Basic principles of gas exchange under Mechanical Ventilation

Lorenzo Del Sorbo, MD

Mechanical Ventilation: From physiology to clinical practice

Toronto, April 14th 2020
Outline

• gas exchange abnormalities
• physiologic principles
• clinical cases
STATE OF THE ART

FIFTY YEARS OF RESEARCH IN ARDS
Gas Exchange in Acute Respiratory Distress Syndrome

Peter Radermacher¹, Salvatore Maurizio Maggiore², and Alain Mercat³

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Hypoxemia and impaired CO2 clearance are characteristics of ARDS.
mechanisms of hypoxemia

• hypoventilation
• ↓ $P_{1}O_2$
• ↓ diffusion
• V/Q mismatch
• shunt
+ ↓ $P_vO_2$

ARDS
V/Q ratio
Shunt

3-5% anatomical shunt

Right-to-left shunt

\( \frac{Q_s}{Q_T} \)

\( P_{V O_2} = 40 \text{ mmHg} \)

\( P_{V CO_2} = 46 \text{ mmHg} \)

\( P_{A O_2} = 40 \text{ mmHg} \)

\( P_{A CO_2} = 46 \text{ mmHg} \)
Higher $V_A/Q$

Higher pleural pressure

Lower $V_A/Q$

Lower pleural pressure

Shunt

Alveolar inhomogeneity
Shunt

\[ C_aO_2 \times Q_T = C_cO_2 \times (Q_T - Q_s) + C_vO_2 \times Q_s \]

\[ \text{Shunt} \ (Q_s/Q_T) = \frac{C_cO_2 - C_aO_2}{C_cO_2 - C_vO_2} \]
<table>
<thead>
<tr>
<th>Treatment</th>
<th>Beneficial Effects</th>
<th>Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>High $F_{O_2}$</td>
<td>Increases in $P_{A_{O_2}}$, $P_{a_{O_2}}$, and $P_{V_{O_2}}$</td>
<td>Resorption atelectasis, Oxygen toxicity</td>
</tr>
<tr>
<td>PEEP</td>
<td>Alveolar recruitment with decrease in $Q_s/Q_T$</td>
<td>Lung overdistension, Decreased $Q_T$</td>
</tr>
<tr>
<td>Spontaneous breathing (mild–moderate ARDS, postacute phase)</td>
<td>Alveolar recruitment, Improved $V_{a}/Q$ matching (redirection of pulmonary blood flow to more aerated regions)</td>
<td>Lung overdistension, VILI</td>
</tr>
<tr>
<td>Recruitment maneuver</td>
<td>Transient recruitment and decreased $Q_s/Q_T$</td>
<td>Transient decrease in $Q_T$, Barotrauma</td>
</tr>
<tr>
<td>Prone position</td>
<td>Homogenization of ventilation distribution (improved aeration in the dorsal regions), Decrease in $Q_s/Q_T$ (unchanged perfusion, predominantly directed to dorsal regions)</td>
<td>—</td>
</tr>
<tr>
<td>Vertical positioning</td>
<td>Alveolar recruitment, Increased lung volume</td>
<td>Unpredictable effect, Decreased $Q_T$</td>
</tr>
<tr>
<td>Inhaled NO</td>
<td>Decrease in $Q_s/Q_T$ (improved perfusion of aerated lung regions with normal $V_{a}/Q$ ratios)</td>
<td>Transient effect, Rebound at withdrawal</td>
</tr>
<tr>
<td>Inhaled PGI₂</td>
<td>Decrease in $Q_s/Q_T$ (improved perfusion of aerated lung regions with normal $V_{a}/Q$ ratios)</td>
<td>Transient effect, Rebound at withdrawal</td>
</tr>
<tr>
<td>Intravenous almitrine</td>
<td>Decrease in $Q_s/Q_T$ (increased pulmonary vascular tone)</td>
<td>Increase in PAP and RV afterload</td>
</tr>
</tbody>
</table>

*Definition of abbreviations: ARDS = acute respiratory distress syndrome; NO = nitric oxide; $P_{A_{O_2}}$ = alveolar oxygen partial pressure; $P_{a_{O_2}}$ = arterial oxygen partial pressure; PAP = pulmonary artery pressure; PEEP = positive end-expiratory pressure; PGI₂ = prostacyclin; $P_{V_{O_2}}$ = mixed venous $O_2$ partial pressure; $Q_s/Q_v$ = intrapulmonary right-to-left shunt; $Q_T$ = cardiac output; RV = right ventricular; VILI = ventilator-induced lung injury.*
Dead space

\[ \text{PaCO}_2 = \frac{V'CO_2}{V'_A} \]

\[ = \frac{V'CO_2}{\text{RR} (V_t-V_d)} \]

\[ = \frac{V'CO_2}{\text{RR} (1-V_d/V_t)} \]

\[ \frac{V_D}{V_T} = \frac{\text{PaCO}_2 - P\text{E}CO_2}{\text{PaCO}_2} \]
Dead space

