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Pathophysiology and Respiratory Disorders

Primary Care Paramedicine

Module: 11

Section: 01

Case Study

- You respond lights and siren to a high rise apartment building for a 18 y/o F patient that is conscious and breathing, short of breath with an altered level of consciousness

Case Study

- Pt is located on the 7th floor
  - Difficult to fit all of your equipment into the elevator
- Arrive at door and greeted by roommate and guided to living room
- Find university aged F student tripoding, tachypnic, with cyanosis to lips
- Blue puffer on coffee table
- Auscultation of lungs reveals very little AW with high pitched inspiratory and expiratory wheezes
- HR 134, BP 160/100, RR 34, hemoglobin saturation is 88%

Life Threats

Upper Airway	Lower Airway
FBAO	COPD
Blunt trauma	Asthma
Penetrating trauma	Pulmonary embolism
Infections	Infections
Angioedema	Pulmonary edema
Cancer	Cancer

Neurological
CVA
Neuromuscular disease (ALS)

Lecture Outline

- Physiology Review
- Pulmonary protective mechanisms
- Pathophysiology

Epidemiology

- In the 2011 Canadian Community Health Survey, 2.5 million Canadians, or 8.6% of the population age 12 and older, reported being diagnosed with asthma
- The leading conditions for which patients were admitted from EDs were respiratory disease (COPD), heart failure and pneumonia. The time spent until decision to admit for these conditions ranged from 10.7 to 11.6 hour and the additional time waiting for an inpatient bed ranged from 25.3 to 26.9 hours

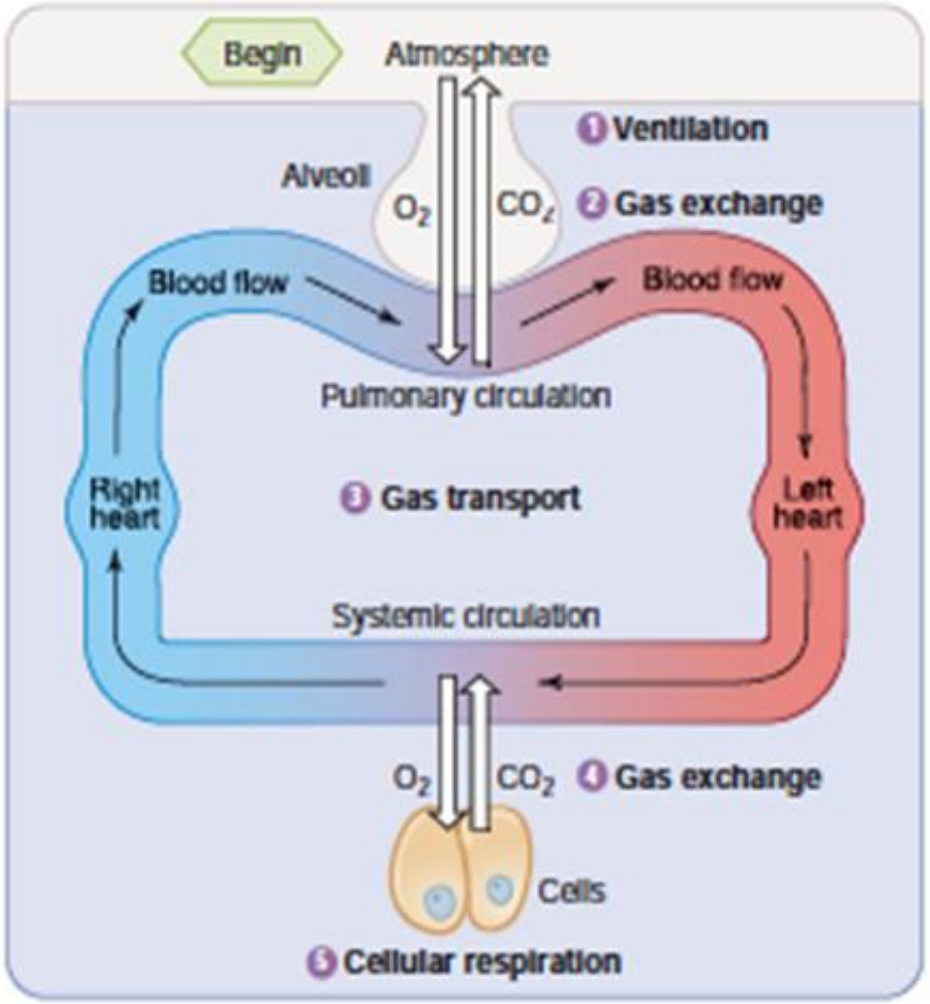
(Canadian Institute for Health Information)

Physiologic Processes

- Gas exchange
- The process by which oxygen is taken in and carbon dioxide is eliminated
  - Ventilation
  - Diffusion
  - Perfusion

Physiologic Processes

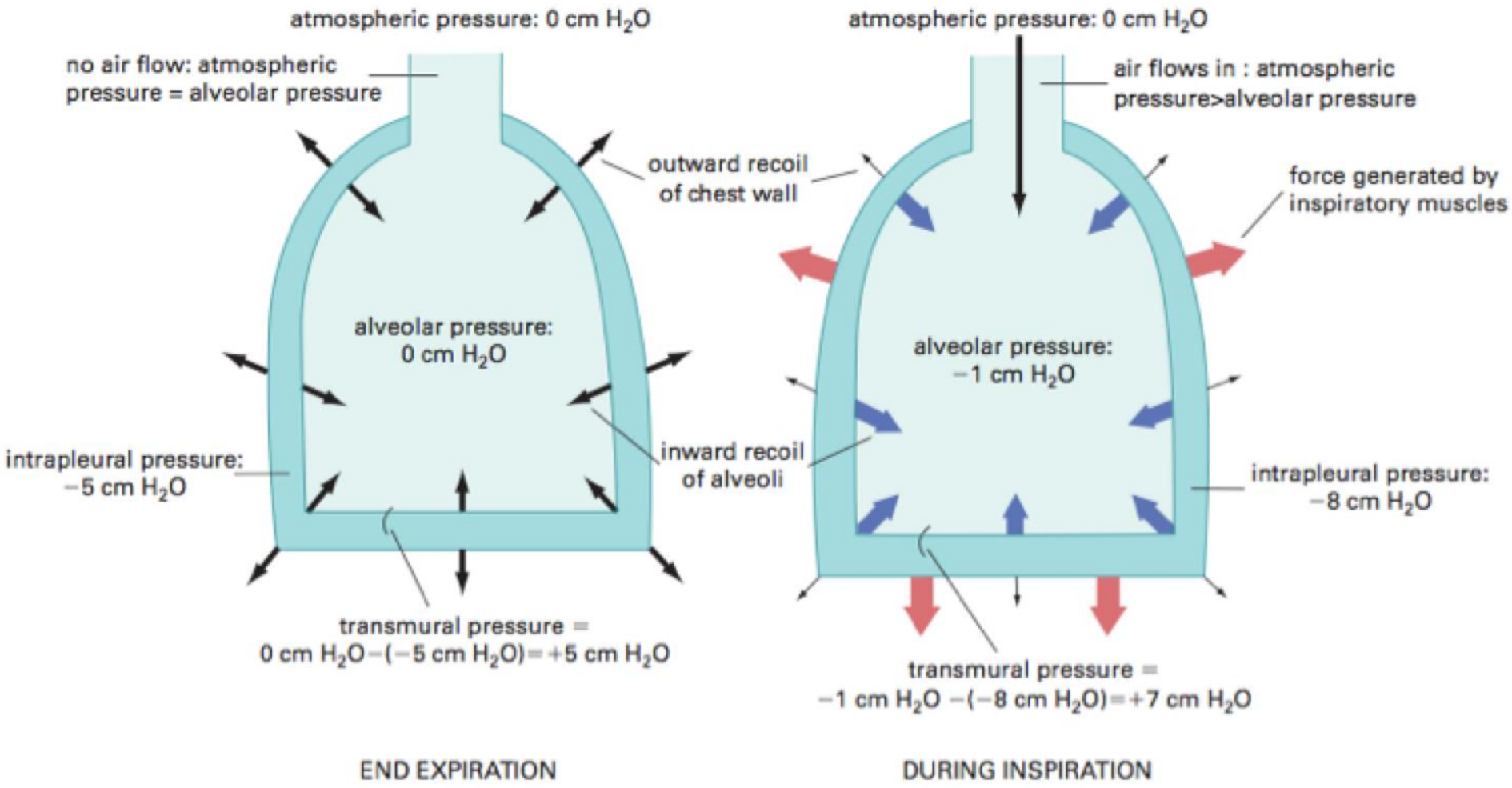
- Ventilation: Exchange of air between atmosphere and alveoli by bulk flow
- Exchange of O_2 and CO_2 between alveolar air and blood in lung capillaries by diffusion
- Transport of O_2 and CO_2 through pulmonary and systemic circulation by bulk flow
- Exchange of O_2 and CO_2 between blood in tissue capillaries and cells in tissues by diffusion
- Cellular utilization of O_2 and production of CO_2



