

Acute Respiratory Failure & ARDS

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Objectives

- Define respiratory failure & discuss types of respiratory failure
- Brief introduction to mechanical ventilation
- Define ARDS, its epidemiology & basic pathophysiology
- Discuss clinical aspects of ARDS
- Discuss treatment of ARDS

Respiratory Failure

- An inability to adequately oxygenate or ventilate
 - $\text{PaO}_2 < 60 \text{ mm Hg}$
 - $\text{PaCO}_2 > 45 \text{ mm Hg}$

Respiratory Failure

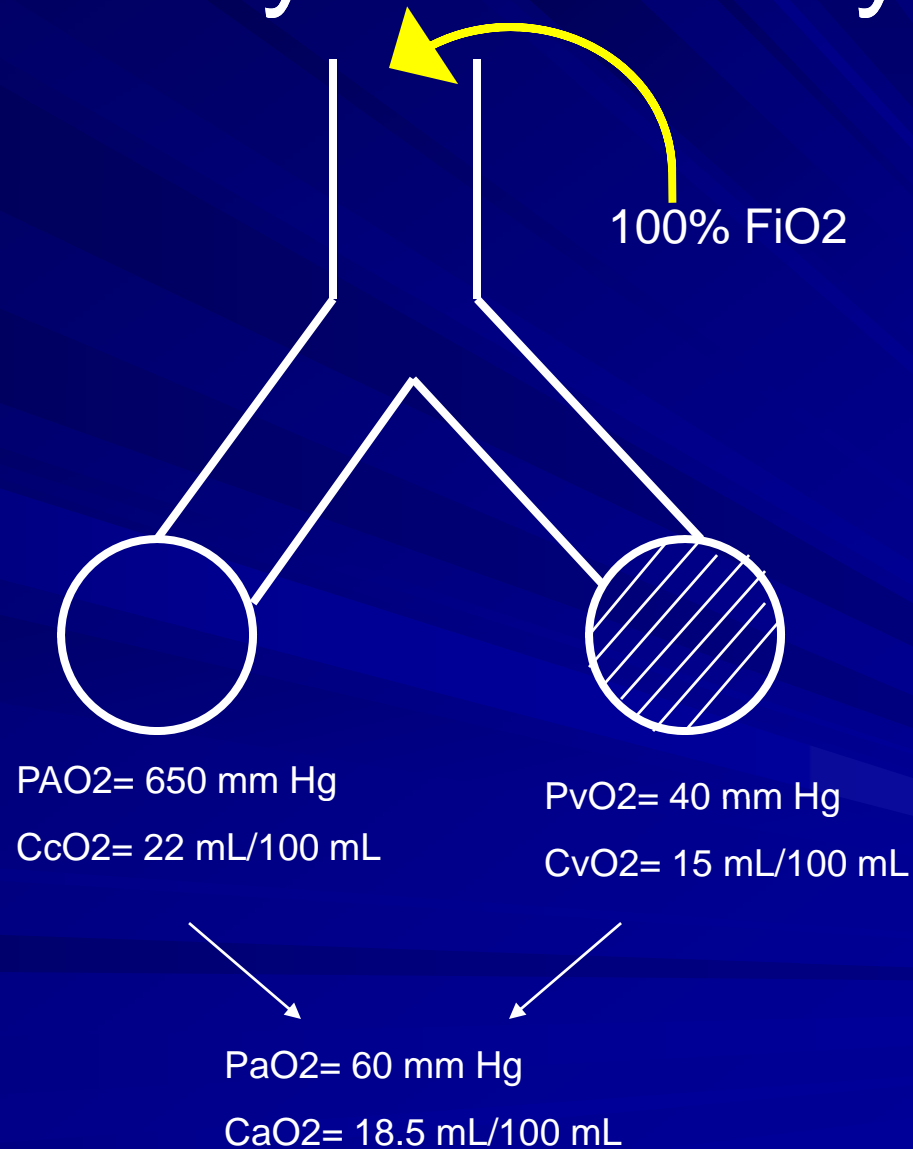
	Type I	Type II	Type III	Type IV
Mechanism	Shunt	Hypoventilation	Atelectasis	Hypoperfusion
Etiology	Alveolar flooding- low or high pressure pulmonary edema	Increased dead space, decreased minute ventilation	Decreased FRC, Increased closing volume	Decreased mixed venous oxygen
Clinical Scenario	ARDS, CHF, Pneumonia, Alveolar hemorrhage	Airway Obstruction, Impaired Lung or Chest Wall Compliance, Neuromuscular weakness, Impaired CNS drive	Postoperative, Obesity	Shock, MI

Respiratory Failure- Type I

Acute Hypoxemic Respiratory Failure

- Cardiogenic
 - “High-pressure” edema
- ARDS
 - “Low-pressure”/increased permeability edema
- Focal lung lesions
 - Pneumonia, Contusion
- Alveolar Hemorrhage syndromes
 - Goodpasture’s, Wegener’s disease
- Miscellaneous