Nuckton T.J., et al. NEJM 2002
Ventilatory ratio

Table E3 Odds ratio for ICU-mortality using univariate logistic regression model for key respiratory variables in the primary dataset. Odds ratio are per unit change of variable unless stated otherwise. * Odds ratio per 0.05 increase in $V_D/V_T$.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilatory Ratio</td>
<td>2.07</td>
<td>1.53 - 2.85</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PaO$_2$/FiO$_2$ Ratio</td>
<td>0.99</td>
<td>0.98 - 0.99</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Oxygenation Index</td>
<td>1.05</td>
<td>1.03 - 1.07</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PEEP (cmH$_2$O)</td>
<td>1.11</td>
<td>1.06 - 1.17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Driving Pressure</td>
<td>1.07</td>
<td>1.03 - 1.12</td>
<td>0.002</td>
</tr>
<tr>
<td>Pulmonary Dead Space</td>
<td>1.37*</td>
<td>1.27 - 1.50</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>$V_E$ corrected</td>
<td>1.09</td>
<td>1.04 - 1.51</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Pratik et al. AJRCCM 2013 & 2019
Table 3. Therapeutic Measures to Correct Hypercapnia

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Beneficial Effects</th>
<th>Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedation ± paralysis</td>
<td>Reduced $V_{CO2}$</td>
<td>Delayed weaning</td>
</tr>
<tr>
<td>Lengthening inspiratory pause</td>
<td>Improved homogeneity of ventilation distribution</td>
<td>Increase in PEEP$<em>i$ and PEEP$</em>{tot}$ (shortening of expiratory time)</td>
</tr>
<tr>
<td>Increasing respiratory rate</td>
<td>Increase in $V_E$</td>
<td>Increase in PEEP$<em>i$ and PEEP$</em>{tot}$ (shortening of expiratory time)</td>
</tr>
<tr>
<td>Decrease of instrumental dead space</td>
<td>Decrease of $V_D/V_T$</td>
<td>—</td>
</tr>
<tr>
<td>TGI</td>
<td>Decrease of $V_D/V_T$ due to reduced airway dead space</td>
<td>Increase in PEEP$<em>i$ and PEEP$</em>{tot}$ Inaccurate $V_T$ measurement Tracheal lesions</td>
</tr>
<tr>
<td>Prone position</td>
<td>Homogenization of ventilation distribution</td>
<td>Unpredictable effect</td>
</tr>
</tbody>
</table>

Definition of abbreviations: PEEP = positive end-expiratory pressure; PEEP$_i$ = intrinsic PEEP; PEEP$_{tot}$ = total PEEP; TGI = tracheal gas insufflation.
Clinical examples
1° case: ARDS

VCV 430 ml X 25 RR, PEEP 15, Pplat 27, FiO₂ 40%, pH 7.31, paCO₂ 38, paO₂ 53
ARDS: hypoxemia

![Graph showing mortality percentage across different PaO2/FiO2 ratios.](image-url)
ARDS: means to decrease hypoxemia

- **Increase FiO\(_2\):** not effective if hypoxemia is only from shunt

 increased risk of O\(_2\) toxicity

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**Figure 1.** Schematic diagram of the precise control of arterial oxygenation concept. A target arterial partial pressure of oxygen or arterial hemoglobin oxygen saturation is selected for each patient (**thick dashed, arrowed line** in the center of curve) around which tight boundaries are delineated that create the therapeutic target range for oxygenation (**thin dashed lines**). Harm is possible if oxygenation strays outside of this selected range. The optimal range for individuals will be dependent upon their specific clinical situation.

