Oxygenation
Assessment and Treatment in COVID-19
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Disclosures / Advertisement

I have a Book ... That I am continually consciously and unconsciously promoting

Friend my book on Facebook:
Landsberg Manual
Major Mistakes and Misconceptions

• CONFUSING Hypoxia (low tissue oxygen levels) with Hypoxemia (low dissolved oxygen concentration in blood)
• Targeting too LOW an SaO₂ (e.g. > 92%), allowing mild hypoxemia (PaO₂ 55-59 mm Hg) to go unnoticed and untreated
• Being reassured about oxygenation because dyspnea is either mild or absent
• Believing that a high FIO₂ and or PaO₂ suppresses ventilatory drive in chronic CO₂ retainers
Hypoxia =
state of low oxygen in TISSUE =
SHOCK

- Represents a failure the circulatory system, primarily cardiac output (CO)
- Causes systemic lactic acidosis
- Treated by increasing CO and vascular tone, if appropriate (e.g. distributive state)
- Increasing PaO₂ > 60 mm Hg does not meaningfully increase oxygen delivery to tissues or decrease lactate

\[
\text{DO}_2 = \dot{Q} \times (\text{Hb} \times \text{SaO}_2 \times 1.34 + (\text{PaO}_2 \times 0.003))
\]

- Oxygen delivery
- Hemoglobin
- Amount of dissolved O₂ in the blood
- Cardiac output
- Arterial O₂ saturation
Hypoxemia = low PaO$_2$

- Represents a failure of the respiratory system, to maintain a PaO$_2$ > 60 mm Hg
- When PaO$_2$ drops acutely to < 60 mm Hg (hypoxemia), individuals experience ......

\[ \text{PaO}_2 \text{ mm Hg} \]

- Mild Hypoxemia
  - 60
- Severe Hypoxemia
  - 55
  - 50

\[ \begin{align*}
\Rightarrow & \text{Confusion} \\
\Rightarrow & \text{Agitation} \\
\Rightarrow & \text{Sinus Tachycardia} \\
\Rightarrow & \text{Tachypnea} \\
\Rightarrow & \text{Dyspnea} \text{ mild or absent} \\
\Rightarrow & \text{Extreme Dyspnea} \\
\Rightarrow & \text{Lactate accumulation / resting tissue hypoxia} \\
\Rightarrow & \text{PEA sinus bradycardic cardiac arrest} \\
\end{align*} \]

Pts with Mild Hypoxemia are at risk for sudden severe hypoxemia.
Assessing Oxygenation and Acute Hypoxemic Respiratory Failure

- **Normal Oxygenation** (at sea level)
  - 21% FiO₂ (room air) → PaO₂ of 75–100 mm Hg
  - 100% FiO₂ → PaO₂ of ~ 660 mm Hg

- **“VQ mismatch”**
  - Perfusion **Too High** relative to Ventilation = Low V/Q ratio
  - PaO₂ Increases with 100% FiO₂

- **Shunt Physiology**
  - Perfusion despite NO ventilation
  - PaO₂ remains low despite 100% FiO₂
    - PaO₂ < 200 mm Hg on 100% implies shunt (physiologic MORE common than anatomic)
  - Treat by Increasing Mean Airway Pressure (best achieved with PEEP), recruits lung to increase PaO₂

- **Hypoxemic Respiratory Failure** *best* defined as a PaO₂ < 60 mm Hg,
  - Occurs when deoxygenated blood mixes directly with oxygenated blood
Low V/Q

Physiologic shunt

Bronchospasm

PAO₂ 80
PAO₂ 30

PA
PV

Mucus plug

PAO₂ 80
PAO₂ 0

PA
PV
Symptoms of Acute Mild Hypoxemia
PaO₂ 54-59 mm Hg

• Tachypnea (hypoxic hyperventilation reflex)
  • ↑ alveolar O₂ by ↓ alveolar CO₂,
  • ↑ work of breathing (WOB)

• Tachycardia
  • Heart rate (HR) ↑ to maintain CO AS:
    • Hypoxic vasoconstriction
    • ↑ pulmonary artery pressure (PAP)
    • ↓ stroke volume (SV)
Symptoms of Acute Mild Hypoxemia (cont.)

