Acute Respiratory Distress Syndrome

(ARDS)

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Acute Respiratory Distress Syndrome

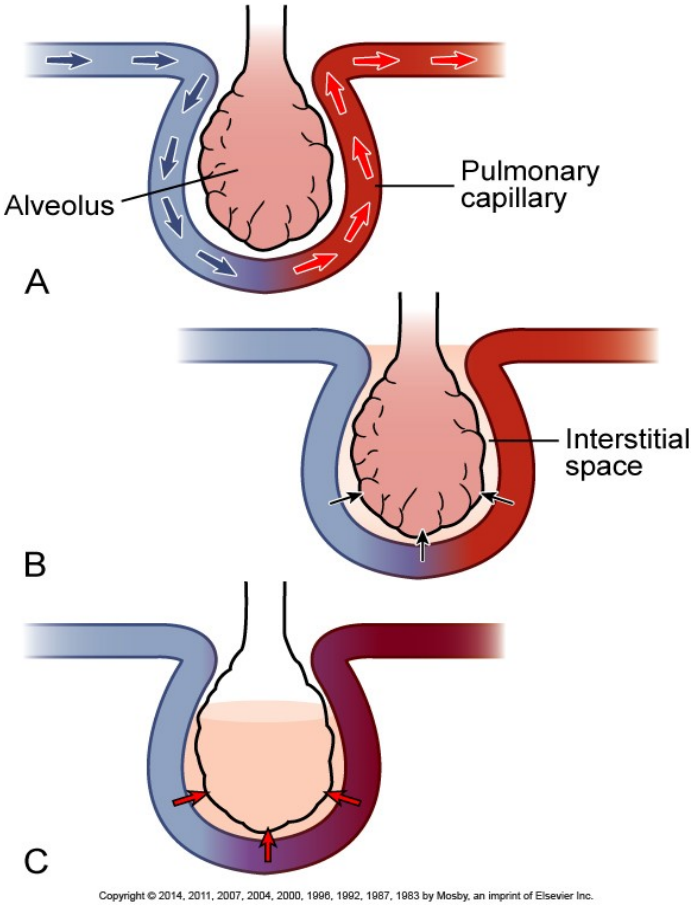
- Sudden, progressive form of acute respiratory failure
- ARDS – PaO_2/FIO_2 ratio is < 200
  - PaO2/FiO2 normal = >400
  - *Normal lungs on RA (21%) (95/.21 = 452)*
  - *ARDS (67/0.55) = 122*
- Alveolar capillary membrane becomes damaged and more permeable to intravascular fluid
  - Alveoli fill with fluid

Acute Respiratory Distress Syndrome

- Severe dyspnea
- Hypoxemia
- ∀↓ Lung compliance
- Diffuse pulmonary infiltrates

- 150,000-200,000 cases annually
- Approximately 50% mortality rate
- 70 – 90% with Gram negative septic shock

Stages of Edema Formation in Acute Respiratory Distress Syndrome



A, Normal alveolus and pulmonary capillary

B, Interstitial edema occurs with increased flow of fluid into the interstitial space

C, Alveolar edema occurs when the fluid crosses the blood-gas barrier_

Fig. 67-9

Etiology and Pathophysiology

- Develop from a variety of direct or indirect lung injuries (p. 1597)
- Exact cause for damage to alveolar-capillary membrane not known
- Pathophysiologic changes of ARDS thought to be due to stimulation of inflammatory and immune systems

Etiology and Pathophysiology

- Neutrophils are attracted to pulmonary interstitium and release mediators producing changes in lungs
  - ∀↑ Pulmonary capillary membrane permeability
    - Destruction of elastin and collagen
    - Formation of pulmonary microemboli
    - Pulmonary artery vasoconstriction

