Oxygenation

Assessment and Treatment in COVID-19

4/7/2020

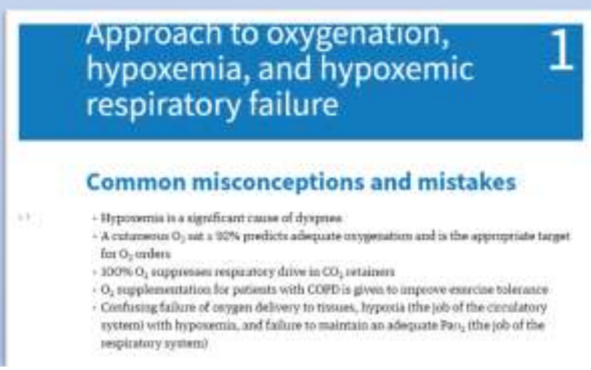
Judd W. Landsberg MD
Clinical Professor of Medicine,
University of California San Diego School of Medicine.
Section Chief for Pulmonary & Critical Care Medicine,
Medical Director for Respiratory Therapy,
Veterans Administration Medical Center, La Jolla

Disclosures / Advertisement

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

Friend my book on Facebook:

Landsberg Manual



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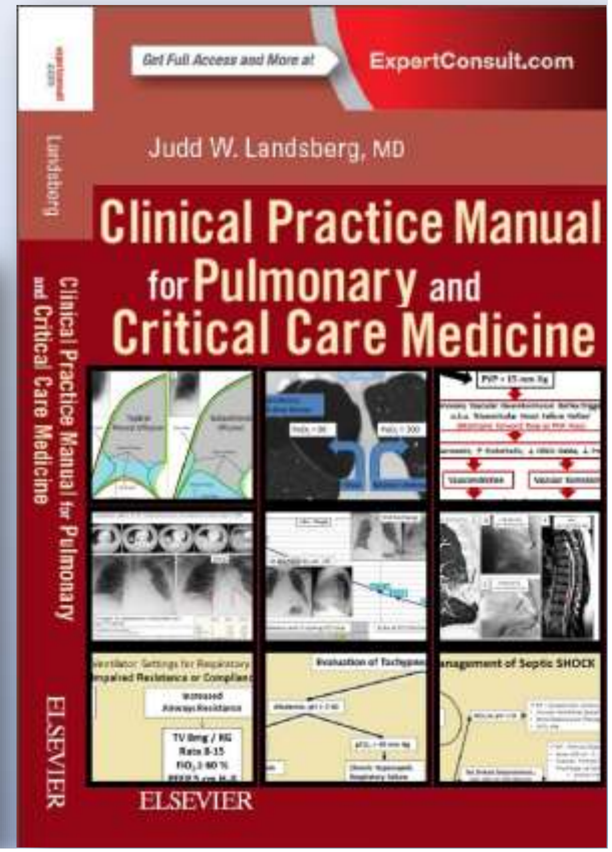
Approach to oxygenation, hypoxemia, and hypoxemic respiratory failure

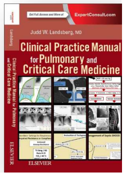
Common misconceptions and mistakes

- Hypoxemia is a significant cause of dyspnea
- A cutaneous O_2 sat ≥ 90% predicts adequate oxygenation and is the appropriate target for O_2 orders
- 100% O_2 suppresses respiratory drive in CO_2 retainers
- O_2 supplementation for patients with COPD is given to improve exercise tolerance
- Confusing failure of oxygen delivery to tissues, hypoxia (the job of the circulatory system) with hypoxemia, and failure to maintain an adequate Pa_{O_2} (the job of the respiratory system)



Major Mistakes and Misconceptions

- CONFUSING Hypoxia (low tissue oxygen levels) with Hypoxemia (low dissolved oxygen concentration in blood)
- Targeting too LOW an SaO_2 (e.g. > 92%), allowing mild hypoxemia (PaO_2 55-59 mm Hg) to go unnoticed and untreated
- Being reassured about oxygenation because dyspnea is either mild or absent
- Believing that a high FIO_2 and or PaO_2 suppresses ventilatory drive in chronic CO_2 retainers

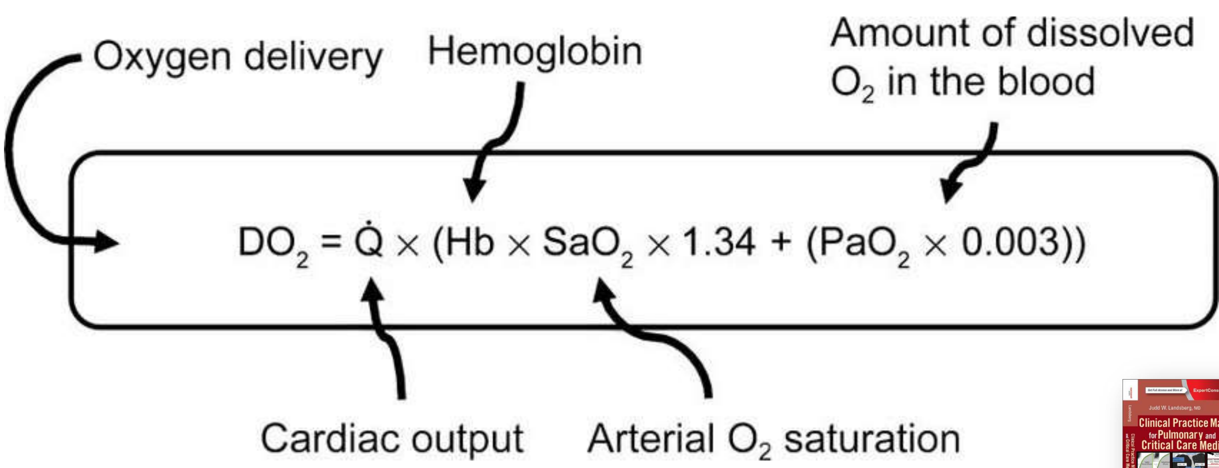


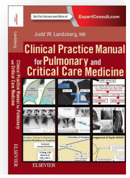
Hypoxia =

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 PaO_2 > 60 mm Hg does not meaningfully increase oxygen delivery to tissues or decrease lactate





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 ……

PaO_2 mm Hg

60

Mild Hypoxemia

55

Severe Hypoxemia

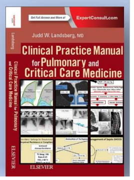
50

- **Confusion**
- **Agitation**
- **Sinus Tachycardia**
- **Tachypnea**

Dyspnea mild or absent

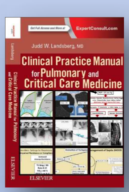
- **Extreme Dyspnea**
- **Lactate accumulation / resting tissue hypoxia**
- **PEA sinus bradycardic cardiac arrest**

