RESPIRATORY REFERENCE

Acute Respiratory Distress Syndrome (ARDS)

Overview

ARDS is a severe form of acute lung injury (ALI) characterized by diffuse inflammation of the alveolar–capillary membrane, increased vascular permeability, and non-cardiogenic pulmonary edema, resulting in severe hypoxemia and decreased lung compliance.

Pathology

↑ Capillary permeability
↓
Pulmonary edema
↓
↓ Surfactant (Type II pneumocyte injury)
↓
Microatelectasis & ↓ Lung compliance
↓
Shunt + V/Q mismatch
↓
Refractory hypoxemia
↓
↑ PVR
↓
Pulmonary hypertension → ↑ RV workload → RV failure

Clinical Features

- Dyspnea, tachypnea, tachycardia, and ↑ work of breathing (WOB).
- Rapid, shallow breathing due to ↓ lung compliance.
- Bilateral inspiratory crackles on auscultation.
- Chest X-ray: Bilateral diffuse infiltrates (“fluffy infiltrates”) with air bronchograms.
- ABG: Moderate–severe refractory hypoxemia, initially respiratory alkalosis with ↑ A–a gradient.
- PCWP ≤18 mmHg, supporting non-cardiogenic pulmonary edema (ARDS) rather than cardiogenic pulmonary edema.

Treatment

- Treat the underlying cause (e.g., sepsis, pneumonia).
- Provide supportive care.
- Mechanical ventilation is the cornerstone of management.

Berlin Definition of ARDS (2012)

Timing	Onset within 1 week of a known clinical insult or new/worsening respiratory symptoms.
Chest Imaging	Bilateral opacities on chest X-ray or CT, not fully explained by pleural effusion, lung collapse, or nodules.
Oxygenation	Mild 201–300 mmHg Moderate 101–200 mmHg Severe ≤100 mmHg

Use to diagnose ARDS and classify its severity

When arterial blood gases are unavailable, the SpO_2/FiO_2 (S/F) ratio may be used as a surrogate for the PaO_2/FiO_2 (P/F) ratio to estimate ARDS severity.

ARDS Protocol

1. Lung-Protective Ventilation
   - VT: 4–6 mL/kg PBW.
   - Pplat: ≤30 cmH_2O.
2. Oxygenation Strategy
   - Titrate PEEP and FiO_2 using PEEP/FiO_2 tables.
   - Target SpO_2: 88–95%.
3. Permissive Hypercapnia
   - Accept elevated $PaCO_2$ provided pH ≥7.30.
4. Adjunctive Therapies (Severe ARDS)
   - Prone positioning: ≥12–16 h/day.
   - Neuromuscular blockade: First 48 hours if indicated.
   - Conservative fluid strategy: Minimize pulmonary edema and facilitate ventilator liberation.

RESPIRATORY REFERENCE

Pneumothorax

Overview

is the presence of air within the pleural space, resulting in partial or complete lung collapse due to loss of negative intrapleural pressure.

Clinical Features

- Sudden pleuritic chest pain
- Acute dyspnea
- Decreased/absent breath sounds
- Hyperresonance to percussion
- ↓ Chest expansion
- Tension pneumothorax: Hypotension, JVD, tracheal deviation (late), tachycardia

Management

- Observation + oxygen (small, stable pneumothorax)
- Needle aspiration (selected spontaneous cases)
- Chest tube (large or symptomatic pneumothorax)
- Tension pneumothorax: Immediate needle decompression followed by chest tube (do not wait for imaging)

Pneumothorax size is determined by measuring the maximum distance between the visceral pleural line and the inner chest wall at the level of the hilum: <2 cm = Small, ≥2 cm = Large (BTS).

Classification

1. Primary Spontaneous → No underlying lung disease.
2. Secondary Spontaneous → Occurs in patients with underlying lung disease (e.g., COPD).
3. Traumatic → Due to blunt or penetrating chest trauma.
4. Tension Pneumothorax → Life-threatening, progressive air trapping causes mediastinal shift and hemodynamic instability.

Tension Pneumothorax

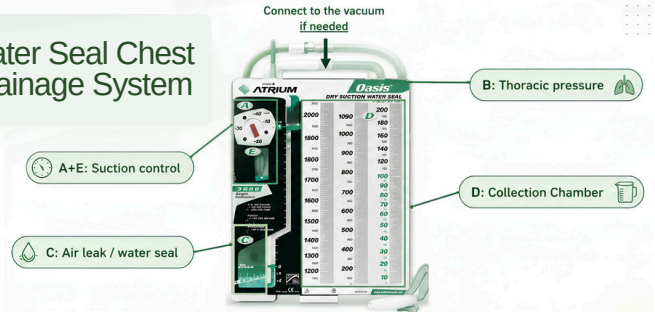
A life-threatening type of pneumothorax caused by a one-way valve mechanism, allowing air to enter the pleural space without escaping. This progressively increases intrathoracic pressure, causing lung collapse, mediastinal shift, reduced venous return, and obstructive shock.

requires immediate needle decompression followed by chest tube insertion—do not delay treatment for imaging.

