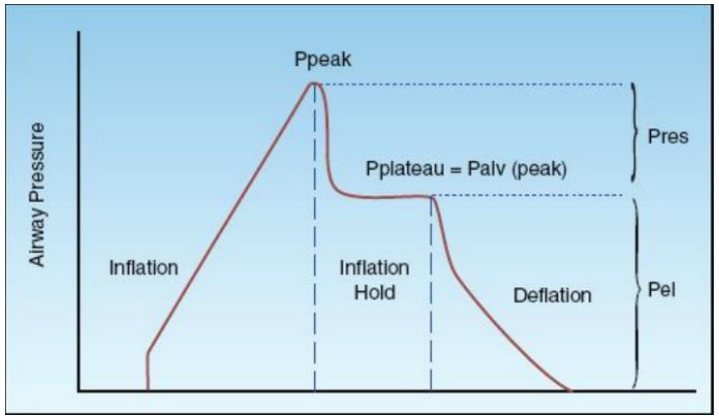
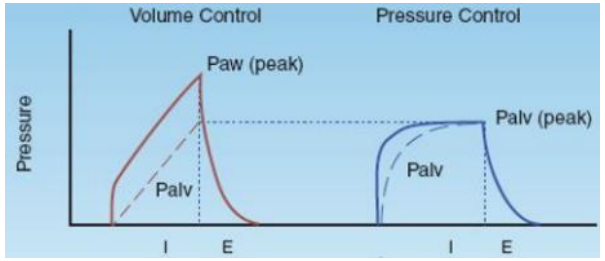
Basics of Mechanical Ventilation



Airway pressure profile for a constant-flow, volume-controlled lung inflation with a brief end-inspiratory occlusion (inflation-hold). **Ppeak** is the peak airway pressure, **Pplateau** is the end-inspiratory occlusion pressure, **Palv (peak)** is the peak alveolar pressure at end-inspiration, **Pres** is the pressure attributed to airway resistance, and **Pel** is the pressure attributed to the elastic recoil force of the lungs and chest wall



Pressure and changes during a single ventilator breath using volume control and pressure control methods of lung inflation at equivalent tidal volumes. Changes in airway pressure (Paw) are indicated by the solid lines, and changes in alveolar pressure (Palv) are indicated by the dashed lines. I = inspiration, E = expiration.

Volume control

- Due to airflow at the end of inspiration, the peak pressure in the airways (Paw) is greater than the peak pressure in the alveoli (Palv)
- The difference (Paw – Palv) is the pressure dissipated by the resistance to flow in the airways.
- The peak alveolar pressure is a reflection of the alveolar volume at the end of lung inflation

Advantages:

- Constant tidal volume despite changes in lung compliance and airway resistance

Disadvantages

- Airway pressures including alveolar pressures may be higher with decreased compliance of lungs due to constant TV

• Volume-targeted: vent delivers a set tidal volume, pressure depends on airway resistance and compliance. Patient remains at risk for barotraumas / volutrauma from high pressures.

Pressure control

- The desired inflation pressure is preselected, and a decelerating inspiratory flow rate provides high flows at the onset of the lung inflation, to attain the desired inflation pressure quickly
- Since there is no airflow at the end of inspiration, the end-inspiratory airway pressure is equivalent to the peak alveolar pressure

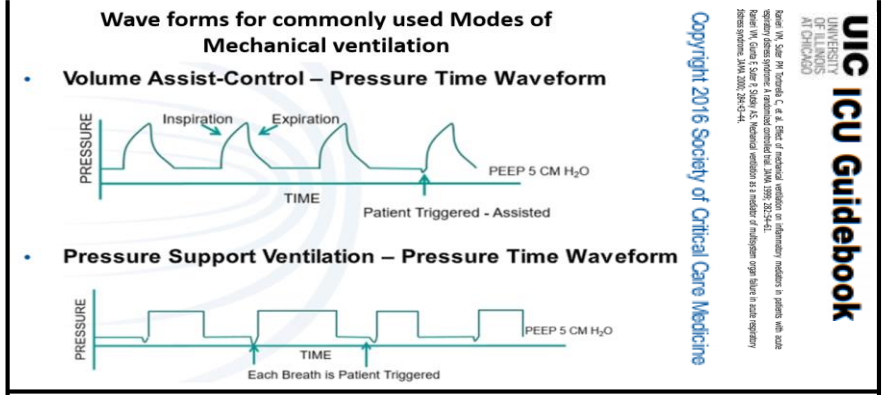
Advantages:

- Major benefit of PCV is the ability to control the peak alveolar pressure, which is the pressure most closely related to the risk of alveolar overdistension and ventilator-induced lung injury

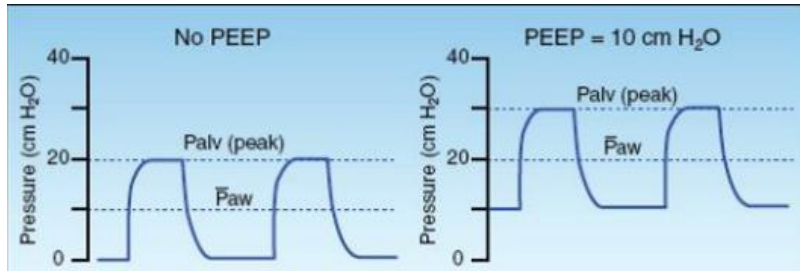
Disadvantages

- Decrease in alveolar volume (and hence ventilation) that occurs when there is an increase in airway resistance or a decrease in lung compliance.

