



# FOR THE LOVE OF OXYGEN

## HOSPITAL RESPIRATORY CASES

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Adult Hospital Medicine Boot Camp  
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<p><b>DISCLOSURES</b></p>	<p><b><u>Relevant Financial Relationships</u></b> None</p> <p><b><u>Off-Label Investigational Uses</u></b> None</p> <p><small>©2021 Mayo Foundation for Medical Education and Research   slide-2</small></p>
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## LEARNING OBJECTIVES

1. Define and classify acute respiratory failure.
2. Review oxygen supplementation techniques.
3. Summarize updates for management of pulmonary embolism.
4. Discuss appropriate use of NPPV.
5. List initial therapeutic strategies for a patient with hemoptysis.
6. Outline the updated CAP guidelines.
7. Diagnose acute respiratory distress syndrome and review the best treatment options for this condition.

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## MRS. KENT

42yo female, with a past medical history of breast cancer, presents to the hospital with a 5 hour history of **chest pain and shortness of breath**.

- PMH: Breast CA s/p R mastectomy (in remission), hypothyroidism
- Medications: Ortho Tri-Cyclen Lo, Levothyroxine
- SH: Smokes ½ pack of cigarettes per day, occasional EtOH use. She just came back from a vacation to Hawaii with her family.
- Vitals: **HR**: 116, **RR**: 30, **BP**: 110/69, **Temp**: 37.5°C, **O2 sat**: 85% on RA
- PE: She is is moderate respiratory distress and clutching her chest. Feels like she “can’t catch her breath”. Lungs sound clear.

**MRS. KENT****ABG**

pH	7.34
PaCO <sub>2</sub>	31
PaO <sub>2</sub>	48
Bicarb	25



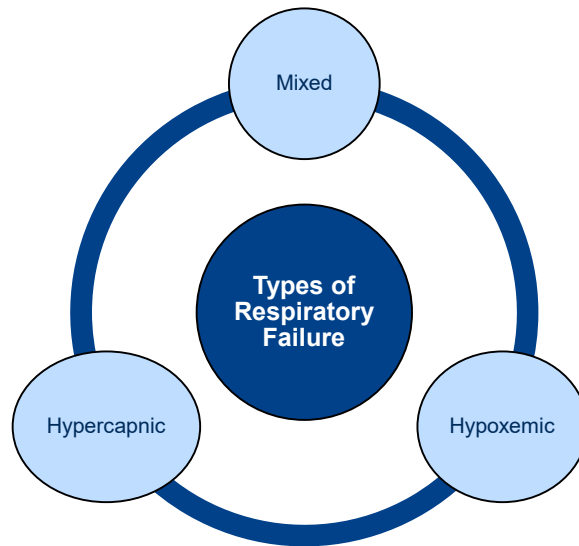
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**WHICH TYPE OF RESPIRATORY FAILURE DOES THIS PATIENT HAVE?**

- A. HYPOXEMIC
- B. HYPERCAPNIC
- C. MIXED
- D. "I HAVE NO IDEA...BUT I'M WORRIED"

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# RESPIRATORY FAILURE



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## HYPOXEMIC RESPIRATORY FAILURE

- **PaO<sub>2</sub> < 80mmHg**
- Abnormal PaO<sub>2</sub>/FiO<sub>2</sub> ratio

**Hypoxia** = state of low O<sub>2</sub> supply  
(high altitude)

**Hypoxemia** = low arterial O<sub>2</sub> tension  
(state of low arterial O<sub>2</sub> supply)

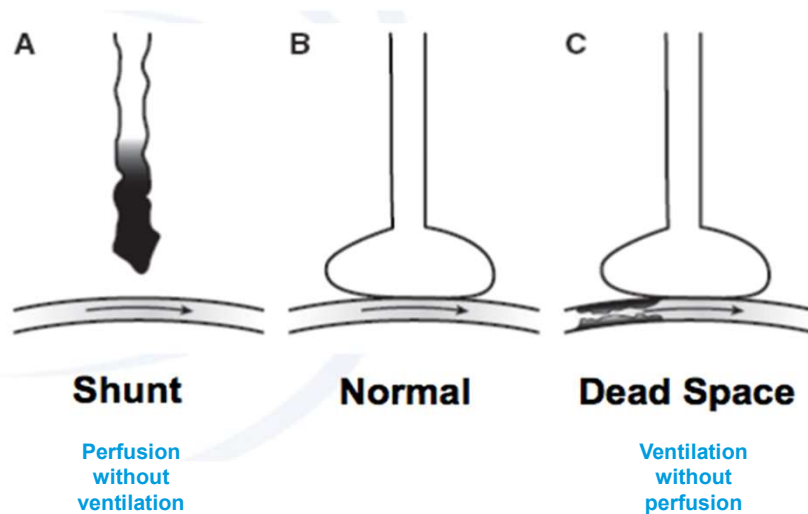
### Common causes of hypoxia:

- High altitude
- **Ventilation/perfusion mismatch**
- Impaired gas diffusion
  - Usually associated with an infiltrate on imaging
- Right to left intra-cardiac shunting
  - Typically doesn't improve with supplemental O<sub>2</sub>
- Hypoventilation
  - Alveolar to arterial (A-a) oxygen gradient should not change



## HYPOXEMIC RESPIRATORY FAILURE

Most common cause of hypoxemia is  
ventilation/perfusion (V/Q) mismatch.