Diagnosis

- Chest X-ray (first-line)
- ultrasound
- CT chest (gold standard if diagnosis is uncertain)

Water Seal Chest Drainage System



Remove Chest Tube

Meeting the following criteria:

- Resolving of the underlying disease
- Wide-open lung in CXR of the chest tube
- Stable clinical condition
- Positive response to the clamping trial (6–24 hrs)
- Decrease drainage to <200 cc or no air leak in 24 hrs

RESPIRATORY REFERENCE

Bronchial Asthma

Overview

Asthma is a chronic inflammatory airway disease characterized by reversible airflow obstruction, airway hyperresponsiveness, and bronchoconstriction.

Pathology

Trigger exposure
↓
Airway inflammation
↓
Bronchoconstriction + mucosal edema + mucus hypersecretion
↓
Reversible airflow obstruction

Clinical Features

Episodic wheezing | dyspnea | chest tightness | cough

Symptoms are variable and reversible.

Diagnosis

- Spirometry: ↓FEV_1 and ↓FEV_1/FVC.
- Bronchodilator reversibility: ↑FEV_1 ≥12% and ≥200 mL confirms asthma.
- PEF variability ≥15% supports the diagnosis.
- Methacholine challenge: Positive if FEV_1 decreases ≥20%, used when spirometry is normal but asthma is suspected.
- FeNO: Elevated in eosinophilic asthma (supportive, not diagnostic).

Asthma Types

1. Allergic (Extrinsic)	IgE-mediated most common type.
2. Non-allergic (Intrinsic)	Not IgE-mediated
3. Exercise-induced	Triggered by physical activity.
4. Occupational	workplace allergens or irritants.
5. Cough-variant	Triggered by aspirin or NSAIDs.

- Asthma is characterized by reversible airflow obstruction, unlike COPD.
- ICS is the cornerstone of long-term management.
- A normal or rising $PaCO_2$ during an acute asthma attack is a sign of impending respiratory failure.
- Methacholine challenge is used when asthma is suspected but spirometry is normal.
- During MV, the priority is to prevent air trapping and auto-PEEP, not to normalize $PaCO_2$.

Treatment

Long-Term Control

- ICS (cornerstone of therapy).
- ICS/LABA for persistent or uncontrolled asthma.
- LAMA or biologic therapy for severe asthma.
- Avoid triggers and provide patient education.

Acute Exacerbation

- Oxygen: Target SpO_2 94–98%.
- SABA (Albuterol): First-line bronchodilator.
- SAMA (Ipratropium): Add in moderate–severe attacks.
- Systemic corticosteroids: Early administration.
- IV Magnesium sulfate: For severe or life-threatening exacerbations.
- Mechanical ventilation: If respiratory failure develops.

RESPIRATORY REFERENCE

Chronic Obstructive Pulmonary Disease (COPD)

Overview

Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable chronic respiratory disease characterized by persistent, progressive airflow limitation resulting from chronic airway and/or alveolar abnormalities

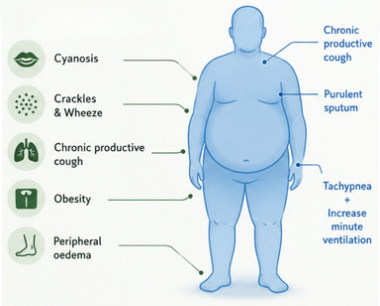
Chronic bronchitis (blue bloater)

A subtype of COPD characterized by chronic inflammation of the bronchi, resulting in mucus hypersecretion, airway narrowing, and persistent airflow limitation. Clinically, it is defined as a productive cough for at least 3 months per year for 2 consecutive years, after excluding other causes.