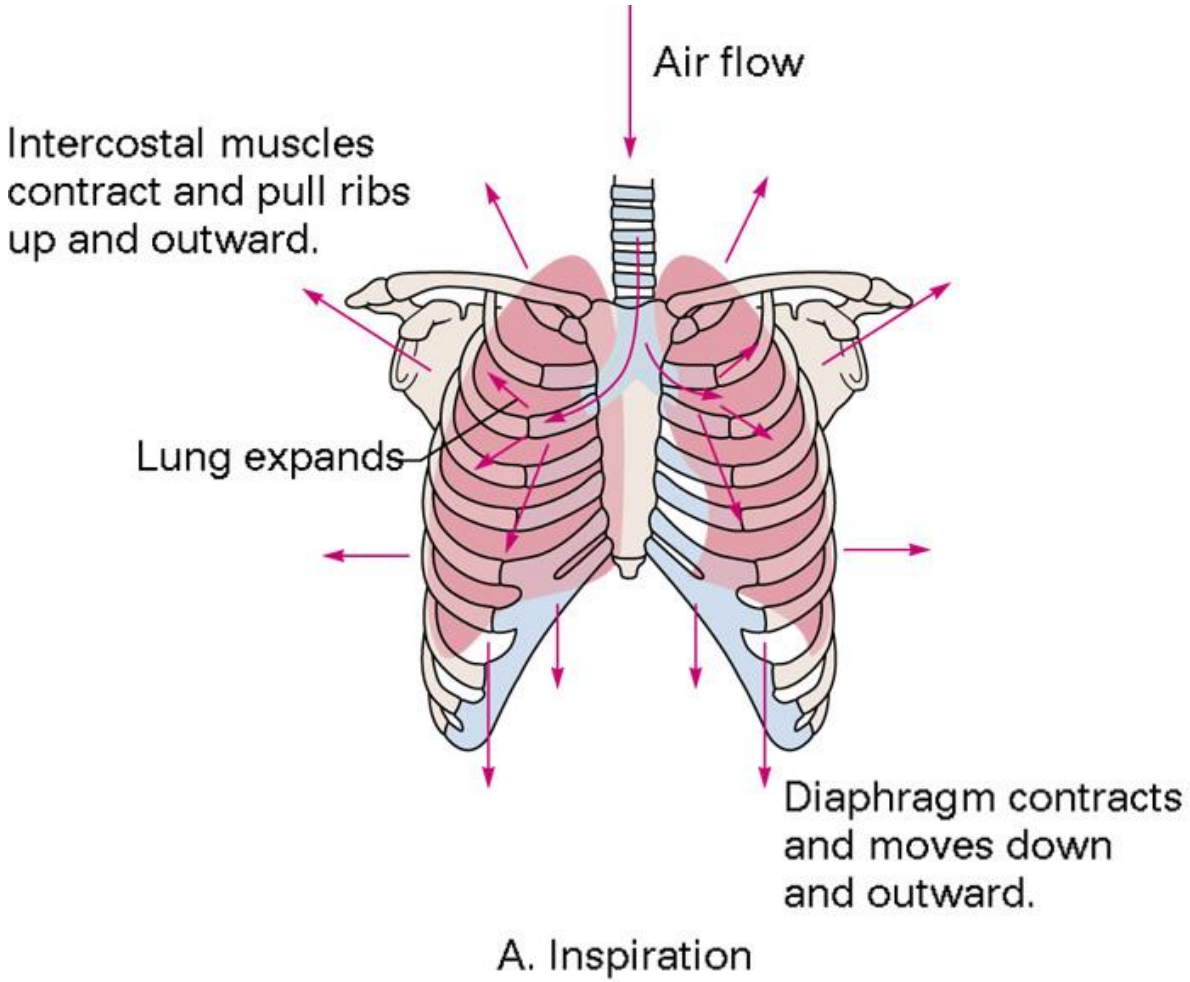
Pressure/Volume Relationship

- Lung structurally attached at the hilum
- Thin layer of pleural fluid between visceral and parietal pleura
- Pressures
  - Atmospheric
  - Intrapleural
  - Alveolar