- Mental status changes
  - Agitation, Confusion, Decreased Sensorium
- ↑ left ventricular end-diastolic pressure (LVEDP)
  a. heart failure) from diastolic dysfunction
  - Hypoxia stiffens the LV, tachycardia shortens diastole, both impair ventricular filling
- ↓ glomerular filtration rate (GFR) from hypoxic renal injury and cardio-renal physiology
- PTs with MILD hypoxemia may suffer life-threatening desaturations
  - Steep portion of the hemoglobin–oxygen dissociation curve

**Dyspnea is NOT a major symptom of Mild Hypoxemia**
Symptoms of Severe Hypoxemia
\( \text{PaO}_2 < 50 \text{ mm Hg} \)

- Extreme Dyspnea, Lactate production
- Cardiac arrest from pulseless electrical activity
  - Sinus Bradycardia, precipitous HR drop 70 ... 60 ... 50 ... 40 ... 30 ... 20
Evaluation of Hypoxemia

- **VQ mismatch** is often attributable to acute process (e.g. Pneumonia), bronchospasm (wheezing)
- **Shunt Physiology** SHOULD BE OBVIOUS on imaging or exam (severe bronchospasm - no air movement)
- Consider **Anatomic Shunt** if **NO radiographic explanation** (e.g. contrast echo looking for early or late left sided bubbles)
Anatomic Shunt (Right to Left shunting)

Intracardiac

more common then

Intrapulmonary

High Right Sided pressures with Low Left Sided pressures (e.g. Pulmonary Embolism with Shock)
Promote Right to Left shunting

Macro pulmonary AVMs
(HHT, Cirrhosis)

Micro pulmonary AVMs
(Cirrhosis)
Screening for and Assessing Hypoxemia

- Pulse oximetry **ESTIMATES** Hb sat (± 3 point error range less reliable at low sats)
- Pulse oximetry should be used as a **screening test**, to ensure that the PaO₂ > 60 mm Hg
- Pulse oximetry > 94% (good wave form) = high probability of PaO₂ ≥ 60 mm Hg
- ‘> 92%’ = most common inpatient oxygen goal = TOO LOW
- Pulse oximeter > 92% (but < 95%) **may mask** a PaO₂ < 60 mm Hg because of alkalosis or error
Pulse oximetry goal of > 92% is TOO LOW to ensure a PaO$_2$ > 60 mm Hg

- Hb binds O$_2$ tight (↑O$_2$ sat) in alkalotic lung and unloads O$_2$ (↓O$_2$ sat) in acidotic muscle

- **Alkalemia ↑ Hb sat**
  - Steepens Hb–O$_2$ dissociation curve
  - ↑ risk of rapid desaturation

- **Acidemia ↓ Hb sat**
  - Flattens Hb–O$_2$ dissociation curve
  - ↓ risk of rapid desaturation
- Patient admitted for heart failure with a preserved ejection fraction (HFpEF)
- Intubated for increased work of breathing and hypoxemia
- Admit CXR with increased interstitial markings, small effusions
- Despite a - L negative fluid balance over the first 24 hours the PT suffers < oxygenation
- CXR on HD # 1 shows worsening pulmonary edema:
  - Perihilar ground glass and interstitial edema with worsening effusions (L > R)
- EKG, troponins and a STAT cardiac echo were unchanged from admission
- Blood pressure overnight 15Q-160/80-85, HR: 60-90 sinus rhythm
Inspection of the flow sheet shows the J in FiO₂ to 60% at 4:30am lead to hypoxemia. Not recognized until a routine ABG was obtained at 6:00am. The hypoxemia was missed because of:
- Pulse oximeter 3 point error despite a good waveform
- Cutaneous O₂ sat 94%, calculated O₂ sat 91%
- Alkalosis shifting the H tr O₂ dissociation curve

