Mechanical ventilation induced or exacerbated right ventricular failure

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Faculty Disclosures

Dr. Hall

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A patient with severe ARDS is sedated, paralyzed with cis-atracurium, and mechanically ventilated on volume assist-control mode. \( \text{PaO}_2 \) is 85 despite an \( \text{FiO}_2 \) of 1.0 (P/F = 85), so PEEP is raised from 5 to 18 cm H\(^2\)O. Coincident with this increase, stroke volume falls from 52 to 43 mL and blood pressure drops. Which of the following mechanisms is most likely to underlie this cardiovascular change?

A. Decreased RV preload  
B. Increased LV afterload  
C. Increased RV afterload  
D. Rightward shift of the interventricular septum
Hemodynamic Effects of PEEP

Normal Heart
Reduced RV preload
Reduced LV preload
Fluid responsive ↓ CO

Acute RV Dysfunction
Increased RV afterload
Septal shift/LV dysfunction
Fluid unresponsive ↓ ↓ CO

LV Dysfunction
Reduced LV afterload
↑ CO
Positive pressure ventilation results in increased intrathoracic pressure and PVR mediated by TV, PEEP, lung and chest wall compliance

Table 1. Characteristics of Right and Left Ventricle

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Right ventricle</th>
<th>Left ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structure</td>
<td>Inflow region, trabeculated myocardium, infundibulum</td>
<td>No infundibulum mitro-aortic continuity</td>
</tr>
<tr>
<td>Shape</td>
<td>From the side: triangular cross-section: crescentic</td>
<td>Elliptic</td>
</tr>
<tr>
<td>Volume (end-diastolic)</td>
<td>49–101 mL/m²</td>
<td>44–89 mL/m²</td>
</tr>
<tr>
<td>Mass (g/m²)</td>
<td>&lt;35 g/m² ≈ 1/6 LV mass</td>
<td>&lt;130 g/m² (men) &lt;100 g/m² (women)</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>40%–68% &gt; 45%&lt;sup&gt;a&lt;/sup&gt;</td>
<td>57%–74% &gt; 50%&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ventricular elastance (mm Hg/mL)</td>
<td>1.30 ± 0.84</td>
<td>5.48 ± 1.23</td>
</tr>
<tr>
<td>Ventricular compliance</td>
<td>Higher compliance than LV</td>
<td>5.0 ± 0.52 × 10⁻²</td>
</tr>
<tr>
<td>Adaptation to disease</td>
<td>Better adaptation to volume overload states</td>
<td>Better adaptation to pressure overload states</td>
</tr>
</tbody>
</table>

Haddad, Anesth Anal 2009
Pressure overload (esp acutely) of the RV results in

- Inc RV volume
- Change in RV shape
- Impingement of the RV on the LV (eventual septal shift to left)
- RV ischemia even in the absence of CAD
- RV systolic dysfunction
- A downward spiral of the above with shock
Manifestations of worsened RVF precipitated by MV

- Elevated RV press
- Right S3
- Tricuspid regurgitation
- Pulsatile liver
- Peripheral edema
- Elevated lfts

- Hypotension
- Inc Cr and dec u/o
- Dec neurologic fn
- Dec SVO2
- Inc lactate
- Inc BNP
Suggestions of RV dysfunction by PAC
Better than these suggestions is echocardiography to dx and titrate ventilator, fluid, vasoactive drug adjustments

RV dilation on 4 chamber TEE view

RV dilation on 4 chamber TTE view
Findings present in diastole and systole and accentuated at end insp (these are end exp views)
### Objective measures of RV compromise

#### TABLE 2  Echocardiographic Parameters of RV Function

<table>
<thead>
<tr>
<th>Parameter</th>
<th>View</th>
<th>Abnormal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV:LV area ratio</td>
<td>ME four chamber</td>
<td>&gt;0.6</td>
</tr>
<tr>
<td>LV eccentricity index</td>
<td>TG midpapillary short axis</td>
<td>&gt;1</td>
</tr>
<tr>
<td>RVFAC</td>
<td>ME four chamber</td>
<td>&lt;35%</td>
</tr>
<tr>
<td>TAPSE</td>
<td>Deep TG RV</td>
<td>&lt;1.6 cm</td>
</tr>
<tr>
<td>Peak velocity of systolic excursion at the annulus</td>
<td>Deep TG RV</td>
<td>&lt;10 cm/s</td>
</tr>
<tr>
<td>Pulmonary artery flow acceleration time</td>
<td>Ascending aortic short-axis</td>
<td>&lt;100 ms</td>
</tr>
</tbody>
</table>