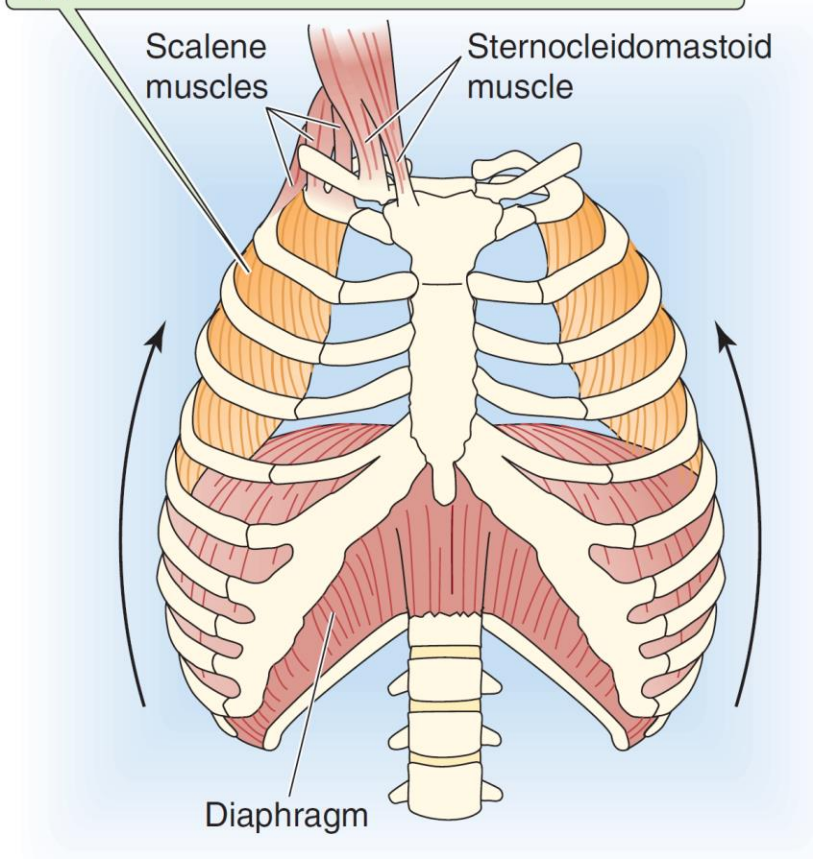
Pressure/Volume Relationship



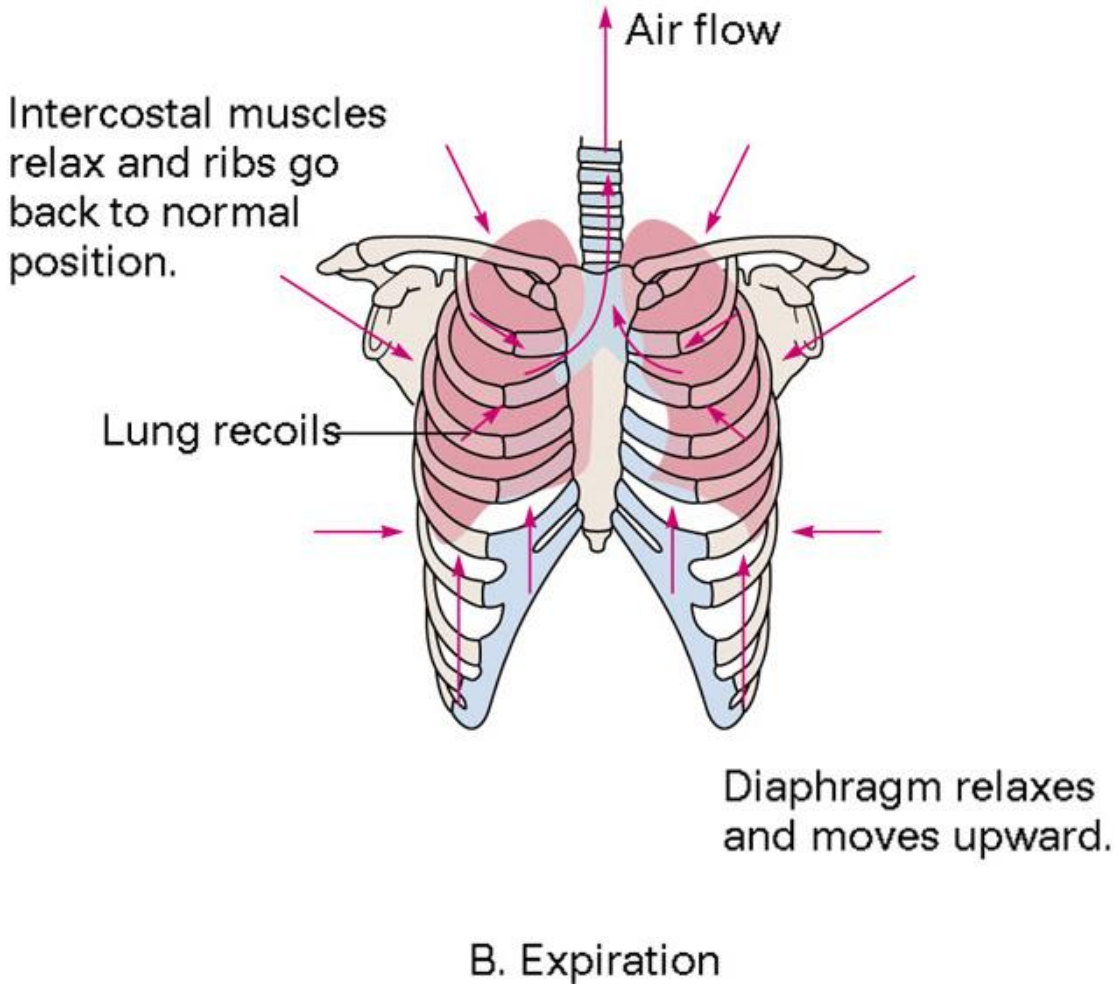
Inspiration



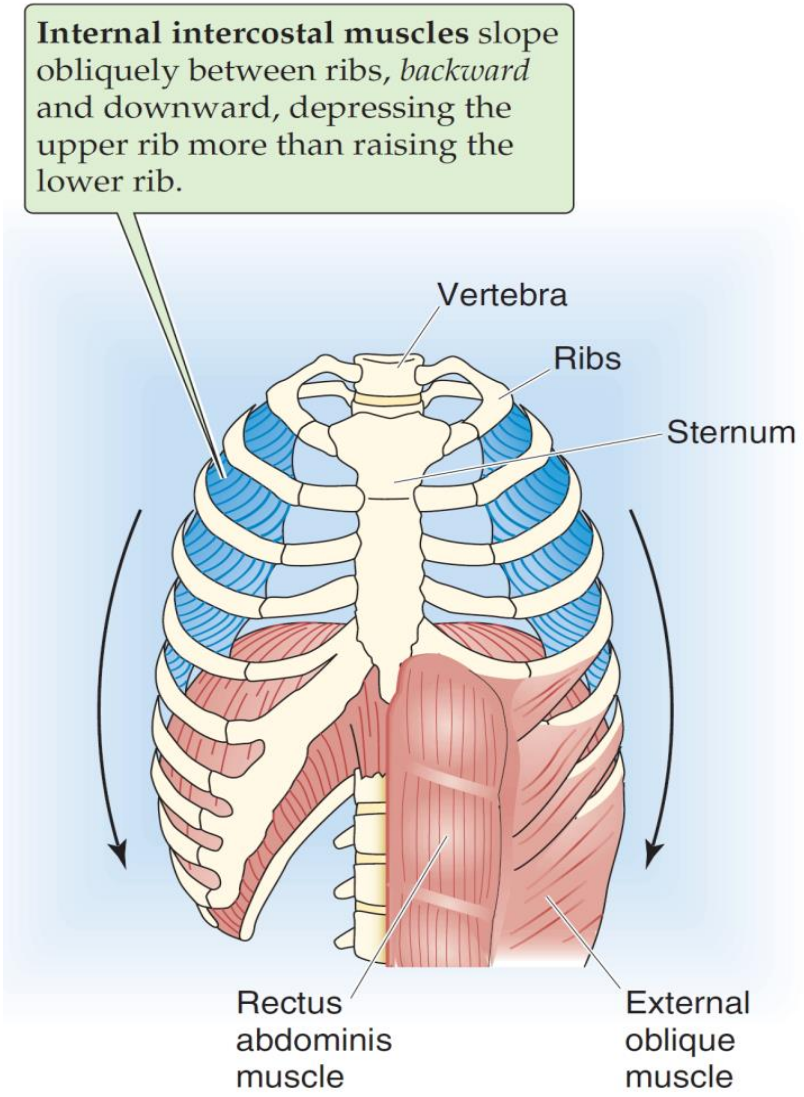
External intercostal muscles slope obliquely between ribs, *forward* and downward. Because the attachment to the lower rib is farther forward from the axis of rotation, contraction raises the lower rib more than it depresses the upper rib.



Expiration



B. Expiration

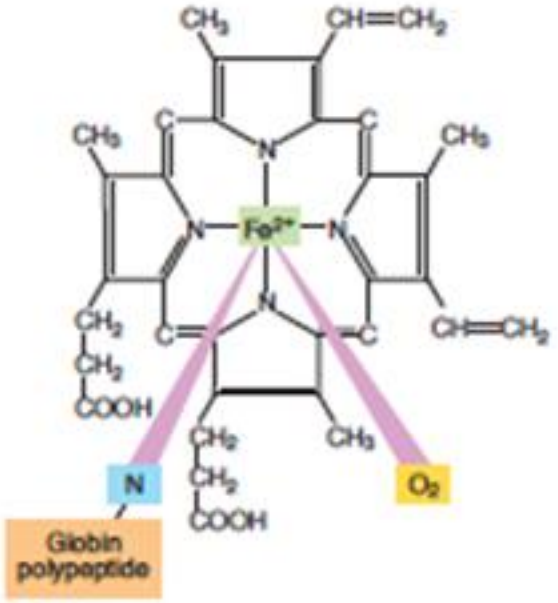


Diffusion

- Process by which gases move between alveoli and pulmonary capillaries
- Gases flow from areas of high to low concentration
- O_2 and CO_2
  - Move across the membrane according to their concentration gradients

Hemoglobin

- Four iron heme and one protein globin molecules
- Oxygen binds to heme molecule
- Carbon monoxide (CO) has an affinity 200x that of oxygen

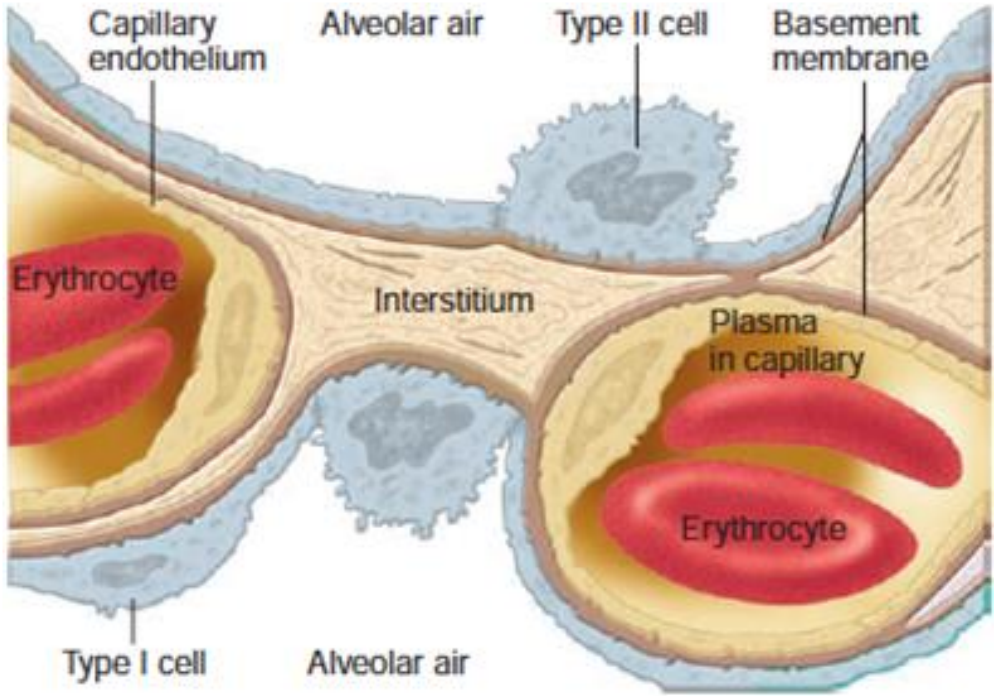


Carbon Dioxide

- Majority transported as bicarbonate ions
  - Transported in red blood cells and released at lungs
- Rest transported
  - Bound to hemoglobin
  - Dissolved in plasma