Martin and Grocott. CCM 2013
ARDS: means to decrease hypoxemia - $\uparrow$ FiO$_2$

Increased risk of reabsorption atelectasis

<table>
<thead>
<tr>
<th>PULMONARY GAS EXCHANGE VARIABLES</th>
<th>Baseline</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30 min</td>
<td>100</td>
</tr>
<tr>
<td>Pa$_{O_2}$, mm Hg</td>
<td>94 ± 24</td>
<td>301 ± 117$^\dagger$</td>
</tr>
<tr>
<td>Pa$_{CO_2}$, mm Hg</td>
<td>41 ± 10</td>
<td>44 ± 12$^\dagger$</td>
</tr>
<tr>
<td>pHa</td>
<td>7.42 ± 0.07</td>
<td>7.38 ± 0.07$^\dagger$</td>
</tr>
<tr>
<td>P$ar{V}_{O_2}$, mm Hg</td>
<td>39 ± 5</td>
<td>46 ± 4$^\S$</td>
</tr>
<tr>
<td>Q$_S$/Q$_T$, % Q$_T$</td>
<td>20 ± 10</td>
<td>23 ± 12</td>
</tr>
<tr>
<td>Shunt, % Q$_T$</td>
<td>16 ± 10</td>
<td>22 ± 11$^\dagger$</td>
</tr>
<tr>
<td>Low V$_A$/Q, % Q$_T$</td>
<td>2.60 ± 2.60</td>
<td>3.70 ± 3.70</td>
</tr>
</tbody>
</table>

Santos C, et al. AJRCCM 2000
2° case: ARDS
VCV 300 ml X 27 RR,
PEEP 15
Pplat 30, FiO₂ 100%,
pH 7.31, paCO₂ 38, paO₂ 40

Courtesy of L. Gattinoni and R. Fumagalli
ARDS

VCV 280 ml X 24 RR, PEEP 20, Pplat 30, FiO₂ 70%, pH 7.35, paCO₂ 38, paO₂ 74

intermediate trans-pulmonary pressure

Recruitment

Courtesy of L. Gattinoni and R. Fumagalli
Excessive trans-pulmonary pressure

ARDS

VCV 320 ml X 35 RR, PEEP 20, Pplat 32, FiO₂ 90%, pH 7.21, paCO₂ 54, paO₂ 74

Recruitment and overdistension

Courtesy of L. Gattinoni and R. Fumagalli
Excessive Trans-pulmonary Pressure
VILI

VCV 320 ml X 35 RR, PEEP 20, Pplat 32, FiO$_2$ 100%, pH 7.21, paCO$_2$ 54, paO$_2$ 64

CO$_2$ clearance

 Courtesy of L. Gattinoni and R. Fumagalli
ARDS: recruitment

Gattinoni L et al., NEJM 2006
ARDS: recruitment
ARDS: recruitment

Adjusted probability of death

$\Delta P/F$ (mm Hg) following initial PEEP modification

$\Delta PEEP > 0$

$\Delta PEEP \leq 0$

Goligher E, et al. AJRCCM 2014
3° case: ARDS

VCV 320 ml X 27 RR, PEEP 15
Pplat 30, FiO$_2$ 100%,
pH 7.31, paCO$_2$ 51, paO$_2$ 70

Gattinoni L, et al. AJRCCM 2013
VCV 320 ml X 27 RR, PEEP 15
Pplat 27, FiO₂ 60%,
pH 7.33, paCO₂ 48, paO₂ 70

Sud S, et al. ICM 2010

**Fig. 4** Effect of prone ventilation on PaO₂ (partial pressure of arterial oxygen)/FiO₂ (inspired fraction of oxygen) on postrandomization calendar days 1–3. Ratio of means = mean PaO₂/FiO₂ in the prone group (in the prone position)/mean PaO₂/FiO₂ in the supine group (at the closest available time). Weight is the contribution of each study to the overall ratio of means. CI confidence interval, P percentage of total variation across studies due to between-study heterogeneity rather than chance.
Prone Position–induced Improvement in Gas Exchange Does Not Predict Improved Survival in ARDS