Pathophysiology

Chronic airway inflammation
↓
Goblet cell & mucus gland hypertrophy
↓
Excess mucus + airway edema
↓
Airway narrowing & mucus plugging
↓
Airflow obstruction → Hypoxemia & hypercapnia

Clinical Features



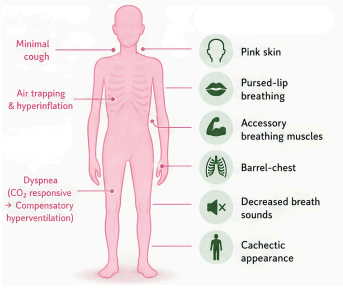
Emphysema (pink puffer)

A subtype of COPD characterized by permanent destruction of the alveolar walls and enlargement of distal airspaces, leading to loss of elastic recoil, air trapping, hyperinflation, and progressive airflow limitation.

Pathophysiology

Alveolar wall destruction
↓
Loss of elastic recoil
↓
Air trapping & hyperinflation
↓
Reduced gas exchange surface area
↓
Progressive dyspnea

Clinical Features



Investigation

- Spirometry (Gold standard): Post-BD $FEV_1/FVC < 0.70$
- CXR / HRCT: Hyperinflation, emphysema, exclude other diseases
- ABG & SpO_2: Assess hypoxemia and hypercapnia
- CBC: Polycythemia or anemia
- Sputum culture: During suspected infection
- α_1-Antitrypsin level: Young patients or family history

Management

Pharmacological treatment:

- Bronchodilators:

B2 Agonists (Short and long acting).
Anticholinergic (Short and long acting).
Theophylline (Short and long acting).

- Corticosteroids:

Phosphodiestrase: (Roflumilast)

Prevention

- Smoking cessation.
- Reduce or prevent air pollution.
- Vaccines.

- Target SpO_2: 88–92%.
- Nasal cannula or Venturi mask.
- If inadequate → NIV (BiPAP).
- If NIV fails → Mechanical Ventilation (MV).

COPD Exacerbation

Acute worsening of COPD symptoms (↑ dyspnea, ↑ cough, ↑ sputum) that requires additional treatment.

Management: Bronchodilators + corticosteroids ± antibiotics + oxygen (SpO_2 88–92%) ± NIV (BiPAP).

Patients with chronic hypercapnic COPD are at risk of oxygen-induced hypercapnia. Therefore, oxygen should be titrated to maintain an SpO_2 of 88–92%, rather than giving high concentrations routinely. Excessive oxygen can worsen V/Q mismatch and the Haldane effect, leading to further CO_2 retention and respiratory acidosis.

RESPIRATORY REFERENCE

Respiratory Failure

Overview

Respiratory failure is the inability of the respiratory system to maintain adequate oxygenation and/or eliminate carbon dioxide, resulting in hypoxemia, hypercapnia, or both.

Respiratory Failure Type 1

PaO_2 <60 mmHg with normal or low $PaCO_2$.

Common Causes

- Pneumonia
- ARDS
- Pulmonary edema
- Pulmonary embolism
- Pneumothorax

Management

- Oxygen therapy (CPAP/PEEP)
- Treat the underlying cause.

Respiratory Failure Type 2

$PaCO_2$ >50 mmHg (usually with PaO_2 <60 mmHg).

Common Causes

- COPD exacerbation
- Severe asthma
- Neuromuscular disorders
- CNS depression (opioids/sedatives)
- Obesity hypoventilation syndrome

Management

- Improve ventilation (NIV/BiPAP)
- Intubation & MV if NIV fails
- Treat underlying cause

Clinical Features

- Dyspnea
- Tachypnea
- Accessory muscle use
- Cyanosis
- Altered mental status
- Tachycardia
- Severe cases: bradypnea, coma, respiratory arrest

Diagnosis

- Arterial Blood Gas (ABG) (Gold standard):

Type I: PaO_2 <60 mmHg
Type II: $PaCO_2$ >50 mmHg

- Pulse oximetry (SpO_2): Assesses oxygen saturation.
- Chest X-ray / CT: Identifies the underlying cause.
- Clinical assessment: Dyspnea, tachypnea, increased work of breathing, cyanosis, altered mental status.

Acute Respiratory Failure	Chronic Respiratory Failure	Acute-on-Chronic Respiratory Failure
• **Sudden onset** • **No renal compensation** • **Low pH (<7.35)** with abnormal ABG • Requires urgent management	• Gradual onset • Renal compensation (↑ HCO_3^-) • Normal or near-normal pH • Common in COPD and neuromuscular diseases	• Acute deterioration in a patient with chronic RF • ↑ **$PaCO_2$** + ↓ **pH** + ↑ **HCO_3^-** • Commonly seen during **COPD exacerbations**

Indications for MV

- Refractory hypoxemia
- Severe hypercapnia with acidosis (pH <7.25)
- Increased work of breathing
- Respiratory muscle fatigue
- Altered consciousness
- Apnea or impending respiratory arrest