Pts with Mild Hypoxemia are at risk for **sudden severe hypoxemia**

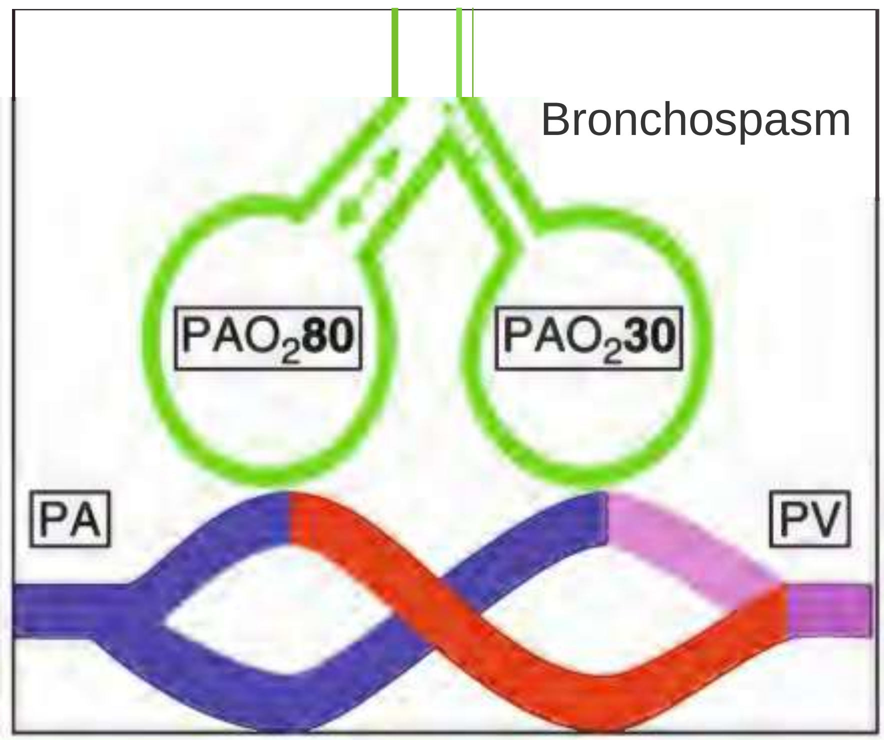


Assessing Oxygenation and Acute Hypoxemic Respiratory Failure

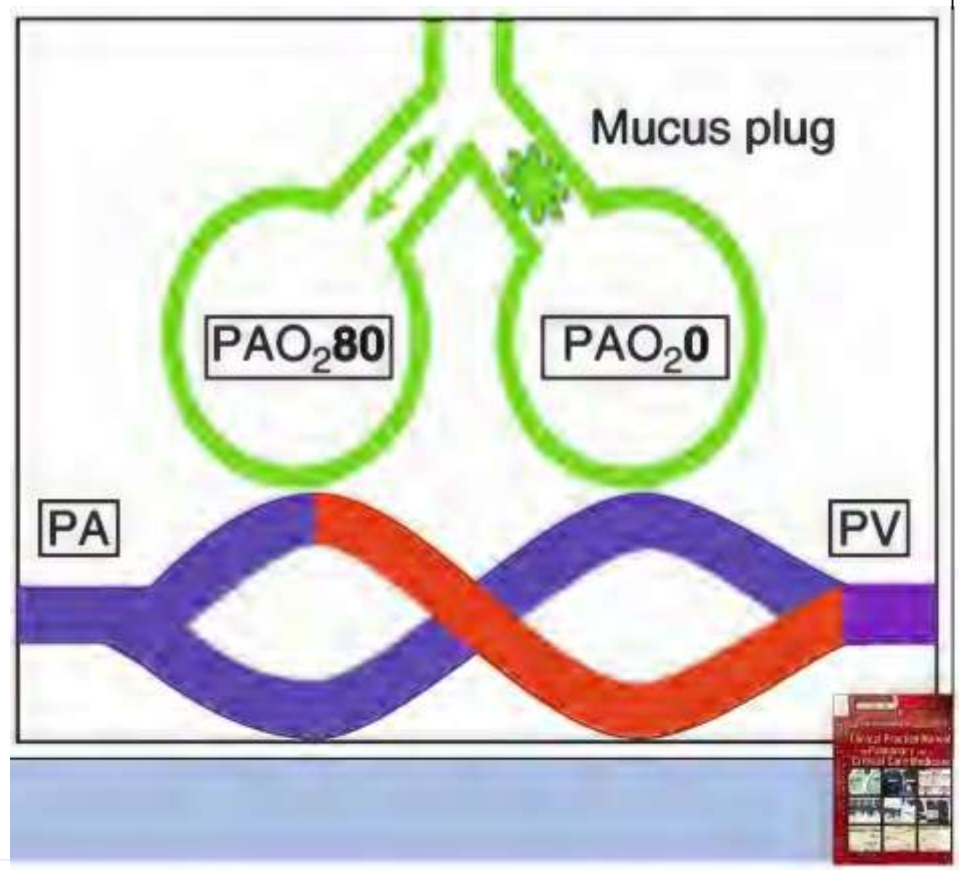
- **Normal Oxygenation** (at sea level)
  - **21% FiO_2** (room air)→ **PaO_2** of **75–100 mm Hg**
  - **100% FiO_2 → PaO_2 of ~ 660 mm Hg**
- **“VQ mismatch”**
  - Perfusion **Too High** relative to Ventilation = Low V/Q ratio
  - PaO_2 Increases with 100% FiO_2
- **Shunt Physiology**
  - Perfusion despite NO ventilation
  - PaO_2 remains low despite 100% FiO_2
    - PaO2 < 200 mm Hg on 100% implies shunt (physiologic MORE common then anatomic)
  - Treat by Increasing Mean Airway Pressure (best achieved with PEEP), recruits lung to increase PaO_2
- **Hypoxemic Respiratory Failure** *best* defined as a **PaO_2< 60 mm Hg,**
  - Occurs when deoxygenated blood mixes directly with oxygenated blood



LowV/Q



Physiologic shunt



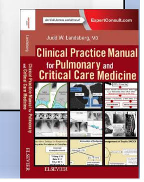
Symptoms of Acute Mild Hypoxemia
PaO_2 54-59 mm Hg

- Tachypnea (hypoxic hyperventilation reflex)
  - ↑ alveolar O_2 by ↓ alveolar CO_2,
  - ↑ 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

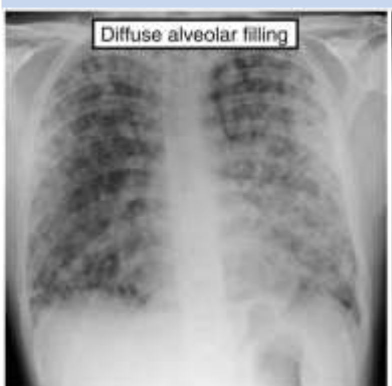


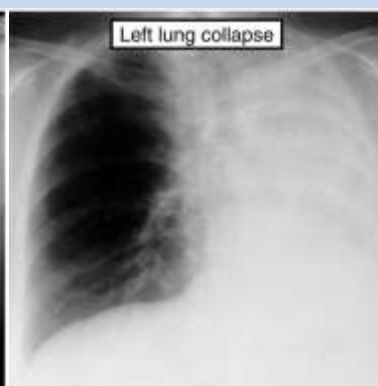
Symptoms of Severe Hypoxemia
$PaO_2 < 50$ mm Hg

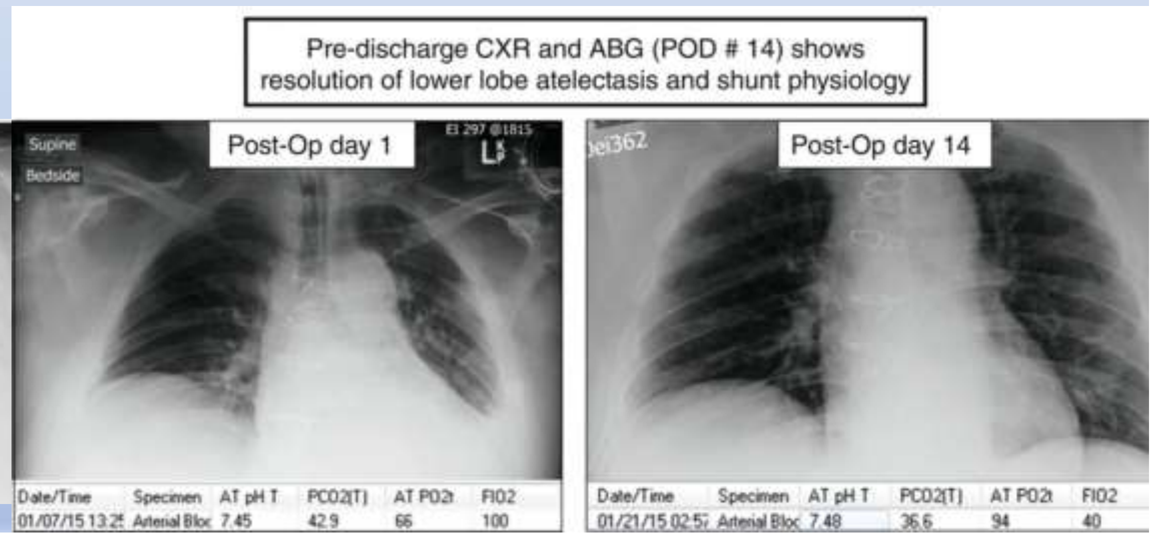
- Extreme Dyspnea, Lactate production
- Cardiac arrest from pulseless electrical activity
  - Sinus Bradycardia, precipitous HR drop **70 … 60 … 50 … 40 … 30 … 20**

