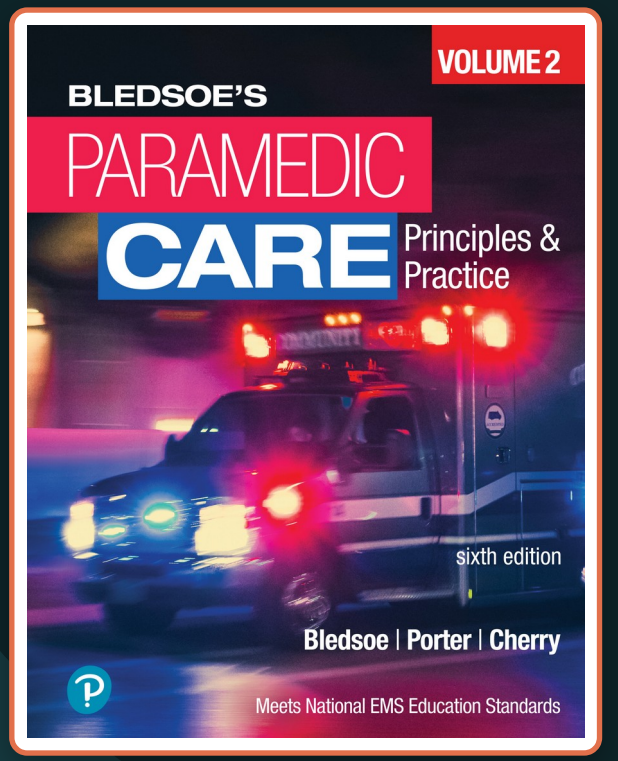
Paramedic Care: Principles & Practice, Sixth Edition

Pulmonology

Chapter 32



Pearson

Chapter Roadmap

01
Respiratory Anatomy & Physiology
Upper and lower airway structures, ventilation, diffusion, and perfusion

02
Pathophysiology
How disease disrupts ventilation, diffusion, and perfusion

03
Assessment
Scene size-up through secondary assessment and diagnostic testing

04
Management Principles
Oxygenation goals and avoiding hyperoxia

05
Specific Respiratory Diseases
Obstructive, infectious, vascular, and neuromuscular causes of dyspnea

06
Summary
Key takeaways for prehospital respiratory care

Introduction

Foundations

- The respiratory system delivers oxygen to the tissues and removes carbon dioxide, the byproduct of metabolism
- Oxygen must be constantly available to convert nutrients into usable energy
- Intrinsic risk factors come from within the patient — genetic predisposition, underlying cardiac or circulatory disease, and stress level can all worsen a respiratory complaint
- Extrinsic risk factors are external — cigarette smoking is the single most important risk factor; environmental pollutants also contribute

01

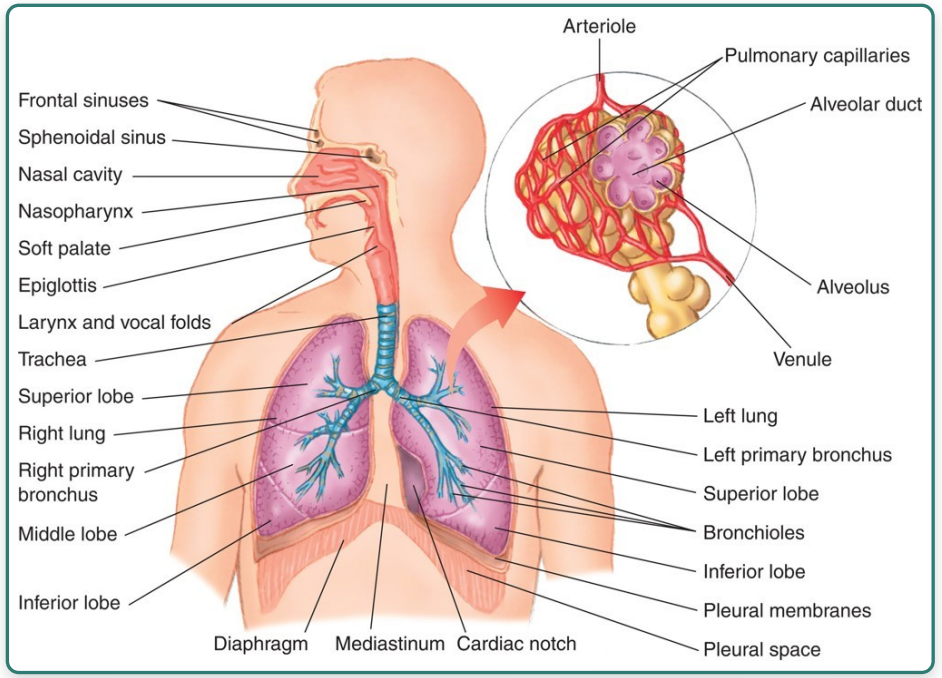
Review of Respiratory Anatomy & Physiology

From nose to alveolus

Upper Airway Anatomy

Anatomy

- Air enters through the nose, passes the external nares, and enters the nasal cavity
- The nasal septum divides the cavity into two chambers; anterior hair follicles trap large dust particles
- Turbinates create turbulence that filters and humidifies incoming air; mucus and cilia sweep debris posteriorly to be swallowed
- Kiesselbach’s plexus, in the lower septum, warms inspired air
- Paranasal sinuses (frontal, ethmoid, sphenoid, maxillary) are air-filled cavities in the skull; the superior nasal cavity holds the olfactory nerve fibers

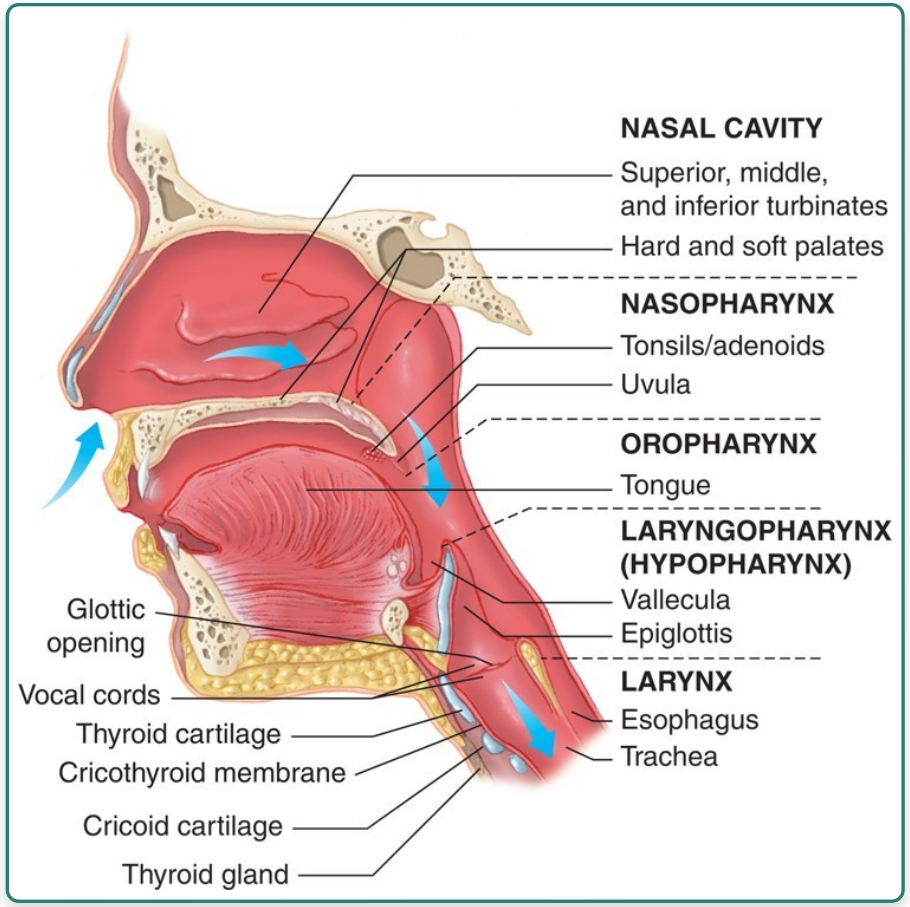


Overview of the upper and lower airways

Upper Airway Anatomy (continued)

Anatomy

- The pharynx is a funnel-shaped structure connecting the nose and mouth to the larynx, with three divisions: nasopharynx, oropharynx, laryngopharynx
- Beyond speech, the larynx filters the digestive and respiratory tracts
- During inspiration the paired laryngeal cartilages stay separated and the epiglottis sits upright, allowing air into the trachea
- During swallowing the epiglottis tips backward and the cartilage pairs close, diverting food into the esophagus instead

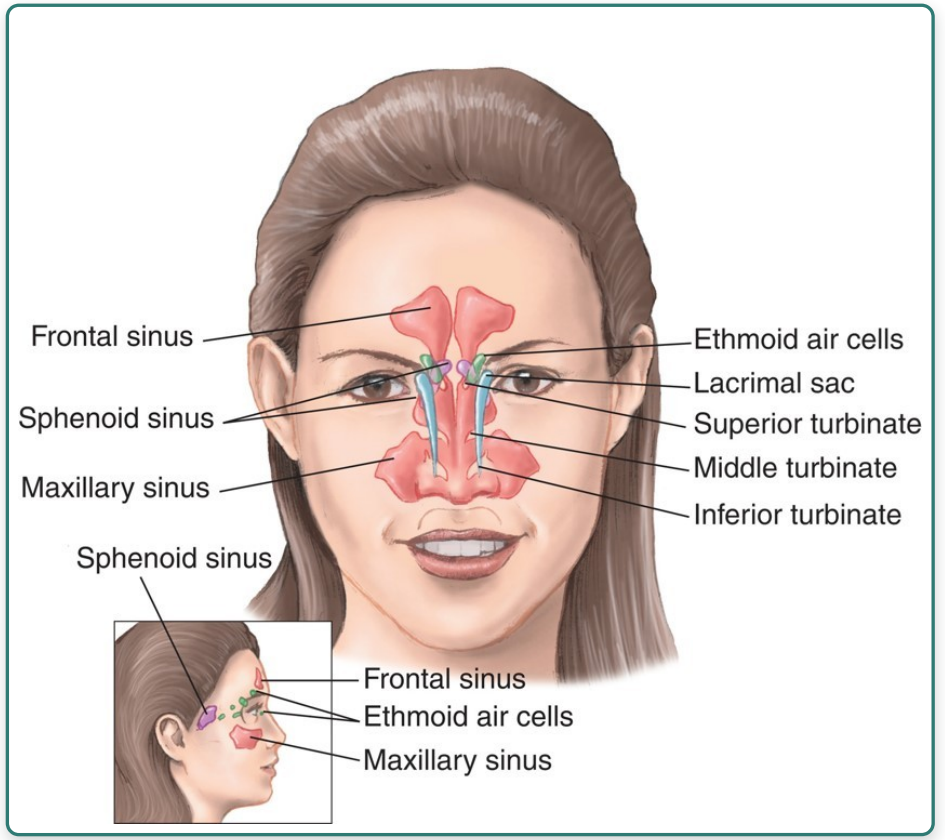


Anatomy of the upper airway

Paranasal Sinuses & Airway Defenses

Anatomy

- In the nose, cilia produce a steady posterior mucus flow that removes trapped particles
- Once mucus and particles reach the nasopharynx, they are swallowed and cleared via the digestive tract
- The paranasal sinuses (frontal, ethmoid, sphenoid, maxillary) lighten the skull and warm/humidify air

