Noninvasive Positive Pressure and Mechanical Ventilation: Clinical Pearls

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Overview

• Basics of Noninvasive Positive Pressure Ventilation (NPPV)
• Mechanical Ventilation
  – ARDS and lung protective ventilation
  – Common clinical challenges
• Weaning
NPPV: Confusing Terminology

• CPAP
  – Continuous positive airway pressure
  – Improves airway patency, V/Q matching

• Bilevel positive airway pressure (BPAP)
  – Combination of pressure support (a.k.a. IPAP) and positive end-expiratory pressure (a.k.a. EPAP)
  – Alveolar ventilation

• BiPAP® (Respironics), BIPAP® (Drager)
Why Bother with NPPV?

- Avoidance of complications of ETI:
  - Increased risk for nosocomial pneumonia/VAP, sinusitis, barotrauma
  - Complications of intubation process
  - Increased requirements for sedation
- Pts can go on/off NPPV easily to eat, talk
- When correctly applied, decreases mortality
  - In some settings, complications and length of stay as well
Contraindications to NPPV

- Impending circulatory collapse or respiratory arrest
- Unable to protect airway/altered mental status
- Excessive secretions or UGI bleeding
- Upper airway obstruction
- Recent facial, upper airway or gastrointestinal surgery
- Unable to get good fit with mask
- Inability to adequately monitor patient
NPPV in COPD Exacerbations: Patient Selection
NPPV in COPD Exacerbations

- Most well-studied acute application of NPPV

- First demonstrated to be effective by Meduri et al. (Chest 1989) and Brochard et al. (NEJM 1990)

- Since then, multiple RCT’s have confirmed significant benefit of NPPV in acute hypercapnic respiratory failure associated with COPD:
  - Compared NPPV to usual/standard care
  - “Failure”: intubation, failure to tolerate NPPV, or death
NPPV in COPD Exacerbations: Meta-analysis Data

- Cochrane Systematic Review 2004
- Decreased mortality
  - RR with NPPV = 0.52 (95% CI 0.35-0.76)
- Decreased intubation
  - RR with NPPV = 0.41 (95% CI 0.37-0.63)
- Decreased rate of complications
- Decreased length of stay

Ram et al, 2004
COPD Patient Selection: Best for Severe Exacerbations?

NPPV in COPD Exacerbations: Patient Selection Summary

• First step: determine if obvious contraindication to NPPV
  – Mild alterations in mental status/level of consciousness likely OK
• Increased severity = increased chance for benefit
  – ? Increased risk of failure
• May have benefit in mild exacerbations
• Initial response after one hour of treatment (improvements in pH, pCO$_2$ and mental status) predictive of success or failure
NPPV in Cardiogenic Pulmonary Edema

- Evidence indicates that NPPV reduces need for intubation and improves cardiopulmonary physiology
- Most studies suggest mortality benefit but data are conflicting
Acute Hypoxemic Respiratory Failure: Conflicting Data

- Meta-analysis of 8 RCT’s suggested benefit
  - Excluded cardiogenic edema
  - 17% absolute risk reduction ICU mortality
  - 23% absolute risk reduction intubation
  - Limited by heterogeneity

- Other studies have demonstrated high failure rate for NPPV in this setting (MGH observational trial, Schettino et al. *CCM* 2008)

- Likely due to heterogeneity of underlying conditions
- May consider its use in this setting, but not as strongly supported by evidence

Keenan et al, *CCM* 2004
A reminder for cautious use of NPPV in the ICU

Noninvasive Positive-Pressure Ventilation for Respiratory Failure after Extubation

Andrés Esteban, M.D., Ph.D., Fernando Frutos-Vivar, M.D.,
Niall D. Ferguson, M.D., Yaseen Arabi, M.D.,
Carlos Apezteguía, M.D., Marco González, M.D., Scott K. Epstein, M.D.,
Nicholas S. Hill, M.D., Stefano Nava, M.D., Marco-Antonio Soares, M.D.,
Gabriel D'Empaire, M.D., Inmaculada Alía, M.D., and Antonio Anzueto, M.D.