FCCS 5<sup>th</sup> Ed. SCCM, 2012, pp 5-7  
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## OXYGEN DELIVERY DEVICES



### Nasal Cannula

Provides 1-6 L/min O<sub>2</sub> flow, 0.24-0.44 FiO<sub>2</sub>



### Face Mask

Delivers humidified O<sub>2</sub>  
6-10 L/min of O<sub>2</sub> flow, 0.4– 0.6 FiO<sub>2</sub>



### Non-rebreather Mask

Up to 15 L/min O<sub>2</sub>, 0.6 – 0.9 FiO<sub>2</sub>



### Face Tent

Up to 15 L/min, 0.4 – 0.5 FiO<sub>2</sub>



### Venturi Mask

Provide a constant, preset level of O<sub>2</sub>  
Up to 15L/min, 0.24-0.6 FiO<sub>2</sub>



## OXYGEN DELIVERY DEVICES

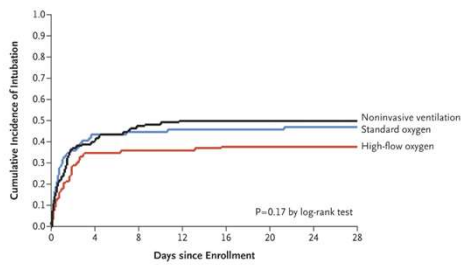
### HIGH FLOW NASAL CANNULA

- Heated & humidified oxygen
- Rates up to **60 L/min** & **1.0 FiO<sub>2</sub>** (100%)
- Improves work of breathing
- Enhances gas exchange
- Provides some positive pressure
- Reduces dead space
- May help improve mucociliary clearance

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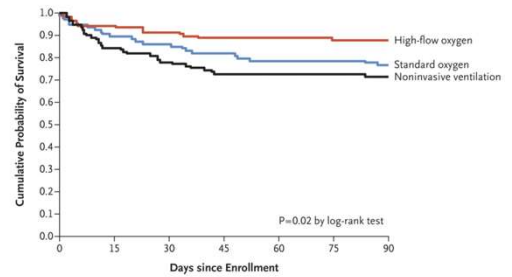
# HIGH FLOW NASAL CANNULA

A Overall Population



No. at Risk	0	4	8	12	16	20	24	28
High-flow oxygen	106	68	67	67	65	65	65	65
Standard oxygen	94	52	50	49	49	49	48	48
Noninvasive ventilation	110	64	57	53	53	53	53	52

No significant difference in intubation rates



No. at Risk	0	15	30	45	60	75	90
High-flow oxygen	106	100	97	94	94	93	93
Standard oxygen	94	84	81	77	74	73	72
Noninvasive ventilation	110	93	86	80	79	78	77

Improved survival with HFNC

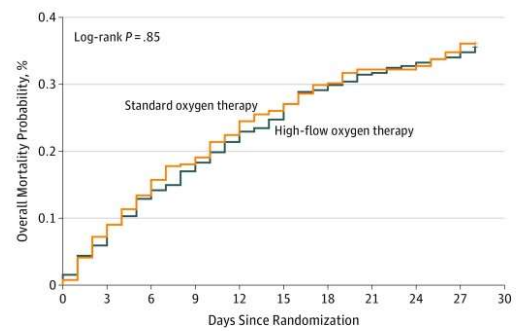
NEJM 2015;372:2185

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## HIGH FLOW NASAL CANNULA

- **HIGH Trial**

- High flow vs standard oxygen in **immunocompromised** patients with acute respiratory failure
- NO difference in mortality, intubation or ICU LOS



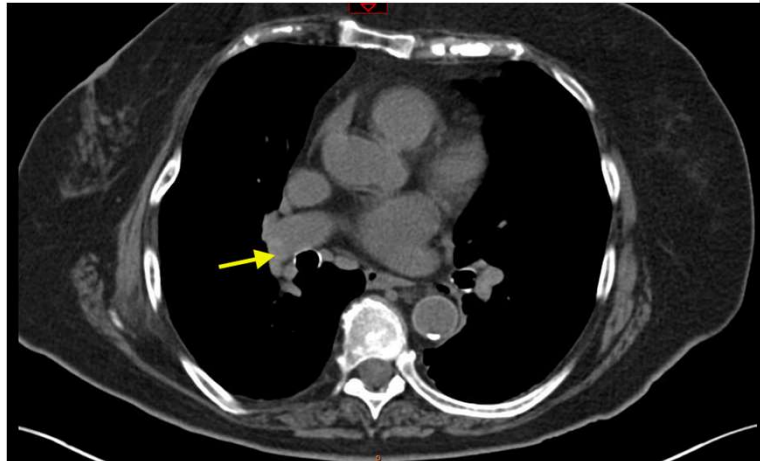
No. at risk	0	3	6	9	12	15	18	21	24	27	30
High-flow oxygen therapy	388	365	338	322	305	292	275	266	261	256	0
Standard oxygen therapy	388	360	336	318	301	287	272	263	263	253	0

JAMA 2018;320(20):2099

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## MRS. KENT

- Diagnosed with an **acute pulmonary embolism**.
- Initially placed on nasal cannula, but with ongoing hypoxia was transitioned to high-flow nasal cannula.
- Heparin drip initiated.



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## UPDATES IN PE TREATMENT

### BIOMARKERS



Use an **age-adjusted** cut-off level for D-dimers (vs. **fixed** cut-off value) for screening



Evaluation of RV function is important for risk assessment

- Can use biomarkers (**troponin, BNP**) and/or echo
- RV dysfunction is associated with ↑ short-term mortality even in hemodynamically stable patients

## UPDATES IN PE TREATMENT

### SETTING



Recommendation to implement PE response teams (PERT)



Outpatient treatment (vs. hospitalization) is recommended in low risk patients with good follow up



All patients with PE should have regular follow up due to:

- ↑ cancer risk (which might not be detectable at the time of PE)
- Risk of bleeding complications
- Risk for developing chronic thromboembolic pulmonary hypertension



# PULMONARY EMBOLISM

## DEFINITIONS

SUBMASSIVE PE	MASSIVE PE
Intermediate-risk PE	High-risk PE
RV dysfunction and/or troponin elevation, but no hypotension	Sustained hypotension (SBP<90 for at least 15 minutes or requiring inotropic support, not due to a cause other than PE), pulselessness, or persistent profound bradycardia

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## UPDATES IN PE TREATMENT

### TREATMENT



#### PE + hemodynamic instability: thrombolytic therapy

- Systemic thrombolytics favored over catheter-directed
- If contraindicated or unsuccessful, consider surgical pulmonary embolectomy or percutaneous catheter-directed therapy
- Thrombolytic therapy should be followed by anticoagulation