• Pressure-targeted: vent delivers volume until a set pressure is achieved. Now, tidal volume is dependent on airway resistance and compliance. Patient remains at risk for low tidal volumes and inadequate minute ventilation.



Positive End Expiratory Pressure



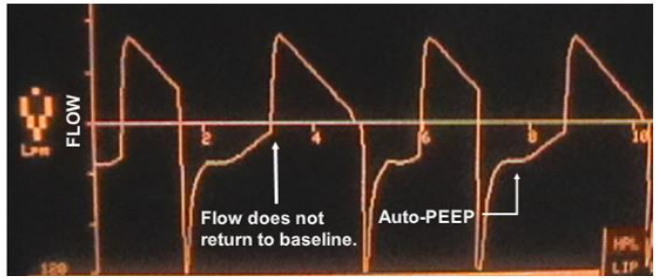
The change in peak alveolar pressure determines the influence of PEEP on alveolar ventilation (hence arterial oxygenation)

The change in mean airway pressure determines the influence of PEEP on cardiac output

Progressive narrowing of the airways during expiration results in collapse of distal airspaces (small airways and alveoli) at the end of expiration.

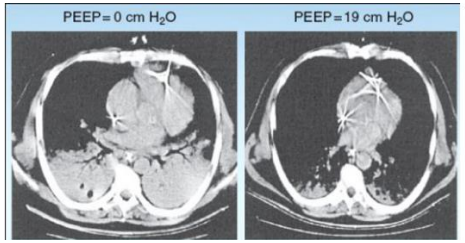
This has two adverse consequences: (a) impaired gas exchange from atelectasis (b) atelectrauma from repetitive closing and opening of the distal airspaces

PEEP is applied to prevent this collapse and reopen distal airspaces that are persistently collapsed

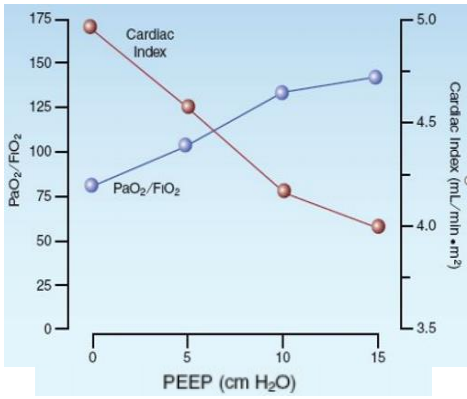


Auto-PEEP can occur when patient has inadequate time to exhale before next breath is delivered

- Consequences
  - ↑Alveolar pressures
  - Hypotension
  - Worsened oxygenation
- Interventions to decrease auto-PEEP
  - ↓Respiratory rate
  - ↓Tidal volume
  - ↑Flow rate



CT images from a patient with ARDS showing the influence of PEEP on lung aeration (alveolar recruitment)



The opposing effects of positive end-expiratory pressure (PEEP) on arterial oxygenation (PaO2 /FIO2) and cardiac index in patients with ARDS

Troubleshooting

• **Low pO2** = **oxygenation issue** = increase FiO2, increase PEEP (to recruit more alveoli).

• **High pCO2** = **ventilation issue** = Increase Minute Ventilation by increasing TV or rate (suction, bronchodilators)

High Peak pressures & High Plateau Pressures (non-compliant lungs)

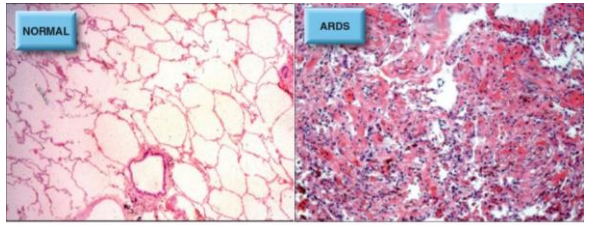
• Pulmonary edema • Worsening consolidation • ARDS • Atelectasis • Mainstem intubation • Tension PTX • Decreased chest wall compliance

High peak pressure low & normal plateau pressure (airway problem)

• Bronchospasm • Mucous plug • Secretions • Obstructed tubing • Patient biting tube

M. Daniyal Hashmi, MD

ARDS - overview

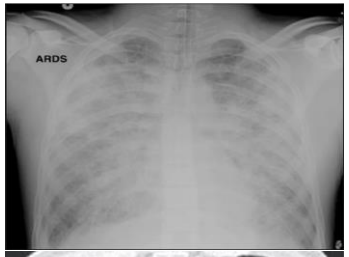


Abraham E. Neutrophils and acute lung injury. Crit Care Med 2003; 31(Suppl): S195–S199

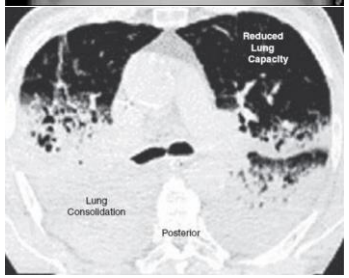
Microscopic images of a normal lung and a lung in the advanced stages of ARDS, which shows a dense infiltration of leukocytes and proteinaceous material that fills and obliterates the normal architecture of the lungs.

Table 2. Risk Factors for ARDS.