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)





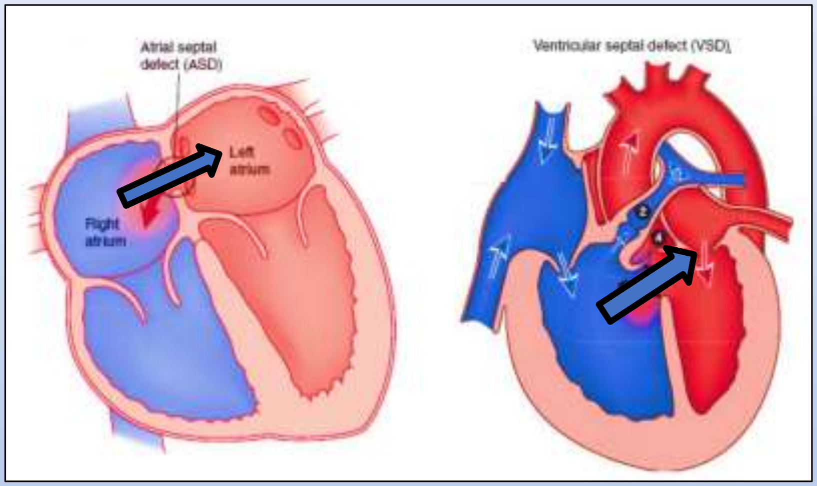


Anatomic Shunt (Right to Left shunting)

Intracardiac

more common then

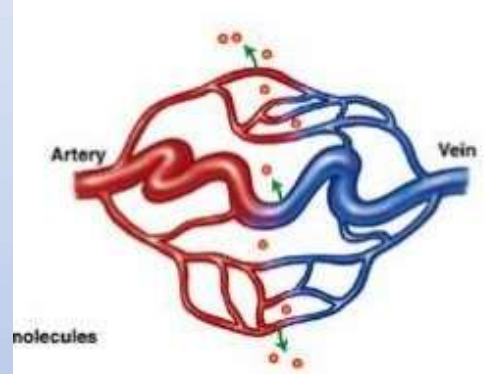
Intrapulmonary



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)



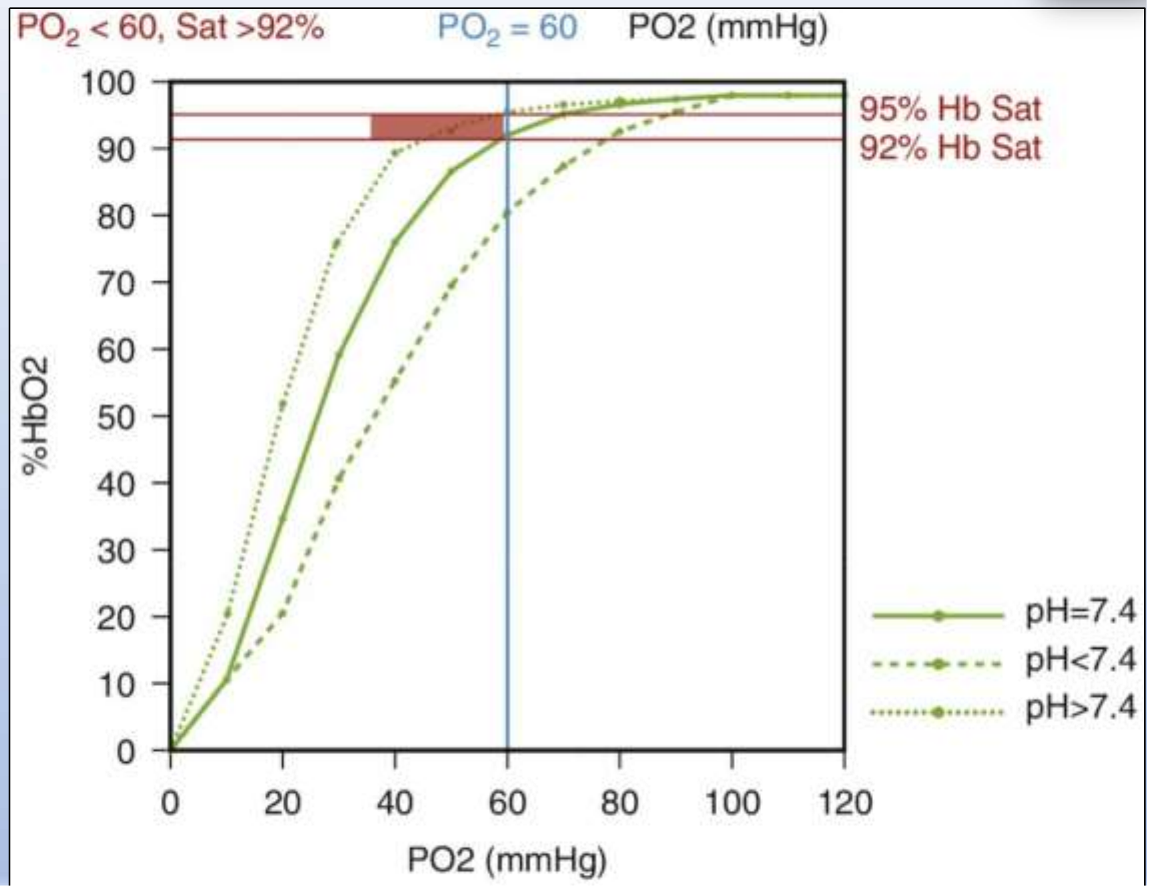
Screening for and Assessing Hypoxemia

- 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 PaO_2> 60 mm Hg
- Pulse oximetry **> 94%** (good wave form) = high probability of $PaO_2 \geq 60$ mm Hg

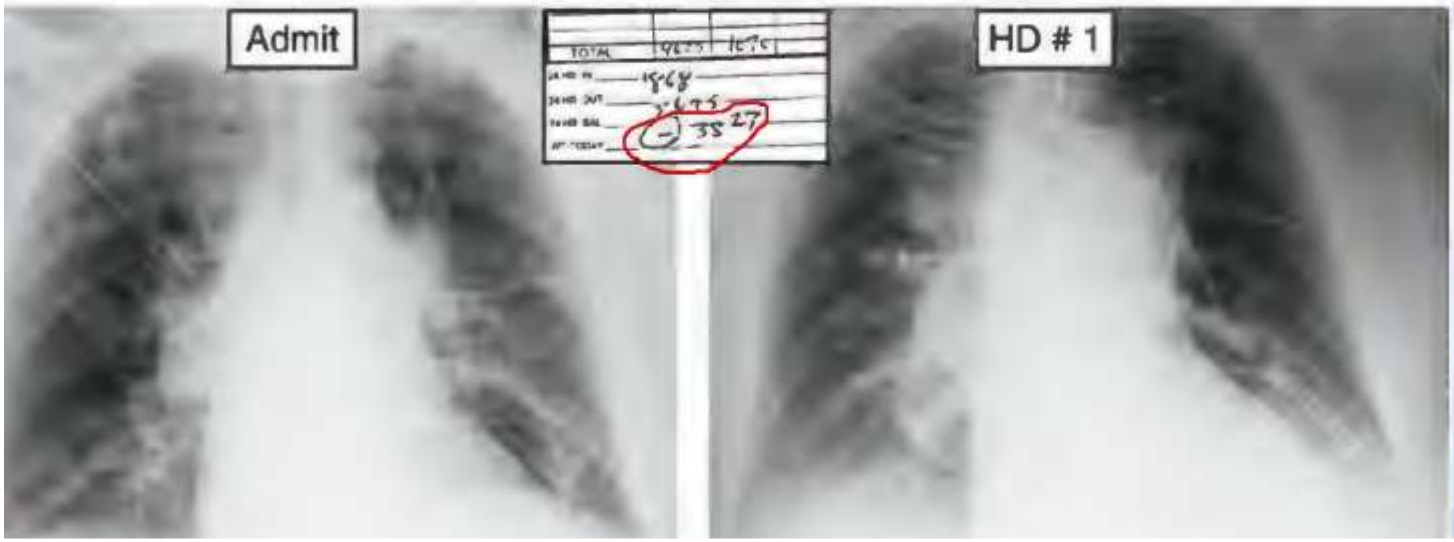
- **'> 92%'** = most common inpatient oxygen goal= **TOO LOW**
- Pulse oximeter > 92% (but < 95%) **may mask** a $PaO_2 < 60$ mm Hg because of alkalosis or error