Alveolar Cells

- Type 1 alveolar cells
- Type 2 great alveolar cells
- Alveolar macrophages



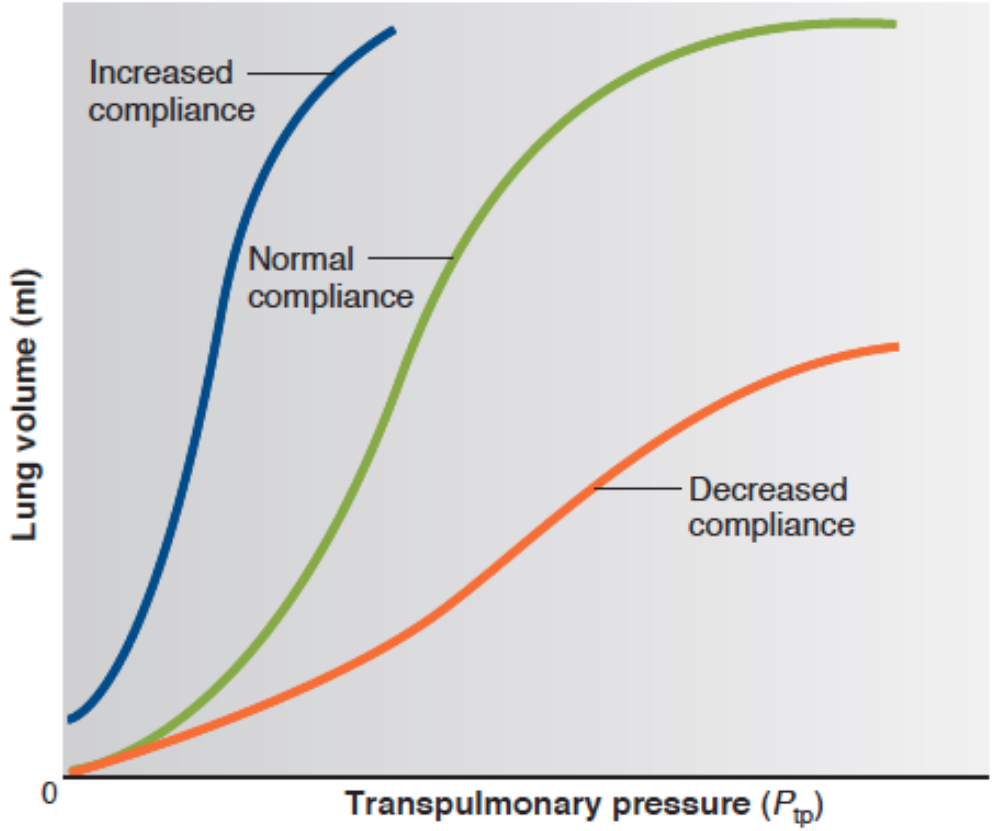
Surfactant and Ventilation

Table 13–4	Some Important Facts About Pulmonary Surfactant

1. Pulmonary surfactant is a mixture of phospholipids and protein.
2. It is secreted by type II alveolar cells.
3. It lowers the surface tension of the water layer at the alveolar surface, which increases lung compliance, thereby making the lungs easier to expand.
4. Its surface tension is lower in smaller alveoli, thus stabilizing alveoli.
5. A deep breath increases its secretion by stretching the type II cells. Its concentration decreases when breaths are small.
6. Production in the fetal lung occurs in late gestation.

Lung Compliance

- The greater the compliance the easier to breathe/ventilate
- The opposite of lung stiffness



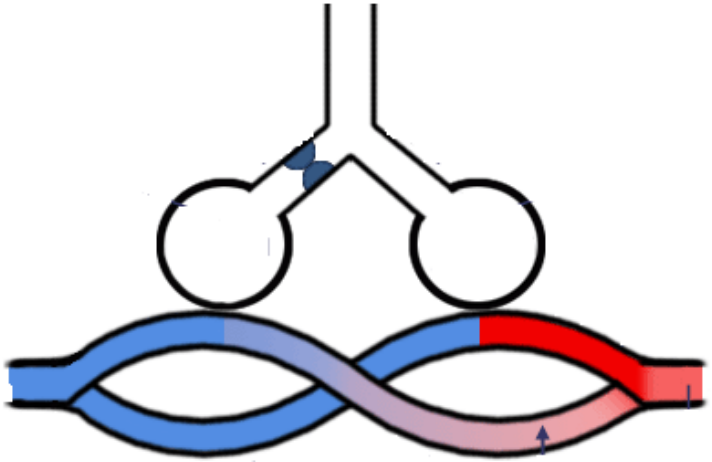
V/Q Mismatch

- V = Ventilation, Q= Perfusion
- Alterations to either side of the equation will cause a shift away from the normal 0.8 ratio
- What would be some potential causes?

V/Q Mismatch

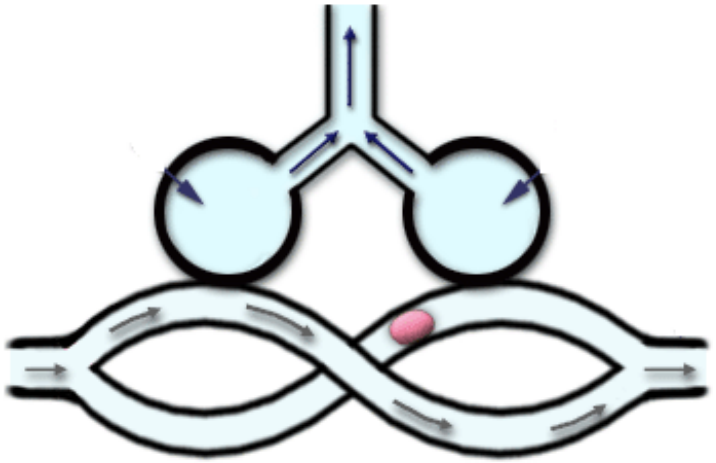
Inadequate Ventilation (Pulmonary Shunt)

- Perfusion exceeds ventilation
- Blood passes through the lung receiving no O_2 at all
- Shunt - producing if V/Q < 0.8