#### Submassive PE: anticoagulation alone

- Thrombolysis offers no immediate survival advantage
- Benefits (improved hemodynamics) appear to be offset by major bleeding (hemorrhagic stroke)

PEITHO Trial

Blood Advances (2020) 4;19:4693-4738  
European Heart Journal (2020), 41; 4: 543-603  
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## UPDATES IN PE TREATMENT

### TREATMENT



1<sup>st</sup> Line Therapy = **Direct-acting Oral Anticoagulants**

Exceptions:

- Severe renal insufficiency
- Antiphospholipid syndrome
- Pregnancy



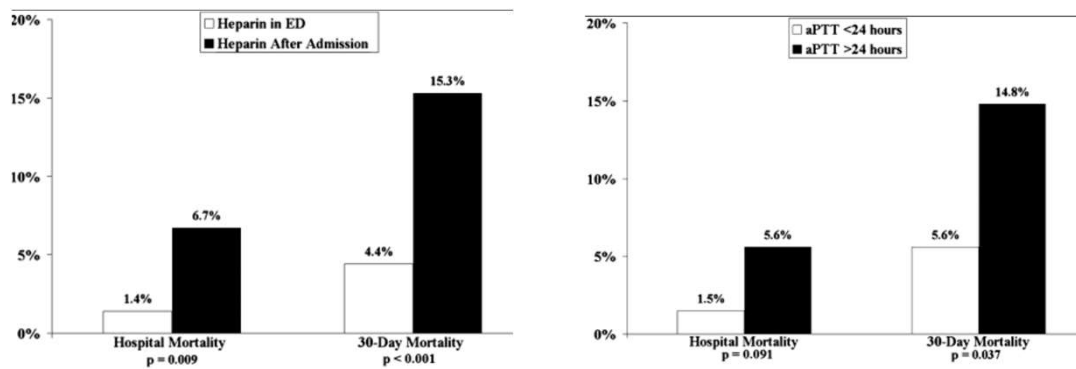
IVC filter should be considered only in patients with absolute contraindications to anticoagulation

- However, they do not appear to reduce the risk of PE recurrence or PE-related mortality

*European Heart Journal (2020), 41; 4: 543–603*

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## PULMONARY EMBOLISM TREATMENT



**Prompt** anticoagulation reduces short-term mortality after PE!

CHEST. 2010;137:1382

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## MR. JONES

75yo male, with a past medical history of **COPD**, type 2 **diabetes**, and HLD presents to the ER with a 3 day history of “**worsening shortness of breath**”.

- Medications: Metformin, Albuterol PRN, Advair Diskus
- SH: 50 pack year history of smoking cigarettes and cigars. Daily EtOH use. He is retired and lives at home with his wife.
- Vitals: **HR**: 105, **RR**: 34, **BP**: 119/75  
**Temp**: 37.8°C **O2 sat**: 87% on RA
- He is in moderate distress, using accessory muscles, and wheezing.

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**MR. JONES**

ABG	
pH	7.36
PaCO <sub>2</sub>	51
PaO <sub>2</sub>	53
Bicarb	33

~~12~~ 325  
~~28~~

135 | 102 | 12  
 3.7 | 29 | 0.8

135



## MR. JONES

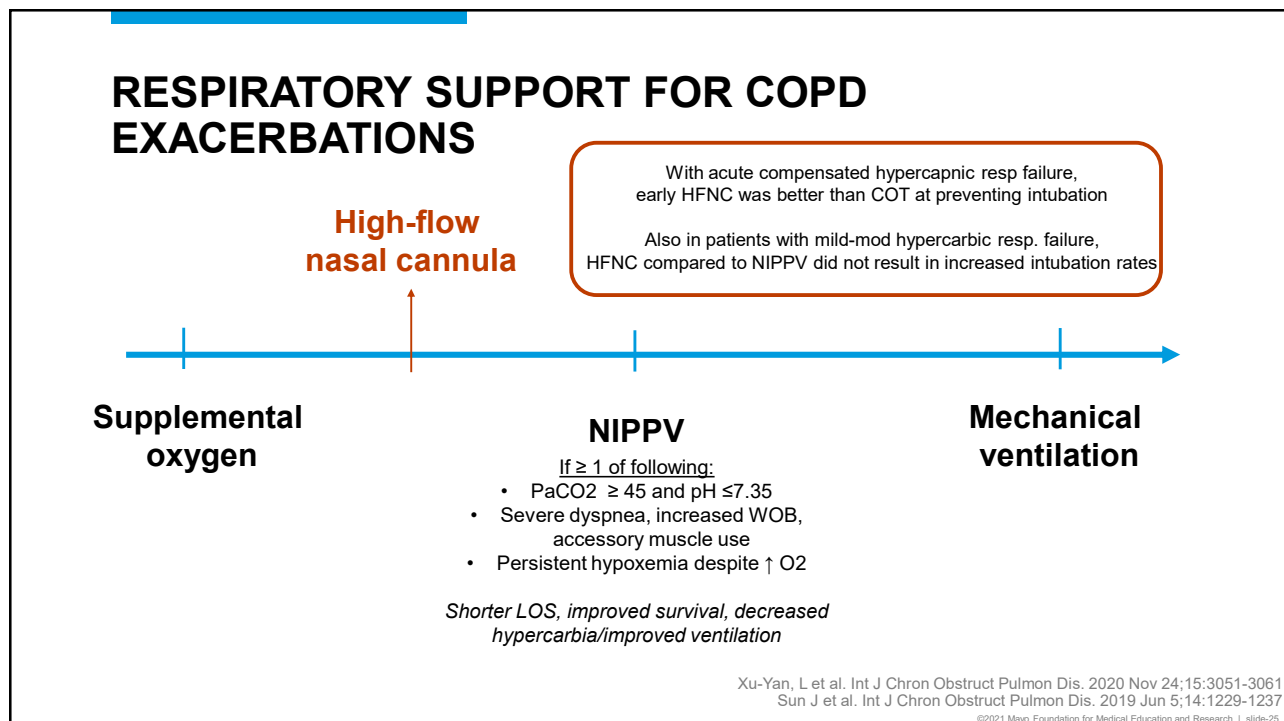
- In the ER, he received:
  - Albuterol/ipratropium nebulizer
  - IV Solu-medrol
  - IV Ceftriaxone + Azithromycin
- Despite this, he continues to be hypoxic. His O2 sat is 83% on 4L NC.