Direct lung-injury risk factors
Pneumonia (bacterial, viral, fungal, or opportunistic)*
Aspiration of gastric contents*
Pulmonary contusion
Inhalation injury
Near drowning
Indirect lung-injury risk factors
Sepsis (nonpulmonary source)*
Nonthoracic trauma or hemorrhagic shock
Pancreatitis
Major burn injury
Drug overdose
Transfusion of blood products
Cardiopulmonary bypass
Reperfusion edema after lung transplantation or embolectomy



Portable chest x-ray showing the classic radiographic appearance of ARDS. The infiltrate has a finely granular or "ground glass" appearance, and is evenly distributed throughout both lungs, with a relative sparing of the lung bases. There is no evidence of a pleural effusion.



Computed tomographic image of lung slices in the region of the hilum from a patient with ARDS. The lung consolidation is confined to the posterior lung regions, which are the dependent regions in the supine position. The uninvolved lung in the anterior one-third of the thorax represents the functional portion of the lung.

Acute Respiratory Distress Syndrome

The Berlin Definition

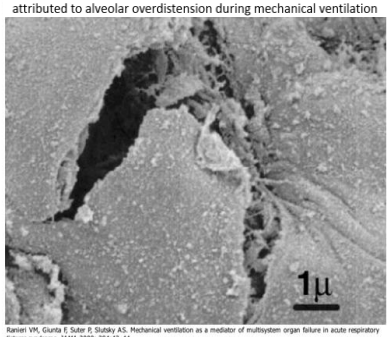
	ACUTE RESPIRATORY DISTRESS SYNDROME		
Timing	Within 1 week of a known clinical insult of new/worsening respiratory symptoms		
Chest Imaging [a]	Bilateral opacities – not fully explained by effusions, lobar/lung collapse, or nodules		
Origin of Edema	Respiratory failure not fully explained by cardiac failure or fluid overload; Need objective assessment (e.g., echocardiography) to exclude hydrostatic edema if no risk factor present		
	Mild	Moderate	Severe
Oxygenation [b]	$200 < PaO_2/FiO_2 \leq 300$ with PEEP or CPAP ≥ 5 cmH_2O [c]	$100 < PaO_2/FiO_2 \leq 200$ with PEEP ≥ 5 cmH_2O	$PaO_2/FiO_2 \leq 100$ with PEEP ≥ 5 cmH_2O

Ventilator induced Lung injury

Excessive inflation of the distal airspaces produce stress fractures in the alveolar capillary interface, and this leads to infiltration of the lung parenchyma and distal airspaces with an inflammatory exudate

- Lung injury that is volume-related is called **volutrauma**
- Pressure-related lung injury is called **barotrauma**
- The decrease in lung distensibility in ARDS can result in the collapse of small airways at the end of expiration. When this occurs, mechanical ventilation can be associated with cyclic opening and closing of small airways, and this process can be a source of lung injury called **atelectrauma**

Electron micrographs showing a tear at the alveolar-capillary interface attributed to alveolar overdistension during mechanical ventilation



Ranieri VM, Giunta F, Suter P, Slutsky AS. Mechanical ventilation as a mediator of multisystem organ failure in acute respiratory distress syndrome. JAMA 2000; 284:43–44.
Muscedere JG, Mullen JBM, Gan K, et al. Tidal ventilation at low airway pressures can augment lung injury. Am J Respir Crit Care Med 1994; 149:1327–1334.
Dreyfuss D, Saumon G. Ventilator-induced lung injury. Am J Respir Crit Care Med 1998; 157:294–323.
Gattinoni L, Protti A, Caironi P, Carlesso E. Ventilator-induced lung injury: the anatomical and physiological framework. Crit Care Med 2010; 38(Suppl):S539–S548.
Ranieri VM, Suter PM Tortorella C, et al. Effect of mechanical ventilation on inflammatory mediators in patients with acute respiratory distress syndrome: A randomized controlled trial. JAMA 1999; 282:54–61.

Positive End-Expiratory Pressure

- **Lung protective ventilation** employs a **PEEP of at least 5 cm H2O** to prevent the collapse of small airways at the end of expiration. The **goal is to reduce the risk of atelectrauma**.
- When a toxic level of inhaled oxygen (FIO2 >60%) is needed to maintain the **target SpO2 of 88–95%,** PEEP levels above 5 cmH2O can be used to improve arterial oxygenation and reduce the FIO2 to safer levels.
- It is important to emphasize that increases in PEEP can reduce the **cardiac output**, and if the goal of increasing PEEP is to maintain the same SpO2 at a lower FIO2 , the reduced cardiac output will **reduce the systemic O2 delivery.**

Bidani A, Tzouanakis AE, Cardenas VJ, Zwischenberger JB. Permissive hypercapnia in acute respiratory failure. JAMA 1994; 272:957–962.

Permissive hypercapnia

- The consequences of low tidal volume ventilation is a decrease in CO2 elimination in the lungs, which can result in hypercapnia and respiratory acidosis
- Because of the benefits of low volume ventilation, hypercapnia is allowed to persist as long as there is no evidence of harm

Non Ventilatory Management

- **Fluid management** avoiding a positive fluid balance will prevent unwanted fluid accumulation in the lungs, which could aggravate the respiratory insufficiency in ARDS.
- Clinical studies have shown that avoiding a positive fluid balance in patients with ARDS can **reduce the time on mechanical ventilation and can even reduce mortality**

The Acute Respiratory Distress Syndrome Network. Comparison of two fluid management strategies in acute lung injury. N Engl J Med 2006; 354:2564–2575.
Murphy CV, Schramm GE, Doherty JA, et al. The importance of fluid management in acute lung injury secondary to septic shock. Chest 2009; 136:102–109.