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



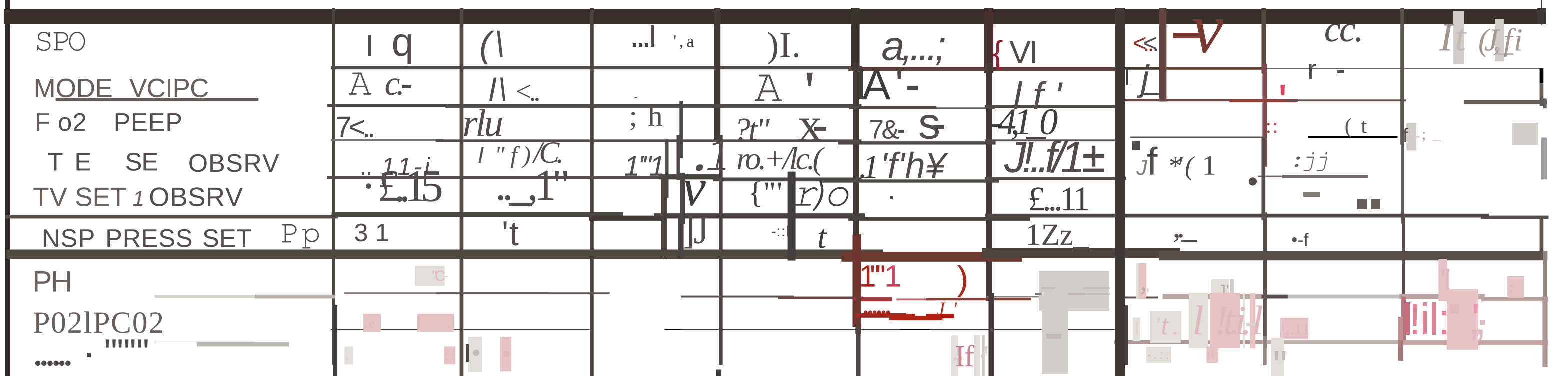
- Patient admitted for heart failure with a preserved ejection fraction (HFpEF)
- Intubated for increased work of breathing and hypoxemia
- Admit CXR with increased interstitial markings, small effusions
- Despite a - L negative fluid balance over the first 24 hours the PT suffers<£ oxygenatio
- CXR on HD # 1 shows worsening pulmonary edema:
  - f 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-160/8o-85, HR: 60- 90 sinus rhythm



Da:e/T 1118	Spec:men	FIO2	pH AT	PCO2 AT	PO2 AT	HCO3 AT	SO2 AT
04/291''' 03:15	Arterial Blood	80	7.39	52.9 H	83	32 H	96

Date/Time	Specimen	FiO2	pH AT	PCO2 AT	PO2 AT	HCO3 AT	SO2 AT
04/30/14 06:07	Arterial Bloo	60	7.47 H	49.5 H	55 L	36 H	91 L

- Inspection of the flow sheet shows the J in Fi0$_2$ to 60°/o 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 wave form
    - Cutaneous 0$_2$ sat 94°/o, calculated 0$_2$ sat 91°/o
  - Alkalosis shifting the H t r 0$_2$ dissociation curve



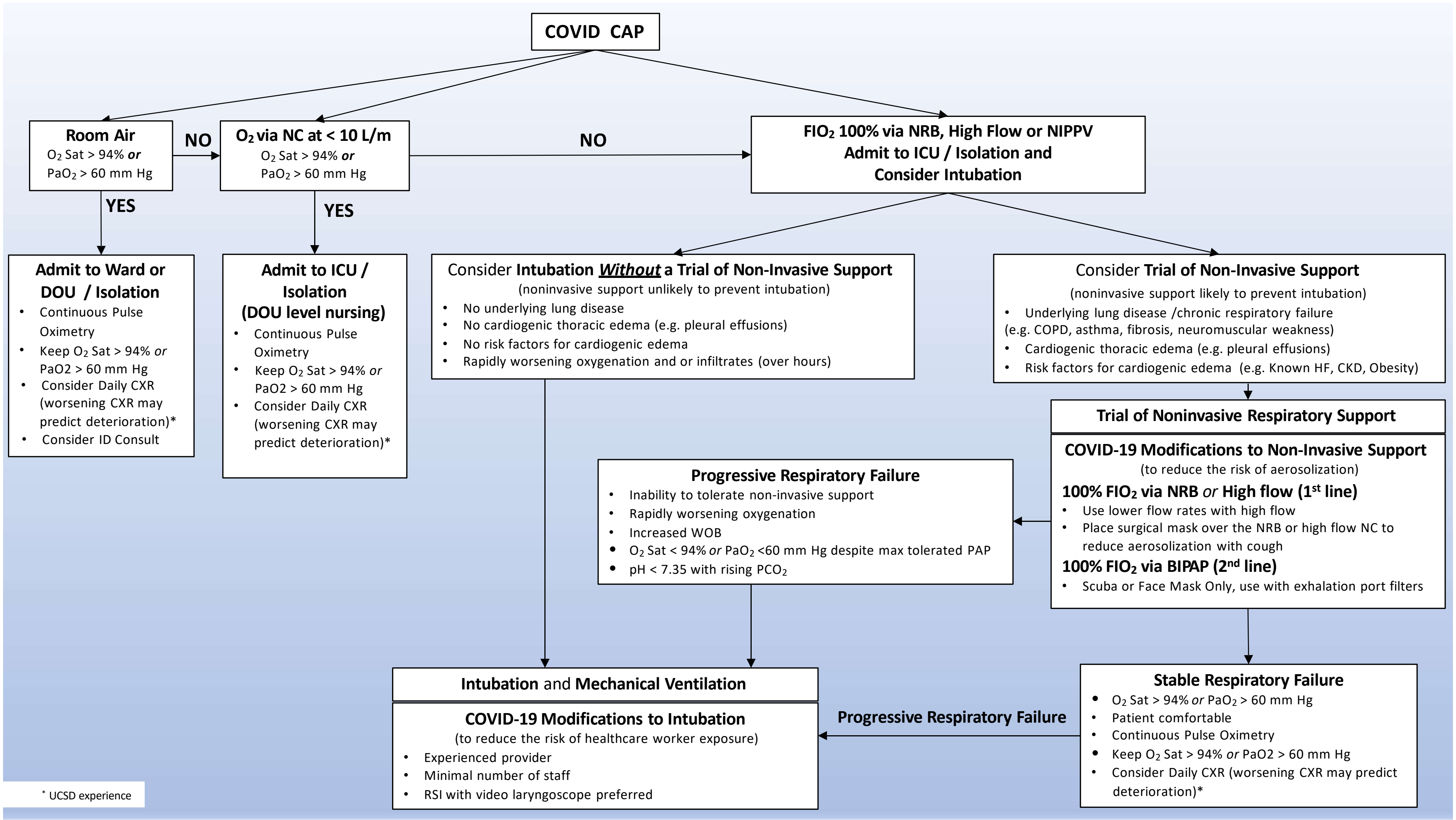
Date/Time	Specimen	FI02	IPHAT	IPC02AT	IP02AT	HCOJAT	IS02AT
04/30/14 06:07	Arteri l Bloo	60	(f 47}3)	49.5H	(59 L:)	36H	(91D