NEJM 2004
Case #1

- 68 yo man
  - PMH: COPD, CAD, HTN
- HPI:
  - 2 day hx increasing dyspnea, cough, low grade fever
  - WBC 11k
  - CXR: flattened diaphragm, no infiltrates
  - O2 sat 88 % on RA
- Dx: likely COPD exacerbation
- Tx: oxygen via NC, abx, steroids, bronchodilators
- Disposition: admitted to step-down unit
Day 2

• Hospital course:
  – Increasing dyspnea, low grade fever, O2 sat 86% on 6l NC

• Treatment options:
  – High flow nasal cannula
  – NPPV
  – Intubation
Day 2

• Treatment:
  – High-flow Nasal Cannula oxygen initiated at 15 LPM with saturation improved to 93%

• One hour later:
  – ABG: 7.25/65/60 and the patient’s mental status is mildly altered
  – Mild-mod accessory muscle use

• Treatment plan?
Case

BiPAP® initiated
NPPV: Clinical Pearls

• What to expect during initiation of BiPAP®
• Keys to selecting patient-mask interface
• Initial settings
• Monitoring
Case (continued)

- 60 minutes after BiPAP® initiation: 
  ABG: 7.21/70/78

- Now what?
  - Increase NPPV support (15/8, 20/8, etc.) vs.
  - Intubation
What is the next step in your treatment plan?

1. Increase BiPAP® level to 15/8 and re-evaluate over next 60 minutes?
2. Intubate trachea and initiate mechanical ventilation?
Summary of practical issues with NPPV

• Patients with acute exacerbations should probably be in ICU (or ED)
• Increased monitoring, particularly at initiation of treatment
• Should be applied early in course of disease
• Improvement in ABG (pH, pCO$_2$) over first 1-2 hours needs to be assessed
  – If pH, pCO$_2$ and/or mental status are worse than pre-NIV, pt probably needs intubation
Case # 2

- 54 yo woman with PMH of HTN, tob use (60 pack-years) admitted with suspected CAP
- Intubated in ED due to hypoxia, altered mental status
- Initial ABG: 7.32/47/89/24 on FiO2 100%, PEEP 10
- Initial vent settings: volume control, 700 ml (~11 ml/kg PBW), rate 16
- CXR: diffuse bilateral infiltrates
- Diagnosis: ARDS
ARDS: a few words on definitions

- Ashbaugh 1967
- AECC 1994
  - Acute onset hypoxemia (PF < 200)
  - Bilateral infiltrates on CXR
  - Absent left atrial HTN
  - Included definition of ALI (P/F < 300)
## ARDS: Berlin 2012

<table>
<thead>
<tr>
<th>Timing</th>
<th>Within one week of known clinical insult</th>
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<tbody>
<tr>
<td>Chest Imaging</td>
<td>Bilateral opacities on CXR or CT</td>
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<tr>
<td>Origin of Edema</td>
<td>Respiratory failure not fully explained by cardiac failure or fluid overload</td>
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<tr>
<td>Oxygenation</td>
<td></td>
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<tr>
<td>Mild</td>
<td>(200 \text{ mm Hg} &lt; \frac{\text{PaO}_2}{\text{FIO}_2} \leq 300 \text{ mm Hg}) with PEEP or CPAP (\geq 5 \text{ cm H}_2\text{O})</td>
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<tr>
<td>Moderate</td>
<td>(100 \text{ mm Hg} &lt; \frac{\text{PaO}_2}{\text{FIO}_2} \leq 200 \text{ mm Hg}) with PEEP (\geq 5 \text{ cm H}_2\text{O})</td>
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<tr>
<td>Severe</td>
<td>(\frac{\text{PaO}_2}{\text{FIO}_2} \leq 100 \text{ mm Hg}) with PEEP (\geq 5 \text{ cm H}_2\text{O})</td>
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*JAMA 2012*
Your patient