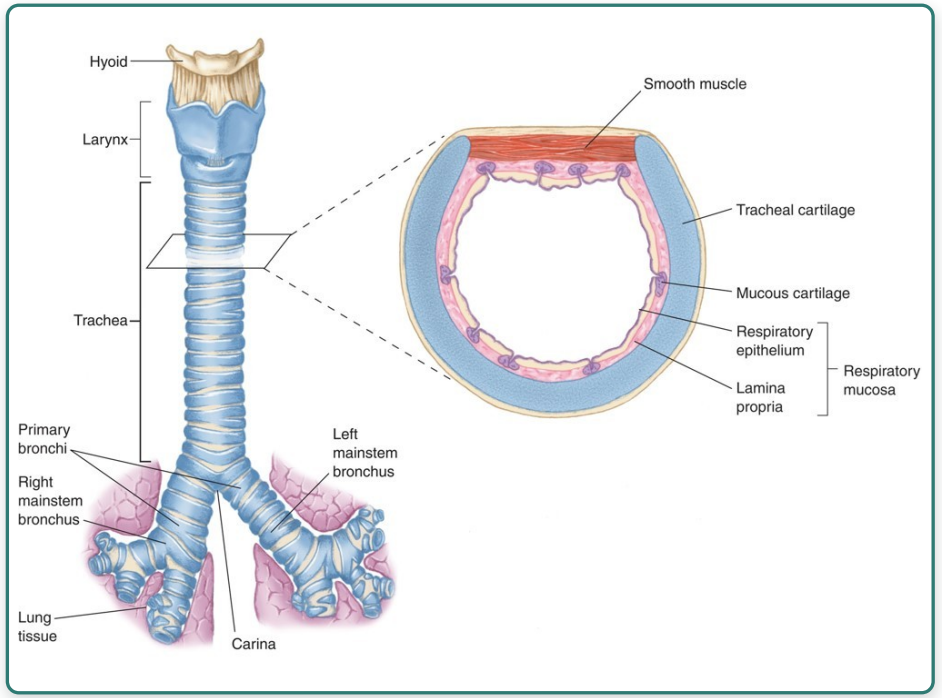


Paranasal sinuses

Lower Airway Anatomy

Anatomy

- During inspiration air exits the upper airway, passes through the larynx, and enters the trachea
- The trachea is about 11 cm long, made of C-shaped cartilaginous rings, and splits into the right and left mainstem bronchi
- Bronchi branch into progressively smaller bronchioles that carry air throughout the lungs
- The right lung has superior, middle, and inferior lobes; the left lung has superior and inferior lobes with a cardiac notch

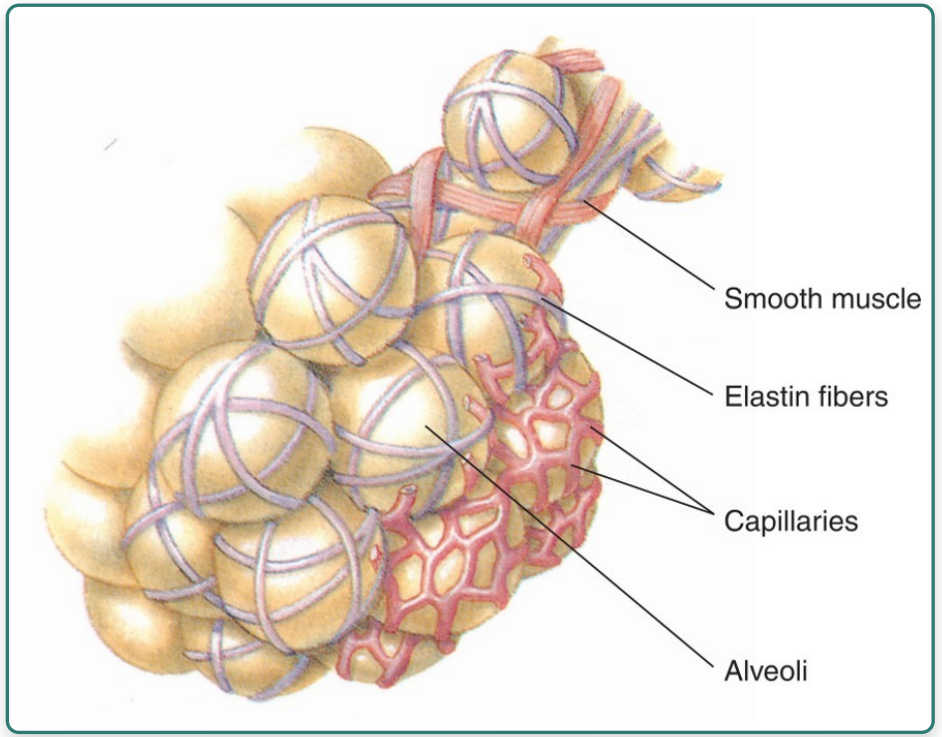


Anatomy of the lower airway

Lower Airway Anatomy (continued)

Anatomy

- Bronchioles terminate at the alveoli — tiny air sacs surrounded by a dense network of pulmonary capillaries where gas exchange occurs
- The alveolar-capillary membrane must stay thin and intact for oxygen and carbon dioxide to diffuse efficiently
- The diaphragm separates the thorax from the abdomen; pleural membranes and the pleural space allow the lungs to move smoothly against the chest wall
- The mediastinum is the space between the lungs, containing the heart and great vessels



The alveoli and the pulmonary capillaries

Physiologic Processes: Ventilation

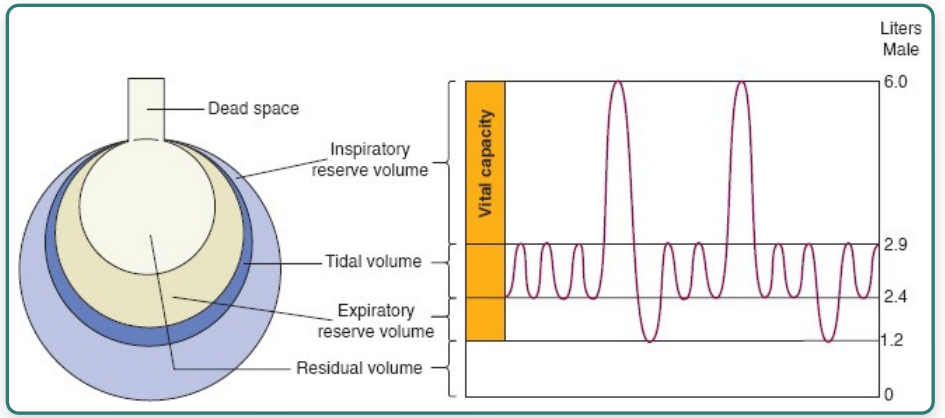
Physiology

- Gas exchange is the respiratory system's core function — oxygen in, carbon dioxide out — and depends on three processes: ventilation, diffusion, and perfusion
- Ventilation is the mechanical movement of air in and out of the lungs; it requires an intact chest wall, nerve pathways, diaphragm, pleural cavity, and brainstem
- Ventilation has two phases — inspiration (always active, requires energy) and expiration (normally passive, driven by elastic recoil)
- Airflow into the lungs depends on the pressure gradient between atmosphere and chest cavity, plus airway resistance and lung compliance (the ease with which the chest expands)

Lung Volumes & Capacities

Physiology

- Tidal volume: ≈500 mL moved in a quiet breath by a 70-kg adult
- Inspiratory / expiratory reserve volume: extra air that can be drawn in or forced out beyond a normal breath
- Residual volume: air that always remains in the lungs, keeping alveoli open
- Inspiratory capacity, functional residual capacity, vital capacity, and total lung capacity are all derived by combining these volumes
- Peak flow measures the maximum rate of airflow during forced expiration; some air (anatomical and alveolar dead space) never reaches functioning alveoli

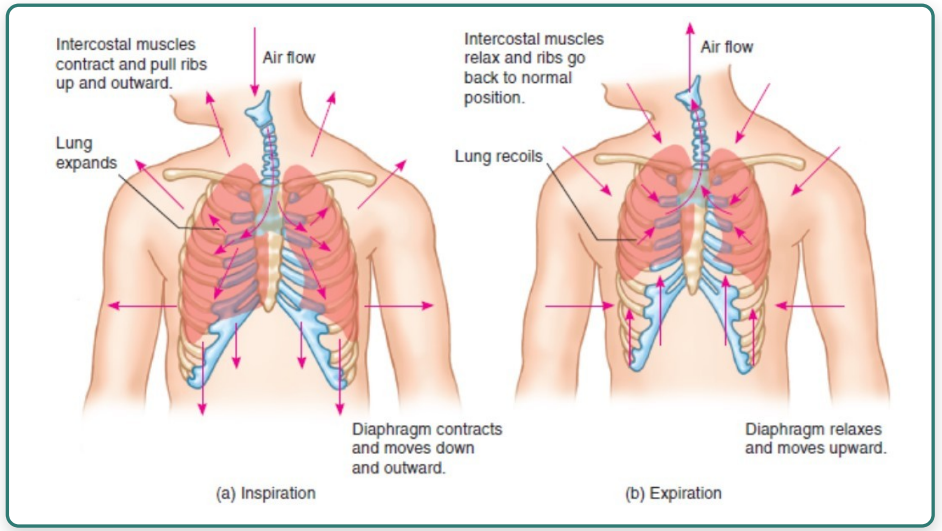


The lung volumes

Regulation of Ventilation

Physiology

- The medulla (lower brainstem) contains the inspiratory and expiratory centers and signals the diaphragm and intercostal muscles via the phrenic and intercostal nerves
- Stretch receptors feed back to the medulla to prevent overinflation (the Hering-Breuer reflex)
- Arterial CO2 (PaCO2) is the primary driver of ventilatory rate — a rise in PaCO2 lowers blood pH, which chemoreceptors detect and answer with an increased ventilatory rate
- In chronic COPD, the body becomes less responsive to changes in arterial CO2, so hypoxic drive can become more important