- 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

pH 7.46/PCO_2 33/PaO_2 60

- ↑FiO_2 (supplemental oxygen)
- Goal PaO_2 > 60 mm Hg (without hyperventilation)
- Target O_2 sat > 94% **OR** ensure PaO_2 > 60 mm Hg (ABG)
- Hypoxemia despite $O_2 \geq$ 10 L/min = Impending hypoxemic arrest → Mandates a trial of 100% FiO_2 and consideration for intubation in COVID-CAP patients
- **DO NOT** withhold 100% FiO_2 fearing CO_2 retention (does not suppress drive)


COVID CAP
Room Air
O_2 Sat > 94% *or*
PaO_2 > 60 mm Hg
NO
O_2 via NC at < 10 L/m
O_2 Sat > 94% *or*
PaO_2 > 60 mm Hg
NO
FIO_2 100% via NRB, High Flow or NIPPV
Admit to ICU / Isolation and
Consider Intubation
YES
YES
Admit to Ward or DOU / Isolation
• Continuous Pulse Oximetry
• Keep O_2 Sat > 94% *or* PaO2 > 60 mm Hg
• Consider Daily CXR (worsening CXR may predict deterioration)*
• Consider ID Consult
Admit to ICU / Isolation (DOU level nursing)
• Continuous Pulse Oximetry
• Keep O_2 Sat > 94% *or* PaO2 > 60 mm Hg
• Consider Daily CXR (worsening CXR may predict deterioration)*
Consider **Intubation *Without* a Trial of Non-Invasive Support**
(noninvasive support unlikely to prevent intubation)
• No underlying lung disease
• No cardiogenic thoracic edema (e.g. pleural effusions)
• No risk factors for cardiogenic edema
• Rapidly worsening oxygenation and or infiltrates (over hours)
Consider **Trial of Non-Invasive Support**
(noninvasive support likely to prevent intubation)
• Underlying lung disease /chronic respiratory failure (e.g. COPD, asthma, fibrosis, neuromuscular weakness)
• Cardiogenic thoracic edema (e.g. pleural effusions)
• Risk factors for cardiogenic edema (e.g. Known HF, CKD, Obesity)
Trial of Noninvasive Respiratory Support
COVID-19 Modifications to Non-Invasive Support
(to reduce the risk of aerosolization)
100% FIO_2 via NRB *or* High flow (1st line)
• Use lower flow rates with high flow
• Place surgical mask over the NRB or high flow NC to reduce aerosolization with cough
100% FIO_2 via BIPAP (2nd line)
• Scuba or Face Mask Only, use with exhalation port filters
Progressive Respiratory Failure
• Inability to tolerate non-invasive support
• Rapidly worsening oxygenation
• Increased WOB
• O_2 Sat < 94% *or* PaO_2 <60 mm Hg despite max tolerated PAP
• pH < 7.35 with rising PCO_2
Intubation and **Mechanical Ventilation**
COVID-19 Modifications to Intubation
(to reduce the risk of healthcare worker exposure)
• Experienced provider
• Minimal number of staff
• RSI with video laryngoscope preferred
Progressive Respiratory Failure
Stable Respiratory Failure
• O_2 Sat > 94% *or* PaO_2 > 60 mm Hg
• Patient comfortable
• Continuous Pulse Oximetry
• Keep O_2 Sat > 94% *or* PaO2 > 60 mm Hg
• Consider Daily CXR (worsening CXR may predict deterioration)*
* UCSD experience

Providing 100% oxygen

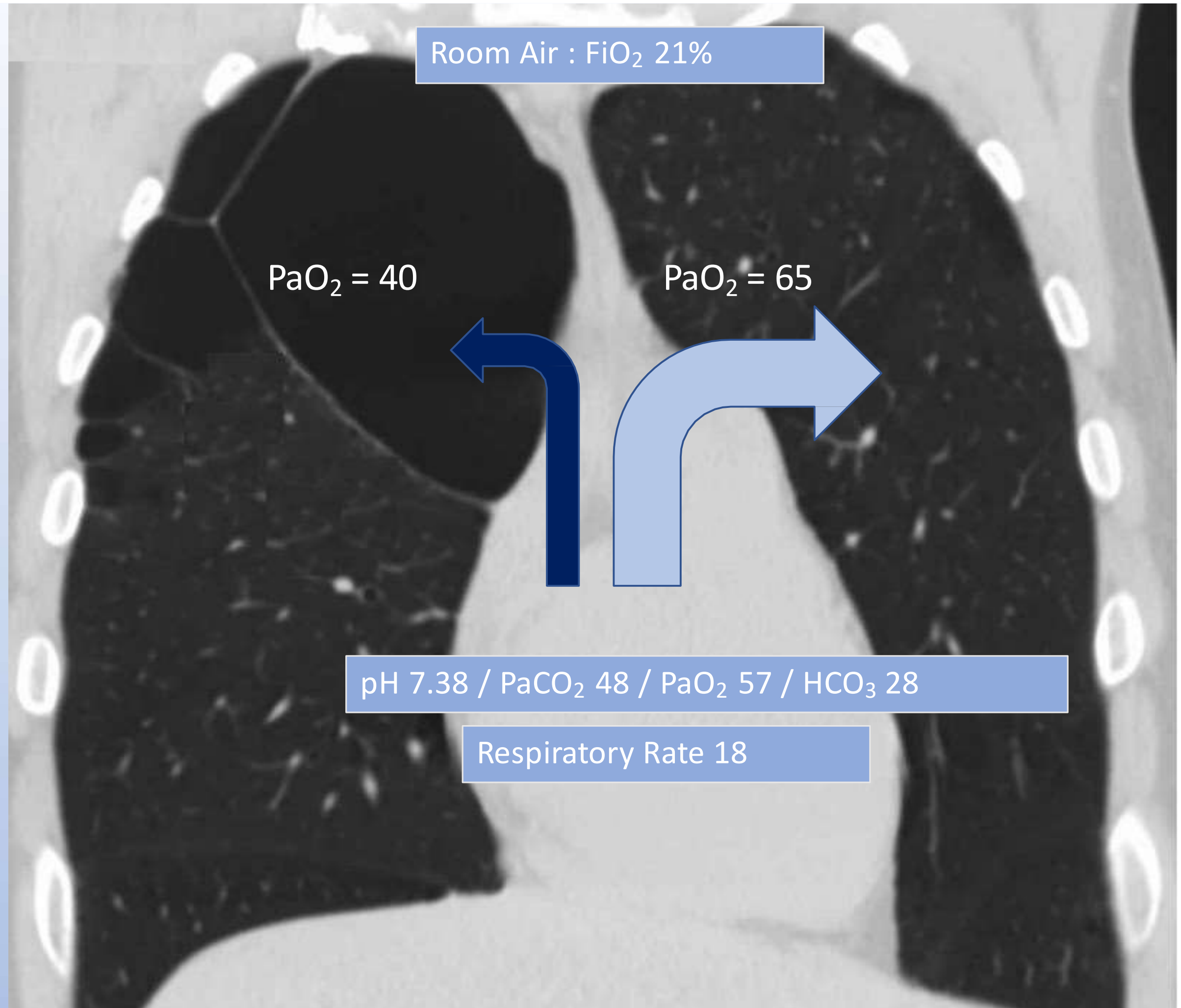
- Achieve 100% FiO_2 via a high-flow system or reservoir device

- Prevents entrainment of room air
  - High minute ventilation dilutes inspired O_2
- $PaO_2 < 60$ mm Hg (100% FiO_2) is life-threatening, mandating mechanical ventilation
  - BiPAP or Intubation