The hypoxemia caused worsening pulmonary edema by provoking diastolic dysfunction.
- Hypoxemia + subendocardial hypoxia causing LV stiffing + impaired filling
- Leading to LVEDP and pulmonary edema despite a negative fluid balance
- Note, increased peak inspiratory pressures occurring during the same time frame indicative of pulmonary edema and worsening pulmonary mechanics
Clinical Approach to Acute Hypoxemic Respiratory Failure

- ↑FiO₂ (supplemental oxygen)
- Goal PaO₂ > 60 mm Hg (without hyperventilation)
- Target O₂ sat > 94% OR ensure PaO₂ > 60 mm Hg (ABG)
- Hypoxemia despite O₂ ≥ 10 L/min = Impending hypoxemic arrest → Mandates a trial of 100% FiO₂ and consideration for intubation in COVID-CAP patients
- **DO NOT** withhold 100% FiO₂ fearing CO₂ retention (does not suppress drive)

pH 7.46/PCO₂ 33/PaO₂ 60
Providing 100% oxygen

• Achieve 100% FiO₂ via a high-flow system or reservoir device
• Prevents entrainment of room air
  • High minute ventilation dilutes inspired O₂
• PaO₂ < 60 mm Hg (100% FiO₂) is life-threatening, mandating mechanical ventilation
  • BiPAP or Intubation
• ↑ mean airway pressure
• Recruits atelectatic lung
CO₂ Retention & High FiO₂

• Hypoxemia NORMALLY stimulates ventilation = Hypoxic Hyperventilation Reflex
  • Alveolar Hypocarbia increases Alveolar O₂
  • May cause diaphragmatic fatigue (unlike all other etiologies of hyperventilation)

• In patients with severe parenchymal disease (baseline VQ mismatch), high FiO₂ & PaO₂ occasionally lead to increased PCO₂ (~6 mmHg)

• **NOT** by inhibition of DRIVE (i.e. Will not lead to progressive central hypercarbic respiratory failure)

• By inhibition of hypoxic vasoconstriction and subsequent adjacent vessel steel, leading to ventilated but relatively unperfused units

• Of little clinical significance (unlike the hypoxemic respiratory arrest)

• Very Rarely high FiO₂ & PaO₂ May impact drive
**Room Air:**
FiO₂ 21%

- PaO₂ = 40
- PaO₂ = 65

**pH:** 7.38
**PaCO₂:** 48
**PaO₂:** 57
**HCO₃:** 28

**Respiratory Rate:** 18
PaO₂ = 90

PaO₂ = 300

pH 7.33 / PaCO₂ 54 / PaO₂ 164 / HCO₃ 28

Respiratory Rate 18

Blunted Ventilatory
Drive AKA Blue Bloater

FiO₂ 100%

Steal

Relative Underperfusion
High Flow

• Provides O2 at flow rates that exceed maximum minute ventilation (e.g. > 60 L/M)
• Increases mean airway pressure/PEEP
• May provide significantly more support at relatively low flow rates (e.g. 40L/M) than 100%NRB, for those suffering RA entrainment
BIPAP for Acute Hypoxemic Respiratory Failure

• PTs in acute hypoxemic respiratory failure deserve a trial of BIPAP before intubation, if they are:
  • Arousalable
  • Able to wear a mask (eg, no facial or scalp wounds)
  • Not requiring continual oral clearance (eg, emesis, copious pulmonary secretions, massive hemoptysis)
• BIPAP provides differential inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP)
• Must Set IPAP, EPAP (aka PEEP) and FIO₂
  • Setting a rate may assist with synchrony BUT does NOT generate breaths (unlike invasive mechanical ventilation)
BIPAP for Hypoxemia