ME = midesophageal; RVFAC = right ventricular fractional area change; TAPSE = tricuspid annular plane systolic excursion; TEE = transesophageal echocardiography; TG = transgastric; TTE = transthoracic echocardiography. See Table 1 legend for expansion of other abbreviations.
Short Axis
Apical 4-Chamber
Bottom line—once a day echo will not suffice—seek continuous or at least frequent monitoring while titrating therapy

- TTE provides adequate imaging in 80-90% of critically ill pts (Chest 2004 126:1592)
- TEE superior for imaging to provide specific parameters of RVF (87% vs 61%, Int Care Med 2013 39:1734)
- Miniature TEE probe has been used for continuous adjustment of vent parameters (Int Care Med 2013 39:629)
- Post cardiac surgery has high level of difficulty of visualization (37% in one series)
RV—MV interactions best described in

- ARDS
- Acute PE
- RV infarction
- Post cardiotomy
- S/P lung resection
- Acute sickle chest
RV Dysfunction in ARDS

• 20 - 50% of pts with severe ARDS will have RV dysfunction

• Defined as:
  – RV enlargement (RVEDA – LVEDA)/LVEDA > 0.6
  – Systolic septal dyskinesia (eccentricity index)

• Made worse by:
  – Increases in mean airway pressure (HFO)
  – Increases in PEEP
  – Fall in pH (acidosis)

# Fluid: RV Dysfunction

<table>
<thead>
<tr>
<th></th>
<th>RVEDP (mmHg)</th>
<th>RVSP (mmHg)</th>
<th>CO (L/min)</th>
<th>BP&lt;sub&gt;sys&lt;/sub&gt; (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embolism</td>
<td>9.4</td>
<td>71.3</td>
<td>1.4</td>
<td>97</td>
</tr>
<tr>
<td>Fluid</td>
<td>11.9</td>
<td>52.1</td>
<td>0.94</td>
<td>65</td>
</tr>
</tbody>
</table>

Ghignone M, et.al: Anesthesiology 1984; 60:132  
Molloy WD, et.al: Am Rev Respir Dis 1984; 130:870  
Krishnan S, Schmidt GA: Chest 2015; 147:835  
ARDS—PEEP effects

- At moderate levels with recruitment, PVR may fall (J Crit Care 1994 9:100)
- In 21 pts taken from PEEP 5 cm H2O to that producing Pplat 30 (avg 13 cm H2O) Qt fell with inc RV end diastolic vol and PVR (Crit Care Med 2010 38:802)
High v Low PEEP

ARDS, Vt 6 mL/kg PBW
Norepi in 16 of 21
PA catheter; TEE
PEEP: 5 to 13
CI fell 13%

Fougères E, et.al: Crit Care Med 2010; 38:802
### Hemodynamic Effect of PEEP

<table>
<thead>
<tr>
<th></th>
<th>PEEP 5</th>
<th>High PEEP</th>
<th>High PEEP + PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pra, mmHg</td>
<td>9</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>RVEDA, cm²</td>
<td>15.9</td>
<td>17.4</td>
<td>19.2</td>
</tr>
<tr>
<td>PVR, dyne•s•sm²/cm⁵</td>
<td>310</td>
<td>385</td>
<td>321</td>
</tr>
</tbody>
</table>

Fougères E, et.al: Crit Care Med 2010; 38:802
# PEEP: Less Recruitment, More Cardiac Impairment

<table>
<thead>
<tr>
<th></th>
<th>Low PEEP</th>
<th>High PEEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Index (L/min/m²²)</td>
<td>2.60</td>
<td>1.89</td>
</tr>
<tr>
<td>RV Stroke Index (cm³/m²²)</td>
<td>22</td>
<td>16</td>
</tr>
<tr>
<td>ES Eccentricity Index</td>
<td>1.10</td>
<td>1.28</td>
</tr>
<tr>
<td>ED RV/LV Area</td>
<td>0.64</td>
<td>1.0</td>
</tr>
</tbody>
</table>