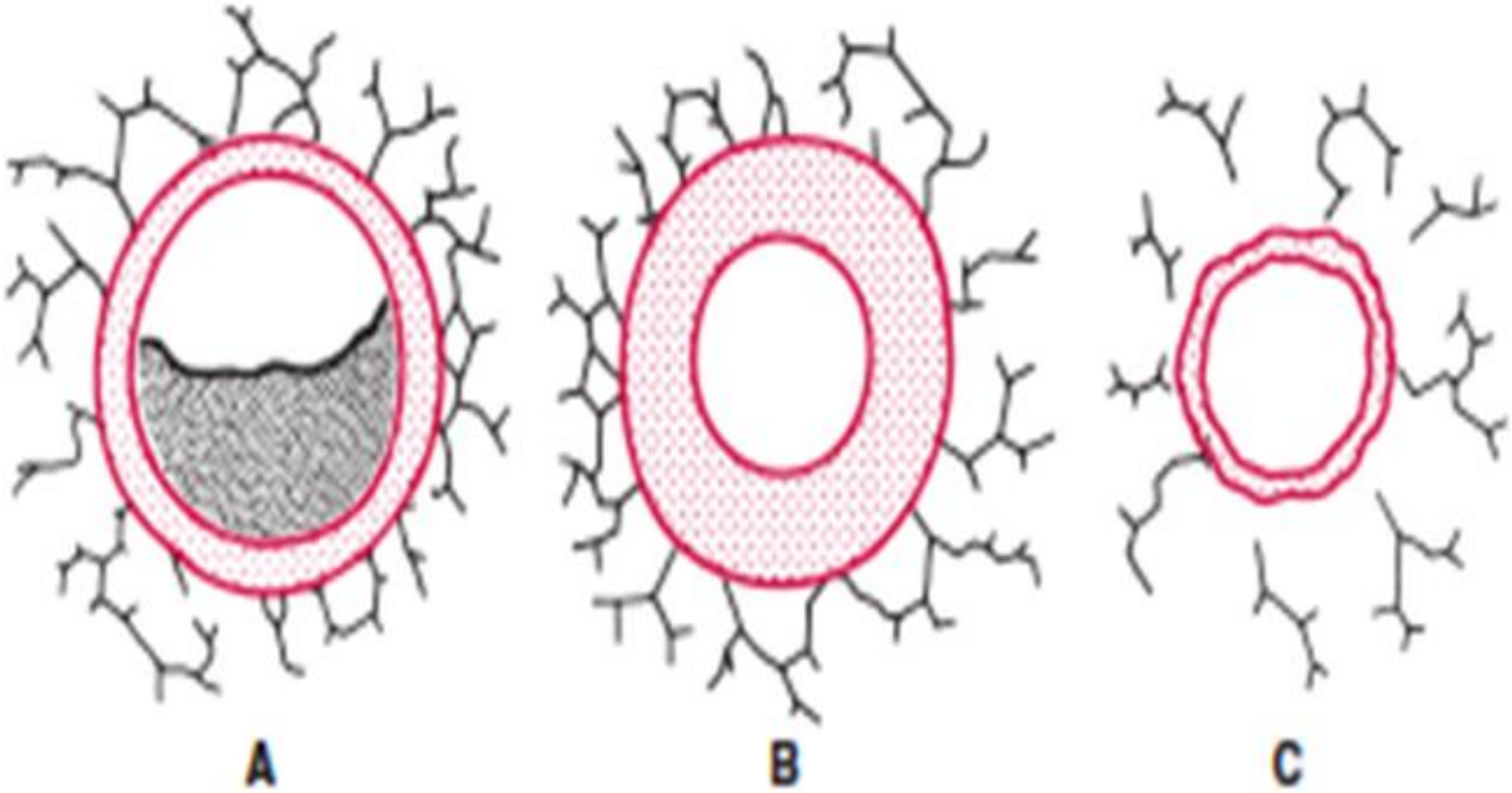
Inadequate Perfusion (Dead Space Ventilation)

- Ventilated exceeds perfusion
- Gas doesn't take part in gas exchange and constitutes an alveolar dead space
- Dead space-producing if V/Q > 0.8



Inadequate Ventilation and Perfusion is a Silent Unit

What Causes AW Resistance?



FEV_1 and FVC

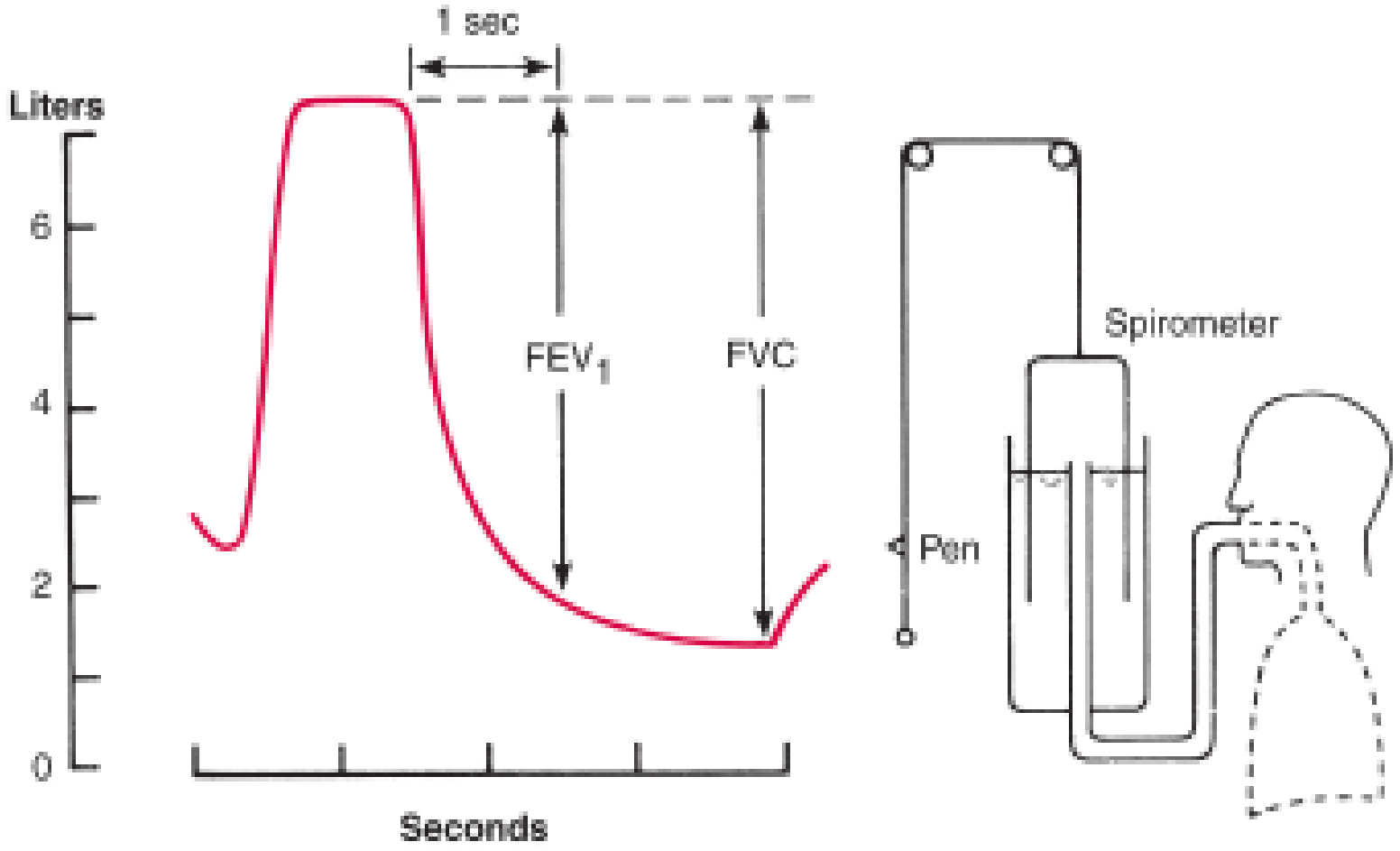


Figure 1-1. Measurement of Forced Expiratory Volume (FEV_1) and Vital Capacity (FVC).