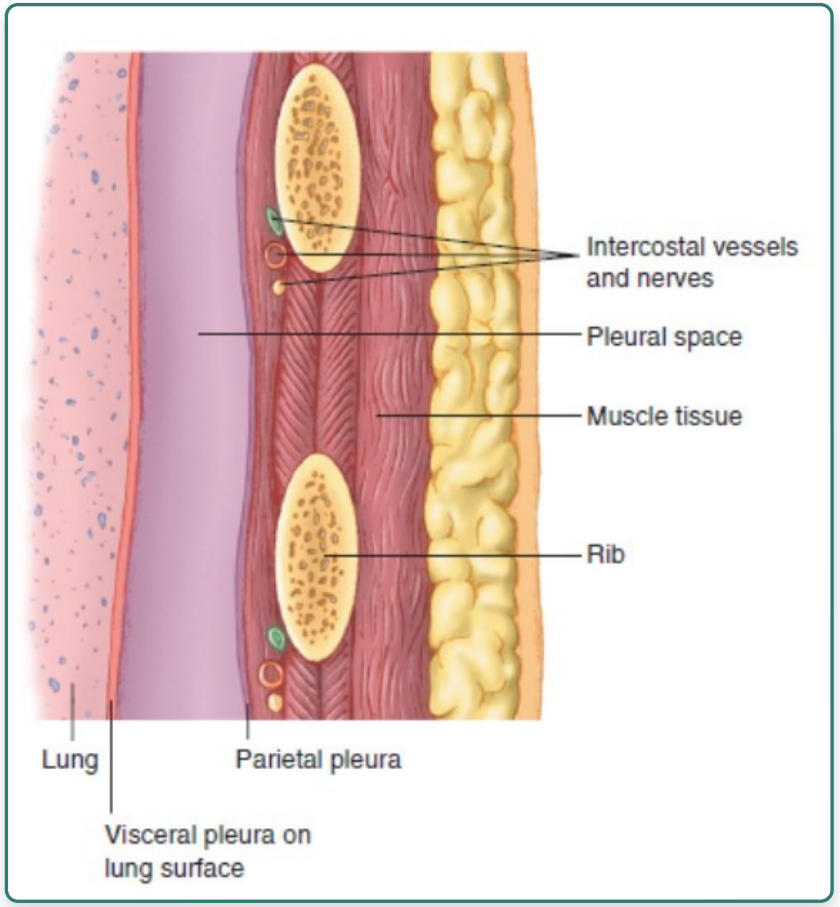


The phases of respiration: (a) inspiration; (b) expiration

Diffusion & Perfusion

Physiology

- Diffusion: gases move between the alveoli and pulmonary capillaries — oxygen into oxygen-poor blood, CO2 out along its concentration gradient
- The respiratory membrane and capillary endothelial lining must both stay intact for efficient gas exchange
- Perfusion is the circulation of blood through the lungs; it depends on adequate blood volume, intact pulmonary capillaries, and efficient cardiac pumping
- Any disease that impairs the pulmonary system can derange ventilation, diffusion, perfusion, or a combination of all three

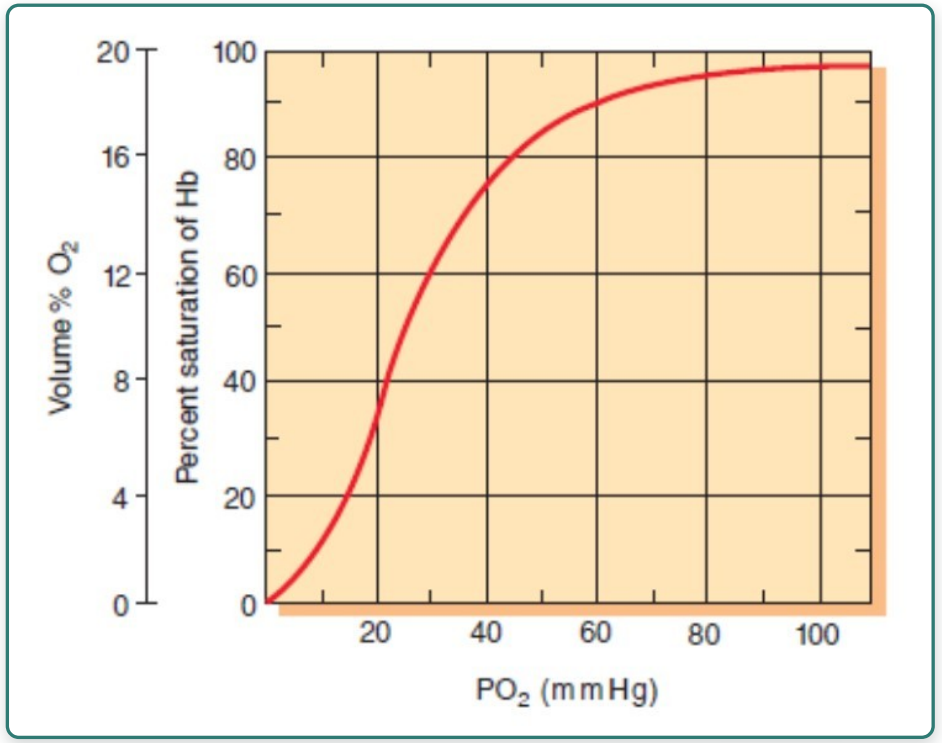


The intercostal vessels and nerves

Oxygen & Carbon Dioxide Transport

Physiology

- Oxygen travels in the blood mostly bound to hemoglobin (oxyhemoglobin) and a small amount dissolved in plasma
- The oxygen dissociation curve describes how readily hemoglobin releases oxygen — the next slide covers what shifts it
- Carbon dioxide is transported three ways: as bicarbonate ion (≈70%), bound to hemoglobin as carbaminohemoglobin (≈23%), and dissolved in plasma (≈7%)
- In the lungs this process reverses — CO2 diffuses into the alveoli and is eliminated on exhalation

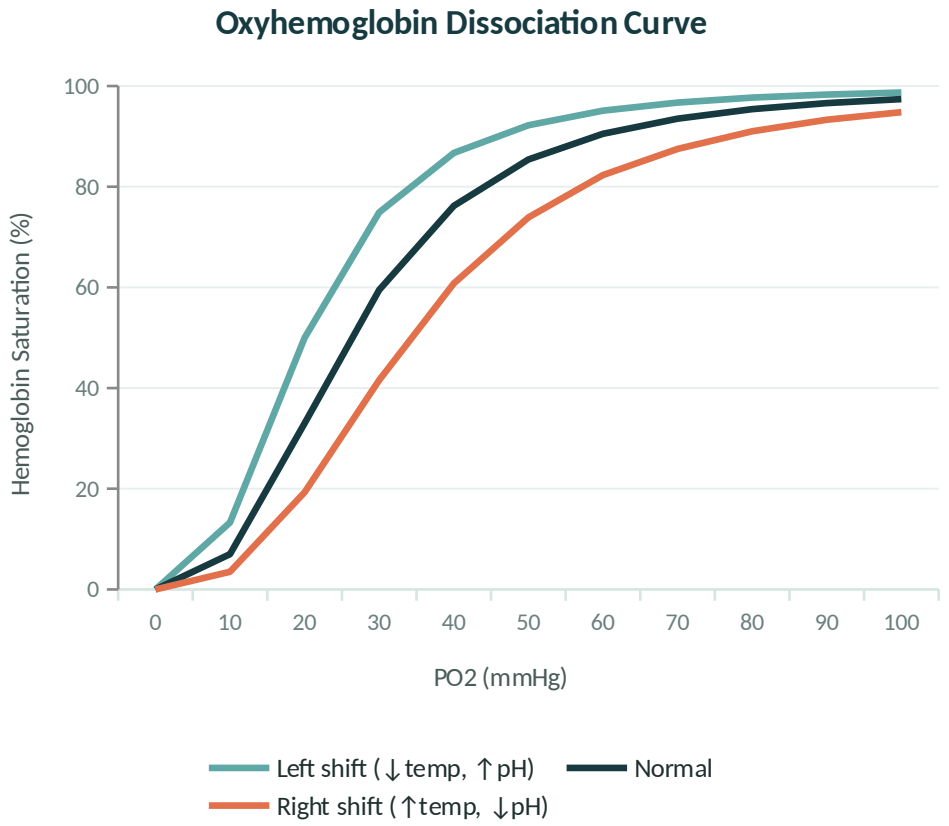


Oxygen dissociation curve

Oxyhemoglobin Curve: Temperature & pH Effects

Physiology

- The oxyhemoglobin dissociation curve plots hemoglobin's oxygen saturation (SaO2) against PaO2 in a characteristic S-shape
- Right shift (Bohr effect): ↑temperature, ↑CO2, ↓pH (acidosis) lower hemoglobin's affinity for oxygen — it releases O2 more readily to tissues (fever, sepsis, exercising muscle)
- Left shift: ↓temperature, ↓CO2, ↑pH (alkalosis) raise hemoglobin's affinity — it holds O2 more tightly and releases less to tissues (hypothermia, hyperventilation)
- Clinical takeaway: interpret pulse oximetry in context — a right-shifted curve can show a lower SpO2 for the same PaO2, while a left-shifted curve can look reassuring even when tissue oxygen delivery is reduced



02

Pathophysiology

How disease disrupts gas exchange

Disruption in Ventilation, Diffusion & Perfusion

Pathophysiology

- Disease of the upper or lower respiratory tract can impair ventilation — anything that narrows the airway or limits chest wall/diaphragm movement reduces airflow
- Diffusion is disrupted whenever the alveolar-capillary membrane thickens or is damaged — fluid, inflammation, or fibrosis all increase the distance oxygen and CO2 must cross
- Perfusion is disrupted when blood flow through the pulmonary capillaries is blocked or reduced (for example, by embolism or shock), even if ventilation is normal
- Most respiratory emergencies involve some combination of ventilation, diffusion, and perfusion problems — identifying which is dominant guides treatment

03

Assessment of the Respiratory System

Scene size-up to diagnostics

Scene Size-Up

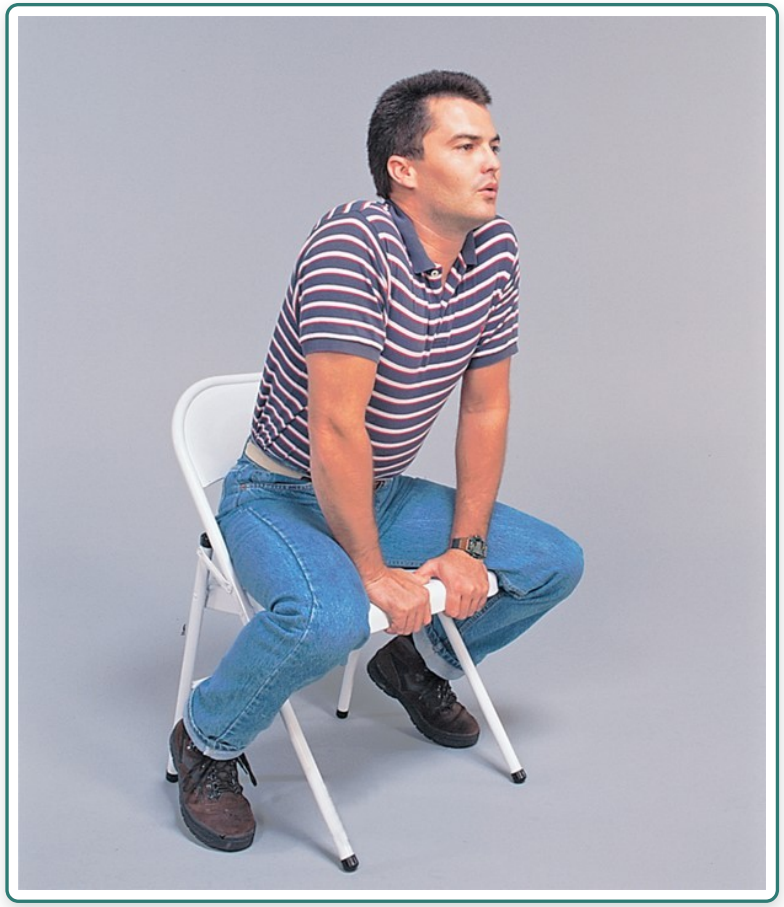
Assessment

- Confirm the scene is safe before approaching — look for visual clues about the patient's complaint
- Toxic gases, dust, and low-oxygen environments (grain silos, enclosed storage, active fires) pose real risk to responders
- Use hazmat suits, SCBA, and supplemental oxygen as needed; call HAZMAT teams through dispatch when indicated
- Your eyes, ears, and nose all provide useful early clues about the nature of the emergency