## WHAT WOULD BE THE NEXT STEP IN YOUR TREATMENT PLAN?

- A. ↑ O<sub>2</sub> to 6L VIA NASAL CANNULA
- B. START HIGH-FLOW NASAL CANNULA
- C. START BIPAP
- D. INTUBATE

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## MR. JONES

- You decide to place Mr. Jones on HFNC and he starts to improve.
- However, a few hours later you get a call that he is more lethargic...

### ABG

pH = 7.21

pCO<sub>2</sub> = 67

pO<sub>2</sub> = 72

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## WHAT WOULD YOU DO NOW?

- A. GO BACK TO NASAL CANNULA
- B. CONTINUE HIGH FLOW NASAL CANNULA
- C. START BIPAP
- D. INTUBATE

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## NPPV

### Advantages

- Reduced need for sedation
- Preservation of airway-protective reflexes
- Avoidance of upper airway trauma
- Decreased incidence of nosocomial sinusitis and pneumonia
- Improved patient comfort
- Shorter length of stays in ICU and hospital
- Improved survival

### Disadvantages

- Claustrophobia
- Increased workload for respiratory practitioner
- Facial/nasal pressure lesions
- Unprotected airway
- Inability to suction deep airway
- Gastric distention
- Delay in intubation

Mechanical Ventilation. FCCS 5<sup>th</sup> Ed. SCCM, 2012, pp 5-3  
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## BILEVEL POSITIVE AIRWAY PRESSURE (BIPAP)

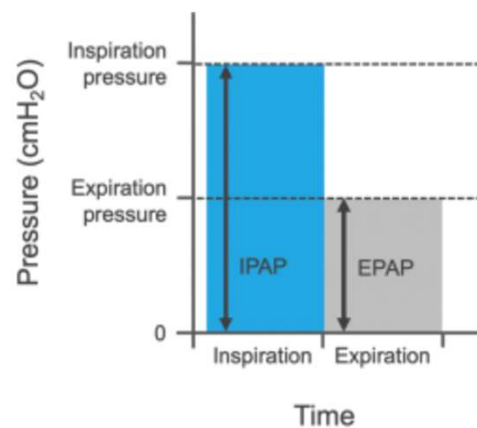
INDICATIONS	CONTRAINDICATIONS
<ul style="list-style-type: none"> <li>• Hypercapnia and acidosis</li> <li>• Cardiogenic pulmonary edema</li> <li>• COPD/asthma exacerbation</li> <li>• Weaning and post-extubation failure</li> <li>• Post surgical period</li> <li>• Obesity hypoventilation syndrome</li> <li>• Neuromuscular disorders</li> <li>• Poor alveolar oxygen exchange</li> </ul>	<ul style="list-style-type: none"> <li>• Cardiac or respiratory arrest</li> <li>• Hemodynamic instability</li> <li>• Inability to protect the airway</li> <li>• Patient who is unable to cooperate</li> <li>• Severe encephalopathy</li> <li>• Significant agitation</li> <li>• High risk of aspiration</li> <li>• Active upper GI hemorrhage</li> <li>• Facial trauma, recent surgery and/or burns</li> </ul>

Liesching, Timothy et al. CHEST , Volume 124 , Issue 2 , 699 - 713  
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## BIPAP

### HOW DOES IT WORK?

- Utilizes two levels of positive airway pressure combining pressure support ventilation (PSV) and continuous positive airway pressure (CPAP)
  - The PSV modality is the **IPAP** (inspiratory positive airway pressure)
  - The CPAP modality is the **EPAP** (expiratory positive airway pressure)
- The difference between these two pressure levels ( $\Delta P$ ) determines tidal volume generated.



Mechanical Ventilation, FCCS 5<sup>th</sup> Ed. SCCM, 2012. pp 5-3  
 Image: <https://www.medmastery.com/guide/noninvasive-ventilation-clinical-guide/deciphering-acronyms-noninvasive-ventilation-niv>  
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## BIPAP

### HOW DOES IT WORK?

- Example for initial BiPAP settings:

- Mode: Spontaneous
- Trigger: Maximum sensitivity
- FiO<sub>2</sub>: 1.0

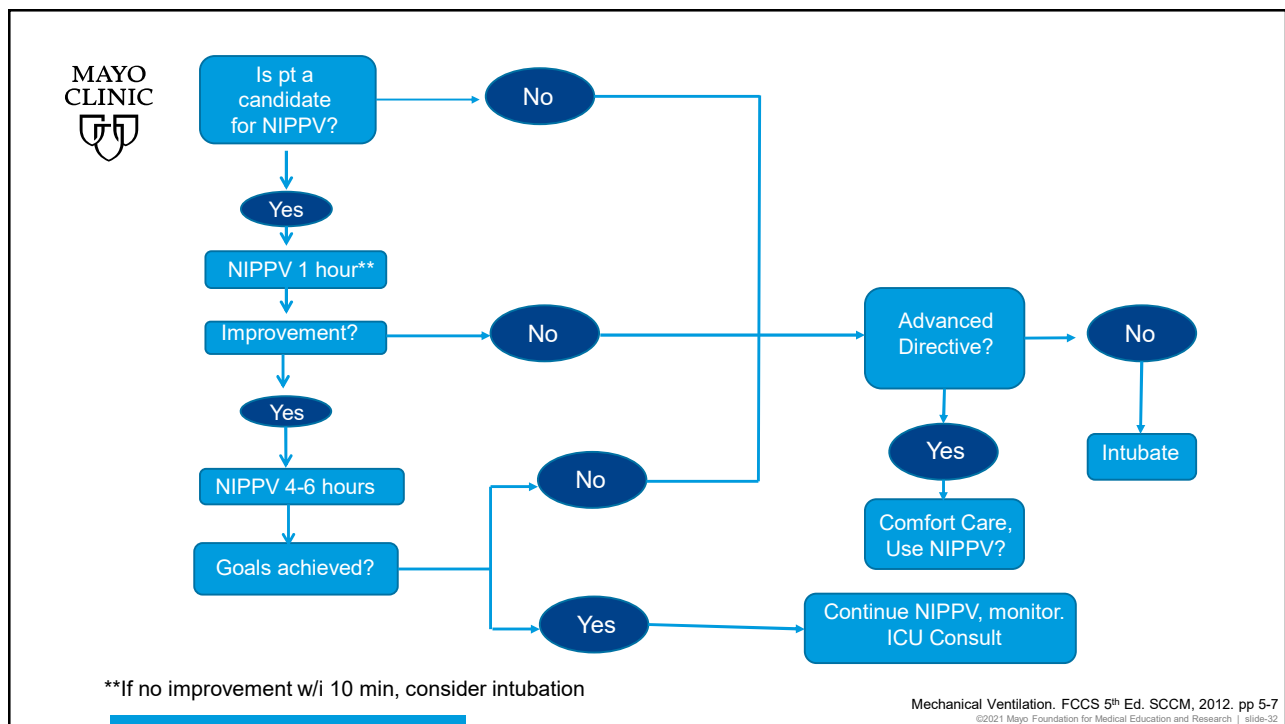
- EPAP: 5 cm H<sub>2</sub>O
- IPAP: 10-15 cm H<sub>2</sub>O

} Adjust  $\Delta$  to achieve an effect  $V_T$  and CO<sub>2</sub> clearance

\*\*if oxygenation needs improving, increase EPAP for alveolar recruitment  
(however, will then need to also adjust IPAP to keep the same  $\Delta$ )

- Backup rate: 6-8/min

Mechanical Ventilation, FCCS 5<sup>th</sup> Ed. SCCM, 2012. pp 5-3  
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## MS. SANDS

- A 28yo female presented as a transfer from an outside hospital with **shortness of breath, cough and occasional hemoptysis.**
- She was recently diagnosed with **SLE** the previous year, but was not on any immunosuppression at this time.
  
- She was hemodynamically stable on arrival. Given IV Solu-Medrol.
- The next day, during the bronchoscopy, she developed massive hemoptysis  
**2/2 diffuse alveolar hemorrhage.**

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## HEMOPTYSIS

Causes of Hemoptysis	
<b>Cryptogenic</b>	
<b>Pulmonary</b>	<ul style="list-style-type: none"> <li>• Airway infections (bronchitis, viral and bacterial PNA, lung abscess)</li> <li>• Bronchial carcinoma/Mets</li> <li>• Bronchiectasis/CF</li> <li>• Pulmonary edema/mitral stenosis</li> <li>• TB</li> <li>• Invasive aspergillosis</li> <li>• Benign bronchial tumors</li> <li>• Vasculitis</li> </ul>
<b>Cardiovascular</b>	<ul style="list-style-type: none"> <li>• Pulmonary artery embolism</li> <li>• Vascular malformations</li> <li>• Idiopathic pulmonary hemosiderosis</li> <li>• Septic embolism/right heart endocarditis</li> <li>• Pulmonary HTN</li> </ul>
<b>Other</b>	<ul style="list-style-type: none"> <li>• <u>Iatrogenic</u>: lung biopsy, R heart cath, CT placement, thoracentesis, radiation therapy</li> <li>• Medications, anticoagulation treatment, thrombolytic therapy</li> <li>• Trauma/lung contusion</li> <li>• Foreign body</li> <li>• Coagulopathy</li> <li>• Thrombocytopenia</li> </ul>

Dtsch Arztebl Int 2017; 114: 371-81  
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## HEMOPTYSIS

- **Massive hemoptysis** = 100 – 600 ml of blood loss in 24h
  - Conservatively treated massive hemoptysis has a **mortality of 50-100%**.
  - Death is usually secondary to asphyxia, as opposed to blood loss/hemorrhagic shock.

## INITIAL MANAGEMENT OF HEMOPTYSIS

- Monitor vital signs closely
- Secure airway first!
  - If intubation is required, use a large diameter ET tube, or consider unilateral intubation if indicated.
- Place patient **bleeding side down**
- Sedation/anxiolysis or paralytics if necessary
- Reverse any coagulopathy - transfuse blood products if indicated.

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## TREATMENT OF HEMOPTYSIS

- Mild - moderate hemoptysis can be treated conservatively
- **Bronchoscopy**
  - Typically first line for diagnostic (localize site of bleeding) and therapeutic intervention
- Bronchial artery embolization
- Surgery

## NEW TREATMENT FOR HEMOPTYSIS?

- **Inhaled tranexamic acid** treatment can be helpful in non-massive hemoptysis
  - Shorter length of stay
  - Required less invasive procedures
  - Reduced recurrence rate at 1 year follow-up
  - The tranexamic acid group didn't have any increased side effects

CHEST, Volume 154, Issue 6, 1379-1384  
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## MR. WILSON

- 60yo male, with a history of HTN, HLD, atrial fibrillation, TIA, and diabetes, presents to the ED with 2 days of cough and fevers.
- Vitals: **HR:** 101, **RR:** 27, **BP:** 110/79  
**Temp:** 38.9 C, **O2 sat:** 87% on RA



## WHAT IS THE MOST APPROPRIATE DIAGNOSIS?

- A. Community-Acquired Pneumonia (CAP)
- B. Ventilator-Associated Pneumonia (VAP)
- C. Hospital-Acquired Pneumonia (HAP)
- D. Healthcare-associated pneumonia (HCAP)

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## CLASSIFICATION OF PNEUMONIA

Community-acquired pneumonia (CAP)

Hospital-acquired pneumonia (HAP)

Ventilator-associated pneumonia (VAP)

\*There is **no longer a healthcare-associated pneumonia (HCAP)** classification.\*

2016 HAP/VAP Clinical Practice Guidelines by the IDSA and ATS. CID 2016:1-43. a  
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## TREATMENT OF CAP

- Ineffective/delayed initial antimicrobial therapy is the most significant predictor of poor outcomes.
- Start empiric antibiotics as soon as diagnosis is made!