Protocol for Lung Protective Ventilation in ARDS

I. Tidal Volume Goal: V_T = 6 mL/kg (predicted body weight)

1. Calculate patient's predicted body weight (PBW):
   Males: PBW = 50 + [2.3 × (height in inches – 60)]
   Females: PBW = 45.5 + [2.3 × (height in inches – 60)]
2. Use volume-controlled ventilation and set initial tidal volume (V_T) to 8 mL/kg (PBW).
3. Set respiratory rate (RR) to match baseline minute ventilation, but not >35 bpm.
4. Set positive end-expiratory pressure (PEEP) at 5 cm H_2O.
5. Reduce V_T by 1 mL/kg every 1 to 2 hours until V_T = 6 mL/kg (PBW)
6. Adjust PEEP and FiO_2 to maintain SpO_2 of 88–95%.

II. Plateau Pressure Goal: PpI ≤30 cm H_2O:

1. If PpI >30 cm H_2O and V_T at 6 mL/kg, decrease V_T in 1 mL/kg increments until PpI falls to ≤30 cm H_2O or V_T reaches a minimum of 4 mL/kg.

III. pH Goal: pH = 7.30–7.45

1. If pH = 7.15–7.30, increase RR until pH >7.30, $PaCO_2$ <25 mm Hg, or RR = 35 bpm.
2. If pH <7.15, increase RR to 35 bpm. If pH remains <7.15, increase in V_T in 1 mL/kg increments until pH >7.15 (PpI target may be exceeded).
3. If pH >7.45, decrease RR, if possible.

The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. New Engl J Med 2000; 342:1301–1308.

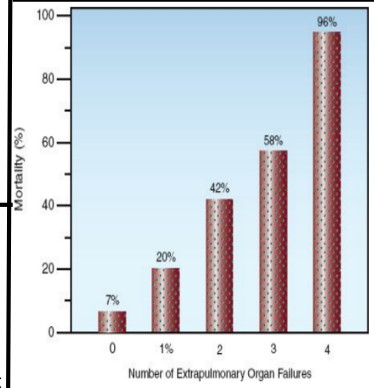
OXYGENATION GOAL:

- PaO2 55-80 mmHg or SpO2 88-95%
- Use a minimum PEEP of 5 cm H2O

Ventilator settings

Brand VM, Suter PM Tortorella C, et al. Effect of mechanical ventilation on inflammatory mediators in patients with acute respiratory distress syndrome: A randomized controlled trial. JAMA 1999; 282:54–61.
Ranieri VM, Giunta F, Suter P, Slutsky AS. Mechanical ventilation as a mediator of multisystem organ failure in acute respiratory distress syndrome. JAMA 2000; 284:43–44.

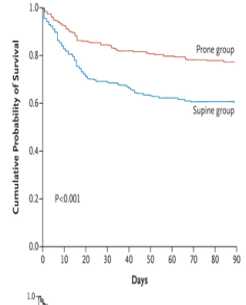
UIC ICU Guidebook

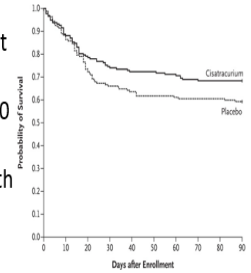


The principal cause of death in ARDS is multiorgan failure, not respiratory failure. The mortality rate is directly related to the number of organs that fail

Refractory Hypoxemia

- **Prone Position** Switching from the supine to prone position can improve pulmonary gas exchange by diverting blood away from poorly aerated lung regions in the posterior thorax and increasing blood flow in aerated lung regions in the anterior thorax and potentially reduces 28 day mortality in severe ARDS
- **Neuromuscular blocking agents** improve ventilator synchrony and may potentially blunt the inflammatory cascade of ARDS and may potentially increase ventilator free days and 90 day survival in severe ARDS
- **ECMO** has had variable success in patients with refractory hypoxemia is a consideration only when other rescue therapies have failed





Summary

There is convincing evidence that mechanical ventilation can damage the lungs in ARDS as a result of overdistension of functional alveoli (volutrauma) and collapse of small airways (atelectrauma). Lung protective ventilation is designed to mitigate the mechanical forces that create ventilator-induced lung injury, and it has been adopted as a standard method of mechanical ventilation in ARDS.

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Shock

- Syndrome of impaired oxygen delivery to tissues
- Mechanisms

-Absolute/relative decrease in oxygen delivery
-Ineffective tissue perfusion
-Ineffective utilization of delivered oxygen

	Cardiac output	Filling pressures	Vascular resistance	$ScvO_2$ SvO_2
Cardiogenic	↓	↑	↑	↓
Hypovolemic	↓	↓	↑	↓
Distributive	↑ or N	↓	↓	↑ or N
Obstructive	↓	↑ or N	↑	↓

Septic shock

Guidelines for the Treatment of Severe Sepsis and Septic Shock from the Surviving Sepsis Campaign.*