Restrictive vs Obstructive Diseases

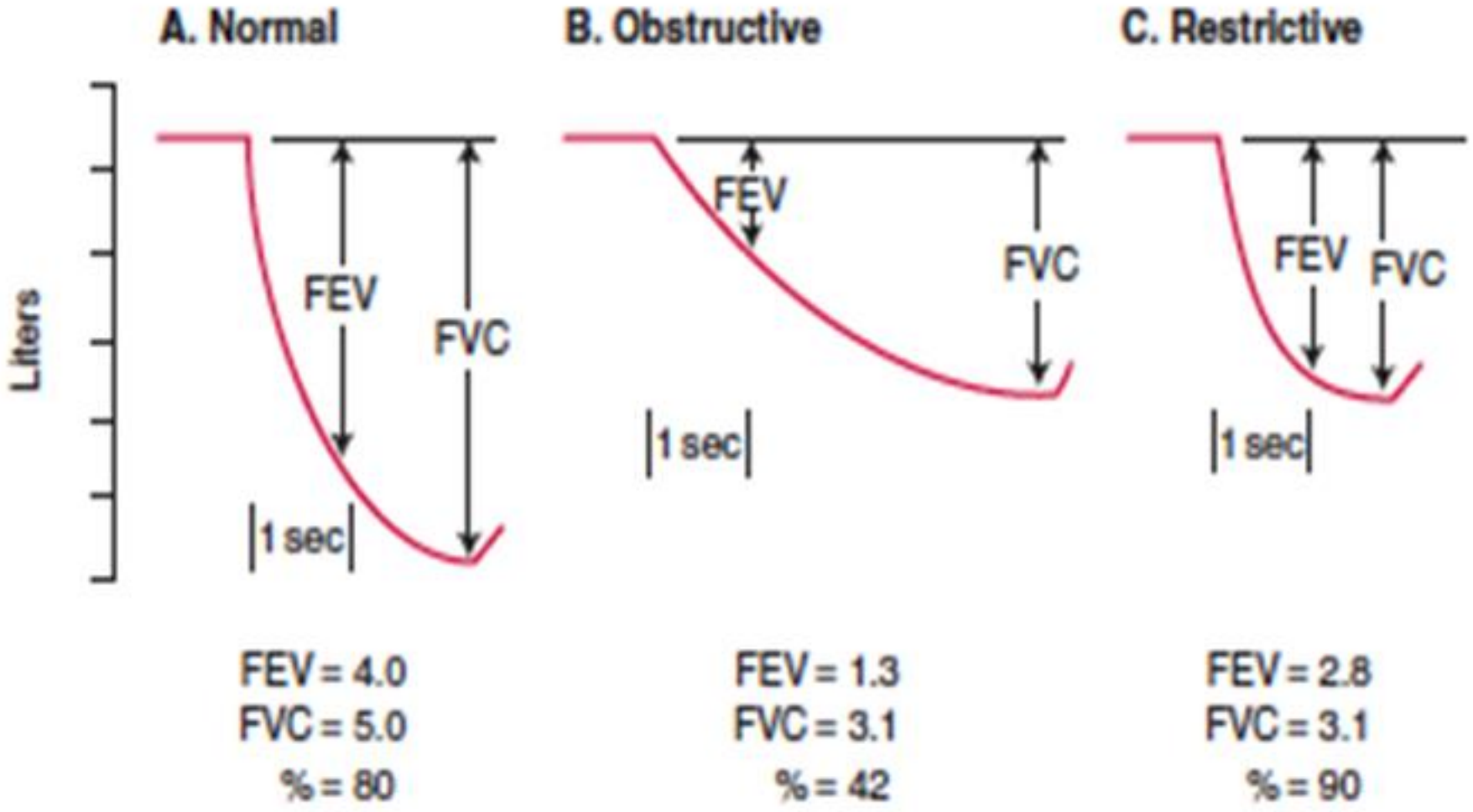


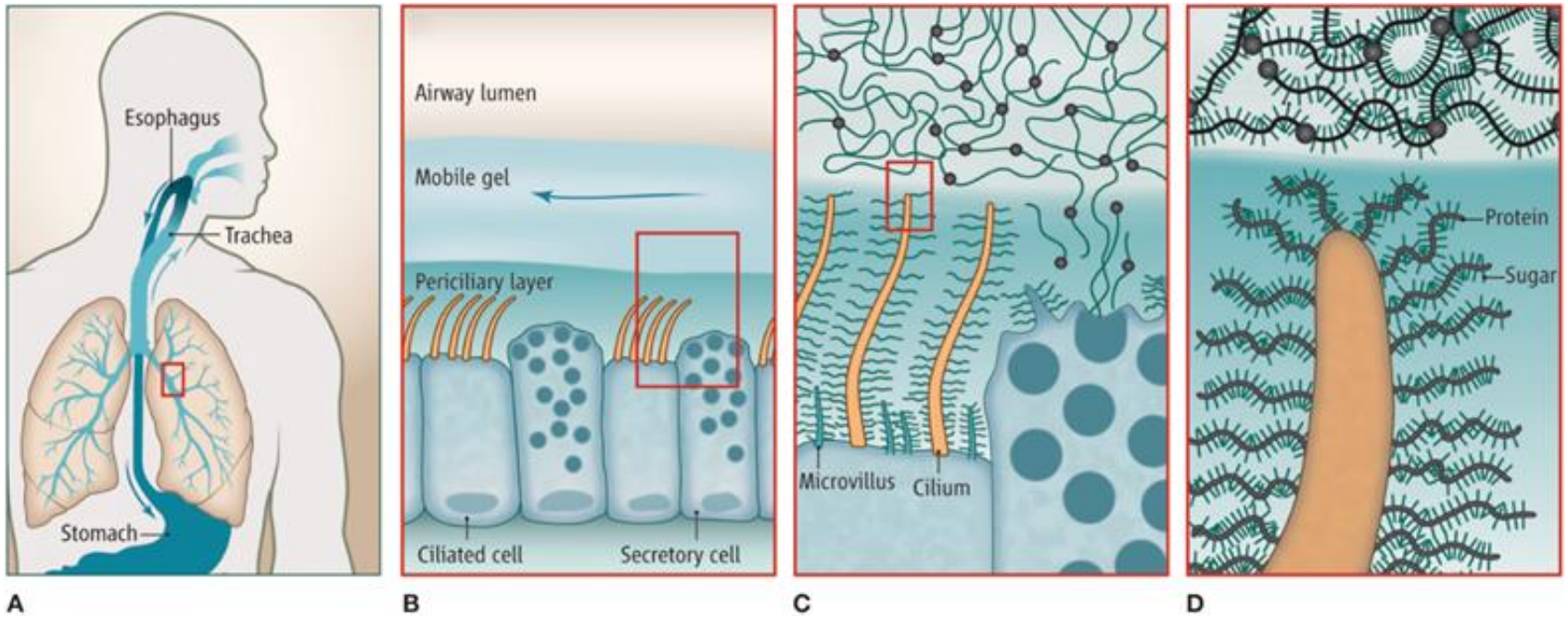
Figure 1-2. Normal, Obstructive, and Restrictive Patterns of a Forced Expiration.

Restrictive vs Obstructive

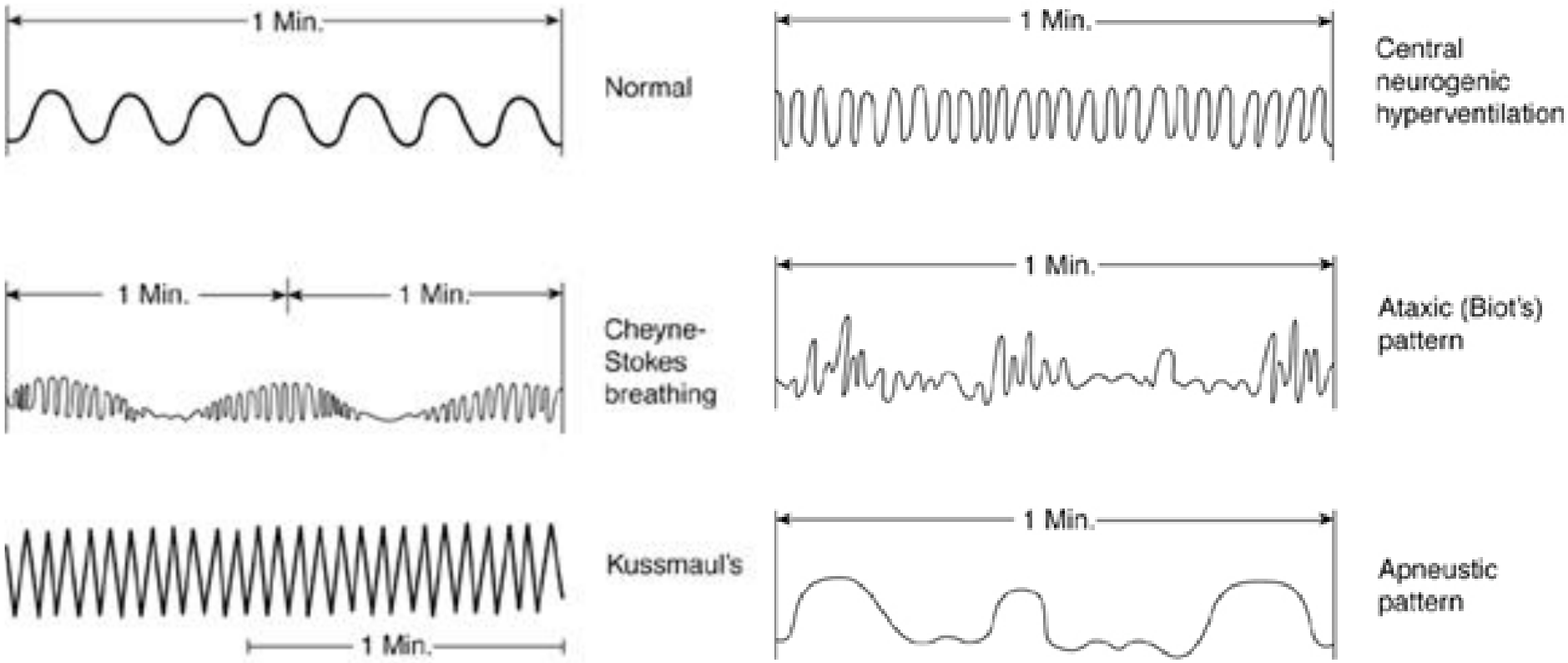
Restrictive	Obstructive
Lung is unable to expand fully	Obstruction to airflow leading to resistance
• Reduced FEV_1, reduced FVC • Reduced lung compliance	• Very reduced FEV_1, FVC • Lung compliance can be normal
• Diseases of the pleura • Diseases of the chest wall • Diseases of neuromuscular apparatus	• Chronic bronchitis • Aspiration of Foreign Material • Asthma • Emphysema (increased compliance)