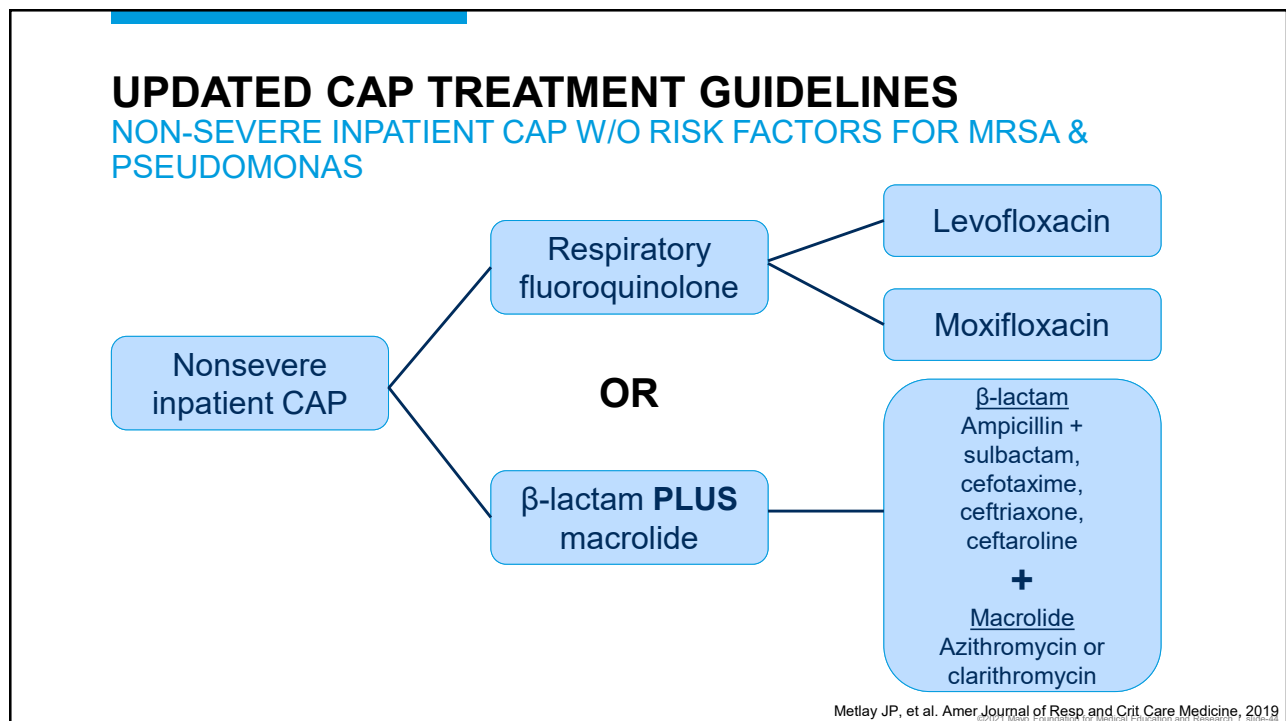


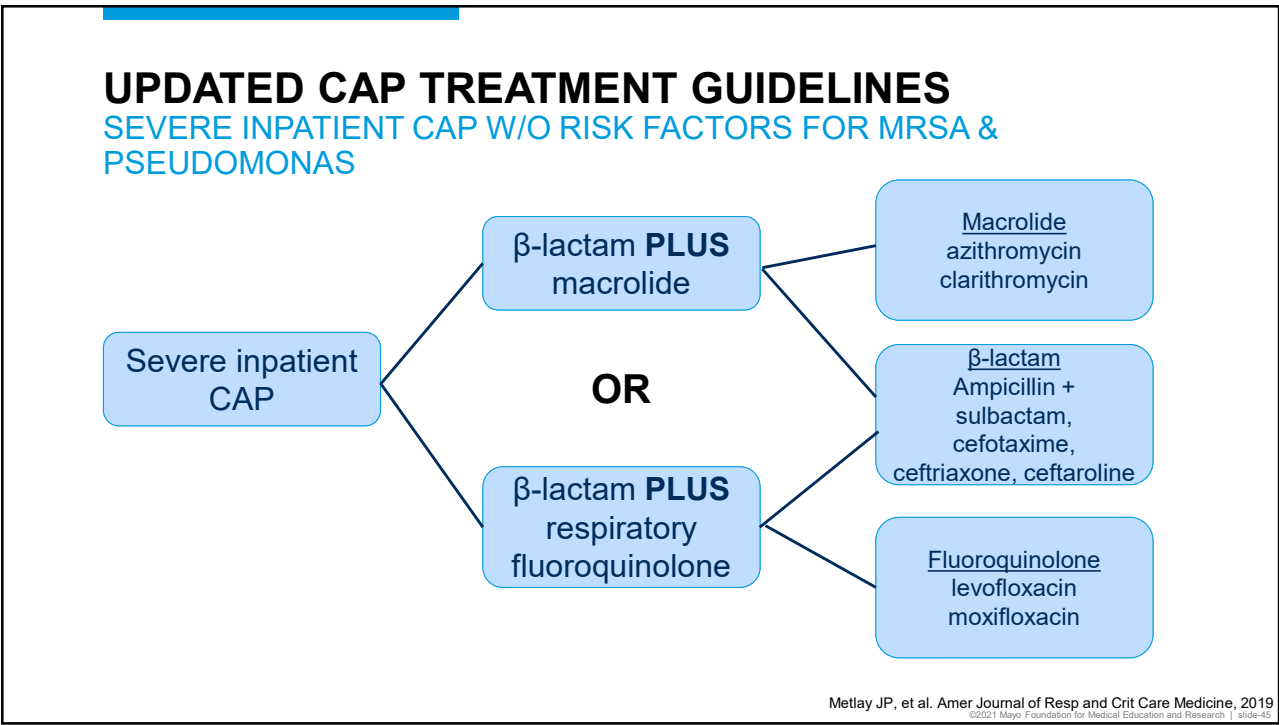
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## WHICH ANTIBIOTICS SHOULD WE START FOR MR. WILSON?