Resuscitation

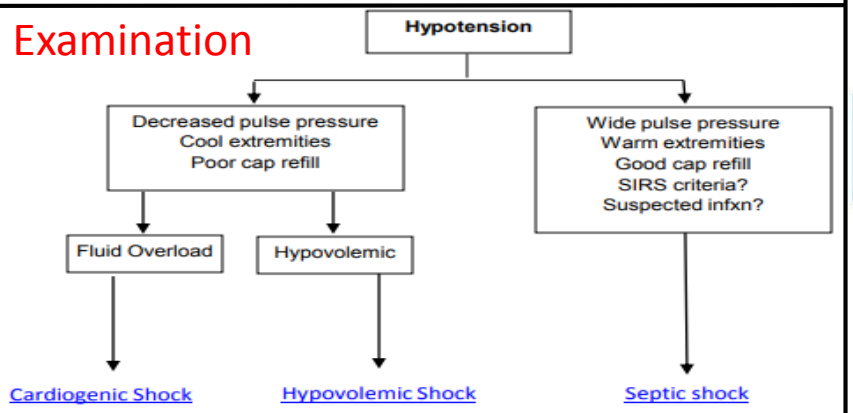
- Begin goal-directed resuscitation during first 6 hr after recognition
- Begin initial fluid resuscitation with crystalloid and consider the addition of albumin
- Consider the addition of albumin when substantial amounts of crystalloid are required to maintain adequate arterial pressure
- Avoid hetastarch formulations
- Begin initial fluid challenge in patients with tissue hypoperfusion and suspected hypovolemia, to achieve ≥30 ml of crystalloids per kilogram of body weight‡
- Continue fluid-challenge technique as long as there is hemodynamic improvement
- Use norepinephrine as the first-choice vasopressor to maintain a mean arterial pressure of ≥65 mm Hg
- Use epinephrine when an additional agent is needed to maintain adequate blood pressure
- Add vasopressin (at a dose of 0.03 units/min) with weaning of norepinephrine, if tolerated
- Avoid the use of dopamine except in carefully selected patients (e.g., patients with a low risk of arrhythmias and either known marked left ventricular systolic dysfunction or low heart rate)
- Infuse dobutamine or add it to vasopressor therapy in the presence of myocardial dysfunction (e.g., elevated cardiac filling pressures or low cardiac output) or ongoing hypoperfusion despite adequate intravascular volume and mean arterial pressure
- Avoid the use of intravenous hydrocortisone if adequate fluid resuscitation and vasopressor therapy restore hemodynamic stability; if hydrocortisone is used, administer at a dose of 200 mg/day
- Target a hemoglobin level of 7 to 9 g/dl in patients without hypoperfusion, critical coronary artery disease or myocardial ischemia, or acute hemorrhage

Interventions for Managing Shock

Component	Intervention
Blood pressure	Fluids, vasopressor, or vasodilator[a]
Cardiac Output	
Preload	Fluids, vasodilator[a]
Contractility	Inotropic agents
Afterload	Vasopressor or vasodilator[a]
Oxygen Content	
Hemoglobin	Blood transfusion
Hemoglobin saturation	Supplemental oxygen, mechanical ventilation
Oxygen demand	Mechanical ventilation, sedation, analgesia, antipyretics

[a]Vasodilator is only indicated when the patient is euvolemic or hypervolemic and the blood pressure is adequate

Examination



diagnosis of SIRS requires at least 2 of the following:

1. Temperature >38°C or <36°C
2. Heart rate >90 beats/min
3. Respiratory rate >20 breaths/min, or arterial PCO_2 <32 mm Hg
4. WBC count >12,000/mm^3 or <4000/mm^3, or >10% immature neutrophils (band forms)