<table>
<thead>
<tr>
<th>Variable</th>
<th>Survived (N = 194)</th>
<th>Died (N = 38)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH, units</td>
<td>7.34 ± 0.10</td>
<td>7.27 ± 0.12</td>
<td>0.004</td>
</tr>
<tr>
<td>Δ from pre-prone positioning</td>
<td>0.01 ± 0.07</td>
<td>0.01 ± 0.05</td>
<td>0.764</td>
</tr>
<tr>
<td>$P_{aCO_2}$, mm Hg</td>
<td>49 ± 14</td>
<td>52 ± 12</td>
<td>0.149</td>
</tr>
<tr>
<td>Δ from pre-prone positioning</td>
<td>-0.5 ± 9.4</td>
<td>-2.7 ± 9.1</td>
<td>0.182</td>
</tr>
<tr>
<td>$P_{aO_2}$, mm Hg</td>
<td>119 ± 65</td>
<td>118 ± 59</td>
<td>0.950</td>
</tr>
<tr>
<td>Δ from pre-prone positioning</td>
<td>38.9 ± 63</td>
<td>38.8 ± 59</td>
<td>0.987</td>
</tr>
<tr>
<td>$FiO_2$, %</td>
<td>74 ± 16</td>
<td>78 ± 17</td>
<td>0.205</td>
</tr>
<tr>
<td>Δ from pre-prone positioning</td>
<td>-3.9 ± 11.4</td>
<td>-6.5 ± 11.6</td>
<td>0.220</td>
</tr>
<tr>
<td>PEEP, cm H$_2$O</td>
<td>12 ± 3</td>
<td>12 ± 2</td>
<td>0.654</td>
</tr>
<tr>
<td>Δ from pre-prone positioning</td>
<td>-1.6 ± 3.0</td>
<td>-1.3 ± 3.2</td>
<td>0.602</td>
</tr>
<tr>
<td>P/F, mm Hg</td>
<td>166 ± 83</td>
<td>152 ± 62</td>
<td>0.321</td>
</tr>
<tr>
<td>Δ from pre-prone positioning</td>
<td>60 ± 79</td>
<td>55 ± 60</td>
<td>0.618</td>
</tr>
<tr>
<td>P/F increase, mm Hg</td>
<td></td>
<td></td>
<td>0.78</td>
</tr>
<tr>
<td>≥ 20, N (%)</td>
<td>123 (85)</td>
<td>25 (17)</td>
<td></td>
</tr>
<tr>
<td>&lt; 20, N (%)</td>
<td>71 (85)</td>
<td>13 (15)</td>
<td></td>
</tr>
<tr>
<td>$P_{aCO_2}$ decrease, mm Hg</td>
<td></td>
<td></td>
<td>0.86</td>
</tr>
<tr>
<td>≥ 1, N (%)</td>
<td>99 (83)</td>
<td>20 (17)</td>
<td></td>
</tr>
<tr>
<td>&lt; 1, N (%)</td>
<td>95 (84)</td>
<td>18 (16)</td>
<td></td>
</tr>
<tr>
<td>Survival by quintile of P/F response after 1 h of prone ventilation, mm Hg, survivors/total patients (%)</td>
<td></td>
<td></td>
<td>0.410</td>
</tr>
<tr>
<td>-86 to -1</td>
<td>39/43 (91)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 to 85</td>
<td>99/121 (82)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>86 to 171</td>
<td>37/47 (78)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>172 to 257</td>
<td>13/15 (87)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>258 to 343</td>
<td>6/6 (100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Survival by quintile of change in $P_{aCO_2}$ after one hour of prone ventilation, mm Hg, survivors/total (%)</td>
<td></td>
<td></td>
<td>0.210</td>
</tr>
<tr>
<td>-30 to -12</td>
<td>13/20 (65)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-11 to +7</td>
<td>160/189 (85)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+8 to +26</td>
<td>16/18 (89)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+27 to +45</td>
<td>4/4 (100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+45 to +64</td>
<td>1/1 (100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Survival by combined P/F and $P_{aCO_2}$ response, survivors/total (%)</td>
<td></td>
<td></td>
<td>0.670</td>
</tr>
<tr>
<td>+P/F response, no $P_{aCO_2}$ response</td>
<td>15/16 (94)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+P/F response, + $P_{aCO_2}$ response</td>
<td>18/23 (78)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No P/F response, no $P_{aCO_2}$ response</td>
<td>80/97 (82)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No P/F response, + $P_{aCO_2}$ response</td>
<td>81/97 (87)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Guerin C, et al. AJRCCM 2014
Prone positioning in ARDS: less VILI

Gattinoni L, et al. ICM 2013


<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Est,cmH2O/L</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td>4.8 (10)</td>
<td>4.6 (3.11)</td>
<td>10 (5)</td>
<td>6 (1)</td>
</tr>
<tr>
<td>Prone</td>
<td>7.4 (19)a</td>
<td>6.3 (1.7)a</td>
<td>18 (7)a</td>
<td>11 (4)a</td>
</tr>
<tr>
<td>EELV (L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td>1.17 (0.41)</td>
<td>ND</td>
<td>1.40 (0.64)</td>
<td>1.03 (0.09)</td>
</tr>
<tr>
<td>Prone</td>
<td>1.29 (0.57)</td>
<td></td>
<td>1.57 (0.72)a</td>
<td>1.48 (0.13)a</td>
</tr>
</tbody>
</table>

Guerin C, et al. ICM 2014
4° case:
ARDS (post-trauma)

HR 135, BP 90/60, Hb 71, Lactate 5.1
SvO₂ 50%
VCV 480 ml X 27 RR, PEEP 16
Pplat 30, FiO₂ 100%,
pH 7.31, paCO₂ 38, paO₂ 40

• Improve hemodynamics (DO₂/V’O₂)