- A. Piperacillin-tazobactam and Vancomycin
- B. Ciprofloxacin
- C. Ceftriaxone and Azithromycin
- D. Azithromycin

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## RISK FACTORS FOR MRSA & PSEUDOMONAS

### MRSA Risk Factors

- End stage renal disease
- IV drug abuse
- Prior antibiotic use

Empiric Treatment  
Vancomycin  
Linezolid

### Pseudomonas Risk Factors

- Prior use of antibiotics (within 90 days)
- H/o Pseudomonas infection w/in 1 year
- Longer hospital stay
- ICU
- Mechanical ventilation
- Immunosuppression
- Cystic Fibrosis
- HIV/AIDS
- Alcohol abuse
- COPD

Empiric Treatment  
Piperacillin-tazobactam  
Cefepime  
Ceftazidime  
Aztreonam  
Meropenem  
Imipenem

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## WHERE DID HCAP GO?

- The Drug-Resistance in Pneumonia (DRIP) score was found to be more effective than the HCAP criteria for identifying risk of drug-resistant pathogens in pneumonia, and the need for broad-spectrum antibiotic use in CAP
  - Combined with the use of nasal MRSA swab for de-escalation, which showed reduction in vancomycin use

ASM journals 2016. 60;5: <https://doi.org/10.1128/AAC.03071-15>  
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## DRUG-RESISTANCE IN PNEUMONIA (DRIP) SCORE

Factors	Points
<b>Major Risk Factors</b>	
Antibiotic use (prior 60 days)	2
Long-term care resident	2
Tube feeding	2
H/o infection with MDR pathogen (prior 12 months)	2
<b>Minor Risk Factors</b>	
Hospitalization (prior 60 days)	1
Chronic pulmonary disease	1
Poor functional status	1
Gastric acid suppression	1
Wound care	1
MRSA colonization (prior 12 months)	1
<b>Total Points Possible</b>	<b>14</b>

**<4** = can be treated without broad-spectrum antibiotics

**≥4** = more likely to require broad-spectrum antibiotics

ASM journals 2016, 60:5; <https://doi.org/10.1128/AAC.03071-15>  
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## WHAT ABOUT ASPIRATION?

- **Anaerobic** antibiotic coverage isn't indicated, unless **lung abscess** or **empyema** is suspected.
  - Most patients who aspirate gastric contents develop aspiration pneumonitis, which typically only requires supportive treatment (without antibiotics) and resolves within 24-48 hours.
  - More recent studies have shown that anaerobes are uncommon in patients hospitalized with suspected aspiration

Metlay JP, et al. Amer Journal of Resp and Crit Care Medicine, 2019  
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## TREATMENT OF CAP

### DURATION OF TREATMENT

- Shorter duration therapy leads to:
  - ↓ antibiotic resistance
  - ↓ antibiotic related complications
  - ↓ cost
  - ↑ patient compliance
- Minimum recommended treatment : **5 days**
  - Applies to patients with severe CAP, as well
- If CAP is due to MRSA or Pseudomonas, treat for 7 days.



Early de-escalation of antibiotics! (after 48 hrs if cultures negative)

## PROCALCITONIN?

- Still controversial! No clear evidence to support better outcomes with procalcitonin guided antibiotic use.
- **Recommendation:** Do not delay initiation of antibiotics regardless of procalcitonin value
  - Procalcitonin can be helpful in de-escalating antibiotic therapy.

Metlay JP, et al. Amer Journal of Resp and Crit Care Medicine, 2019  
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## STEROIDS?

- Routine use of steroids in non-severe or severe CAP is **not** recommended
  - Exceptions:
    - Refractory shock (consistent with Surviving Sepsis Campaign recommendations)
    - If concomitant COPD/asthma exacerbation or autoimmune illness

Metlay JP, et al. Amer Journal of Resp and Crit Care Medicine, 2019  
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## SEVERE CAP

- Late admission to ICU significantly ↑ 30 day mortality
- Severe CAP =  
1 Major or 3+ Minor Criteria

### Major Criteria

- Need for invasive mechanical ventilation
- Septic shock with need for vasopressors

### Minor Criteria

- Respiratory rate  $\geq 30$  breaths/min
- PaO<sub>2</sub>/FiO<sub>2</sub> ratio  $\leq 250$
- Multilobar infiltrates
- Confusion/disorientation
- Uremia (BUN  $\geq 20$ )
- Leukopenia (WBC  $< 4,000$ )
- Thrombocytopenia (Platelets  $< 100,000$ )
- Hypothermia (Core temp  $< 36^{\circ}\text{C}$ )

CID 2007:44 (Suppl 2)

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## BRONCHOSCOPY

- When should you consider bronchoscopy?
  - Immunocompromised host
  - Non-resolving pneumonia
  - Nodular/cavitary lesions on imaging
- Can be both diagnostic and therapeutic
- Consider risk of airway/respiratory compromise in patients with high O<sub>2</sub> requirement.
- Risks of Bronchoscopy:
  - Difficult to truly assess
  - Operator and patient dependent
  - Risks increase when biopsies are performed



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## 2007 VS. 2019 CAP GUIDELINES

**Table 2.** Differences between the 2019 and 2007 American Thoracic Society/Infectious Diseases Society of America Community-acquired Pneumonia Guidelines