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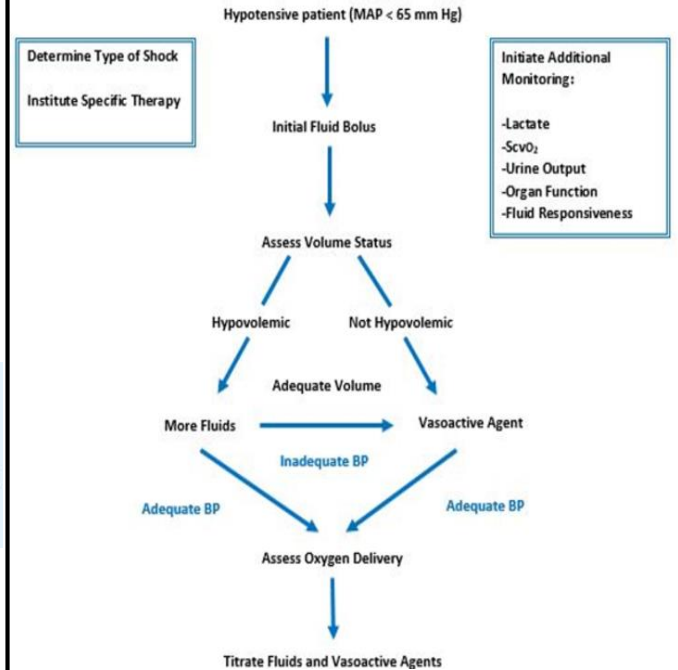
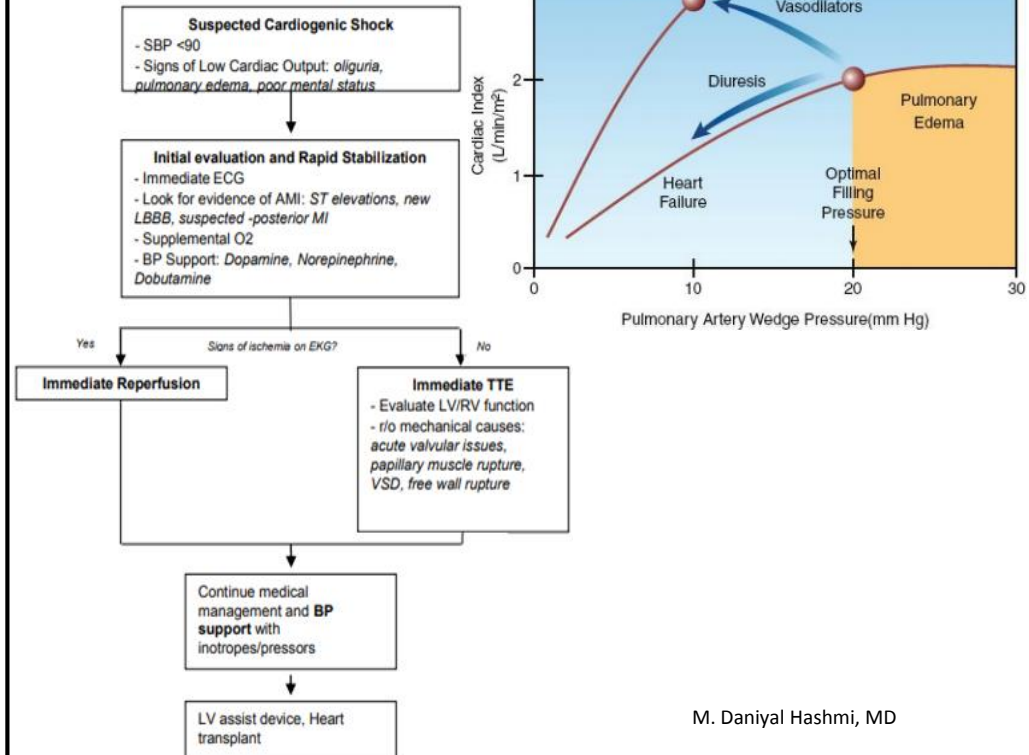
Management of Hypotension



Cardiogenic shock



M. Daniyal Hashmi, MD

Surviving Sepsis Campaign: Guidelines on the Management of Critically Ill Adults with Coronavirus Disease 2019 (COVID-19)

Infection Control and Testing:
For healthcare workers performing **aerosol-generating procedures*** on patients with COVID-19 in the ICU, we **recommend** using **fitted respirator masks (N95 respirators, FFP2, or equivalent)**, as opposed to surgical/medical masks, in addition to other personal protective equipment (i.e., gloves, gown, and eye protection, such as a face shield or safety goggles)
We **recommend** performing **aerosol-generating procedures** on ICU patients with COVID-19 in a negative pressure room.
For intubated and mechanically ventilated adults with suspicion of COVID-19: For diagnostic testing, we **suggest** obtaining lower respiratory tract samples in preference to upper respiratory tract (nasopharyngeal or oropharyngeal) samples.
For intubated and mechanically ventilated adults with suspicion of COVID-19: With regard to lower respiratory samples, we **suggest** obtaining endotracheal aspirates in preference to bronchial wash or bronchoalveolar lavage samples.

Hemodynamics:
In adults with **COVID-19 and shock**, we **suggest** using dynamic parameters skin temperature, capillary refilling time, and/or serum lactate measurement over static parameters in order to assess fluid responsiveness.
For the **acute resuscitation** of adults with **COVID-19 and shock**, we **suggest** using a conservative over a liberal fluid strategy.
For the **acute resuscitation** of adults with **COVID-19 and shock**, we **recommend** using crystalloids over colloids.
For the **acute resuscitation** of adults with **COVID-19 and shock**, we **suggest** using buffered/balanced crystalloids over unbalanced crystalloids.
For adults with **COVID-19 and shock**, we **suggest** using norepinephrine as the first-line vasoactive agent, over other agents.
For adults with **COVID-19 and shock**, we **suggest** adding vasopressin as a second-line
For adults with **COVID-19 and shock**, we **suggest titrating** vasoactive agents to target a MAP of 60-65 mmHg, rather than higher MAP targets.
For adults with **COVID-19 and shock with evidence of cardiac dysfunction and persistent hypoperfusion despite fluid resuscitation and norepinephrine**, we **suggest** adding dobutamine, over increasing norepinephrine dose.

Ventilation
In adults with COVID-19, we **suggest** starting supplemental oxygen if the peripheral oxygen saturation (SPO_2) is < 92%, and **recommend** starting supplemental oxygen if SPO_2 is < 90%
In adults with COVID-19 and **acute hypoxemic respiratory failure on oxygen**, we **recommend** that SPO_2 be maintained no higher than 96%.
For adults with COVID-19 and **acute hypoxemic respiratory failure** despite conventional oxygen therapy, we **suggest using** HFNC over conventional oxygen therapy.
In adults with COVID-19 and **acute hypoxemic respiratory failure**, we **suggest** using HFNC over NIPPV.
In adults with COVID-19 and **acute hypoxemic respiratory failure**, if HFNC is not available and there is no urgent indication for endotracheal intubation, we **suggest** a trial of NIPPV with close monitoring and short-interval assessment for worsening of respiratory failure.
In adults with COVID-19 receiving NIPPV or HFNC, we **recommend** close monitoring for worsening of respiratory status, and early intubation in a controlled setting if worsening occurs.
In mechanically ventilated adults with COVID-19 and ARDS, we **recommend** using low tidal volume (Vt) ventilation (Vt 4-8 mL/kg of predicted body weight), over higher tidal volumes (Vt>8 mL/kg).
For mechanically ventilated adults with COVID-19 and **ARDS**, we **recommend** targeting plateau pressures (Pplat) of < 30 cm H_2O.
For mechanically ventilated adults with COVID-19 and moderate to severe ARDS, we **suggest** using a higher PEEP strategy, over a lower PEEP strategy.
For mechanically ventilated adults with COVID-19 and **moderate to severe ARDS**, we **suggest** prone ventilation for **12 to 16 hours**, over no prone ventilation.
For mechanically ventilated adults with COVID-19 and **moderate to severe ARDS**: We **suggest** using, as needed, intermittent boluses of neuromuscular blocking agents (NMBA), over continuous NMBA infusion, to facilitate protective lung ventilation.
For mechanically ventilated adults with COVID-19 and hypoxemia despite optimizing ventilation, we **suggest** using recruitment maneuvers, over not using recruitment maneuvers.