Respiratory Failure- Type I



Respiratory Failure- Type I

Starling Equation

$$* J_v = K_f [(P_c - P_i) - \sigma(\pi_c - \pi_i)]$$

K_f = filtration coefficient

P_c = hydrostatic capillary pressure

P_i = interstitial capillary pressure

π_c = oncotic capillary pressure

π_i = oncotic interstitial pressure

σ = reflection coefficient

Respiratory Failure- Type II

$$Pa_{CO_2} = \frac{\dot{V}_{CO_2} \times k}{\dot{V}_A}$$

- Pa CO₂ rises if:
 - CO₂ production increases
 - Alveolar ventilation decreases

Strength/ Drive

CNS Drive

Sedation
Metabolic encephalopathy
OHS

NM Transmission Impaired

ALS
Guillain-Barre Syndrome
Paralytic

Muscle Weakness

Malnutrition
Fatigue
Electrolyte
Hypoperfusion



Load

Resistive

Bronchospasm
OSA
Secretions

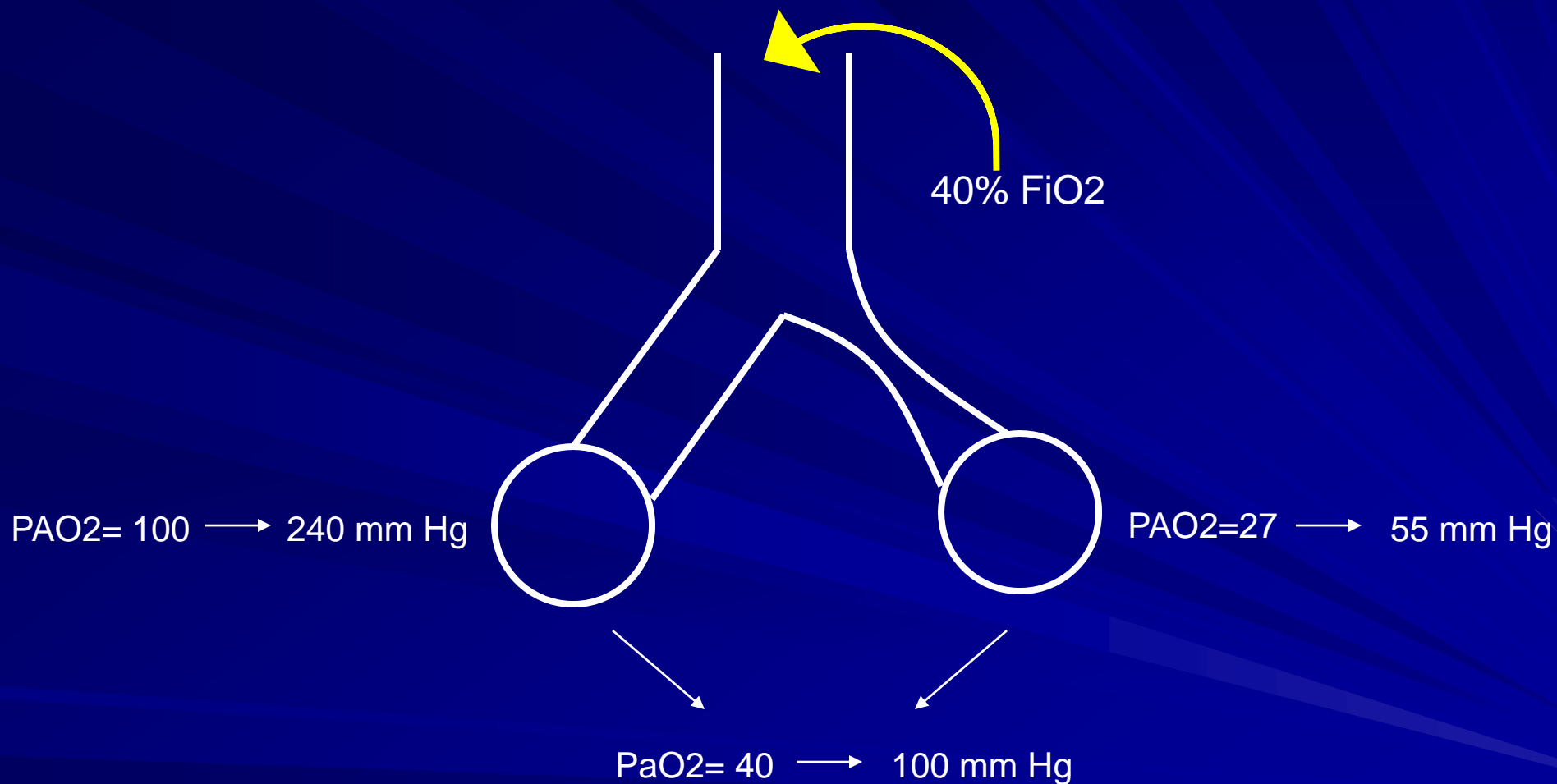
Lung & Chest Wall Elastic

Pneumonia
Pulmonary Edema
Pleural Effusion
Ascites

Minute Ventilation

Sepsis
Pulmonary Embolism
Metabolic Acidosis

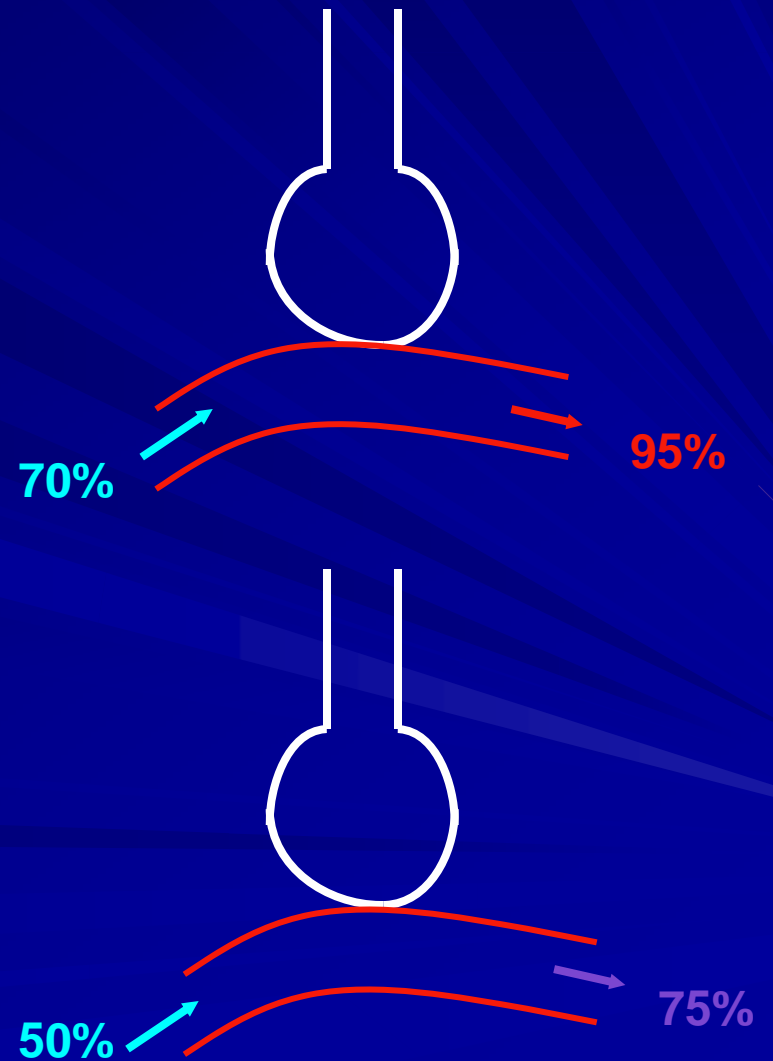
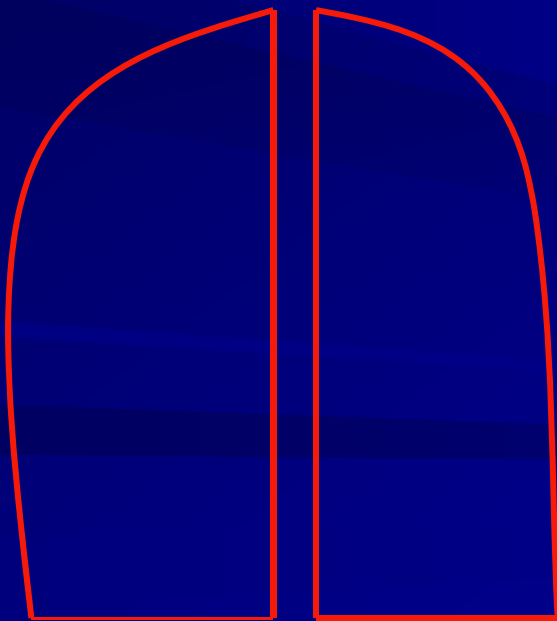
Respiratory Failure- Type II



* PaO₂ corrects readily with supplemental oxygen

Respiratory Failure- Type IV

- Hypoperfusion
- Cardiac output “steal”



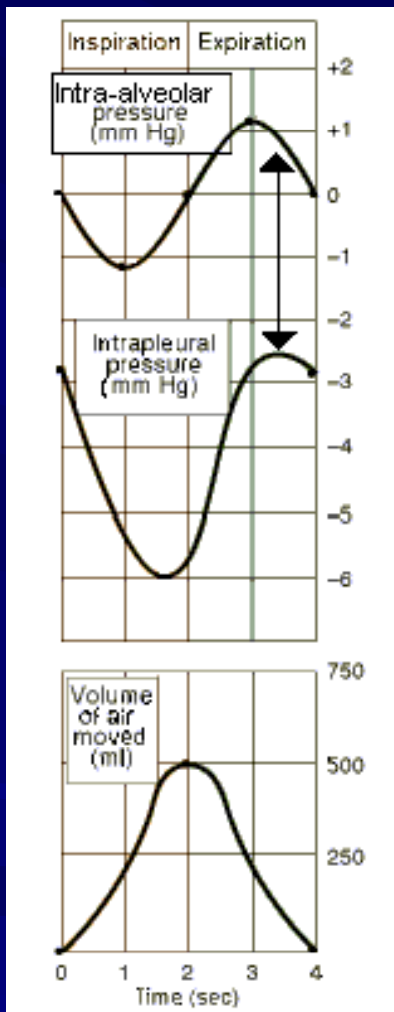
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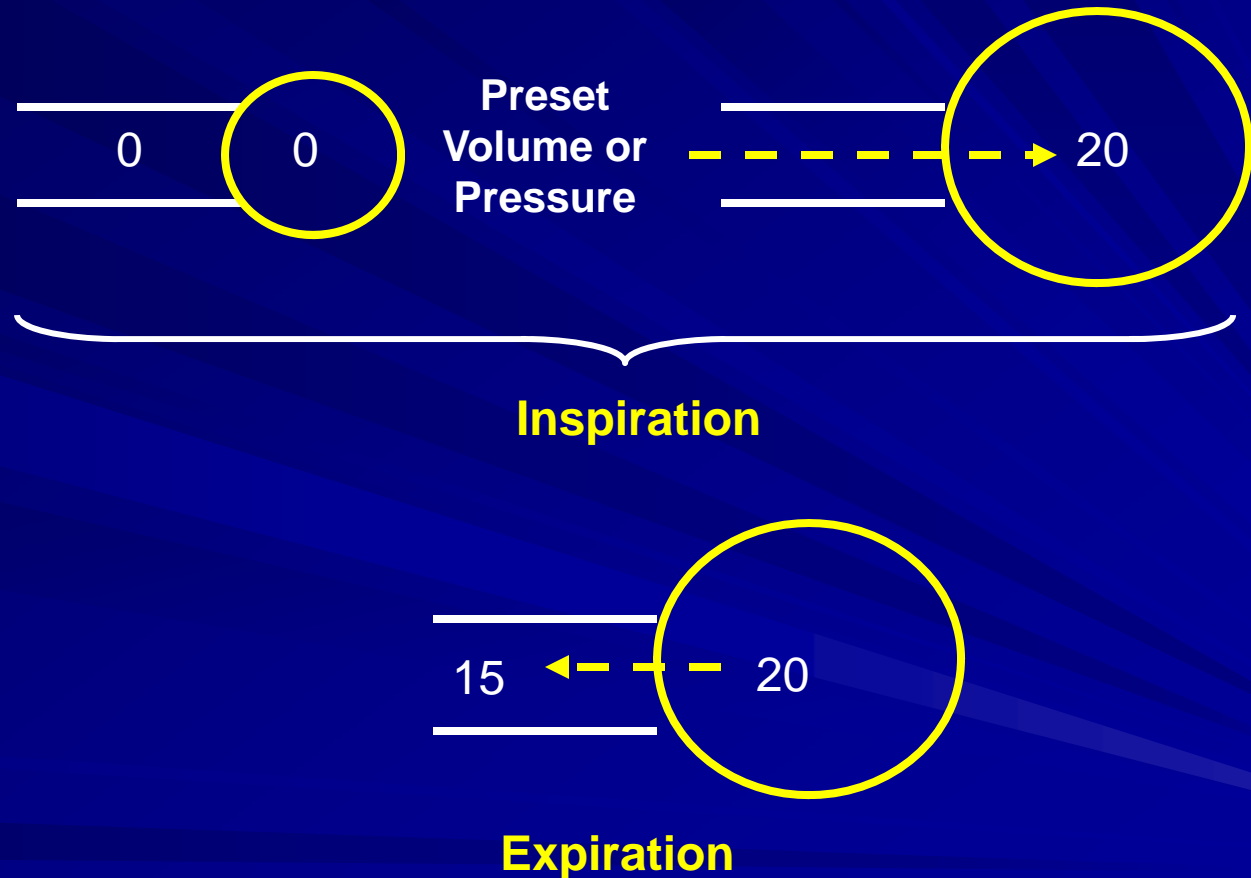
Indications for Mechanical Ventilation