ARDS

- Higher incidence of RVF in era of higher TV (Crit Care Med 1985; 13:952)
- With lPVs, incidence of RVF defined as dilated RV with septal dyskinesia (or PFO right to left flow in other studies) reported at 22-30% (Int Care Med 2013 39:1934; Int Care Med 2013 39:1725)
- Higher with HFOV (Crit Care Med 2012 40:1539)
- Prone positioning reduced PVR and enhance Qt in fluid responsive pts (Chest 2007 132:1440; Am J Resp Crit Care Med 2013; 188: 1428)

**Pulmonary vascular resistance (dynes·s/cm³/m²)**

**Cardiac index (L/min/m²)**
The blood gases matter

- Hypoxia clearly a risk for HVC and worsened RFV
- When titrating PEEP higher and maintaining Pplat, the reduction in TV often causes permissive hypercapnea, and even modest levels (PaCO2 60-70) have been shown to increase PVR and decrease RV function (Int Care Med 2009 35:1850)

RFV, Pplat, 260-09

Fig. 1 Tested ventilatory strategies. HME heat-and-moisture exchanger, HH heated humidifier, RR respiratory rate, Plat plateau pressure, PEEP positive end-expiratory pressure, LP low PEEP, HP/HR high PEEP and high respiratory rate, HP/LR high PEEP and low respiratory rate
Principles of MV for patients with or at risk for RVF

- Limit tidal volume and PEEP
- Avoid hypercapnea and acidosis
- Avoid hypoxia and associated hypoxic vasonconstriction
- Note that the second and third injunctions above likely conflict with the first
Adjunctive treatments

• Avoiding excessive fluids worsening RVF and considering diuresis
  – Dynamic predictors of volume responsiveness (PPV, SPV) should not be used
  – PLR with a measure to SV best way to go

• Inhaled pulmonary vasodilators

• Inotropes

• Vasoconstrictors to maintain right coronary perfusion
iNO Improves Oxygenation

## iNO and ARDS Outcomes

### Mortality

<table>
<thead>
<tr>
<th>Study</th>
<th>Treatment n/N</th>
<th>Control n/N</th>
<th>Mortality RR (95% CI Fixed)</th>
<th>Weight %</th>
<th>RR (95% CI Fixed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dellinger</td>
<td>35/120</td>
<td>17/54</td>
<td>74.6</td>
<td>0.93</td>
<td>[0.57, 1.50]</td>
</tr>
<tr>
<td>Troncy</td>
<td>9/15</td>
<td>8/15</td>
<td>25.4</td>
<td>1.12</td>
<td>[0.62, 2.11]</td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>44/135</td>
<td>25/69</td>
<td>100</td>
<td>0.98</td>
<td>[0.66, 1.44]</td>
</tr>
</tbody>
</table>

Chi-squared 0.24 (df=1) Z=0.12

### LOS

<table>
<thead>
<tr>
<th>LOS</th>
<th>Treatment n/N</th>
<th>Control n/N</th>
<th>LOS RR (95% CI Fixed)</th>
<th>Weight %</th>
<th>RR (95% CI Fixed)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number in intensive care unit, at 30 days</td>
<td>17/93</td>
<td>12/87</td>
<td>100%</td>
<td>1.33 [0.67, 2.61]</td>
</tr>
<tr>
<td></td>
<td>Chi-squared  0.00 (df=0) Z=0.81</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number in hospital, at 30 days</td>
<td>12/93</td>
<td>14/87</td>
<td>100%</td>
<td>0.80 [0.39, 1.64]</td>
</tr>
<tr>
<td></td>
<td>Chi-squared 0.00 (df=0) Z=0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

PGI₂ vs iNO

Zwissler B: Am J Respir Crit Care Med 2006; 154:1671
Vasoactive Drugs

- Few data are available
- Norepinephrine: RV inotropy, enhance coronary perfusion
- Dobutamine: RV inotropy, lowers filling pressures
- Levosimendan: RV inotropy, pulmonary vasodilator

And all of the therapies above guided by bedside echocardiography!
Figure 1. Mean pulmonary artery pressure (mPAP) and oxygen saturation in the pulmonary artery ($SvO_2$) versus time after initiation of venovenous extracorporeal membrane oxygenation in six patients in whom $SvO_2$ was available. Mean ± SE. $SpO_2$ = peripheral oxygen saturation.
Veno-Arterial ECLS

- Supports circulation
- Unloads the RV

Gaffney AM: BMJ 2010; 341:982