Waleed Alhazzani[1,2], Morten Hylander Møller[3,4], Yaseen M. Arabi[5], Mark Loeb[1,2], Michelle Ng Gong[6] Kathryn Maitland[20], Fayez Alshamsi[21], Emilie Belley-Cote[1,22], Massimiliano Greco[16,17], Matthew Eddy Fan[7], Simon Oczkowski[1,2], Mitchell M. Levy[8,9], Lennie Derde[10,11], Amy Dzierba[12], Bin Du[13], Laundy[23], Jill S. Morgan[24], Jozef Kesecioglu[10], Allison McGeer[25], Leonard Mermel[8], Manoj J. Michael Aboodi[6], Hannah Wunsch[14,15], Maurizio Cecconi[16,17], Younsuck Koh[18], Daniel S. Chertow[19], Mammen[26], Paul E. Alexander[2,27], Amy Arrington[28], John Centofanti[29], Giuseppe Citerio[30,31], Bandar Baw[1,32], Ziad A. Memish[33], Naomi Hammond[34,35], Frederick G. Hayden[36], Laura Evans[37], Andrew

General facts

Spread: Droplet spread, survives 2-3 hours on most surfaces, 2 days on smooth metal/plastic

Incubation: 2-14 days

1st week: Fever, cough, headache, fatigue, myalgias, pharyngitis

2nd week: Resolves in 80%, Viral pneumonia 20%

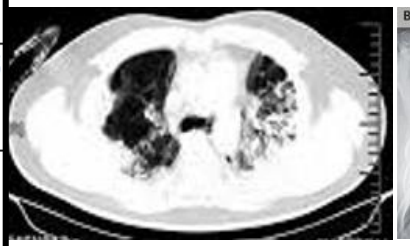
Risk increased: Heart/lung disease, immunosuppression, poorly controlled DM

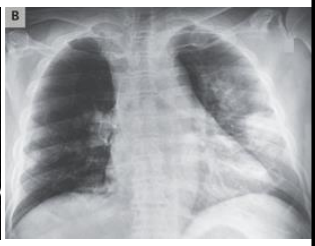
Exam: Non specific

Labs: Lymphopenia with normal WBC count or relative leukopenia, Elevated Ferritin/CRP/D-Dimer is negative prognostic indicator

Mortality: Due to oxygenation failure or septic shock/multiorgan failure

Imaging:





Testing: CBC, CMP, ABG, Troponin, G6PD, Rapid flu testing and bacterial sputum and blood cultures (coinfection with BACTERIAL respiratory pathogens unlikely), Coronavirus PCR testing, CRP, Ferritin, D-Dimer

Treatment: Symptomatic support for stable patients otherwise refer to guidelines for critical care support -Currently under investigation (Plaquenil, Azithromycin and Remdesivir)