- “Revised” diagnosis: Severe ARDS
- Treatment:
  - Mechanical Ventilation
  - Ventilator Mode?
Initial Ventilator Settings

• ARDSNet approach:
  – Select vent mode: AC/VC vs. AC/PC
  – You start with AC/VC, Vt 8 ml/kg, FiO₂ 100%, PEEP 10
  – Goals:
    • Tidal Volume: 6 ml/kg
    • Rate: adjust to achieve baseline minute ventilation, keep ≤ 35
    • plateau pressure: < 30 cm H₂O
    • pH: 7.30 -7.45
    • PaO₂ goal 55 – 80 mmHg
But now…

• Plateau pressure is 35 on 8 ml/kg
• What next?
  – Attempt to reduce to 6 ml/kg
• Now, $P_{\text{plat}}$ is 29 mmHg and ABG:
  – 7.34/47/89 on FiO$_2$ 1.0 and PEEP 10
• What should you do about FiO$_2$ and PEEP?
### FiO2/PEEP Scales: ARDSnet recs

#### Lower PEEP/higher FiO2

<table>
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<tr>
<th>FiO₂</th>
<th>0.3</th>
<th>0.4</th>
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<th>0.6</th>
<th>0.7</th>
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<th>0.8</th>
<th>0.9</th>
<th>0.9</th>
<th>0.9</th>
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<tr>
<td>PEEP</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>16</td>
<td>18</td>
<td>18-24</td>
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</table>

#### Higher PEEP/lower FiO2

<table>
<thead>
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<th>FiO₂</th>
<th>0.3</th>
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<tr>
<td>PEEP</td>
<td>5</td>
<td>8</td>
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<td>12</td>
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<td>16</td>
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<table>
<thead>
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<th>FiO₂</th>
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<th>0.5-0.8</th>
<th>0.8</th>
<th>0.9</th>
<th>1.0</th>
<th>1.0</th>
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<tbody>
<tr>
<td>PEEP</td>
<td>18</td>
<td>20</td>
<td>22</td>
<td>22</td>
<td>22</td>
<td>24</td>
</tr>
</tbody>
</table>
3 hours later

• RT: “Wanted to let you know that the peak pressure is in mid-40s. I’ve decreased the flow rate without much improvement. Would you come look at the patient and vent with me?”
Components of Inflation Pressure

Begin Inspiration

Begin Expiration

\[ P_{aw} \text{ (cm H}_2\text{O)} \]

\[ P_{IP} \]

\[ P_{plateau} \text{ (Palveolar)} \]

\{ Transairway Pressure (P_{TA}) \}

Expiration

Time (sec)
Compliance & Pressures

- Peak inspiratory pressure (PIP)
  - reflects dynamic compliance
- Plateau Pressure ($P_{\text{plat}}$)
  - static compliance
  - Indication of transpulmonary P (airway P @ end-inspir pause - pleural pressure)
- The PIP – $P_{\text{plat}}$ gradient relates to airway resistance
High pressures

**PIP**
- High airway resistance
  - Bronchospasm
  - Secretions
  - ETT kinking
- Pneumothorax
- High flow

**Plateau Pressure**
- Poor compliance secondary to edema
- ARDS with fibrosis
PIP versus $P_{\text{plat}}$

- **Normal**: PIP, Pplat

- **High airway resistance**: Kinked ETT, Bronchospasm, Secretions

- **Poor Compliance**: Edema, Fibrosis, Stiff chest wall, Turbulent flow
Interventions

- Examine patient
- Consider CXR or US
- Consider disconnecting patient from ventilator and manually ventilating
  - *Pass suction catheter and confirm tube patency, remove secretions*
- Consider bronchodilator
Next steps

- Sedation: propofol gtt plus fentanyl prn
  - Other options
- Hemodynamics: acceptable without need for aggressive fluid resuscitation or vasopressor support
Early going … no problems
And…more trouble