- Inadequate Oxygenation
- Inadequate Ventilation
- Shock
- Airway Protection
- Elective (e.g. Sx)

Mechanical Ventilation



Normal Respiration



Positive Pressure Ventilation

Principles of Mechanical Ventilation

■ Equation of Motion

– Driving Pressure

■ = **Resistive load + Elastic load**

■ = **Airways Resistance + (lung and chest wall) Elastance**

Mechanical Ventilation

■ Ventilation

- Volume or Pressure Modes
- Compliance determines:
 - Alveolar pressure
 - Tidal volume

■ Oxygenation

- PEEP & FiO₂

Mechanical Ventilation

■ GOALS

- Maximal Rest
- Meet minute ventilatory requirements
- Patient-Ventilator Synchrony

■ Avoid

- Respiratory Alkalosis
- Barotrauma/Volutrauma
- Auto-PEEP

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ALI & ARDS Definition

- Acute onset
- Bilateral infiltrates
- $\text{PaO}_2/\text{FiO}_2$
 - < 300 mm Hg for ALI
 - ≤ 200 mm Hg for ARDS
- No evidence of pulmonary venous congestion
 - $\text{PCWP} \leq 18$ mm Hg

ALI & ARDS Epidemiology

- 64 to 86.2 cases/100,000 person-years
- ~142,000 - 191,000 annual cases
- Risk factors
 - EtOH abuse
 - Poor nutritional status
 - Increased age
 - Increased APACHE score

Rubinfeld GD, Caldwell E, Peabody E, et al. *NEJM* 2005;353:1685-93.

MacCallum NS; Evans TW. *Curr Opin Crit Care* 2005;11(1):43-9

From Hall, Schmidt & Wood, eds: *Principles of Critical Care*, 3rd ed. New York, McGraw-Hill 2005.

ARDS Causes

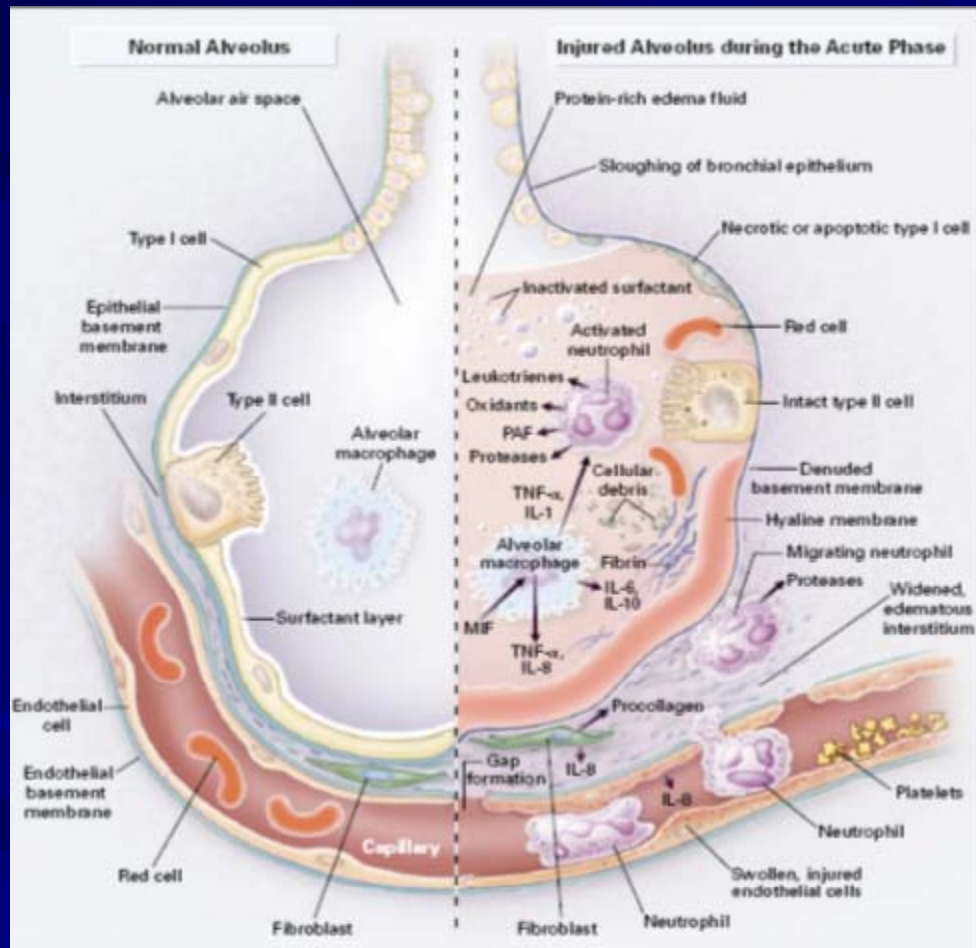
Direct

- Pneumonia
- Aspiration
- Inhalational injury
(e.g. heroin/crack)
- Lung contusion
- Near-drowning