Primary Assessment: General Impression

Assessment

- Note patient positioning — severe distress often produces the tripod position
- Watch for pallor, diaphoresis, nasal flaring, pursed lips, tracheal tugging, and accessory muscle use
- Cyanosis is a late finding and may be absent even with significant hypoxia
- Assess mental status: restlessness/agitation suggests hypoxia; confusion suggests hypoxia and hypercarbia; severe lethargy signals imminent respiratory failure
- Rambling or incoherent speech, or the inability to speak in full sentences, indicates significant distress



Tripod position

Primary Assessment: Airway, Breathing & Circulation

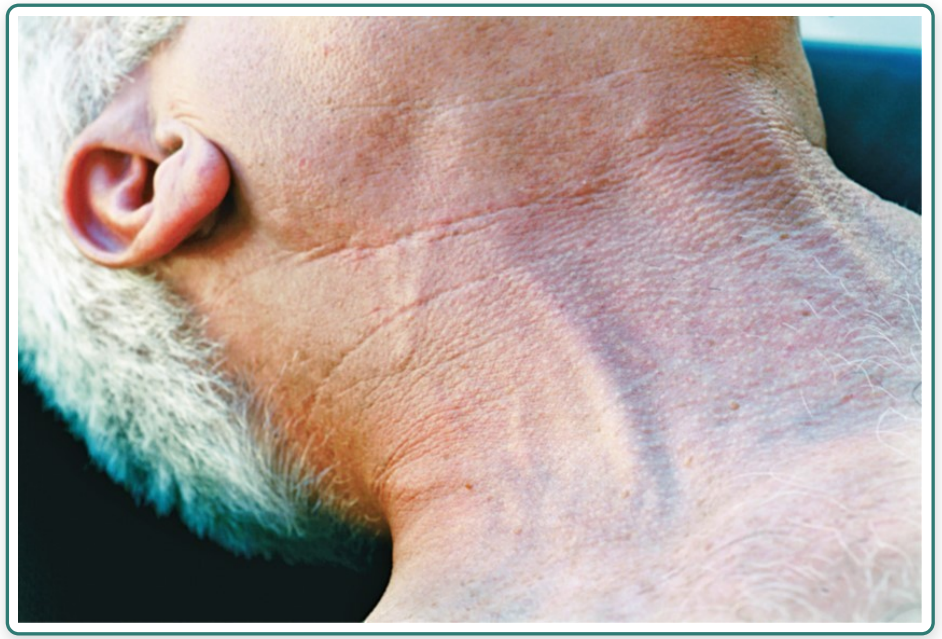
Assessment

- Airway: any significant abnormality can be life-threatening — noisy breathing usually means partial obstruction, but obstruction is not always noisy; the brain survives only minutes without oxygen
- If the airway is obstructed, act immediately — do not delay for equipment or help
- Breathing: watch for altered mental status, severe central cyanosis, absent breath sounds, audible stridor, and one- to two-word dyspnea
- If any of these signs are present, resuscitate and transport immediately
- Circulation: assess central and peripheral pulses, evaluate skin condition, and determine transport priority

Secondary Assessment: History

Assessment

- History and physical exam are guided by the chief complaint — obtain a SAMPLE history and ask OPQRST questions
- Ask about prior hospitalizations, intubations, and known respiratory disease; determine whether ventilation, diffusion, or perfusion is affected
- Medication history is essential — ask about nebulizers, cardiac medications, and allergies
- Increased sputum suggests infection; look at the jugular veins for evidence of distention



Jugular vein distention

Secondary Assessment: Physical Exam

Assessment

- Follow the standard sequence: inspection, palpation, percussion, auscultation
- Inspection: chest shape and anterior-posterior dimensions
- Palpation: tenderness, crepitus, subcutaneous emphysema
- Percussion: reserved for suspected pneumothorax or pulmonary edema
- Auscultation: listen first without a stethoscope for loud stridor, wheezing, or cough, then auscultate the full chest

Inspection of the chest

Breath Sounds

Assessment

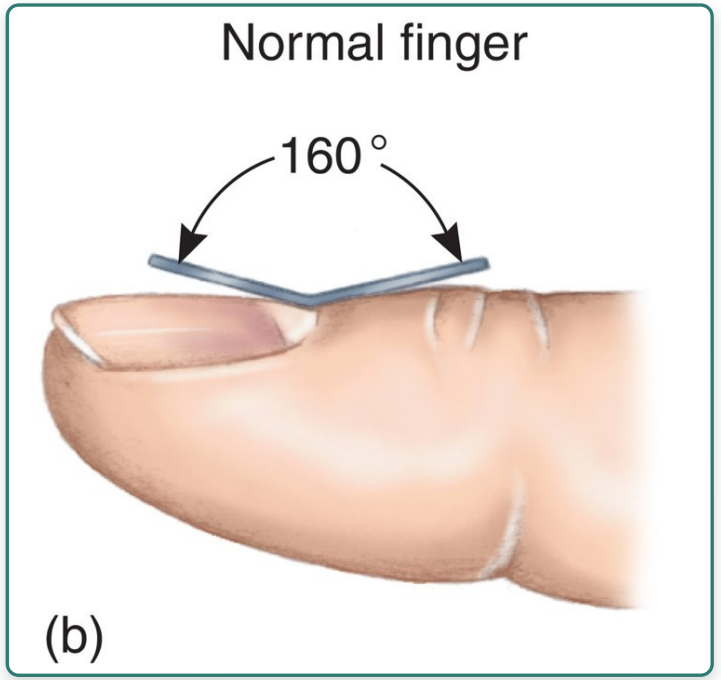
- Normal: bronchial (loud, high-pitched, over the trachea), bronchovesicular (medium-pitched, over the mainstem bronchi), vesicular (soft, low-pitched, in the lung periphery)
- Abnormal: snoring, stridor, wheezing, rhonchi, crackles (rales), pleural friction rub
- Examine the extremities for peripheral cyanosis, swelling/redness/a firm cord (possible pulmonary embolism), clubbing (chronic hypoxemia), or carpopedal spasm (hyperventilation)

The chest should be auscultated

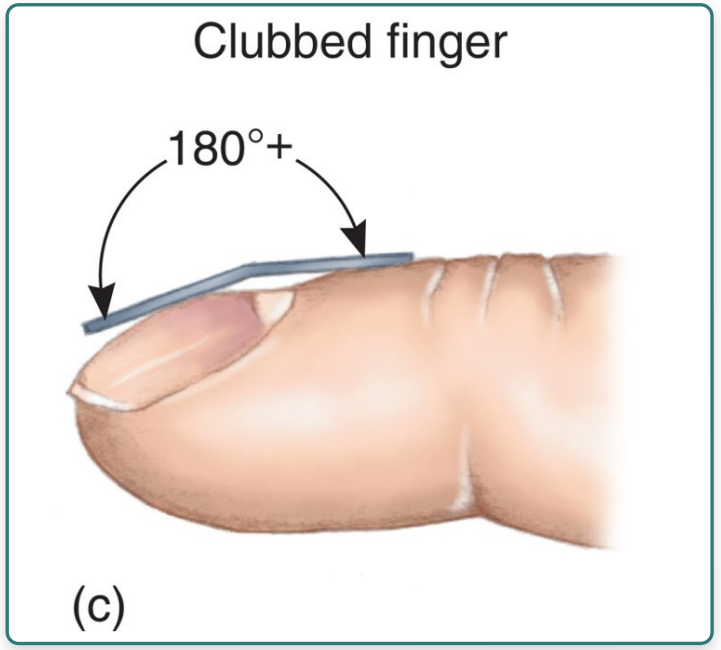
Clubbing & Vital Signs

Assessment

- Finger clubbing — enlarged fingertips and loss of the normal nail-bed angle — signals chronic hypoxemia
- Tachycardia can indicate hypoxia; pulsus paradoxus is associated with COPD and cardiac tamponade
- Tachypnea: respiratory rate over 20/min; bradypnea: under 12/min — a persistently slow rate warns of impending respiratory arrest



Normal finger — 160°



Clubbed finger — 180°+

Pulse Oximetry

Assessment

- Pulse oximetry offers a rapid, noninvasive estimate of oxygen saturation, applied to a finger or earlobe
- It continuously displays pulse rate and the percentage of hemoglobin saturated with oxygen (SpO2)
- Pulse oximeters cannot distinguish normal from abnormal hemoglobin — some newer devices can noninvasively measure total hemoglobin (SpHb)
- A handheld peak-flow meter (Wright spirometer) measures peak expiratory flow rate (PEFR) in liters per minute, compared against norms for age, sex, and height

Sensing unit for pulse oximetry

Capnography & Capnometry

Assessment

- Capnometry measures the level of expired CO2; capnography displays that reading graphically over time as a waveform (capnogram)
- End-tidal CO2 (ETCO2) is the CO2 concentration at the end of exhalation, which approximates arterial CO2 (PaCO2)
- CO2 is produced by metabolism, carried by the venous system to the right heart, pumped to the pulmonary capillaries, diffused into the alveoli, and exhaled
- Decreased ETCO2: shock, cardiac arrest, pulmonary embolism, bronchospasm, airway obstruction
- Increased ETCO2: hypoventilation, respiratory depression, hyperthermia

Modern prehospital patient monitors allow continuous monitoring of multiple physiologic parameters

Continuous Waveform Capnography

Assessment

- Colorimetric devices can detect the presence of CO2 but cannot quantify hypercarbia or hypocarbia
- Electronic (infrared) capnography detectors measure CO2 quantitatively as a continuous waveform
- The waveform includes a respiratory baseline, upstroke, plateau, and inspiratory phase — pattern changes help identify specific conditions such as bronchospasm
- Continuous waveform capnography confirms airway placement and monitors ventilation in intubated patients, is useful in nonintubated patients, and helps guide CPR quality

Capnography devices provide a digital waveform (capnogram) reflecting the entire respiratory cycle

04

Management of Respiratory Disorders

Oxygen: enough, not too much

Management Principles

Management

- Airway always has first priority
- Any patient with hypoxia — or whose illness/injury suggests possible hypoxia — should receive oxygen until pulse oximetry is available
- Strive for normoxia; avoid both hypoxia and hyperoxia whenever possible
- Administering too much oxygen can worsen outcomes — excess oxygen promotes toxic free radicals that cause oxidative stress and tissue damage
- Give just enough oxygen to correct hypoxia without inducing hyperoxia

Excess oxygen (hyperoxia) has been associated with worsened outcomes in critically ill patients

05

Specific Respiratory Diseases

Recognition and field management

Upper Airway Obstruction

Disease

- Common causes: a relaxed tongue, food, dentures, or other foreign bodies; also facial/neck trauma, upper airway burns, or allergic swelling
- Severe signs: silent cough, cyanosis, inability to speak or breathe
- Conscious adult with severe obstruction: rapid, sequential abdominal thrusts until the obstruction clears
- Unconscious adult: attempt to open the airway, begin CPR, and if ventilation still fails, visualize with a laryngoscope and remove the object with Magill forceps
- Laryngeal edema (e.g., anaphylaxis): open the airway, give oxygen, attempt BVM ventilation, start an IV, and give IM epinephrine and diphenhydramine