HbO₂ = (50 +100)/2 = 75%
PaO₂ = 40 mmHg

Courtesy of R. Fumagalli
ARDS (post-trauma)

HR 100, BP 110/60, Hb 95, Lactate 2
SvO\textsubscript{2} 80%
VCV 460 ml X 23 RR, PEEP 18
Pplat 28, FiO\textsubscript{2} 80%,
pH 7.37, paCO\textsubscript{2} 46, paO\textsubscript{2} 60

Post fluid resuscitation and PRBC transfusion

Better DO\textsubscript{2}/V’O\textsubscript{2}

HbO\textsubscript{2} = (80 + 100)/2 = 90%
PaO\textsubscript{2} = 60 mmHg

Courtesy of R. Fumagalli
ARDS (post-trauma)

HR 100, BP 110/60, Hb 95, Lactate 2
SvO₂ 80%
VCV 460 ml X 23 RR, PEEP 18
Pplat 28, FiO₂ 80%,
pH 7.37, paCO₂ 46, paO₂ 60

Better DO₂/V’O₂
Post fluid resuscitation and PRBC transfusion

Improving perfusion in ventilated alveoli
Better V/Q
Failed prone position
VCV 320 ml X 27 RR, PEEP 15
Pplat 30, FiO$_2$ 100%,
pH 7.31, paCO$_2$ 38, paO$_2$ 40

Improving perfusion in ventilated alveoli
INHALED NITRIC OXIDE FOR THE ADULT RESPIRATORY DISTRESS SYNDROME

Rolf Rossaint, M.D., Konrad J. Falke, M.D., Frank López, B.S., Klaus Slama, M.D., Ulrich Pison, M.D., and Warren M. Zapol, M.D.

NEJM 1993
6° case: COPD exacerbation

Vt  450 ml
RR  27
iPEEP 18
Ppeak 61
FiO₂ 30%
pH 7.25
paCO₂ 80
paO₂ 50
Main issue: hypoxemia/hypercapnia?

Vt  450 ml
RR  27
iPEEP 18
Ppeak 61
FiO\textsubscript{2} 30%
pH  7.25
paCO\textsubscript{2} 80
paO\textsubscript{2} 50

Effects of \(\uparrow\)FiO\textsubscript{2}

\(\uparrow\)  \(P_{A}O_{2}\)
\(\uparrow\)  \(P_{a}O_{2}\)
Change in Hypoxic pulmonary vasoconstriction
Haldane effect
Change in V/Q
Decrease respiratory drive
\(\uparrow\)  \(P_{a}CO2\)
Expiratory flow limitation: $V'_{\text{exp}(t)} = \frac{V(t)}{R^*C(\tau)}$

Main issues:

Dynamic hyperinflation: $V_{(\text{trapped})} = \int \frac{V_T \times \tau}{T_E}$

Vt 450 ml
RR 27
iPEEP 18
Ppeak 61
FiO₂ 30%
pH 7.25
paCO₂ 80
paO₂ 50
7° case: pulmonary embolism

Vt 450 ml, RR 35, FiO₂ 50%, pH 7.48, paCO₂ 30, paO₂ 70

PaCO₂ may increase or decrease according to the response of the respiratory drive to increase dead space and hypoxemia

Acute pulmonary hypertension (also from high intra-thoracic pressure during MV) may aggravate hypoxemia by low MvO₂ or by intra-cardiac right-to-left shunting

8° case: refractory respiratory failure

Pplat 35, FiO₂ 100%, pH 7.11, paCO₂ 64, paO₂ 40

Excessive Trans-pulmonary Pressure

VILI

Courtesy of L. Gattinoni and R. Fumagalli
ARDS

HbO₂ 75%

HbO₂ (75% + 75%)/2 = 75%

SvO₂ 50%

HbO₂ 75%

PaO₂ = 40 mmHg

Courtesy of R. Fumagalli
**VV-ECMO**
- Pre-pulmonary
- Increased SvO₂
- CO₂ clearance

HbO₂ \( \frac{90\% + 90\%}{2} = 90\% \)

\[ \text{PaO}_2 = 60 \text{ mmHg} \]

Courtesy of R. Fumagalli
Conclusions
Conclusions

Hemodynamics ($\text{DO}_2/\text{V’O}_2$)

Physiologic interpretation of the effects of the bedside maneuvers

ARDS is an hypoxemic syndrome, **but**

Carbon Dioxide elimination is the true target of ventilation  
(BAD CO$_2$ ELIMINATION = risk of VILI)
Conclusions

In refractory cases.....

......think out of the BOX

Extra-Corporeal (gas exchange) Life Support
Thank you

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