• **Start 15/5**

• **Minimum IPAP** = **15 cm H₂O**
  • *Increasing IPAP* can ↓ WOB and ↑ O₂ (↑ mean Airway pressure)
  • Maximum tolerated / deliverable IPAP 20-25 cm H₂O

• **Minimum EPAP** (aka PEEP) = **5 cm H₂O** (physiologic)
  • *Increasing EPAP* (a.k.a. PEEP) may
    • ↑ EPAP can redistribute fluid to the edges of the alveolar space and ↑ O₂
    • Aid in recruitment
    • High EPAP is uncomfortable for patients
    • EPAP should be increased slowly (increments of 2 cm H₂O) to avoid precipitating intolerance
End of slide deck presented on 4/7/2020

Further / Additional slides on mechanical ventilation included below
INACION OF BIPAP FOR ACUTE HYPERCAPNIC OR HYPOXEMIC RESPIRATORY FAILURE

Screen for contraindications:
- Otitis media (1130/30)
- Inability to tolerate a mask (wounds)
- Need for oral intubation

0 Massive hemoptysis
- Copious pulmonary edema

Start BIPAP
- IPAP 15 cm H₂O, EPAP 5 cm H₂O, FiO₂ 1

Initial tolerance:
- Tolerated
- Intolerant

First step:
- PAP b.i.d. (to a max of 25 cm H₂O)
- EPAP Z (to a max of 15 cm H₂O)

Objectives:
- Improved
- No change
- Worse

Worst case:
- May have to leave the bedside III this part while a ABO (but not polar)
- Prednisone, should be continued
Mechanical Ventilation for Acute Respiratory Failure

Your insurance won't cover a ventilator any longer, so Bob here will be giving you mouth to mouth for the next several days.
Invasive Mechanical Ventilation: Necessary Evil

- Mechanical ventilation is capable of causing life-threatening injury by causing either (or both):
  - Noncardiogenic pulmonary edema/ARDS (by alveolar overdistension and trauma)
  - Pneumothorax (PTX)
- Most likely to occur when:
  - **Lung volumes** are high (> 8 mg/kg)
  - **Peak airway pressures** are high (> 40 cm H₂O)
  - **Plateau pressures** (P_{plat}) are high (> 30 cm H₂O)
Lung Protective Ventilation

- **Goal** of mechanical ventilation is to provide adequate respiratory support without causing lung injury.
- **Adequate** as opposed to full support prioritizes low lung volumes over a normal pH and pCO₂.
- **Lung-protective ventilation** prioritizes Tidal Volume (TV) of 6–8 mg/kg IBW.
- Low lung volumes improve survival in individuals with ARDS (diffuse infiltrates, poor compliance, shunt physiology).

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<th>Male Tidal Volume (mL) 6 mg/kg</th>
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Permitting Hypercapnia and Acidosis to Maintain LOW Lung Volumes

• Permissive hypercapnia = tolerating hemodynamically asymptomatic respiratory acidosis (Often pH values < 7.25)

• Prioritizing low lung volumes means that minute ventilation is increased via ↑respiratory rate (RR), NOT tidal volume
  • Max RR (breaths/min) is limited by exhalation time, varies widely based on pulmonary mechanics (~15 for obstruction vs ~ 35 for restriction)
  • Practically speaking, max rate is determined by examining the expiratory flow waveform and ensuring that flow returns to zero before the next breath is delivered

![Basic Ventilator Wave Form Analysis](image1)

![Breath Stacking](image2)
Low lung volumes and Hypercapnia Are Uncomfortable (acidosis and air hunger)