ARDS / Noncardiogenic Pulmonary Edema

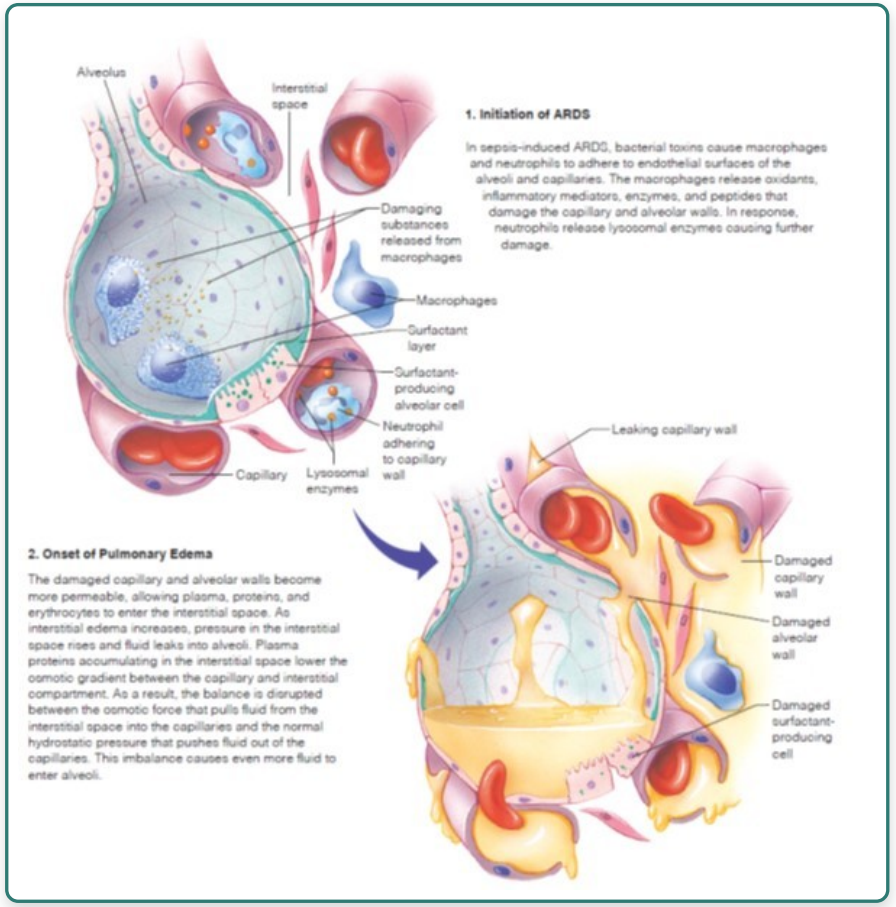
Disease

- A life-threatening condition in which fluid accumulates in the interstitial space, driven by increased vascular permeability and reduced fluid clearance
- Mortality is high (≈70%), often from respiratory failure or multi-organ failure (liver, kidneys)
- Increased capillary permeability and osmotic forces thicken the respiratory membrane and limit oxygen diffusion
- Presentation: gradual decline in respiratory status, dyspnea, confusion, agitation, tachypnea, tachycardia, and bilateral crackles; pulse oximetry shows low saturation as disease advances

ARDS: Disease Progression (1–2)

Disease

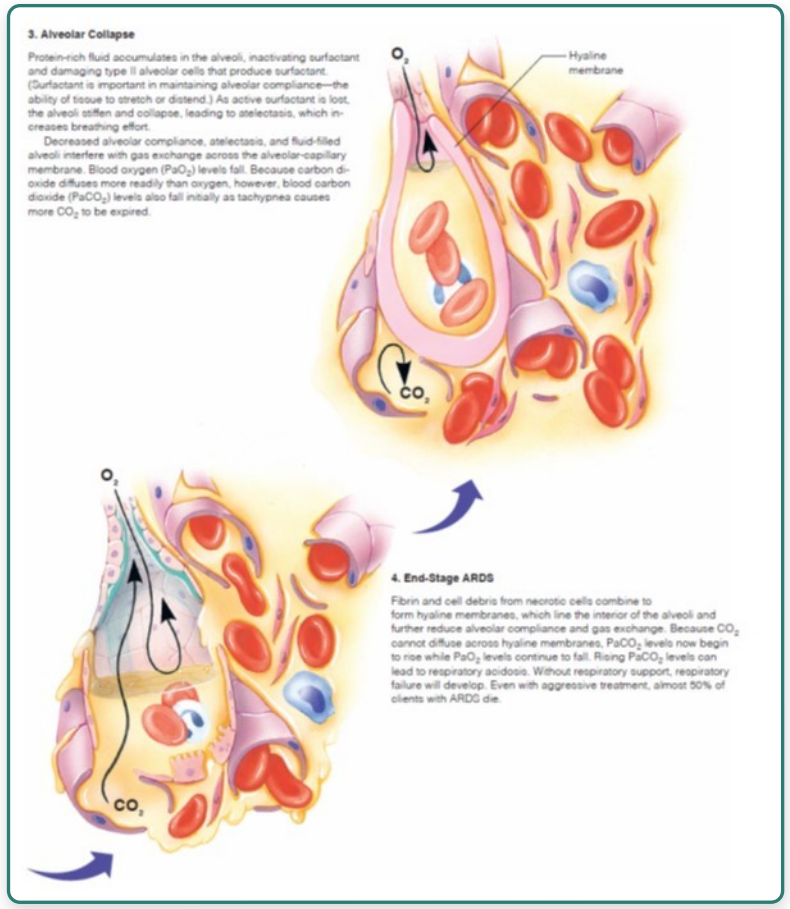
- Initiation of ARDS: in septic ARDS, bacterial toxins trigger inflammation that damages the alveolar and capillary walls
- Onset of pulmonary edema: the damaged, more-permeable walls let plasma and fluid leak into the interstitial space and then into the alveoli



Stage 1: Initiation of ARDS — Stage 2: Onset of pulmonary edema

ARDS: Disease Progression (3–4)

Disease



Stage 3: Alveolar collapse — Stage 4: End-stage ARDS

- Alveolar collapse: protein-rich fluid damages surfactant-producing cells; without surfactant the alveoli stiffen and collapse (atelectasis)
- End-stage ARDS: fibrin and cell debris form hyaline membranes that further reduce alveolar compliance and gas diffusion, driving respiratory failure

ARDS: Management

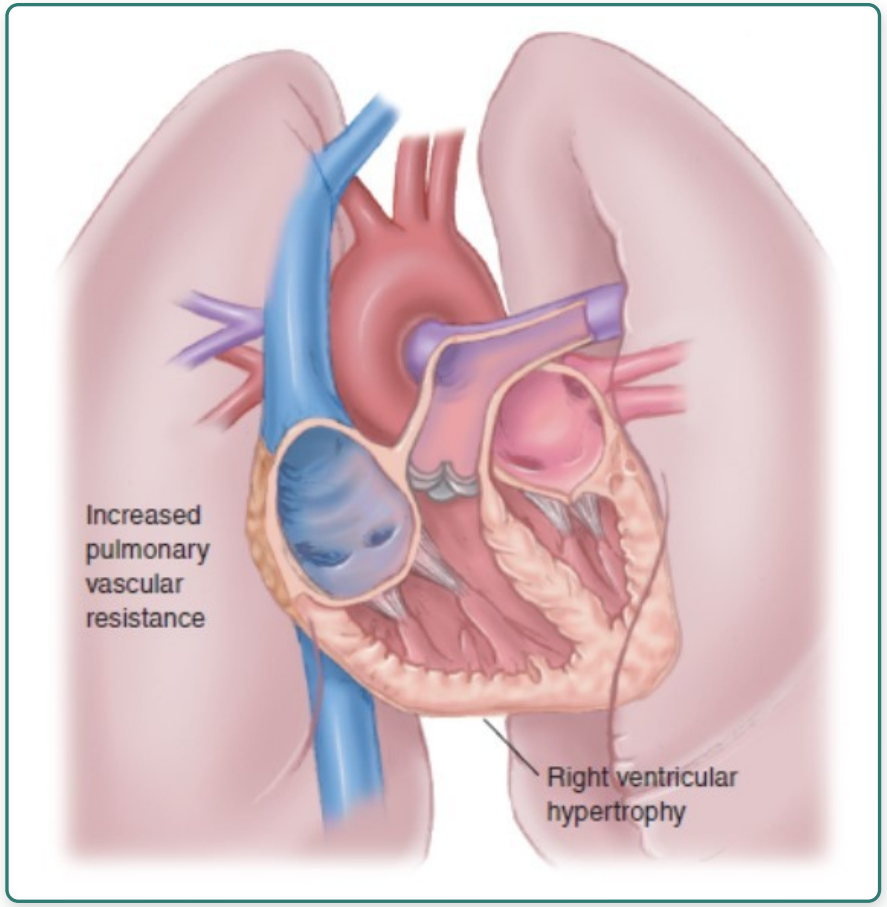
Disease

- Treat the underlying cause — appropriate antibiotics for gram-negative sepsis, removal from the inciting toxin, or rapid descent for high-altitude pulmonary edema (HAPE)
- Establish IV access; give fluids only if hypovolemia is present, since overhydration worsens pulmonary edema
- Establish cardiac monitoring and pulse oximetry; suction secretions as needed
- CPAP can avoid the need for intubation and mechanical ventilation in appropriate patients
- Transport to a facility capable of advanced hemodynamic monitoring and mechanical ventilation support

Obstructive Lung Diseases: Overview

Disease

- Umbrella term covering emphysema, chronic bronchitis, and asthma — collectively COPD plus asthma
- COPD is caused directly by cigarette smoking and environmental toxins, with some genetic predisposition
- Shared features: abnormal ventilation, bronchiolar obstruction, bronchospasm, and increased mucus production from goblet cells
- Inflammation of the bronchial passages leads to fluid accumulation and further airway narrowing



Long-standing COPD can cause pulmonary hypertension, which can lead to cor pulmonale

Emphysema

Disease

- Destruction of alveolar walls distal to the terminal bronchioles — more common in men; driven by smoking and environmental toxin exposure
- Reduced alveolar surface area causes diffusion defects and increases resistance to pulmonary blood flow, ultimately leading to pulmonary hypertension, cor pulmonale, and right-heart failure
- Weakened bronchiole walls lose recoil, trapping air; patients breathe through pursed lips to create positive pressure similar to PEEP and prevent alveolar collapse
- Patients are prone to acute respiratory infections and cardiac arrhythmias, and depend on bronchodilators, corticosteroids, and supplemental oxygen