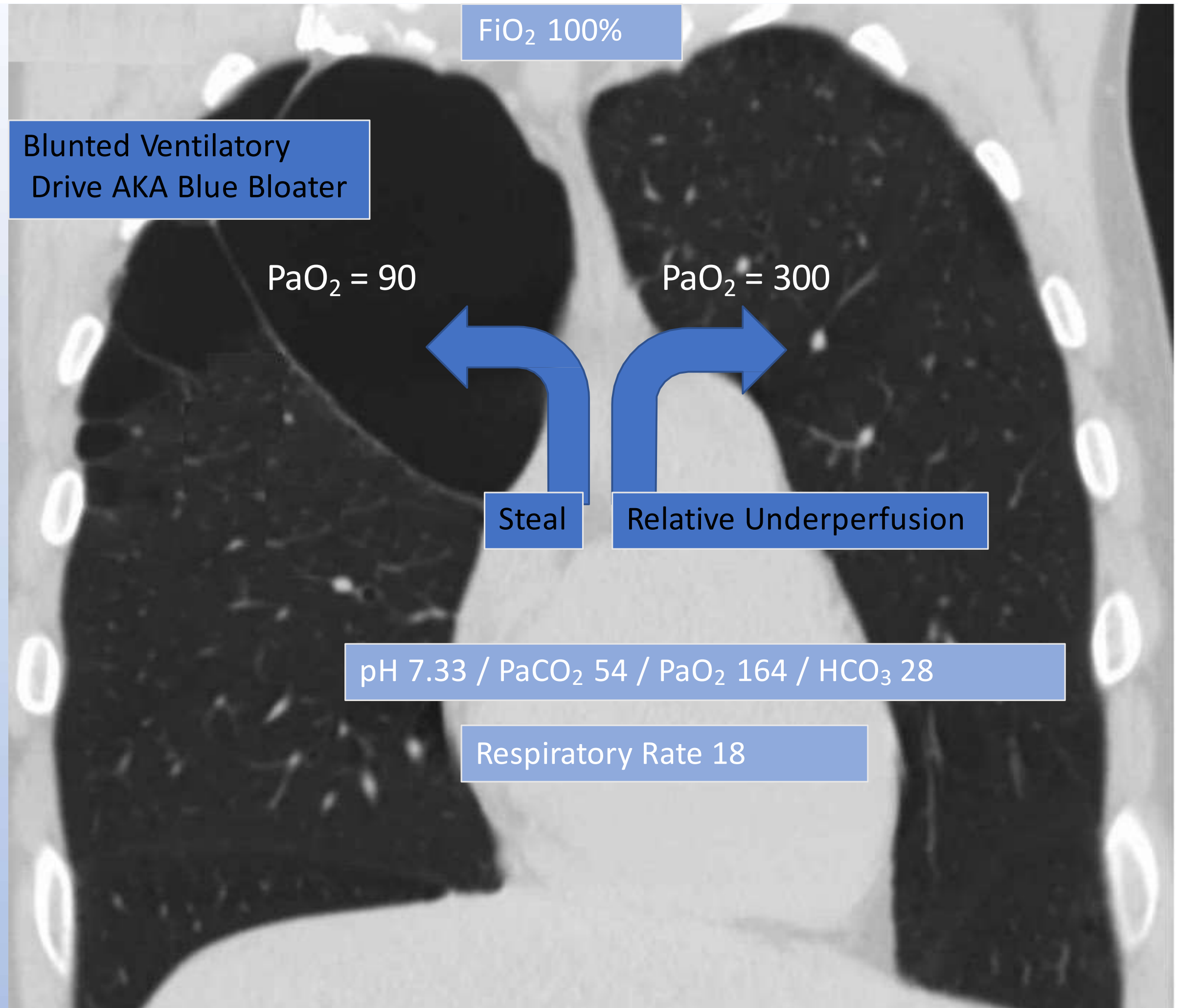
- ↑mean airway pressure
- Recruits atelectatic lung

CO_2 Retention & High FiO_2

- Hypoxemia NORMALLY stimulates ventilation = Hypoxic Hyperventilation Reflex
  - Alveolar Hypocarbia increases Alveolar O_2
  - May cause diaphragmatic fatigue (unlike all other etiologies of hyperventilation)
- In patients with severe parenchymal disease (baseline VQ mismatch), high FiO_2 & PaO_2 *occasionally* lead to increased PCO_2 (~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 FiO_2 & PaO_2 May impact drive

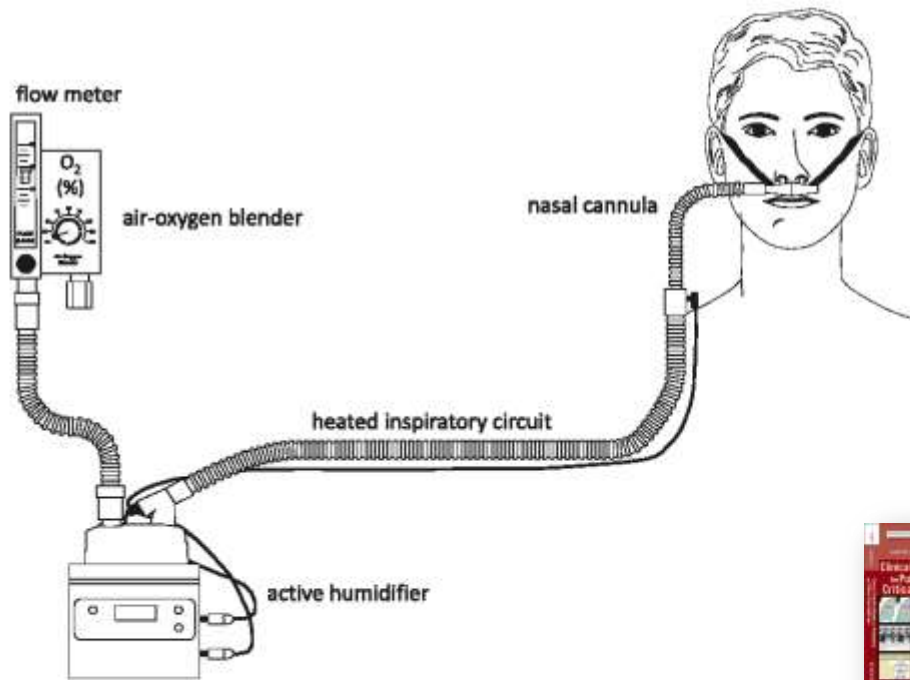

Room Air : FiO_2 21%
PaO_2 = 40
PaO_2 = 65
pH 7.38 / $PaCO_2$ 48 / PaO_2 57 / HCO_3 28
Respiratory Rate 18


Clinical Practice Manual for Pulmonary and Critical Care Medicine


FiO_2 100%
Blunted Ventilatory Drive AKA Blue Bloater
$PaO_2 = 90$
$PaO_2 = 300$
Steal
Relative Underperfusion
pH 7.33 / $PaCO_2$ 54 / PaO_2 164 / HCO_3 28
Respiratory Rate 18

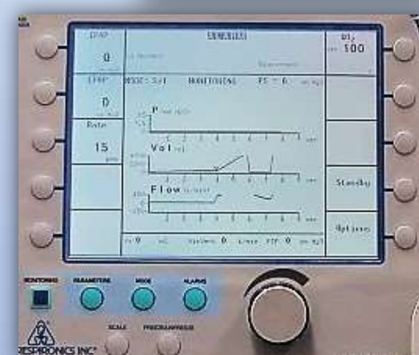
High Flow

- 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

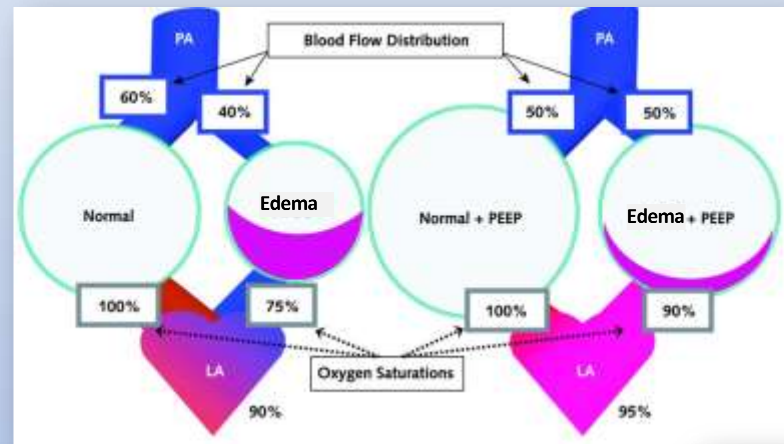
- PTs in acute hypoxemic respiratory failure deserve a trial of BiPAP before intubation, if they are:
  - Arousable
  - Able to wear a mask (eg, no facial or scalp wounds)
  - Not requiring continual oral clearance (eg, emesis, copious pulmonary secretions, massive hemoptysis)
- BIPAP provides differential inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP)
- Must Set IPAP, EPAP (aka PEEP) and FIO_2
  - 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 H_20**
  - **Increasing IPAP** can ↓ WOB and ↑ O_2 (↑ mean Airway pressure)
  - Maximum tolerated / deliverable IPAP 20-25 cm H_20
- **Minimum EPAP** (aka PEEP) = **5 cm H_2O** (physiologic)