Protective Lung Functions

- Coughing
- Airway surface liquid (ASL)
- Alveolar macrophages

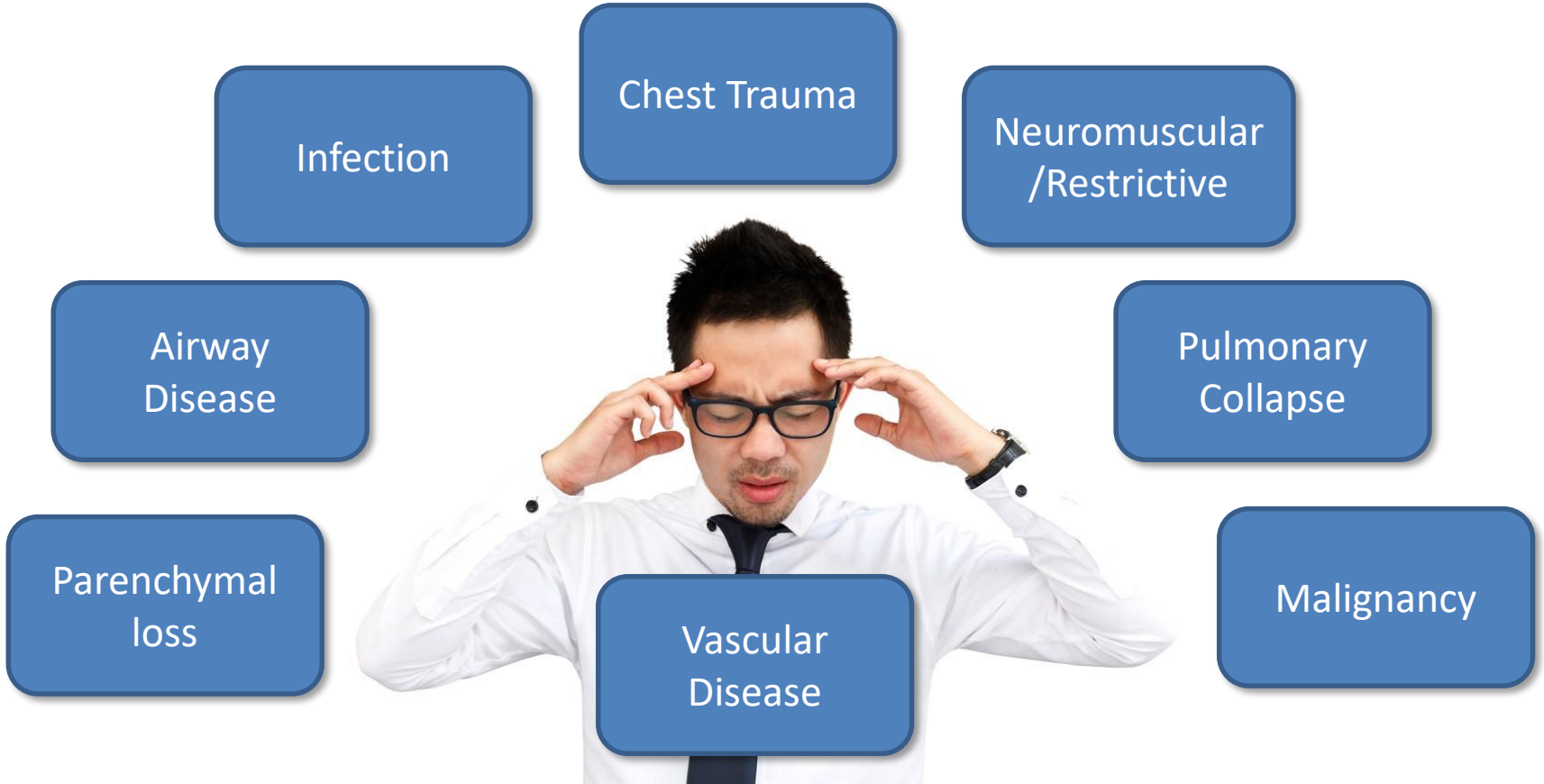


Abnormal Respiratory Patterns



Pathophysiology

- Not as simple as asthma, COPD and pneumonia



Differential Diagnosis

Chest Trauma	Airway Disease	Vascular Disease	Pulmonary Collapse
Flail chest	Asthma	Pulmonary embolism	Pneumothorax
Ruptured diaphragm	COPD		Significant atelectasis
	FBAO		
	Fibrosis		

Infection	Malignancy	Neuromuscular	Parenchymal loss
Bronchitis	Metastasis	ALS	Pulmonary edema
Pneumonia	Lung cancer	Ankylosing spondylitis	Sarcoidosis

Pathophysiology

- Hyperventilation syndrome
- COPD
- Asthma
- Pneumonia
- Pulmonary Edema
  - ARDS
- PE & DVT
- Pleural Effusion
- SARS

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Pathophysiology and Respiratory Disorders

HYPERVENTILATION SYNDROME

Hyperventilation Syndrome

- Ventilation in excess of metabolic needs
- Controversial
  - Some suggest removing this condition
- Etiology
  - Organic
    - CNS
    - Acidosis
  - Panic
    - Hyperventilation due to panic is controllable
      - Voluntary vs Involuntary

Hyperventilation Syndrome

Signs and Symptoms	Management

• Light-headedness
• Paresthesias
• Palpitations
• Diaphoresis
• Carpopedal spasm
	• Reassurance
	  • Coached breathing
	  • Data suggests just as effective, it not more effective than sedative medications
	• Sedatives

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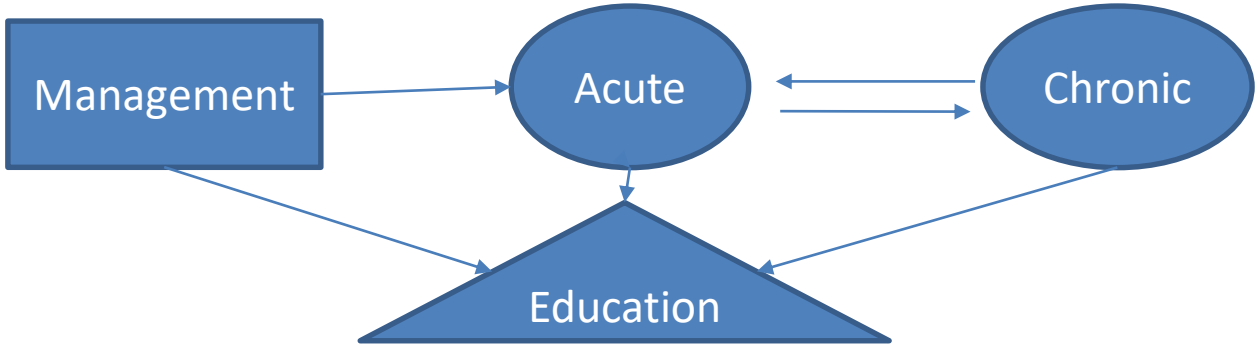
Pathophysiology and Respiratory Disorders