Emphysema: Presentation

Disease

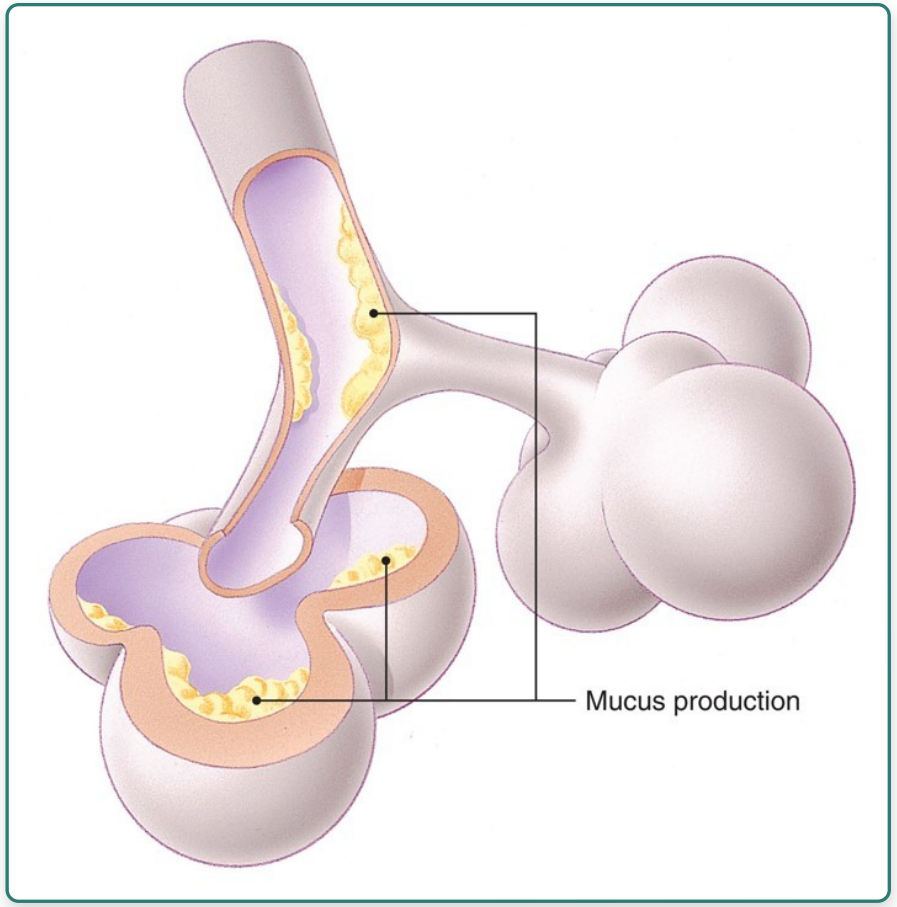
- Weight loss, increased dyspnea on exertion, and limited activity tolerance; cough is uncommon
- Barrel chest from increased anterior-posterior diameter; hypertrophied accessory muscles
- Patients tend to stay “pink” (polycythemia compensates) and are sometimes called “pink puffers”
- Clubbing is common; breath sounds are diminished
- Signs of right-heart failure may appear: jugular vein distention, peripheral edema, hepatic congestion

Typical appearance of a patient with emphysema, showing accessory muscle use and suprasternal retraction

Chronic Bronchitis

Disease

- Increased goblet (mucus-secreting) cells in the respiratory tree, producing large quantities of sputum, usually after prolonged smoke exposure
- Alveoli are relatively spared, so diffusion stays fairly normal; gas exchange falls mainly because alveolar ventilation is lowered — causing hypoxia and hypercarbia
- Diagnosis: productive cough for 3+ months per year, 2+ consecutive years
- Patients are often overweight and cyanotic (“blue bloaters”) with rhonchi from mucus-plugged airways, and may show signs of right-heart failure



Chronic mucus production and airway plugging in chronic bronchitis

Chronic Bronchitis: Management

Disease

- Goal: relieve hypoxia and reverse bronchoconstriction
- Supplemental oxygen at a low flow rate, targeting SpO2 90–95%; support ventilation with BVM assistance or CPAP as needed
- Intubation if CPAP fails and respiratory failure is imminent
- Saline lock, fluids for dehydration, and nebulized bronchodilators (albuterol, levalbuterol, metaproterenol) per medical direction

Asthma

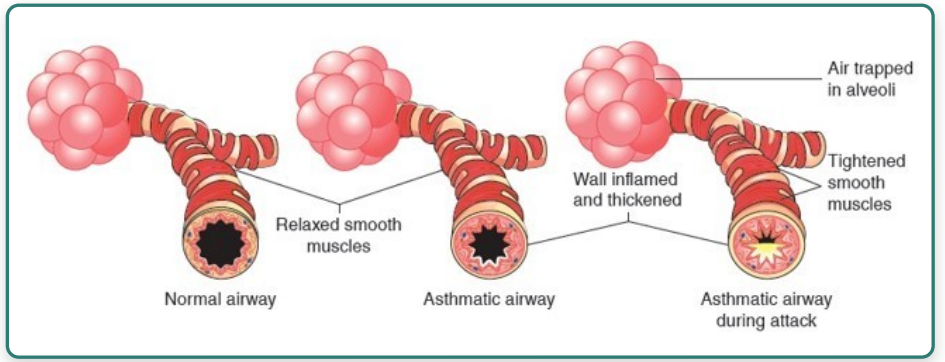
Disease

- A chronic inflammatory airway disorder with reversible airflow obstruction and hyperresponsiveness, provoked by triggers — allergens, cold air, exercise, irritants, stress, certain medications
- Early-phase reaction: histamine and other mediators cause bronchial smooth-muscle contraction, capillary leakage, bronchoconstriction, and edema — the “asthma attack”
- Attacks often resolve spontaneously in 1–2 hours or respond to inhaled bronchodilators such as albuterol
- A late-phase reaction can follow 6–8 hours after exposure, driven by inflammatory cell infiltration and typically unresponsive to beta-agonists alone — corticosteroids are required

Asthma: Assessment

Disease

- Symptoms: dyspnea, wheezing, cough, chest hyperinflation, tachypnea; agitation and anxiety increase as hypoxia develops
- History clues: prior intubation, continuous corticosteroid therapy — both mark a high-risk patient
- Rising respiratory rate is often the earliest warning sign; falling PEFR indicates worsening severity
- Continuous waveform capnography can help identify asthma and gauge airflow obstruction severity



Normal airway, asthmatic airway, and asthmatic airway during an attack

Phases of Acute Asthma Exacerbation

Disease

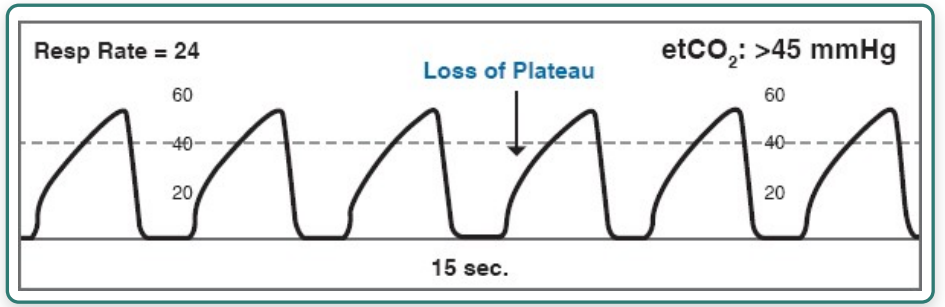
Phase	Clinical Assessment	ETCO2 (mmHg)
Mild	Hyperventilating	< 35
Moderate	Tiring	35–50
Severe	Tired	> 50

A severe exacerbation produces a “shark fin” capnogram configuration; patients hyperventilate to maintain oxygenation.

Asthma: Management

Disease

- Correct hypoxia with supplemental oxygen; establish IV access and continuous ECG monitoring
- Inhaled beta-agonists (albuterol or levalbuterol) combined with ipratropium bromide, via small-volume oxygen-powered nebulizer
- Treat inflammation with a corticosteroid; earlier treatment works better — delay reduces bronchodilator effectiveness
- Status asthmaticus: a severe, prolonged attack unresponsive to repeated bronchodilator doses — a distended chest, absent wheezing, exhaustion, and severe acidosis signal impending respiratory arrest
- Prepare for endotracheal intubation and transport immediately
- Pediatric asthma: pathophysiology and treatment mirror the adult presentation, adjusted for pediatric medication dosages



Comparison of normal and asthmatic capnogram waveforms

Upper Respiratory Infection (URI)

Disease

- Can worsen existing pulmonary disease or cause direct pulmonary infection; good hand hygiene and covering coughs/sneezes are the best defense
- Most URIs are viral; Streptococcus accounts for about 30% of bacterial cases; illnesses are usually self-limiting and resolve in days
- Common symptoms: fever, chills, myalgias, fatigue
- No specific field intervention is usually required, except in children with epiglottitis or complicated infections where pus threatens the airway — give oxygen for hypoxia, avoiding hyperoxia

Locations & Signs/Symptoms of Upper Respiratory Infections

Disease

Structure	Infection	Symptoms	Signs
Nose	Rhinitis	Runny nose, congestion, sneezing	Rhinorrhea
Pharynx	Pharyngitis	Sore throat, pain swallowing	Erythematous pharynx, tonsil enlargement, pus, cervical nodes
Middle ear	Otitis media	Ear pain, decreased hearing	Red/bulging eardrum, pus, nearby node enlargement
Larynx	Laryngitis	Sore throat, hoarseness, pain speaking	Red pharynx, hoarse voice, cervical nodes
Epiglottis	Epiglottitis	Sore throat, drooling, ill appearing	Upright position, drooling, ill appearing
Sinuses	Sinusitis	Headache, congestion	Sinus tenderness, worse leaning forward, yellow discharge

URI: Treatment

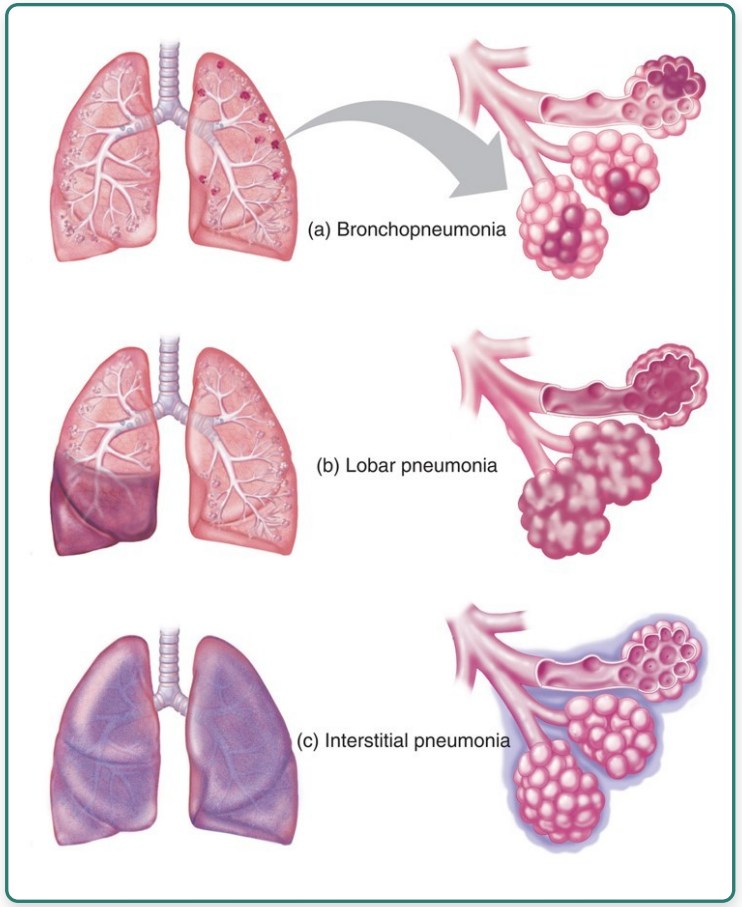
Disease

- Diagnosis and treatment are based on history and physical findings
- Acetaminophen or ibuprofen for fever, headache, and myalgias; encourage fluids
- Saltwater gargles for throat discomfort; decongestants/antihistamines to reduce mucus secretion
- Encourage patients on antibiotics to complete the full course