  Increasing EPAP (a.k.a. PEEP) may
  - ↑ EPAP can redistribute fluid to the edges of the alveolar space and ↑ O_2
  - Aid in recruitment
  - High EPAP is uncomfortable for patients
  - EPAP should be increased slowly (increments of 2 cm H_2O) to avoid precipitating intolerance



End of slide deck presented on 4/7/2020

Further / Additional slides on mechanical ventilation included below

INmAnON OF BIPAP FOR ACUTE HYPERCAPNIC OR HYPOXEMIC RESPIRATORY FAILURE

Screen for conlnllncllcadon •
0 OtiiiRiatlon (111arousable)
0 lnabillly to - a mask (wounds)
0 Need lor oonlinUII cnl clearance:
- Massive llemCJil(ysls
- Copioua pulmonary IIIICAIliona
- Ongoing emesis

Start BIPAP
IPAP 15em Hp, EPAP 5 em lfaO, FI02 • 1

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Tolerant

Intolerant

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lnl..-aeda - a n t of tnetlllng on BIPAP
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ObjeciiYt dlla: TV,MV,O,. . %

Improved

No ctw1ga o r -

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P A P b J H c m H_2O*
(toa mu ol20-25 em Hp. or intolerance)
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tnc,_EPAPZ- H_2O*
(toa mu oi1G-15 em Hp, or intolerance)

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Mechanical Ventilation for Acute Respiratory Failure

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 H_2O)**
  - **Plateau pressures** (P_{plat}) are high **(> 30 cm H_2O)**

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 pCO_2
- **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)



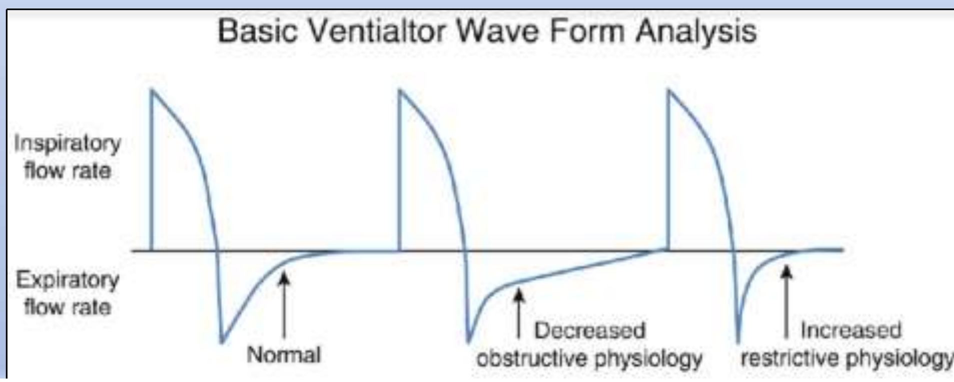

TABLE 19.1 Safe Tidal Volumes Based on Height (Ideal Body Weight)

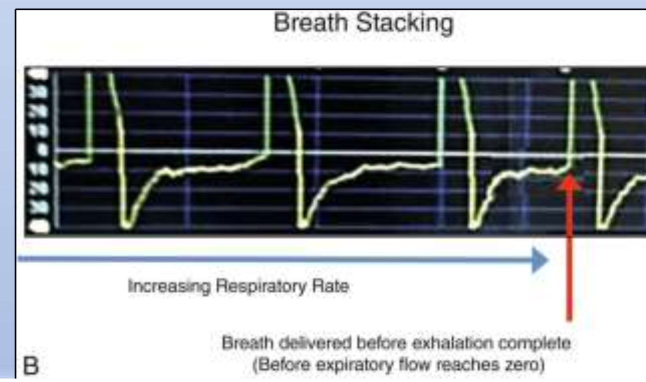
Height (ft)	Inches	Centimeters	Female Tidal Volume (mL) 6 mg/kg	Female Tidal Volume (mL) 8 mg/kg	Male Tidal Volume (mL) 6 mg/kg	Male Tidal Volume (mL) 8 mg/kg
5'	60	152	270	360	300	400
5'3	63	160	310	420	340	455
5'6	66	168	350	480	380	510
5'9	69	175	400	530	420	560
6'	72	182	430	580	460	610
6'6	78	198	520	690	550	730
6'10	82	208	575	770	600	800




Permitting Hypercapnia and Acidosis to Maintain LOW Lung Volumes

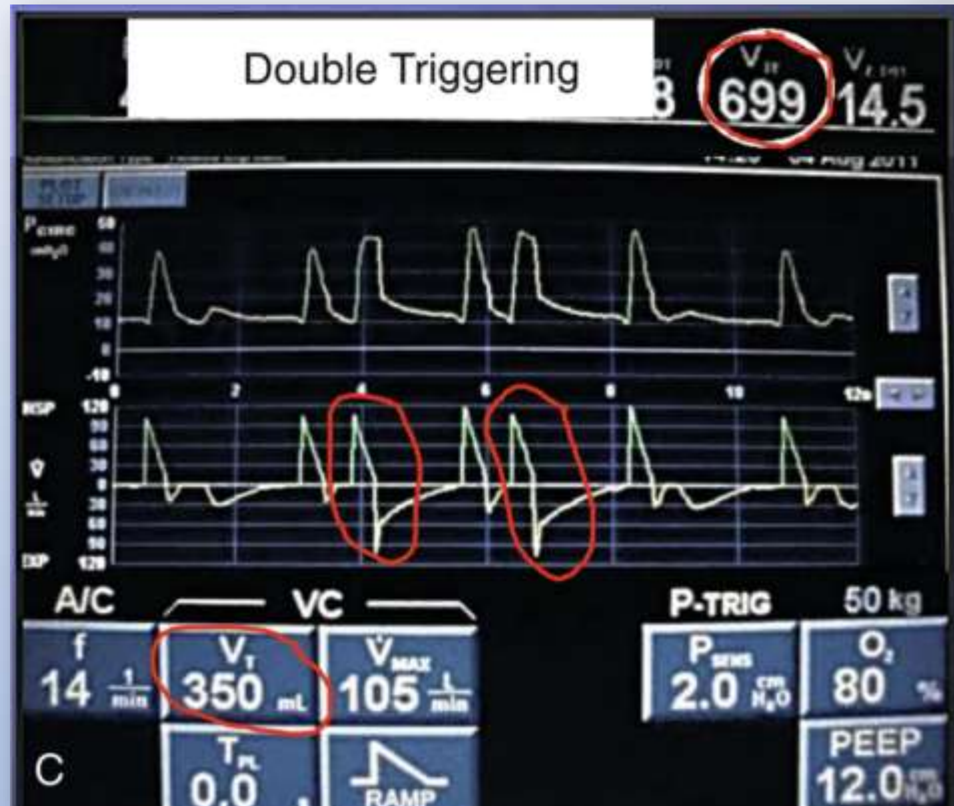
- Permissive hypercapnia = tolerating hemodynamically asymptomatic respiratory acidosis (Often pH values < 7.25)
- Prioritizing low lung volumes means that minute ventilation is increased via ↑respiratory rate (RR), **NOT** tidal volume
  - Max RR (breaths/min) is limited by exhalation time, varies widely 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