- RT to you: “Things are going poorly. She’s not tolerating this mode. She’s completely asynchronous. I’m not sure she really has ARDS. Can we just switch to pressure control and give her larger tidal volumes?”
Patient / Ventilator Aysynchrony

- Breath-stacking
  - Large tidal volume
Possible options

• Continue with AC/VC
  – Consider adjusting inspiratory flow rate
• Re-evaluate sedation regimen
  – ? need for additional narcotic
  – In situations of severe hypoxia or vent asynchrony, consider temporary use of neuromuscular blockade (e.g. single dose cisatracurium)
Interventions

• O2 sat 84%, so after ensuring adequate amnesia, you gave cisatracurium 12 mg IV x 1 ➔ much better!
  – O2 sat 96%

• Fentanyl bolus plus increase in gtt rate
  – Hemodynamics acceptable, so no problem at this point.

• Cisatracurium metabolized, pt breathing slightly above set RR of 25
More trouble

• 24 hours later
• New issue with patient-ventilator interaction
Intrinsic or Auto-PEEP

• Unintentional (and often unmeasured) dynamic hyperinflation
• Characterized by interplay between elastic forces of lung and resistive forces of airways
• Measure with end-expiratory pause

Mughal et al. Clev Clinic J of Med 2005
Brochard L 2009
Causes of Auto-PEEP

- Increased resistance to expiration
  - COPD or asthma
- High minute ventilation (large volume and/or respiratory rate)
- Extrinsic obstruction to exhalation
- Dynamic airway collapse
Intrinsic Positive End-Expiratory Pressure

- **Normal**: 
  - End Inspiration: $P_{AO} = 20 \text{ cmH}_2\text{O}$, $P_{ALV} = 20 \text{ cmH}_2\text{O}$
  - End Expiration: $P_{AO} = 0 \text{ cmH}_2\text{O}$, $P_{ALV} = 0 \text{ cmH}_2\text{O}$

- **Airflow obstructed**: 
  - End Inspiration: $P_{AO} = 35 \text{ cmH}_2\text{O}$, $P_{ALV} = 35 \text{ cmH}_2\text{O}$
  - End Expiration: $P_{AO} = 0 \text{ cmH}_2\text{O}$, $P_{ALV} = 15 \text{ cmH}_2\text{O}$

For Expiratory hold, $P_{AO} = 15 \text{ cmH}_2\text{O}$
Auto-PEEP

Consequences

- Respiratory effects
  - Increased WOB
  - Vent asynchrony
  - Risk of pneumothorax

- Hemodynamic effects
  - Decreased venous
  - Decreased cardiac output
  - Increased right heart strain

Treatment

- Disconnect patient from ventilator temporarily if auto-PEEP suspected
- Reduce pt demand
- Reduce resistance
- Consider use of external PEEP
24 hours later…

- Hypoxia worsening
- Current status:
  - AC/VC, 6 ml/kg, FiO₂ 1.0, PEEP 20, RR 25
  - ABG: 7.36/44/44/51
- Options for intervention?
Refractory Hypoxemia: Overview of Options

**Ventilator Options**
- Recruitment Maneuvers
- High PEEP
- APRV
- Oscillator-based strategies

**Non-Vent Options**
- Proning
- iNO
- ECLS
Details & Evidence
Do recruitment maneuvers work?

• Goal: Increase aerated lung tissue (↓ shunt and V/Q mismatch)

• Technique:
  – Large sustained manual breaths
  – Sustained (e.g 30 -40 secs) CPAP at 30 – 40 mmHg

• Outcomes:
  – Three major RCTs plus one large meta-analysis demonstrate consistent but *transient* improvement in oxygenation
  – Low incidence of serious adverse events
  – No demonstrated mortality benefit

• Cannot be routinely recommended or discouraged

High PEEP

- ALVEOLI, EXPRESS, LOV Trials
- No mortality benefit demonstrated
- EXPRESS and LOV both demonstrated improvement in secondary endpoints
  - Increase vent free days
  - Better oxygenation
  - Decreased need for adjunctive therapies or “rescue”
Airway Pressure Release Ventilation