Indirect

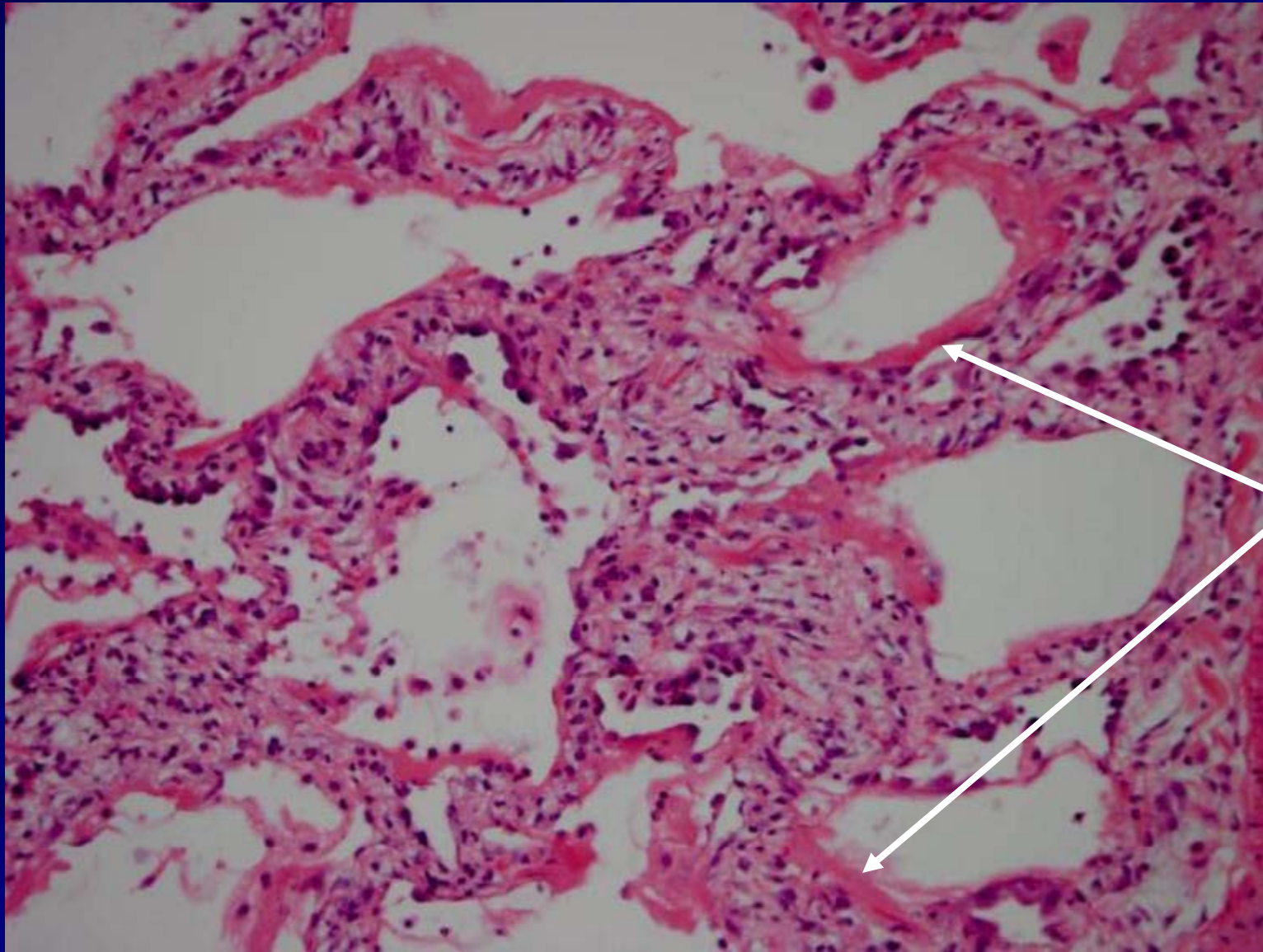
- Sepsis
- Trauma
- Pancreatitis
- Burns
- Air, Amniotic fluid or Fat
Emboli
- Drug Reaction
- Transfusion of Blood
Products
- D.I.C.

ARDS- Basic Pathophysiology



- Alveolar flooding
 - \uparrow permeability alveolar-capillary barrier
 - Endothelial & epithelial injury
 - Surfactant depletion
- Inflammatory injury
 - TNF α , IL1, IL6
- Coagulation abnormalities

ARDS- Histopathology



Hyaline
Membranes

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ARDS- Clinical Presentation

■ History:

- Acute onset: 4-48 hrs

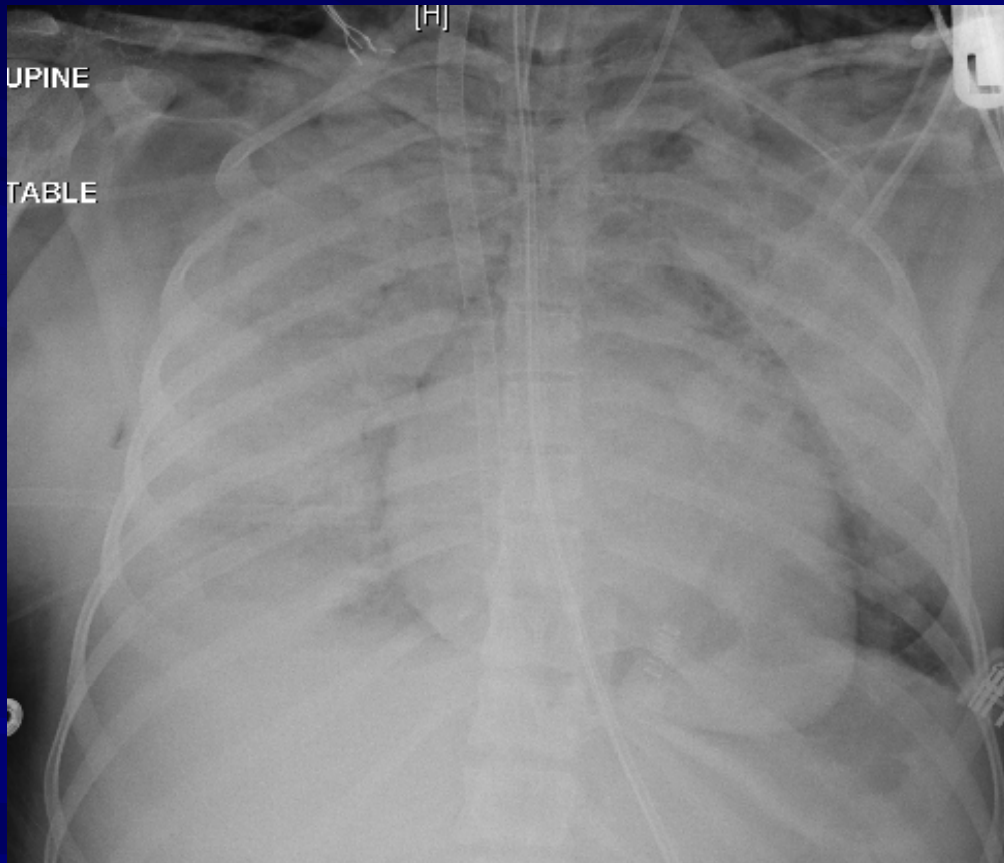
■ Symptoms:

- Tachypnea, Dyspnea

■ Exam

- Severe, refractory hypoxemia
- Diffuse “wet” crackles on lung exam

ARDS- Radiographically



- Bilateral infiltrates
- Consolidation
 - May be patchy
 - Often dependent
- Kerley B lines absent
- +/- pleural effusions & atelectasis

ARDS- Radiographically



ARDS- Differential Diagnosis

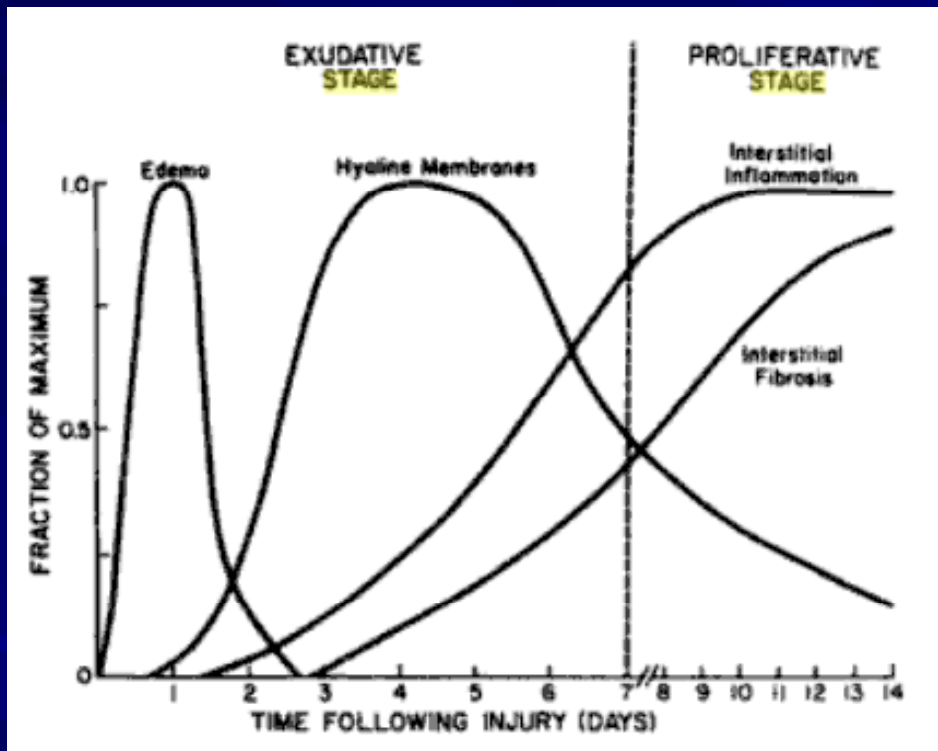
- Congestive heart failure
- Diffuse alveolar hemorrhage
- Acute eosinophilic PNA
- Acute interstitial PNA

- Less commonly:
 - Pulmonary alveolar proteinosis
 - Hypersensitivity pneumonitis
 - Cryptogenic organizing PNA

ARDS- Dx Evaluation

- Basic Labs (CBC, BMP)
- Chest X-ray
- ECG & Echocardiogram
- Bronchoalveolar lavage

ARDS- Clinical Course



■ Exudative Stage

- Refractory hypoxemia
- Intrapulmonary shunt
- Decreased compliance

■ Proliferative Stage

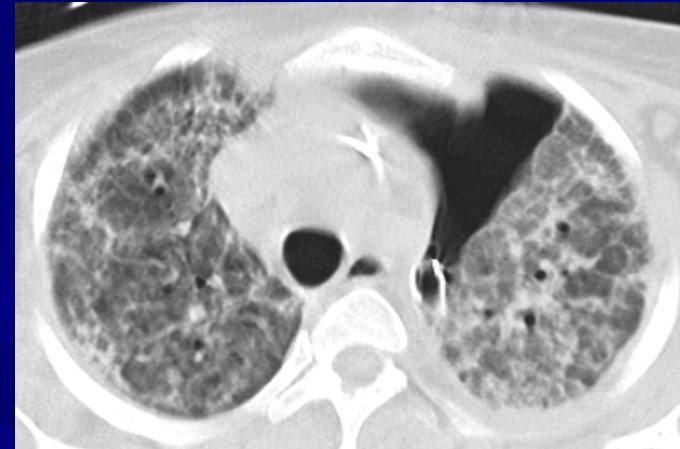
- Increased dead space & V_E
- Pulmonary HTN