Low lung volumes and Hypercapnia Are Uncomfortable (acidosis and air hunger)

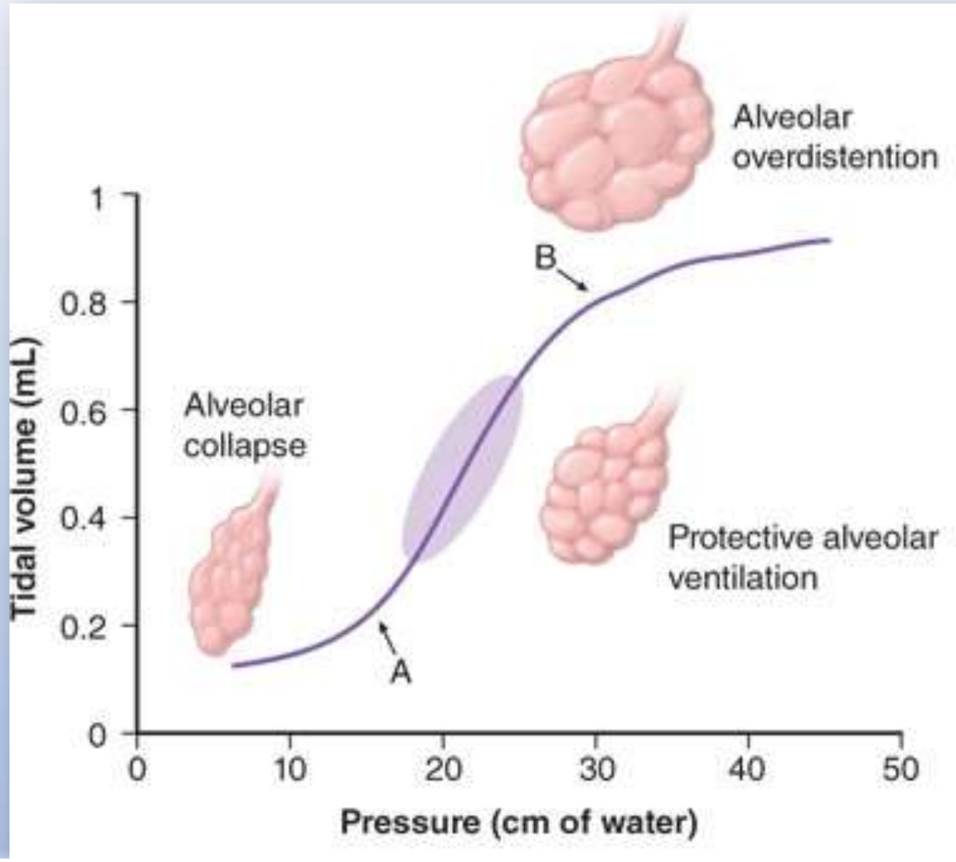
- Lung-protective ventilation often requires deep **sedation** and **paralysis** to avoid **dyssynchrony**, which can:
  - **Prevent effective ventilation** via high airway pressures from patient struggling
  - **Cause breath stacking**
  - **Cause double triggering**
    - PT triggers a breath immediately 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 FiO_2 to 100% can improve hypoxemia from low V/Q physiology
- Shunt physiology, common with extremely poor compliance (eg, ARDS), requires an FiO_2 of 100% and high PEEP to attain a PaO_2 > 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 H_2O 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**, **FiO_2 100%, PEEP 5-10 cm H_2O**
(Consult ID and consider clinical trial eligibility, off label / new antivirals, and immune mediated therapies)

Ventilator Assessment, Obtain:
- Peak Inspiratory Pressure (**PIP**)
- Plateau Pressure (P_{plat})
- ABG (Consider A-line)

Important Alarm = PIP
- **PIP** alarm set > 40 cm H_2O

PIP > 35 cm H_2O OR P_{plat} > 30 cm H_2O
→ **↓ Tidal Volume** (by 50-100 ml)

pH
- **pH < 7.35*** → **↑ Rate** to maximum ǂ (by 2-4 bpm) BEFORE **↑Tidal Volume up to 8 ml/kg)** (by 50-100 ml)
- **pH > 7.44** → **↓Tidal Volume (6 ml /kg)** BEFORE **↓ Rate**

PaO_2
- **PaO_2< 60 mm Hg** → **↑FiO_2 to 100 %** AND **↑ PEEP** (by 2-5 cm H_2O)
- **PaO_2> 80 mm Hg** → **↓ FiO_2 ≤ 60 %** THEN **↓ PEEP** (by 2-5 cm H_2O) ӝ

***** Minimize acidosis to avoid dyspnea, patient discomfort, and increased sedation needs
ǂ **Maximum Rate** = As fast as possible without breath stacking (typically 12-15 bpm for Obstructive Disease, 25-35 bpm for ARDS)
ӝ In ARDS **wean PEEP slowly** (i.e. decrease by 2-5 cm H_2O q 12-24 hrs) to avoid derecruitment, In Cardiogenic edema PEEP may be weaned more quickly

Trouble shooting High Peak Inspiratory Pressure

- Examine the patient (check for dyssynchrony, agitation, breath stacking)
- Obtain peek and plateau pressures
  - High peak pressures ***WITH*** low plateau pressures (Δ > 15) = increased resistance
  - Commonly seen with occluded ET tube or airway (e.g. biting, mucus plug, blood clot, bio film), or bronchospasm
  - High peak ***AND*** plateau pressures (Δ < 10) = worsening compliance
  - Commonly seen with dyssynchrony, extremely high auto PEEP (aka breath stacked), edema (HF or ARDS), collapse, pneumothorax or intrusive 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 proregression to severe ARDS (see mechanical ventilation of severe ARDS)

Improving Oxygenation in Mild to Moderte ARDS on Mechanical Ventilation

- **↑ PaO_2 by ↑ FIO_2 to 100% and then ↑ positive end expiratory pressure (PEEP)**
- **↑ PEEP 1st maneuver for ARDS and hypoxemia despite 100% FiO_2**
  - 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)
  - 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 H_2O 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. Norepi, Dopamine, Dobutamine)
  - ↓BP from high PEEP rapidly responds to ↓ PEEP (making PEEP up-titration safe)
  - Obese patients often need a PEEP > 20 cm H_2O 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 cmH_2O, PEEP 10-20 cmH_2O, Rate 20-30 bpm, FIO_2 : 100%

(Consult ID and consider clinical trial eligibility, off label / new antivirals, and immune mediated therapies)

Important Alarms

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

Obtain

- **Tidal Volume (TV)**
- **Plateau Pressure (P_{plat})**
- **Arterial Blood Gas (ABG)**

TV > 6 mg /kg OR **P_{plat} > 30 cm H_20** → **↓ Driving Pressure**

pH < 7.20 -7.25* → **↑ Rate** (to maximum ǂ) **BEFORE** **↑Driving pressure ⁂**

PaO_2< 60 mm Hg → **↑ PEEP** (to maximum †)

ǂ **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 refectory Hypotension)

† **Maximum PEEP =** As high a PEEP as possible without hypotension (impaired Venous return), typically 15–20 cm H_2O

⁂ **Keep Peek Air Way Pressure** (Driving Pressure + PEEP) **< 40-50 cm H_2O** (to avoid pneumothorax)

Trouble shooting Low exhaled TV / Low minute ventilation alarm

- Examine the patient (check for dyssynchrony, agitation, breath stacking) – ensure paralysis
- Commonly seen with dyssynchrony, extremely high auto PEEP (aka breath stacked), edema (HF or ARDS), collapse, pneumothorax or intrusive abdominal 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)**
- **↑ PaO_2 by ↑ positive end expiratory pressure (PEEP)**
- **↑ PEEP 1st maneuver for ARDS and hypoxemia despite 100% FiO_2**
  - 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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  - PEEP > 12 H_2O may ↓ venous return, CO and BP (worse in RV dysfunction and hypovolemia)
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    - 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
- **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