- Inverse ratio, pressure controlled, intermittent mandatory ventilation
- Allows for spontaneous breathing (with optional pressure support)
- No clear mortality benefit, including its use in patients with lung injury
- Confusion exists about terms and definitions
  - APRV, BiLevel, Bi-Vent, etc.
APRV

• Advantages
  – Improved oxygenation
  – Better alveolar recruitment
  – Decreased sedation needs
  – ? Lung protective

• Disadvantages
  – No clear mortality benefit
  – Impaired hemodynamics
Is there evidence for APRV?

• Very limited
• Best known study: Putensen et al. *AJRCCM* 2001
  – 30 patients
  – APRV vs. PCV
  – Only 20% in APRV group ultimately determined to have ARDS vs. 74% in PCV group
  – Decreased ICU LOS and duration of vent support
More recent data

- Maxwell et al. *J Trauma* 2010
  - RCT, 63 patients, needing mech vent > 72 hours
  - APRV vs. low tidal volume ventilation
  - No significant difference in vent days, ICU stay, VAP, or mortality
  - Trend toward increased sedation needs with APRV
  - Higher APACHE II scores in APRV group
APRV Settings

- P High, P Low
- T High, T Low
- Pressure support for spontaneous breathing
What Is APRV?

![Diagram](image-url)

- Spontaneous Breaths
- CPAP
What Is APRV?

If This...

Why Not This?

Why Not This?
What Is APRV?

• Mimics CPAP... but with release
• Preserves spontaneous breathing
• Substantial improvements for spontaneous breathing
• better synchronization with spontaneous breathing
Prone positioning

- Better matching of ventilation and perfusion
- Opening of dependent collapsed lung segments
- RCTs demonstrated improved oxygenation but no mortality benefit
  - Gattinoni et al. *NEJM* 2001
  - Guerin et al. *JAMA* 2004
  - Recent meta-analysis (Sud et al. *ICM* 2010) found mortality benefit in patients with severe hypoxemia (P/F < 100)
Physiologic Benefits

Supine: end-inspir

Prone: end-inspir

Pelosi et al. Eur Respir J 2002
• Inhaled selective pulmonary vasodilator
• Physiologically sensible but no mortality benefit
Possible negative effects

- Methemoglobinemia
  - Usually at higher doses
- Increase risk of renal dysfunction
- Rebound pulmonary HTN
- ? worsening of oxygenation at higher doses (> 40 ppm)
When all else fails…
ECLS
ECLS & respiratory failure: early literature

• Hill et al. *NEJM* 1972
  – First case report of successful use of ECLS for young man with respiratory failure

• Zapol et al. *JAMA* 1979
  – RCT of 90 pts with ARDS: ECMO vs. conventional ventilation (pre-lung protective ventilation, HIGH mortality rates, > 90%)

• Morris et al. *AJRCCM* 1994
  – RCT of IRV + ECMO for CO₂ removal vs. conventional tx; no benefit
CESAR Trial

Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial

Giles J Peck, Miranda Mugford, Ravindranath Tiruvoipati, Andrew Wilson, Elizabeth Allen, Mariam M Thalanany, Clare L Hibbert, Ann Truesdale, Felicity Clemens, Nicola Cooper, Richard K Firmin, Diana Elbourne, for the CESAR trial collaboration

• ECMO vs conventional ventilation
• 180 pts
• 63% six month survival without disability in ECMO group vs. 47% in conventional
• Many caveats
When to consider ECMO?