- Lung-protective ventilation often requires deep **sedation** and **paralysis** to avoid **dyssynchrony**, which can:
  - Prevent effective ventilation via high airway pressures from patient struggling
  - **Cause breath stacking**
  - **Cause double triggering**
    - PT triggers a breath immediately after the last breath (before exhalation), leading to ~ 2 x the set TV
Improving Oxygenation by Recruiting Alveoli by Increasing mean airway pressure, with PEEP

• Increasing FiO₂ to 100% can improve hypoxemia from low V/Q physiology

• Shunt physiology, common with extremely poor compliance (eg, ARDS), requires an FiO₂ of 100% and high PEEP to attain a PaO₂ > 60 mm Hg

• Increasing PEEP
  • Recruits uninjured alveoli and protects injured alveoli from atelectasis
  • Forces intraalveolar fluid to the edges of the air sac, improving diffusion and thus oxygenation
  • Prevents and resolves segmental / lobar atelectasis (as seen obesity)

• Optimal PEEP (where most lung units are inflated but none are overdistended), is different in every patient and changes over time
  • Increasing PEEP will increase PIP variably based on the stiffness of the lung and the degree of lung inflation

• PEEP > 12 cm H₂O may decrease venous return and cardiac output, causing hypotension (worse in PTs with abnormal RV function and hypovolemia)
Improving Oxygenation by Recruiting Alveoli by Increasing mean airway pressure, with PEEP
Volume controlled vs Pressure controlled Modes

• All individuals requiring mechanical ventilation can be appropriately managed with either volume-controlled (VC) or pressure-controlled (PC) ventilation

• Volume controlled
  • TV is set and fixed
  • Airway pressures vary based on airway resistance and lung compliance

• Pressure controlled
  • Peak airway pressure is set and fixed
  • TV varies based on airway resistance and lung compliance
Volume-controlled (VC) ventilation

• **VC** is *preferred* for PTs with **normal to moderately abnormal** pulmonary mechanics:
  - Prioritizes control of tidal volume, the hallmark of a lung-protective ventilation
  - Most commonly used mode, making it the safest
    • Staff familiarity makes it easiest mode to troubleshoot
  - More comfortable than PC, requiring less sedation
• VC is only problematic in PTs with severely abnormal pulmonary mechanics (ie, ↑↑ airway resistance or ↓↓ compliance)
  • Takes time to find the minimally acceptable TV (based on airway pressure alarming), delaying adequate support increasing the **risk for barotrauma**
Pressure-controlled ventilation (PC)

- **PC** is preferred for PTs with **SEVERELY abnormal pulmonary mechanics**
- PC protects PTs with severe obstruction or poor compliance from barotrauma, rapidly establishing the minimally effective/safe TV
- Protection from barotrauma **RISKS underventilation**, as TV is sacrificed to avoid high airway pressures
  - PIPs are fixed; therefore TV and MV drop when mechanics worsen
- **Low exhaled TV and low MV alarms** are the most important alarms in PC signaling a change in mechanics (akin to the peak airway alarm in VC)
  - Settings reflect the lowest TV & MV you would tolerate (eg, 350 mL in pt w/ TV target of 450 mL)
Mechanical Ventilation of Mild to Moderate ARDS: Initial ventilator settings and Adjustments
Tidal Volume (TV) 6–8 ml / kg IBW, Respiratory Rate (RR) 20-25 bpm, FiO2 100%, PEEP 5-10 cm H2O
(Consult ID and consider clinical trial eligibility, off label / new antivirals, and immune mediated therapies)

**Trouble shooting High Peak Inspiratory Pressure**
- Examine the patient (check for dysynchrony, agitation, breath stacking )
- Obtain peak and plateau pressures
  - High peak pressures WITH low plateau pressures (Δ > 15) = increased resistance
  - Commonly seen with occluded ET tube or airway (e.g. biding, mucus plug, blood clot, bio film), or bronchospasm
  - High peak AND plateau pressures (Δ < 10) = worsening compliance
  - Commonly seen with dysynchrony, extremely high auto PEEP (aka breath stacked), edema (HF or ARDS), collapse, pneumothorax or intravascular abdominal physiology (e.g. ileus, ascites)
- Obtain an ABG and a CXR
- Consider change to pressure control (to avoid barotrauma) while troubleshooting
- Worsening compliance, not related to superimposed problems (see above) suggests progresstion to severe ARDS (see mechanical ventilation of severe ARDS)