Pathophysiology of ARDS

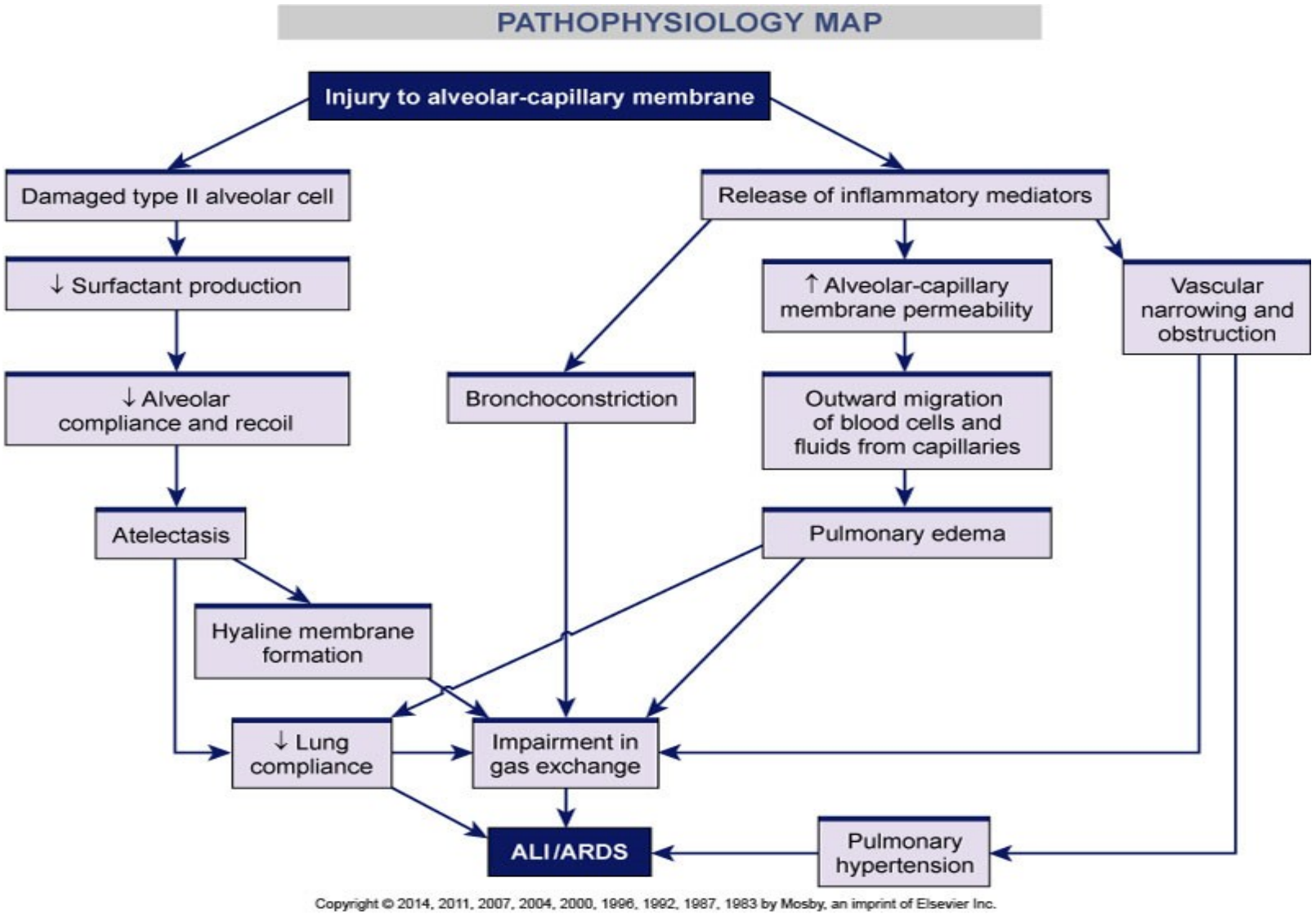


Fig. 67-10

Etiology and Pathophysiology

- **Injury or exudative phase (I)**
  - 1-7 days after direct lung injury or host insult
  - Neutrophils adhere to pulmonary microcirculation
    - Damage to vascular endothelium
    - Increased capillary permeability

Etiology and Pathophysiology

- Fluid crosses into alveolar space
- Alveolar cells type I and II are damaged
  - Surfactant dysfunction → atelectasis
- Hyaline membranes form and line alveoli, limiting diffusion
  - Contribute to atelectasis and fibrosis

Etiology and Pathophysiology

- **Reparative or proliferative phase (II)**
  - 1-2 weeks after initial lung injury
  - Fibroblast proliferation
  - Lung becomes dense and fibrous
  - Lung compliance continues to decrease

Etiology and Pathophysiology

- Hypoxemia worsens
- If reparative phase persists, then widespread fibrosis results
- *If phase is arrested, lesions resolve*

Etiology and Pathophysiology

- **Fibrotic or chronic/late phase (III)**

  -2-3 weeks after initial lung injury

  -Lung is completely remodeled by sparsely collagenous and fibrous tissues (diffuse scarring and fibrosis)

  -Pulmonary Hypertension results from pulm. vascular destruction & fibrosis

Clinical Progression

- Early diagnosis and treatment (phases I-II) improves survival of ARDS
- Poorer outcomes for those who reach phase III.