Pneumonia

Disease

- An infection of the lungs and a leading cause of death, especially in the elderly and those with HIV; risk factors include alcoholism, smoking, and cold exposure
- Caused by a variety of infectious agents (bacterial and viral most common) that impair mucus production and ciliary clearance
- Infection starts in one area of the lung and can spread; fluid and inflammatory cells collect in the alveoli, primarily disrupting ventilation
- Presentation: acute illness, fever with chills, malaise, deep productive cough with yellow-brown or blood-streaked sputum, and possible pleuritic chest pain

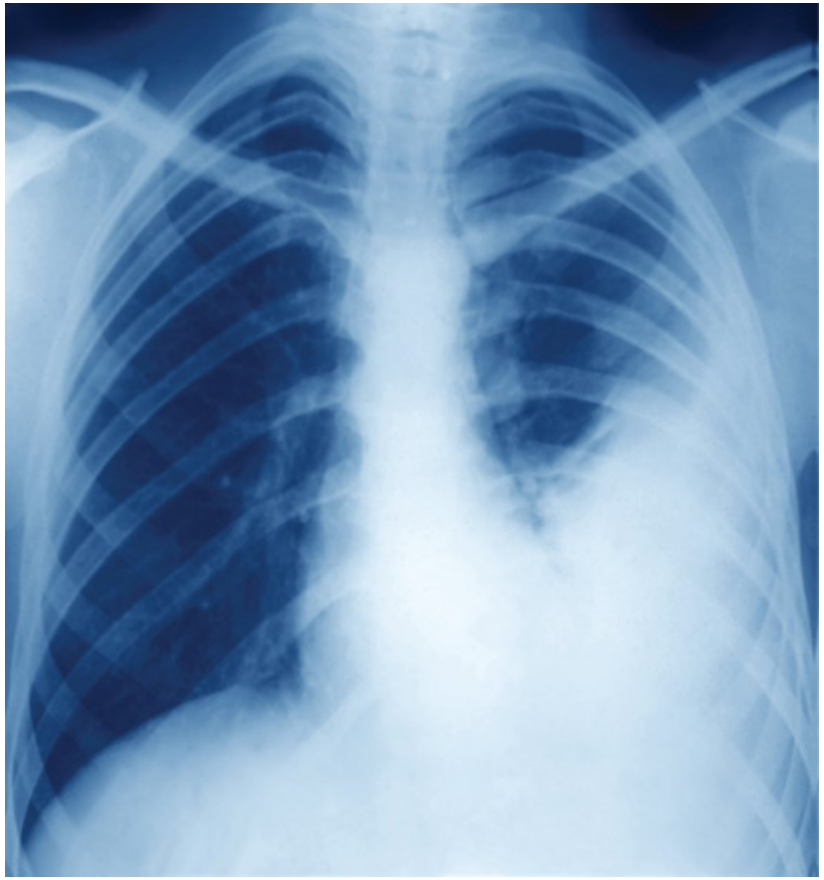


Types of pneumonia: bronchopneumonia, lobar pneumonia, and interstitial pneumonia

Pneumonia: Management

Disease

- Diagnosed by physical exam, chest X-ray, and lab cultures; antibiotics are the primary treatment
- Give supplemental oxygen for hypoxia; severe cases may need ventilatory assistance or intubation
- Establish IV access; treat dehydration with fluids, but avoid overhydration, which can worsen the respiratory condition
- Antipyretics (acetaminophen or ibuprofen) reduce high fever
- Patients over 65 have high mortality and complication rates — transport to a facility equipped to manage them



Pneumonia chest X-ray

Severe Acute Respiratory Syndrome (SARS)

Disease

- A viral respiratory illness (SARS-CoV) first identified in southern China in 2002; spreads by close person-to-person contact with a 2–7 day incubation
- Contagious as long as symptoms persist — use appropriate PPE on every call or as directed by local health authorities
- Symptoms progress from sore throat, rhinorrhea, chills, myalgias, and headache to cough, sputum production, and respiratory distress or failure
- Manage as suspected pneumonia: supplemental oxygen, IV access, and ventilatory support/intubation for severe cases; notify the receiving hospital for isolation precautions

MERS & COVID-19

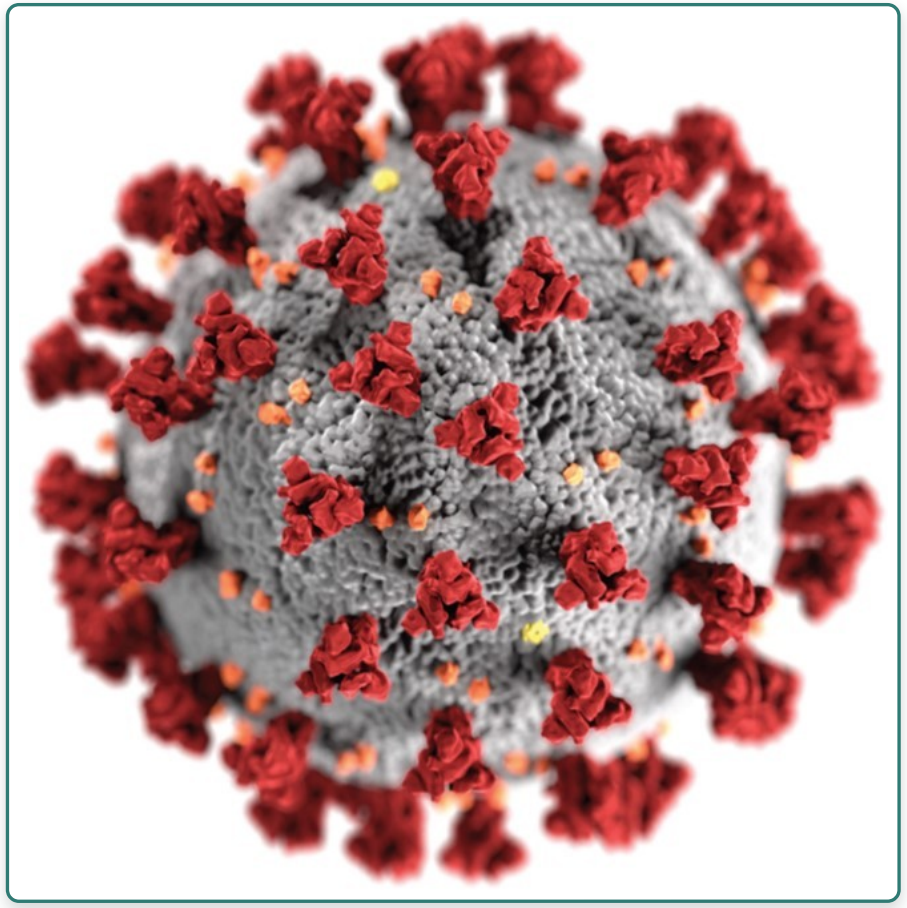
Disease

- MERS: first reported in Jordan and Saudi Arabia in 2012; fever, cough, and shortness of breath, with a case-fatality rate near 30–40%; spreads through close contact and can progress to pneumonia and renal failure
- COVID-19: caused by a novel coronavirus related to SARS; produces fever, dry cough, and malaise, with roughly 20% hospitalization and disproportionate fatality at the age extremes
- COVID-19 spreads via respiratory droplets and continues to evolve through new variants
- Standard respiratory-illness precautions apply to both — PPE, isolation notification, and supportive respiratory care

COVID-19

Disease

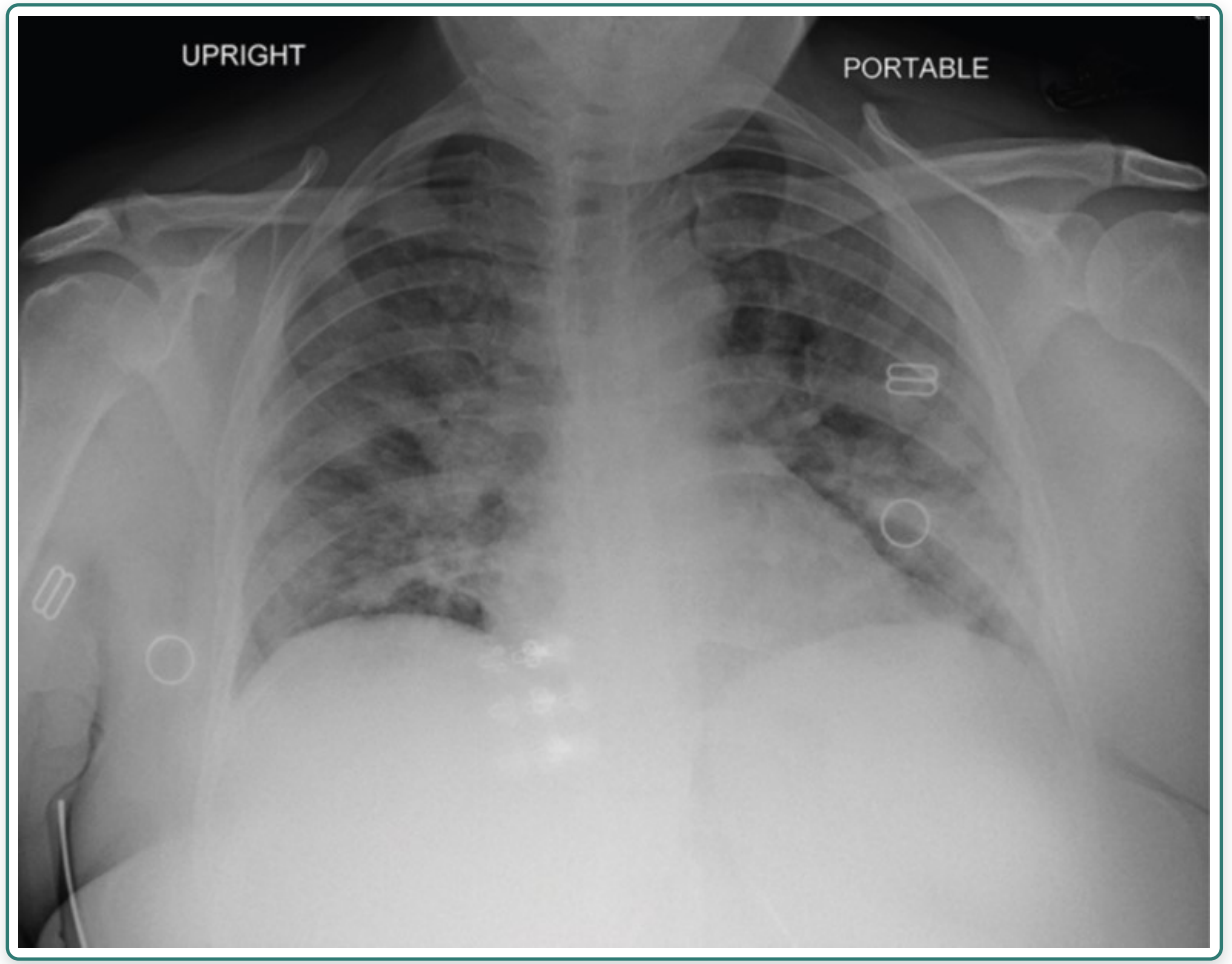
- SARS-CoV-2 causes illness ranging from mild upper respiratory symptoms to severe viral pneumonia and ARDS
- Supplemental oxygen for hypoxia; escalate to CPAP or ventilatory support for severe respiratory distress
- Follow current local/agency protocols for PPE and patient isolation, since guidance evolves with the pandemic response