**Improving Oxygenation in Mild to Moderate ARDS on Mechanical Ventilation**
- ↑ PaO2 by ↑ FiO2 to 100% and then ↑ positive end expiratory pressure (PEEP)
- ↑ PEEP 1st maneuver for ARDS and hypoxemia despite 100% FiO2
  - Recruits and protects unjured alvei, improves diffusion in injured alveoli
  - Prevents and resolves lower lobe atelectasis (as seen in the obese)
  - Optimal PEEP = the PEEP where alveoli are inflated but not over distended (determined empirically)
  - Increasing PEEP increases PIP variably based on lung stiffness and the degree of recruitability
    - When PIP increases 1 for 1 with PEEP it is concerning for overdistension
    - When PIP stays the same despite increased PEEP it suggests recruitment
  - PEEP > 12 H2O may ↓ venous return, CO and BP (worse in RV dysfunction and hypovolemia)
  - Tolerate asymptomatic ↓ CO to prevent a PaO2 < 60 mm Hg
- Symptomatic ↓ CO (e.g. ↓ BP):
  - Euvolemic or hypovolemic Pts can be Rx with NS Boluses 250-500 ml up to 1-2 L (screening for edema)
  - Volume overloaded Pts with ↓ CO and ↓ BP related to high PEEP should have a trial of inotropes to improve RV functioning (e.g. Norop, Dopamine, Dobutamine)
  - ↓ BP from high PEEP rapidly responds to ↓ PEEP (making PEEP up-titration safe)
  - Obese patients often need a PEEP > 20 cm H2O to resist the collapsing force of their thoracic wall
- Ensure ventilator synchrony with deep sedation and PRN paralytic administration
- Aggressive Treatment and Prevention of Volume Overload
  - Edematous Patients:
    - Goal I/O negative 1-2L Daily
    - Rx poor urine output with Loop Diuretic
  - Euvolemic – Dry Patients:
    - Goal I/O even with Loop Diuretics
    - Rx poor urine output with high dose Loop Diuretics 1st and if fails Rx with NS Boluses 250-500 ml up to 1-2 L
Severe ARDS Initial Ventilator Settings and Adjustments:

PRESSURE CONTROL: Driving Pressure 20 cmH₂O, PEEP 10-20 cmH₂O, Rate 20-30 bpm, FIO₂ : 100%
(Consult ID and consider clinical trial eligibility, off label / new antivirals, and immune mediated therapies)

- Tidal Volume (TV)
- Plateau Pressure (Pplat)
- Arterial Blood Gas (ABG)

Important Alarms
- Low exhaled TV, set for 300-400 mls
- Low minute ventilation alarm set for < 4 L/m

TV > 6 mg/kg
OR
Pplat > 30 cm H₂O
↓ Driving Pressure

pH < 7.20 - 7.25*
PaO₂ < 60 mm Hg

↑ Rate (to maximum †)
↑ PEEP (to maximum †)

† Maximum Rate = As fast as possible without breath stacking (typically 25-35 b/m)
* Tolerate a lower pH if no symptoms (i.e. No Supraventricular Tachycardia or refractory Hypotension)
† Maximum PEEP = As high a PEEP as possible without hypotension (impaired Venous return), typically 15–20 cm H₂O
* Keep Peak Air Way Pressure (Driving Pressure + PEEP) < 40-50 cm H₂O (to avoid pneumothorax)