  -May require long-term mechanical ventilation

Nursing Assessment

- **Early S/S:** ***See table 67-3, p. 1593***
  - **Restlessness, Agitation**
  - **Shallow breathing with increased respiratory rate**
  - **Abnormal breath sounds***
  - **Use of accessory muscles**
  - **Pale, cool, clammy or warm, flushed skin**

Nursing Assessment

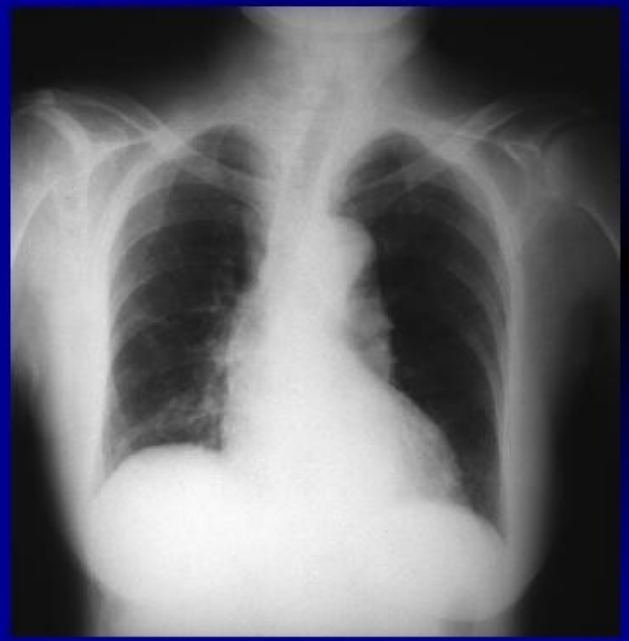
- **Later S/S:**
  - Tachycardia progressing to bradycardia
  - Absent/diminished breath sounds
  - Hypertension progressing to hypotension
  - Somnolence, confusion, delirium

Assessment - Diagnostic Findings

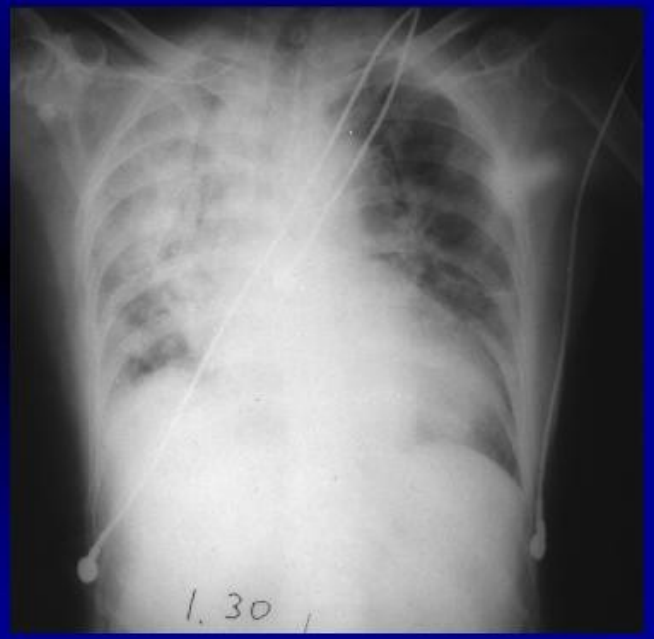
- Changes in ABG's
- Abnormal chest x-ray, CBC, chemistry values
- Changes in pressure limits on ventilator
- Abnormal $ScvO_2$ (SvO_2)
- PaO_2/FIO_2 ratio </= 200