- No well-accepted criteria
- Criteria from CESAR trial:
  - Lung injury score > 3, or
  - Hypercapnic respiratory failure with pH<7.2
- Other key criteria:
  - Respiratory failure judged to be reversible
  - Early in course of disease
  - No contra-indication to anticoagulation
- When you are already considering other “rescue” therapies
Ventilator Weaning
Difficult to wean patient

• Back to our first patient
  – 68 yo man with COPD, intubated after failing NPPV
  – Mechanical ventilation, day # 6
  – Current settings:
    • AC/VC 500, Rate 12 (14-22)
    • FiO₂ 40 %, PEEP 8
    • Failed SBT x 3 secondary to tachypnea
    • Most recent ABG on AC/VC: 7.38/49/95/32
What’s a spontaneous breathing trial?

• Spontaneous breathing trial (SBT)
  – Variably defined
  – Generally, breathing through an ETT with either minimal support (low level PSV, CPAP, or ATC) or no support (T-piece)
  – No evidence to support one approach over another

• SBTs are often protocol-driven and do not require physician order
  – Multiple studies support this approach
    • Ely et al. *NEJM* 1996
    • Blackwood et al. *BMJ* 2011 (Cochrane Review)
Weaning Predictors

- Generally, not very helpful
- Rapid Shallow Breathing Index (RSBI)
  - RR/TV
  - RSBI ≥ 105 = “negative” RSBI
  - Index is better at predicting which patients will fail
- Maximal Inspiratory Pressure (MIP) or Negative Inspiratory Force (NIF)
  - More negative than 30 cm H20 may predict successful liberation
Readiness for SBT

- Reversal of underlying cause of respiratory failure
- Adequate oxygenation and pH
- Hemodynamics are acceptable
- Ability to initiate an inspiratory effort
- *Normothermic, adequate hemoglobin, reasonable mental status*

MacIntyre et al. *CHEST* 2001
Diaphoresis and nasal flaring indicate increased patient effort

Cyanosis is not a reliable physical sign

Tachypnea determined over the course of a full minute is a sensitive sign of failure

Paradoxical motion of the abdomen is also evidence of increased patient effort

Heightened sternomastoid activity is evidence of increased patient effort

Recession may be seen in the suprasternal and supraclavicular spaces

Intercostal space recession also indicates increased patient effort

Tachycardia is an indicator of severe cardiopulmonary distress
Tolerance of SBT

- Gas exchange (ABG)
- Hemodynamics
- RR and TV (Minute Ventilation)
- Mental Status
- Discomfort
- Work of Breathing
Spontaneous Breathing Trials

- Recommend once daily trials
- Recommended length of SBT: 30 – 120 minutes
- Return to higher levels of support if patient “fails” SBT
- Search for causes of failure
- If successful SBT, is the patient appropriate for extubation?
  - Can the pt protect his or her airway?
  - Will the airway remain patent?
What to do about our difficult to wean patient with COPD?

Is there a role for NPPV?
NPPV for Ventilator Weaning

- Ferrer et al, *AJRCCM* 2003
- 43 mechanically ventilated patients who had failed conventional weaning x 3 days
  - 77% chronic lung disease, mostly COPD
- Stopped early after interim analysis
NPPV for Ventilator Weaning

Days

Invasive Vent  ICU Stay  Hospital Stay

NIV  Control

(p≤0.003 for all)

Ferrer AJRCCM 2003
NIV for Ventilator Weaning

- ICU Mortality
- PNA
- Sepsis
- Trach

\( p \leq 0.05 \) for all

Ferrer AJRCCM 2003
NPPV and Ventilator Weaning: Cochrane Analysis 2010

• 12 trials, 530 patients
  – Majority of patients included in trials had COPD

• NPPV decreased:
  – Mortality
  – VAP
  – ICU LOS
  – Hospital LOS
  – Ventilators days

Burns et al. The Cochrane Library 2010
Key points

• Do not extubate and wait for failure before initiating NPPV
• This is a planned approach to use NPPV immediately after extubation
• Regimens can vary but close monitoring is essential
Summary

• NPPV is an important tool in select patient populations
• Use lung protective ventilation
• Consider a variety of ventilator and non-ventilator options for the patient with refractory hypoxemia
• Perform daily evaluations to determine extubation readiness