Trouble shooting Low exhaled TV / Low minute ventilation alarm

- Examine the patient (check for dysynchrony, agitation, breath stacking) – ensure paralysis
- Commonly seen with dysynchrony, extremely high auto PEEP (aka breath stacked), edema (HF or ARDS), collapse, pneumothorax or intrinsically abnormal physiology (e.g. ileus, ascites)
- Obtain an ABG and a CXR
- Worsening compliance, not related to superimposed problems, suggests worsening ARDS (see worsening ARDS)

Improving Oxygenation in Severe ARDS on Mechanical Ventilation

- Sedate and Paralyze (ensure ventilator synchrony)
- ↑ PaO₂ by ↑ positive end expiratory pressure (PEEP)
- ↑ PEEP 1st maneuver for ARDS and hypoxemia despite 100% FiO₂
  - Recruits and protects uninjured alveoli, improves diffusion in injured alveoli
  - Prevents and resolves lower lobe atelectasis (as seen in the obese)
  - Optimal PEEP = the PEEP where alveoli are inflated but not over distended (determined empirically)
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    - When PIP stays the same despite increased PEEP it suggests recruitment
  - PEEP > 12 H₂O may ↓ venous return, CO and BP (worse in RV dysfunction and hypovolemia)
  - Tolerate asymptomatic ↓ CO to prevent a PaO₂ < 60 mm Hg
  - Symptomatic ↓ CO (e.g. ↓ BP):
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    - Volume overloaded PTs with ↓ CO and ↓ BP related to high PEEP should have a trial of inotropes to improve RV functioning (e.g. Nor keypad, Dobutamine, Dopamine)
  - ↓ BP from high PEEP rapidly responds to ↓ PEEP (making PEEP up-titration safe)
  - Obese patients often need a PEEP > 20 cm H₂O to resist the collapsing force of their thoracic wall

- Aggressive treatment and Prevention of Volume Overload
  - Edematous Patients
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- Refractory Hypoxemia / Worsening ARDS (despite PEEP, paralysis and diuresis) Consider:
  - Prone Positioning for 12-16 H
  - Traditional recruitment maneuvers (transient high PEEP)
  - Inhaled Pulmonary Vasodilators (e.g. NO) NOT safe in the presence of cardiogenic pulmonary edema
  - Trial of glucocorticoids for possible AEP, AIP, or OP
  - Referral to UCSD ECMO (cannulation evaluation occurs on site)
Troubleshooting

TROUBLESHOOTING CHANGES IN PULMONARY MECHANICS OCCURRING ON MECHANICAL VENTILATION

- **Lung compliance** or **Airways resistance** = O₂ desaturation

**Volume control**
- Worse mechanics = Increased peak inspiratory pressure

High peak airway pressure alarm
- Obtain
  - Peak inspiratory pressure (PIP)
  - Plateau pressure (Pₚₑₚ)
  - Arterial blood gas (ABG)
  - Chest X-ray

**Pressure control**
- Worse mechanics = Decreased tidal volume

Low exhaled TV or Low minute ventilation alarm
- Obtain
  - Arterial blood gas (ABG)
  - Chest X-ray

**Lung compliance** or **Airways resistance**

- Dyssynchrony
- Breath stacked
- Edema
- Collapse
- Pneumothorax
- Intrusive abdomen

- **Lung compliance**
  - PIP - Pₚₑₚ < 10 cm H₂O
  - Sedate (paralyze)
  - Consider disconnect from ventilator circuit*
  - Diuresis, paracentesis

- Suction
  - Consider bronchoscopy*
  - Sedate (paralyze)
  - Bronchodilator
  - Steroids

- **Airways resistance**
  - PIP - Pₚₑₚ > 15 cm H₂O
  - Sedate (paralyze)
  - Consider bronchoscopy*
  - Consider disconnect from ventilator circuit*
  - Bronchodilator
  - Steroids
  - Diuresis, paracentesis

* Disconnection from the ventilator and bronchoscopy causes PEEP loss and possible derecruitment