>Sepsis syndrome/extrapulmonary infection</td>
<td>1 (2%)</td>
<td>—</td>
<td>9 (11%)</td>
<td>3 (4.5%)</td>
<td>13 (3.8%)</td>
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<tr>
<td>Poor pulmonary toilet</td>
<td>1 (2%)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1 (0.3%)</td>
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<tr>
<td>Acute neurological event</td>
<td>—</td>
<td>—</td>
<td>10 (12%)</td>
<td>—</td>
<td>10 (2.9%)</td>
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<tr>
<td>Transfusion-associated lung injury</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2 (3.0%)</td>
<td>2 (0.6%)</td>
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<tr>
<td>Other</td>
<td>—</td>
<td>—</td>
<td>9 (13%)</td>
<td>—</td>
<td>9 (2.6%)</td>
</tr>
<tr>
<td>No apparent pulmonary complication</td>
<td>18 (41%)</td>
<td>17 (11%)</td>
<td>10 (12%)</td>
<td>—</td>
<td>45 (13%)</td>
</tr>
</tbody>
</table>

*Some ventilator-associated events were attributed to multiple etiologies: hence the percentages exceed 100%.
Three major approaches to prevent VAEs:

(1) Avoid intubation: … Use of NIV
(2) Minimize duration of MV
(3) Target the specific conditions that most frequently trigger VAEs
1. Minimize Sedation

Sedation protocol (RASS Scales, Frequent controls)

Decrease the use of benzodiazepines vs
No sedation, propofol, remifentanil
and dexmedetomidine

Agitated delirium
Self-extubations
Staffing requirements
Emergency reintubations

Pneumonia risk
Time to extubation
2. Daily Spontaneous Awakening Trials and Breathing Trials

Weaning protocol

30 min to 2 h of SBT or Pressure Support Ventilation

Reconnect Ventilation for 1 h before extubation
3. Programs of Early Exercise and Mobility

- Physiotherapists
- Mobilization protocol
- Nurse training
- Family collaboration

ABCDE package

Awakening and Breathing Coordination
Delirium monitoring and management
Early exercise and Mobility
Family collaboration

**Family Presence Behaviors**

**Touch**
- Hand Holding
- Stroking arms, shoulders, head-face
- Massaging feet, head, shoulders
- Affectionate Touch hugging, kissing

**Talk**
- Encouraging
- Coaching
- Normalizing
- Interpreting
- Questioning asking about symptoms or progress

**Surveillance**
- Interpretive
- Protective

**Happ MB. Heart Lung. 2007**
4. Appropriate ventilation strategy

Lung protective strategy

Appropriate ventilation strategy

- Low tidal volumes (6-7 ml/Kg)
- Higher frequencies
- Adjust PEEP
- Low plateau pressure (<27cmH2O) and driving pressure (<17cmH2O)
- Permissive, but controlled hypercapnia
- Meassures to prevent VAP
- Avoid $F_1O_2 > 0.6$  Prone position
5. Conservative Fluid Management

20–40% of VAEs are attributable to fluid overload including congestive heart failure, pulmonary edema and new pleural effusions

Physical examination, CVP, PCWP, extravascular lung volume, mean arterial pressure, urinary output, cardiac index, IVC, E/E’
Central Venous Pressure and Fluid Responsiveness

A Systematic Review

Marik PE et al. Chest 2008
Pulse Pressure Variation with respiration

Michard F. Am J Respir Crit Care Med 2000
Pulse Pressure Variation (PPV)

Conditions:

- Mechanical ventilation
- No arrhythmia
- No spontaneous breathing
- Constant Vt ≥7 ml/kg
- RR < 30
- No RVF

Mahjoub Y et al Br J Anesthesia 2013
Passive Leg Raising

• PLR and LVOT VTI (TTE) ↑ 12%

• PLR and SV (TTE) ↑ 12.5% predicts
  SV ↑ 15% after volume load
  (Sens. 77%; Spec. 100%)

Monnet X. Intensive Care Med 2008
Lamia: Intensive Care Med 2007
Other methods:

End-expiratory occlusion test

15s occlusion at end-expiration PP or PCA-CO ↑5%
Sens 87%, Spec 100% for response to 500 ml

Minifluid challenge

100 ml of colloid/1 min: LVOT VTI ↑ 10%
Sens 95%, Spec 78% for response to volume

Monnet X, Crit Care Med 2012  Muller L, Anesthesiology 2011
6. Conservative Blood Transfusion Thresholds
7. Ventilator Associated Pneumonia Prevention

Oral care with chlorhexidine
Subglottic secretion drainage

Elevating the Head of the Bed
Hand washing
Disposable gloves
Sterile aspiration

Unlikely
Likely

Campaigns: Pneumonia Zero, Bacteremia Zero, Resistance Zero

VAP: from 15/1000 days MV to 5/1000 days MV
Characteristics of Patients With Complicated ACS Requiring Prolonged Mechanical Ventilation

267 patients

Zahger D. Am J Cardiol 2005
IABP Shock Trial

45 patients AMI-PCI shock randomized to IABP

**COMPLEMENTARY EFFECTS OF MV AND IABP**

**Prondzinsky R et al, Crit Care Med 2010**
COMPLEMENTARY EFFECTS OF MV AND IABP

CONCLUSIONS

MV is a good friend that has saved millions of lives

In patients with CS it should be used in cases with severe respiratory failure or altered mental status that can not be managed by other ways

The appropriate use of the technique targeted to shorten the duration of MV and avoiding ventilator events is essential to sustain this friendship
Thank you for your attention