■ Resolution

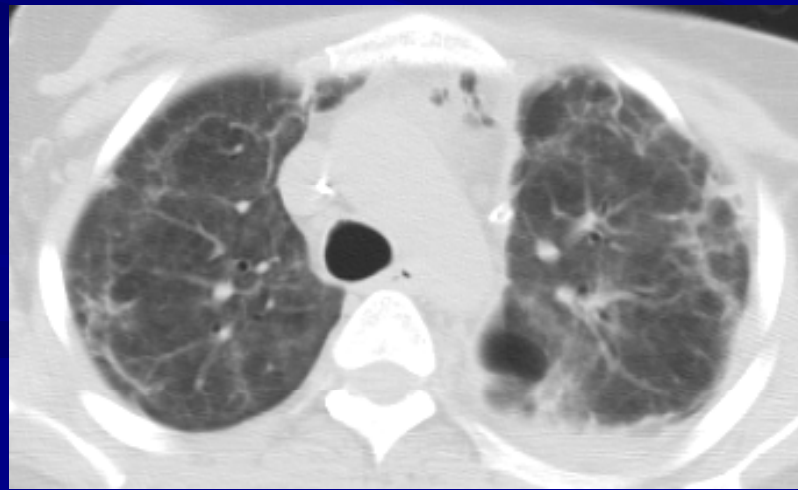
ARDS- Clinical Course



Exudative



Fibroproliferative



Resolution

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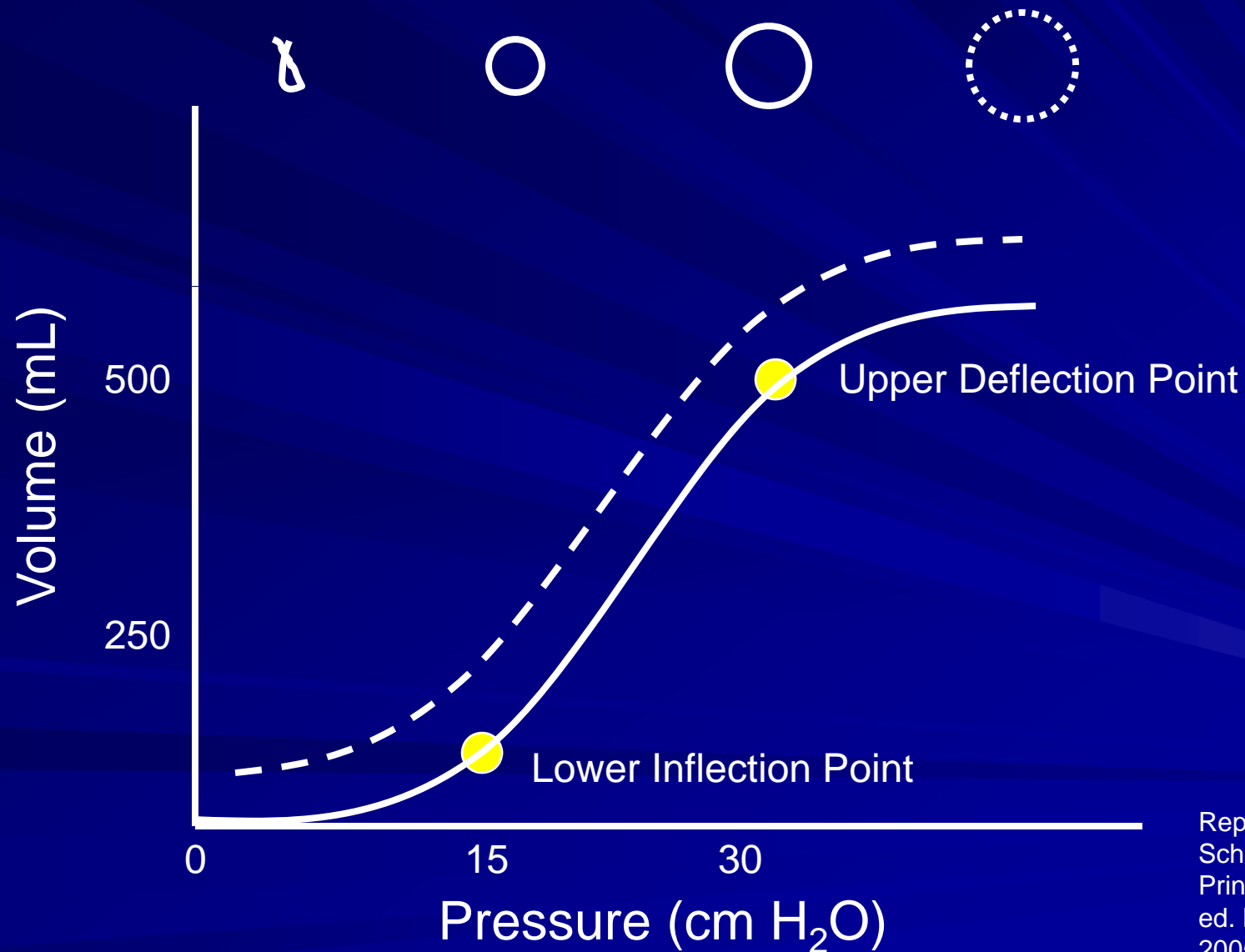
ARDS- Treatment

Treat the Underlying Cause!!!