Recommendation	2007 ATS/IDSA Guideline	2019 ATS/IDSA Guideline
Sputum culture	Primarily recommended in patients with severe disease	Now recommended in patients with severe disease as well as in all inpatients empirically treated for MRSA or <i>Pseudomonas aeruginosa</i>
Blood culture	Primarily recommended in patients with severe disease	Now recommended in patients with severe disease as well as in all inpatients empirically treated for MRSA or <i>P. aeruginosa</i>
Macrolide monotherapy	Strong recommendation for outpatients	Conditional recommendation for outpatients based on resistance levels
Use of procalcitonin	Not covered	Not recommended to determine need for initial antibacterial therapy
Use of corticosteroids	Not covered	Recommended not to use. May be considered in patients with refractory septic shock.
Use of healthcare-associated pneumonia category	Accepted as introduced in the 2005 ATS/IDSA hospital-acquired and ventilator-associated pneumonia guidelines	Recommend abandoning this categorization. Emphasis on local epidemiology and validated risk factors to determine need for MRSA or <i>P. aeruginosa</i> coverage. Increased emphasis on deescalation of treatment if cultures are negative
Standard empiric therapy for severe CAP	$\beta$ -Lactam/macrolide and $\beta$ -lactam/fluoroquinolone combinations given equal weighting	Both accepted but stronger evidence in favor of $\beta$ -lactam/macrolide combination
Routine use of follow-up chest imaging	Not addressed	Recommended not to obtain. Patients may be eligible for lung cancer screening, which should be performed as clinically indicated

*Definition of abbreviations:* ATS = American Thoracic Society; CAP = community-acquired pneumonia; IDSA = Infectious Diseases Society of America; MRSA = methicillin-resistant *Staphylococcus aureus*.

Metlay JP, et al. Amer Journal of Resp and Crit Care Medicine, 2019

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## MR. WILSON

- 2 days after admission, you get a page from his nurse:
  - “Mr. Wilson has increased WOB, please come evaluate ASAP”



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## MR. WILSON

- Vitals:  
**HR: 112, RR: 32, BP: 108/73, Temp: 37.6**  
**O2: 83% on 6L NC**
- ABG: pH = 7.37, pCO<sub>2</sub> = 35, pO<sub>2</sub> = 40
- Echo (from earlier in the day): EF 65%,  
1/4 diastolic dysfunction, normal RV  
function, L atrial enlargement



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## WHAT IS THE MOST APPROPRIATE DIAGNOSIS?

- A. PNEUMONIA
- B. PULMONARY EDEMA
- C. ARDS
- D. "I HAVE NO IDEA...BUT I'M VERY WORRIED"

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## ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

### Berlin Criteria

- **Acute onset**
- **Bilateral opacities** on CXR or CT within 24 hours
- No evidence of left heart failure or **fluid overload**
- Moderate to severe impairment of oxygenation ( **$\text{PaO}_2/\text{FiO}_2 \leq 300$** )
- Presence of a **predisposing condition**

ARDS Definition Task Force. ARDS. JAMA 2012; 307:2526-2533  
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## ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

Severity of ARDS	PaO <sub>2</sub> /FiO <sub>2</sub> (mmHg)
Mild	200 – 300
Moderate	100 – 200
Severe	≤100

ARDS Definition Task Force. ARDS. JAMA 2012; 307:2526-2533  
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## PATHOPHYSIOLOGY

Acute, diffuse inflammatory lung or systemic injury

Damage to pulmonary capillary endothelial cells and alveolar epithelial cells

Increased vascular permeability and decreased production and activity of surfactant

Pulmonary edema and alveolar collapse

Hypoxemia/ARDS

ARDS Definition Task Force. ARDS. JAMA 2012; 307:2526-2533  
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## CAUSES OF ARDS

### SYSTEMIC

- Sepsis
- Shock
- Trauma
- Blood transfusions
- Burns
- Drug overdose
- Cardiopulmonary bypass

### PULMONARY

- Severe pneumonia
- Aspiration
- Lung contusion
- Toxic inhalation
- Near-drowning
- Pulmonary embolus

\*If idiopathic, it is considered **Acute Interstitial Pneumonia\***

ARDS Definition Task Force. ARDS. JAMA 2012; 307:2526-2533  
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## TREATMENT OF ARDS

- Identify the initial systemic or pulmonary insult, and treat underlying cause

### Supportive Care

- Corticosteroids
- Conservative fluid strategy
- Lung protective ventilation (low tidal volumes, high PEEP)
- Prone positioning
- +/- ECMO (in select patients)

Griffiths MJD, et al. BMJ Open Respiratory Research 2019.  
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**ONE LAST THING  
BEFORE I GO...**



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## LUNG POINT OF CARE ULTRASOUND (POCUS)

- Lung US can assess for:
  - Pulmonary edema
  - Consolidation/pneumonia
  - Pleural effusions
  - Pneumothorax

	Sensitivity	
	CXR	US
Pulmonary edema	56.9%	85-92%
Pneumonia	38-64%	85-96%
Pneumothorax	39-50%	78-90%

Lung ultrasound can provide the correct diagnosis  
in **90.5%** of cases.

Lichtenstein DA, Mezière GA. Relevance of Lung Ultrasound in the Diagnosis of Acute Respiratory Failure. Chest. 2008;134(1):117-125. doi:10.1378/chest.07-2800.  
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## TAKE HOME POINTS

- When a patient is in respiratory distress, first determine if it is hypoxic, hypercapnic, or mixed respiratory failure.
- Use the most appropriate form of supplemental O<sub>2</sub>.
- Consider high-flow nasal cannula, even in COPD exacerbations (under the right conditions).
- NPPV can be an extremely helpful tool when used in the right clinical setting.
- With hemoptysis, turn patient bleeding side down, and secure an airway first.
- There is no longer a “healthcare-associated” classification of pneumonia. Use the DRIP score to assess need for broad-spectrum antibiotics in CAP.
- In a patient with refractory hypoxemia, consider ARDS in your differential – and try to recognize and treat as quickly as possible.

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**QUESTIONS?**

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