Images

Chest X-ray of ARDS patient

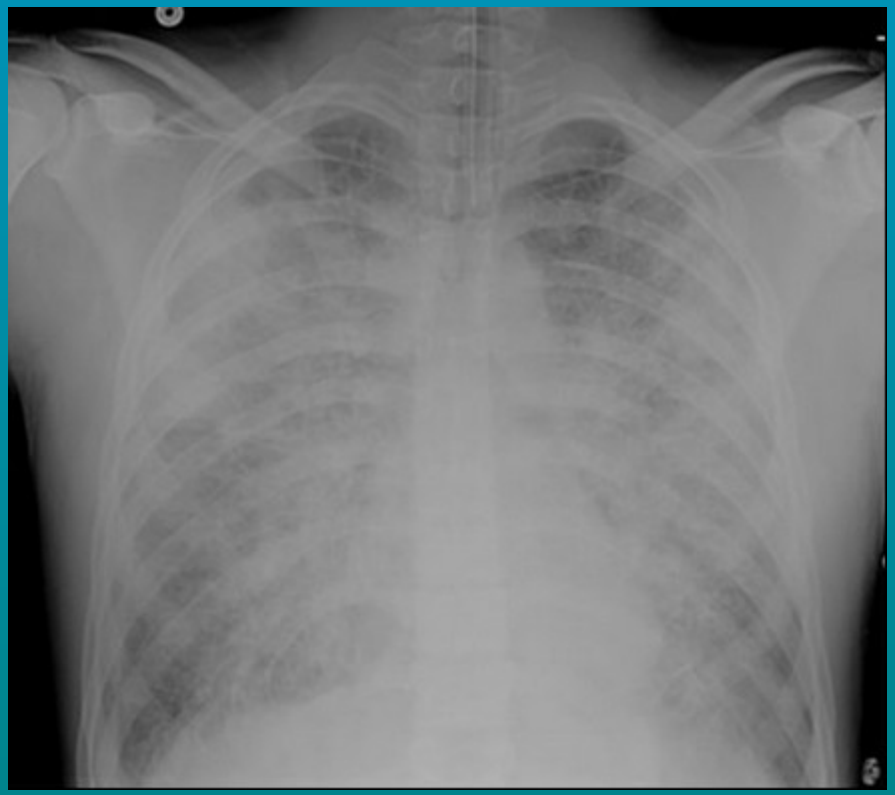


Normal



ARDS

Images



Interventions- Nursing/Medical

- Treat the underlying cause
- Oxygen
  - SpO_2 continuously monitored > 90%
  - Give lowest FiO2 that results in PaO_2 60 mmHg or greater
  - $ScvO_2$ = 60% to 80%

Interventions

- Mechanical ventilation
  - Often necessary to maintain FIO_2 at 60% or greater to maintain PaO_2 at 60 mmHg or greater
  - Risk for O_2 toxicity increases when FIO_2 exceeds 60% for more than 48 hours

Interventions

- PEEP at 5 cm H_2O to compensate for loss of glottic function
  - Opens collapsed alveoli
  - **Increasing levels will be needed**

Interventions

- Additional pressures from PEEP can compromise venous return to right side of the heart
  - Decreases preload, CO, and BP

Interventions

PEEP cont.

- **Can cause hyperinflation of alveoli, compression of capillary bed, reduction in blood return to left side of heart, and reduction in blood pressure**
- **Can result in barotrauma & volu-pressure trauma**

Interventions

- Pressure-control ventilation, reverse I:E ratio
- Prone positioning
  - Plan for immediate repositioning for cardiopulmonary resuscitation
- Lateral rotation therapy to provide continuous, slow, side-to-side turning

Interventions

- Maintenance of cardiac output and tissue perfusion
  - IV fluids, pressors
  - Continuous monitoring (VS, ECG, SpO2)
  - Hemodynamic monitoring (Aline, CO, SvO2)
  - UO hourly

Interventions

- Inotropic/vasopressor medications
- Sedation/analgesia
- Neuromuscular blockade
- Transfuse if Hgb < 8 with SaO_2 ≦90%
- Diuretics as needed & mild fluid restriction
- ECMO
- Monitor daily weights, I&O's to assess fluid status

Complications

Ventilator-Associated Pneumonia

-Risk factors

-Strategies for prevention

-Infection control measures

-Elevating HOB 30-45 degrees

-Mouth care & oral hygiene

-PPI

-Daily sedation holiday

-Evaluation for weaning

Complications

- **Barotrauma** ***(Too much pressure)***
  - **Rupture of overdistended alveoli during mechanical ventilation**
  - **To avoid, ventilate with smaller tidal volumes**
    - **Results in higher $PaCO_2$**
    - ***Permissive hypercapnia***
    - **Keep pH >/= 7.2**

Complications

- **Volutrauma** ***(Too much volume)***
  - Occurs when large tidal volumes used to ventilate noncompliant lungs
    - Alveolar fractures/tears and movement of fluids and proteins into alveolar spaces
  - Avoid by using smaller tidal volumes or pressure control ventilation

Complications

- **Stress Ulcers**
  - Bleeding from stress ulcer occurs in 30% of patients with ARDS on PPV
  - Management strategies include correction of predisposing conditions, prophylactic antiulcer agents, and early initiation of enteral nutrition

Complications

- **Renal Failure**
  - Occurs from decreased renal tissue oxygenation from hypotension, hypoxemia, or hypercapnia
  - May also be caused by nephrotoxic drugs used for infections associated with ARDS
- **Pressure ulcers**

Other – see pg 1600 table 67-7

Goals (Expected Outcomes) of Care

Overall goals for patient with ARDS:

- **PaO_2 >/= 60 mmHg**
- **Adequate lung ventilation to maintain normal pH**

Goals for patient recovering from ARDS

- **PaO_2 normal for age/baseline with FIO_2 21%**
- **SaO_2 >/= 90% on decreasing O2 support**
- **Maintain patent airway**
- **Clear lungs/breath sounds throughout**

CARDIOHELP

- https://www.youtube.com/watch?v=OOjKEPTKSpg
- https://www.youtube.com/watch?v=JCVcSEg7M-w
- https://www.youtube.com/watch?v=MZGgJohtI2g