ARDS- Treatment

- Maintain “adequate” oxygenation
 - O_2 Sat ~ 88-90%
- Avoid “toxic” $F_I O_2$ exposure
- **Lung protective ventilation**

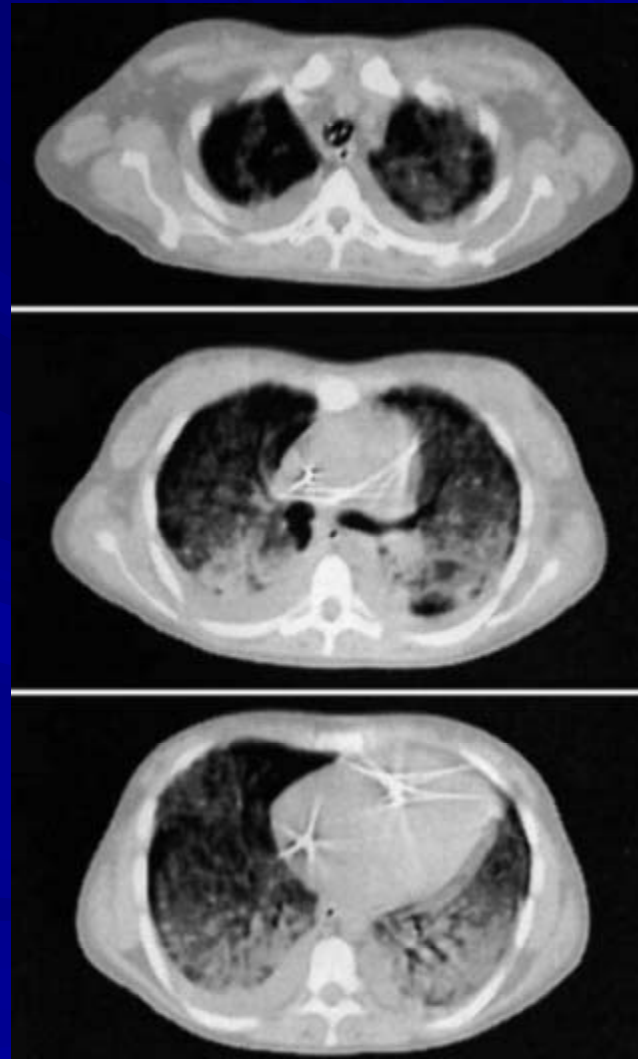
ARDS- PEEP & Oxygenation



Reproduced from Hall, Schmidt & Wood, eds: Principles of Critical Care, 3rd ed. New York, McGraw-Hill 2005.

ARDS- “Volutrauma”

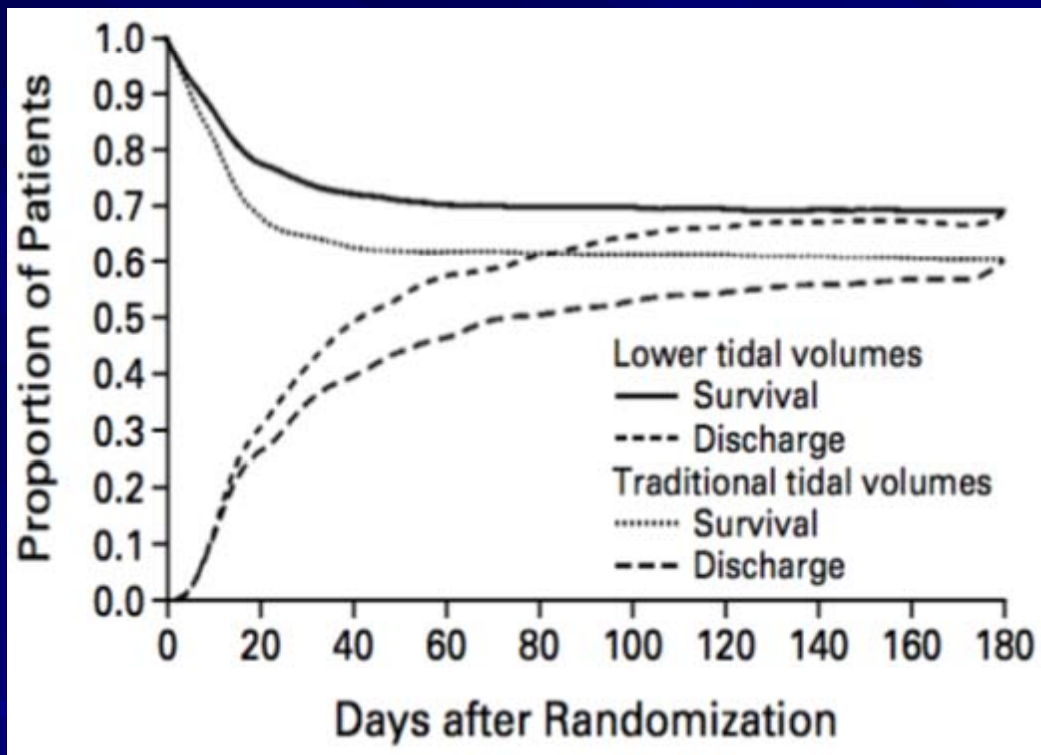
- High-tidal volumes lead to ALI
- “Baby Lungs”



Gattoni L, Pesenti A. *Inten Care Med* 2005 31:776-784

From Hall, Schmidt & Wood, eds: *Principles of Critical Care*, 3rd ed. New York, McGraw-Hill 2005.

ARDS- Low tidal volume ventilation



- 9% reduction in mortality
- Lower IL-6 levels
- > days without nonpulmonary organ failure

ARDS Mortality & Prognosis

Mortality

- Underlying Dz
- Multiorgan failure
- < often due to refractory hypoxemia

**Mortality
34-58%**

Long-Term Sequelae

- Neurocognitive deficits
- Neuromuscular weakness
- Neuropsychologic effects
- Decreased HRQL

Risk Factors for Death

- Age
- > physiologic severity of illness
- + Shock on admit
- Immunosuppression

Pulmonary Function

- Decreased diffusing capacity
- Obstructive & Restrictive Deficits observed

Thank you
Questions. . . ?