Per présentation by Dr. Leon Liang-Yu Lai, MD

M. Daniyal Hashmi, MD

Flowchart for choosing respiratory support

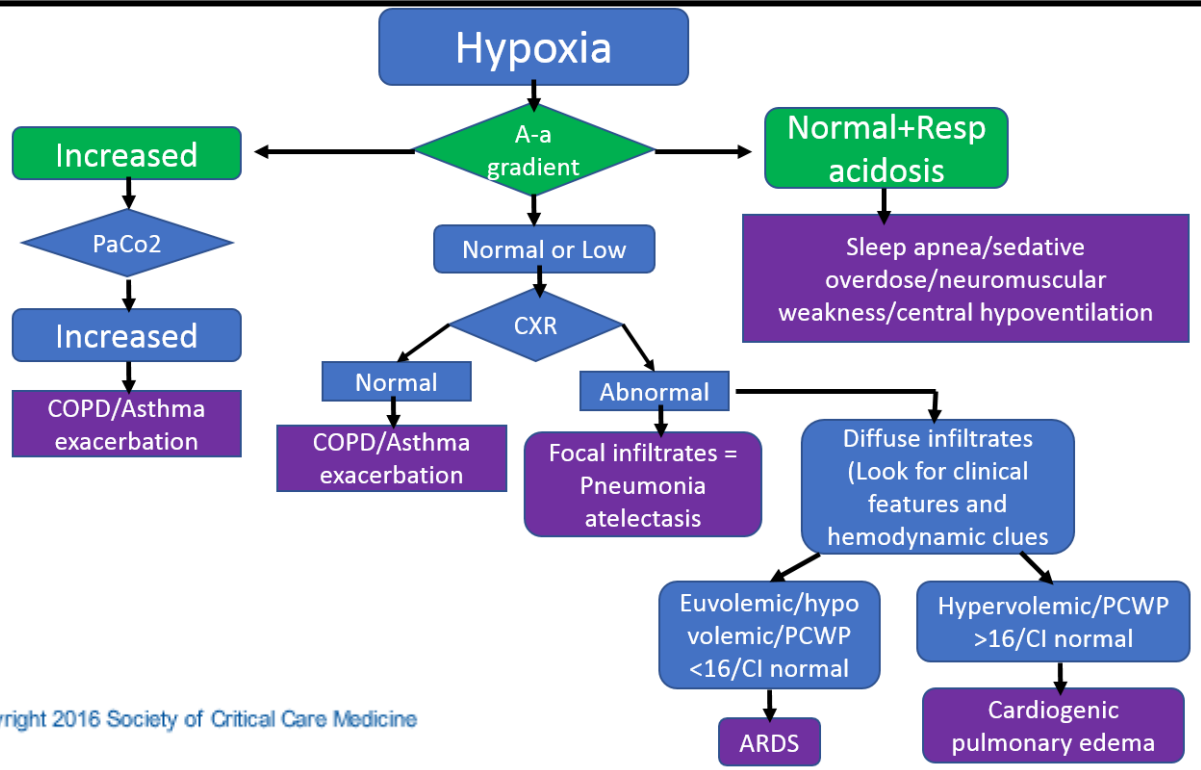
- If adequate spontaneous breathing:
  - Provide oxygen supplementation
- Manual assisted ventilation (NIPPV, bag mask ventilation) if:
  - Apnea
  - Inadequate spontaneous tidal volumes
  - Excessive work of breathing
  - Hypoxemia with poor ventilation
  - Hypoventilation with hypercarbia

→

- Indications for endotracheal intubation:
  - Airway protection
  - Relief of obstruction
  - Need for mechanical ventilation to improve oxygenation
  - Respiratory failure
  - Shock
  - Hypoventilation
  - Increased work of breathing
  - Need to facilitate suctioning/pulmonary toilet – inability to clear secretions

Supplemental Oxygenation

- **Nasal cannula**
  - Fio2 provided = 21+(L oxygen x 4)
- **Masks**
  - Venturi and Non-rebreather masks (Upto 15L flow, Non-rebreather actual delivery <75%)



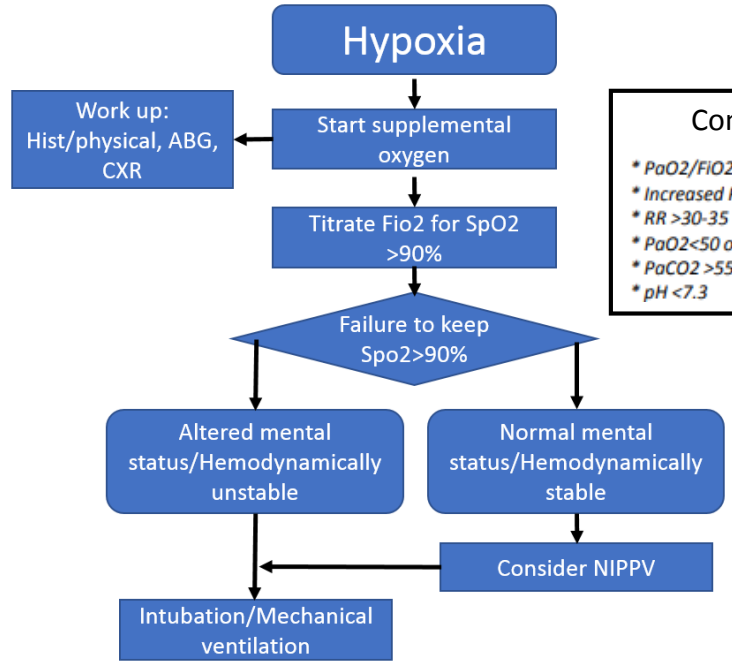
Differential diagnosis for Hypoxia

- **Hypoxemia**
  - -Room air PaO2 <60mmHg
  - -Abnormal PaO2/FiO2 ratio
- **Hypercapnea**
  - -PaCo2>45 mmHg with pH <7.35

- Normocapneic
  - Ventilation-perfusion mismatch
  - Acute respiratory distress syndrome
  - Aspiration
  - Pulmonary edema
  - Pulmonary embolus
  - Pneumonia
  - Sepsis
  - Pulmonary contusion
  - Pneumothorax

- Hypercapneic
  - Hypoventilation
  - Obstructive sleep apnea
  - Medications (Sedatives)
  - Neurological causes
  - Flail chest
  - Chest wall burns
  - Large pleural effusion
  - Morbid obesity
  - Laryngeal obstruction (epiglottitis, croup)
  - Chronic obstructive pulmonary disease
  - Asthma (marked obstruction

General management



Concerning levels on ABG

- *PaO2/FiO2 <300-200*
- *Increased PaCO2 + tachypnea*
- *RR >30-35*
- *PaO2<50 on 50% or greater FiO2*
- *PaCO2 >55 w/ nL lung fxn (I.e no COPD, fibrotic lung dz)*
- *pH <7.3*

M. Daniyal Hashmi, MD