The COVID-19 virus

COVID-19 Pneumonia

Disease



COVID-19 can result in serious illness including pneumonia

Lung Cancer

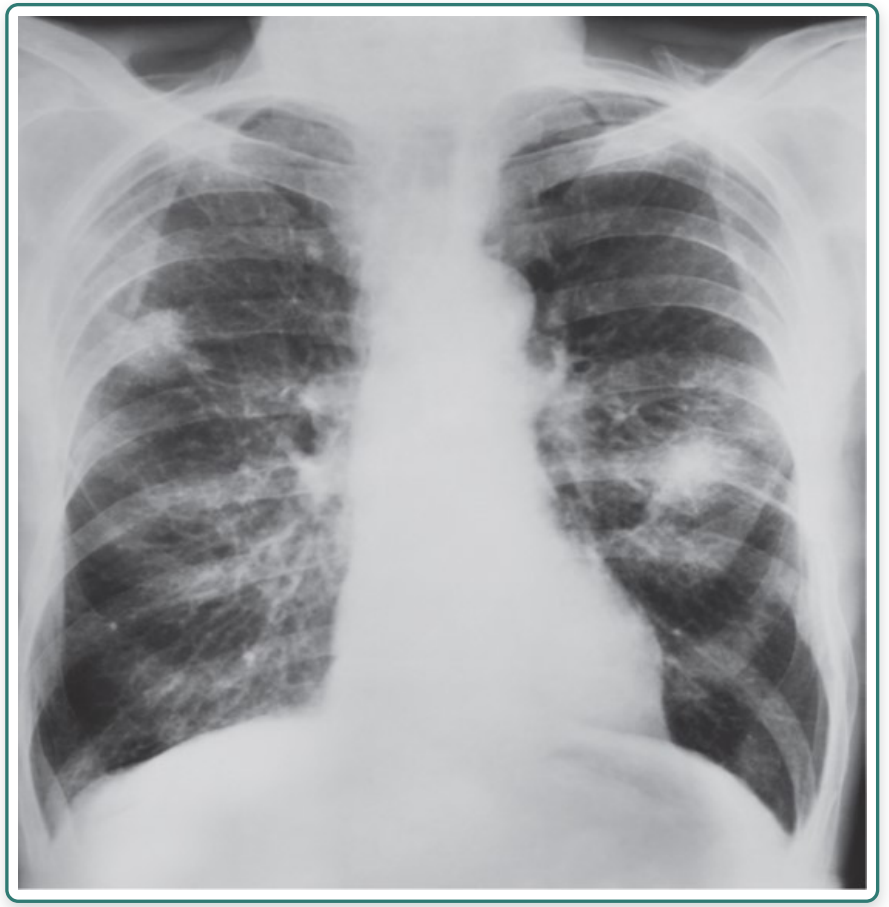
Disease

- The leading cause of cancer-related death in the U.S. for both men and women, typically ages 55–65
- Vast majority of cases are caused by cigarette-smoke carcinogens; other risk factors include asbestos, hydrocarbons, radiation, metal-production fumes, and home radon exposure
- Roughly 20% of cases are confined to lung tissue, 35% have spread to the lymphatic system, and 45% have distant metastases at diagnosis
- Four main cell types: adenocarcinoma (most common), small-cell (“oat cell”), epidermoid, and large-cell carcinoma

Lung Cancer: Presentation & Care

Disease

- Prognosis is poor — most patients die within a year of diagnosis
- Presentation: cough, dyspnea, hoarseness, vague chest pain, and hemoptysis, which can become severe and life-threatening
- Metastatic symptoms: headache, seizures, bone pain, abdominal pain, nausea, malaise, profound weight loss/cachexia, and adventitious breath sounds
- Give supplemental oxygen, watch for a DNR order or advance directive, and follow local protocol; IV normal saline for dehydration, respecting any indwelling catheter access



Chest X-ray showing lung cancer

Toxic Inhalation

Disease

- Causes pain, inflammation, and destruction of pulmonary tissue — consider in any dyspneic patient
- Causes: superheated air, toxic combustion products, chemical irritants, steam inhalation
- Severe inhalation can disrupt the alveolar-capillary membrane, producing life-threatening pulmonary edema
- Several inhalants form corrosive acids/alkalis: ammonia, nitrogen oxide, sulfur dioxide, sulfur trioxide, and chlorine
- Assess exposure duration and enclosure; look for facial/mouth/throat burns or particulate matter; wheezing suggests bronchospasm, crackles suggest pulmonary edema

Toxic Inhalation: Management

Disease

- Ensure rescuer safety first, then remove the patient from the hazardous environment
- Establish and maintain an open airway
- Administer humidified oxygen to correct hypoxia
- Place a saline lock for venous access and transport promptly

Carbon Monoxide Inhalation

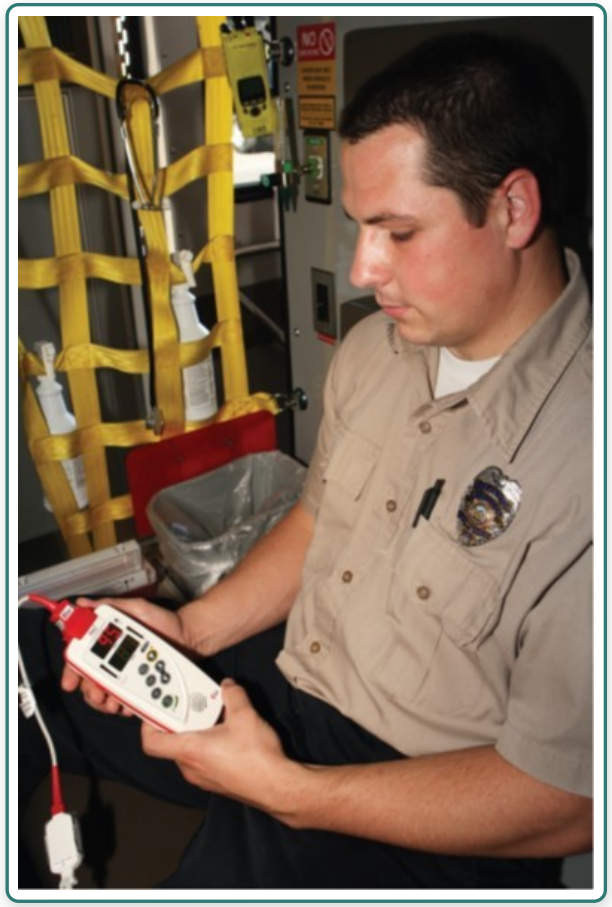
Disease

- An odorless, tasteless, colorless gas from incomplete combustion — the #1 cause of poisoning in industrialized countries
- CO binds hemoglobin far more readily than oxygen, forming carboxyhemoglobin and blocking oxygen delivery to tissues — producing cellular hypoxia and eventually metabolic acidosis
- Symptoms: headache, nausea/vomiting, confusion, agitation, loss of coordination, chest pain, loss of consciousness, seizures; skin may appear cyanotic or cherry red
- CO-oximetry noninvasively detects carboxyhemoglobin, methemoglobin, oxyhemoglobin, and deoxyhemoglobin in the field

Carbon Monoxide: Management

Disease

- Ensure rescuer safety and remove the patient from the exposure source
- Ensure and maintain the airway; give the highest possible concentration of supplemental oxygen via a tight-fitting nonrebreather
- Consider CPAP for moderate-to-severe exposures and assist respirations as needed
- Treat shock if present; prompt transport is essential — the benefit of hyperbaric oxygen therapy remains unclear



The CO-oximeter rapidly and noninvasively monitors carboxyhemoglobin levels in the field

Pulmonary Embolism

Disease

- A thrombus or other particle lodges in a pulmonary artery, blocking blood flow and potentially causing hypoxemia — responsible for roughly 1 in 5 cases of sudden death
- Risk factors: immobility, venous pooling in pregnancy, cancer, infection, thrombophlebitis, atrial fibrillation, sickle cell disease
- Sources include air, fat, or amniotic fluid emboli, in addition to blood clots — the main derangement is perfusion, since the affected segment is still ventilated (ventilation-perfusion mismatch)
- Acute presentation: sudden unexplained severe dyspnea, often with recent immobilization history, labored breathing, tachypnea, tachycardia, and signs of right-heart failure

Pulmonary Embolism: Assessment & Management

Disease

- Always examine the extremities — up to 50% of cases show deep venous thrombosis: a warm, swollen limb with a palpable cord, pain on palpation, or petechiae
- First priorities remain the ABCs; a large embolism can cause cardiac arrest — perform CPR if needed
- Assist ventilations, give high-concentration oxygen, and consider endotracheal intubation
- Place a saline lock, monitor vital signs and cardiac rhythm, and transport quickly — this diagnosis carries a high complication rate and significant mortality

Spontaneous Pneumothorax

Disease

- Occurs without blunt or penetrating trauma, more common in males; risk factors include a tall, thin build, smoking history, and age 20–40; incidence rises with COPD
- Loss of the normal negative pleural pressure prevents proper lung expansion, producing sudden sharp pleuritic chest or shoulder pain and dyspnea
- Tachypnea, diaphoresis, and pallor are common; cyanosis is rare
- Give supplemental oxygen guided by symptoms and pulse oximetry; patients needing positive-pressure ventilation are at risk for tension pneumothorax

Hyperventilation Syndrome

Disease

- Rapid breathing with chest pain, numbness, and other anxiety- or stress-related symptoms; carpopedal spasm (hand/foot cramping) can occur
- Treat as a possible serious medical problem until proven otherwise — a seizure history can be precipitated by a hyperventilation episode
- Primary treatment is reassurance and coaching the patient to voluntarily slow their breathing
- Check oxygen saturation — pulmonary embolism and acute MI can mimic this presentation

CNS & Neuromuscular Causes of Respiratory Failure

Disease

- CNS dysfunction is relatively rare but should be considered in any dyspneic patient — causes include head trauma, stroke, brain tumors, and drugs
- Spinal cord, nerve, or respiratory muscle dysfunction reduces the mechanical ability to ventilate, leading to hypoventilation and hypoxemia (spinal trauma, polio, ALS, myasthenia gravis, viral infection, tumors)
- These patients are at increased risk for pneumonia — always ask about recent falls or injuries
- Establish and maintain the airway, support ventilation (including mechanical ventilation if breathing is depressed or absent), give supplemental oxygen, and provide spinal motion restriction if injury is possible

Key Takeaways

Recognize

- Respiratory emergencies are among the most common prehospital calls
- Every respiratory disorder produces some derangement in ventilation, diffusion, or perfusion

Act

- Establish and maintain the airway, assist ventilation as needed, and give supplemental oxygen
- Follow local protocol for additional pharmacological agents

Target normoxia

- The primary responsibility never changes: keep the airway open and breathing adequate to maintain normoxia
- Avoid both hyperoxia and hypoxia — both carry real danger
- Never let technology replace thorough physical assessment, sound judgment, and common sense