COPD

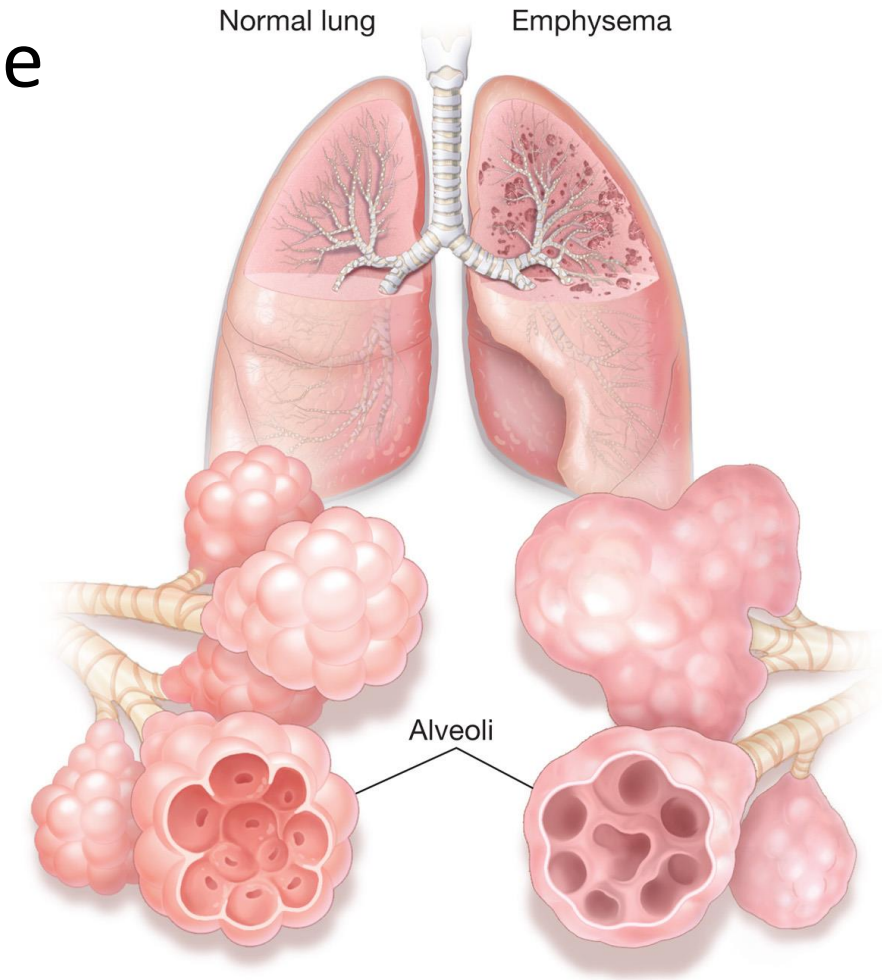
Chronic Obstructive Pulmonary Disease (COPD)

- In 2005 it was estimated that 4.4% (700,000) of Canadians aged 35 years or older have probable COPD.
- The prevalence of COPD among men is 3.9% and 4.8% among women.
- Highest rates for admission and readmission to hospital due to chronic disease in Canada
- Most minor-moderate severity patients die from cardiac causes and lung cancer as a result of COPD



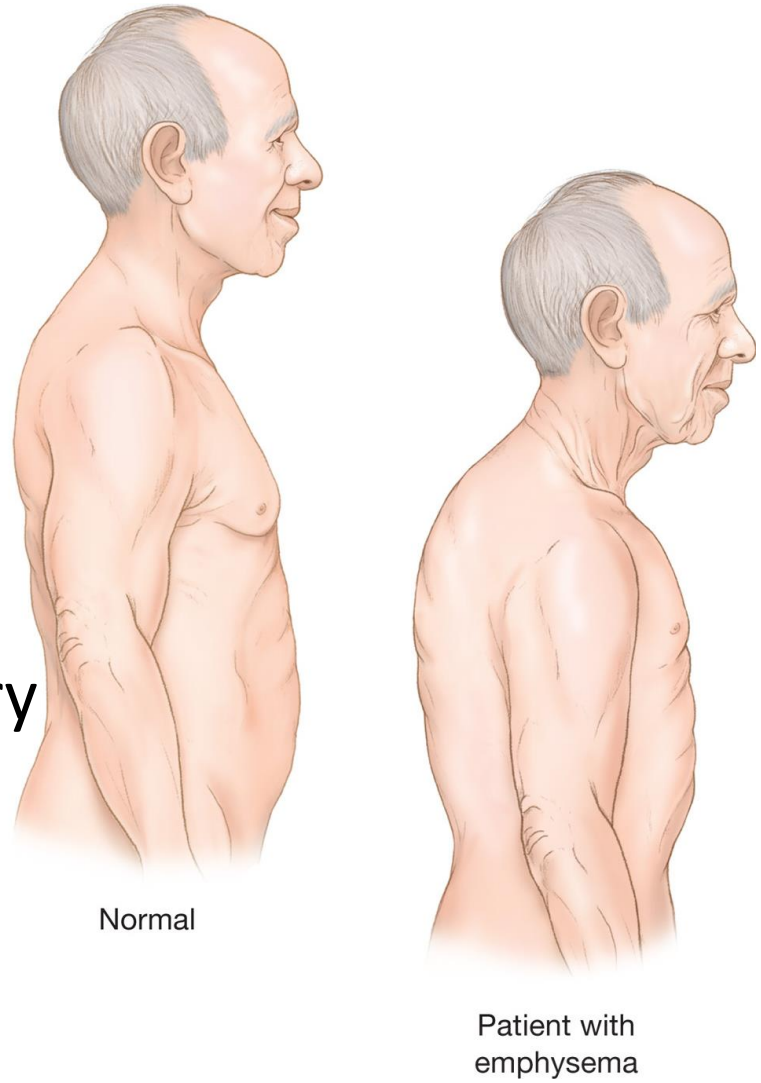
Emphysema

- Destruction of alveolar walls distal to the terminal bronchioles
- Contributing factors
  - Heredity
  - Cigarette smoking
  - Environmental factors



Pathophysiology

- Weakening of alveolar walls
  - Loss of elastic recoil
  - Air trapping
  - Pursed lipped breathing
  - Barrel chest
- Unable to expel carbon dioxide
  - Chronic increased respiratory rate and accessory muscle use
  - SOB OE
  - Polycythemia

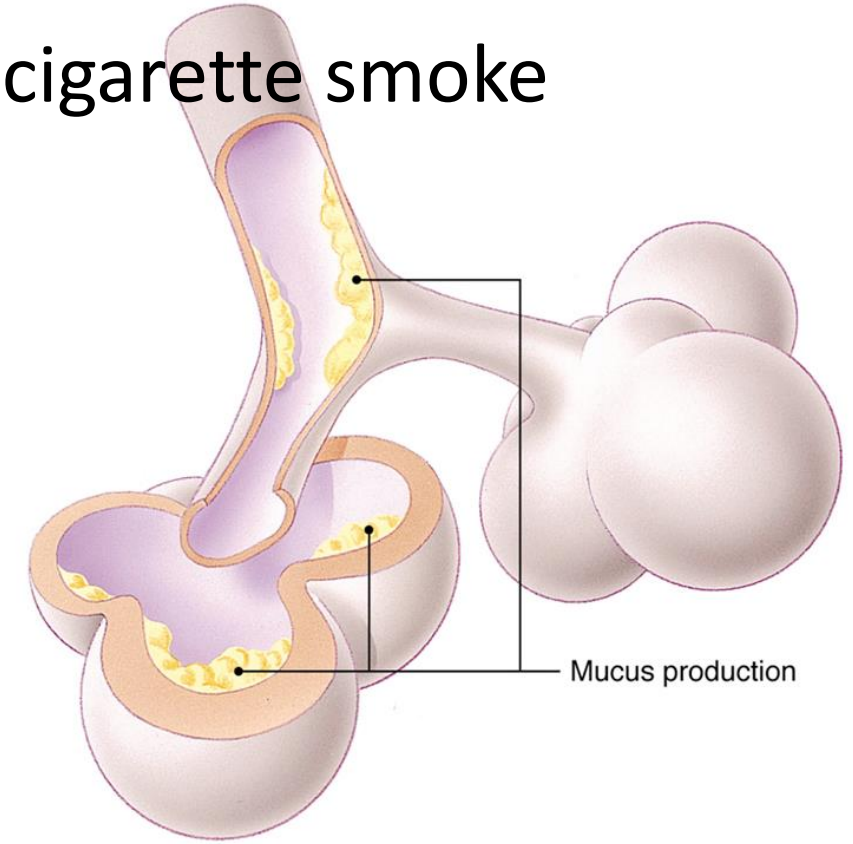


Assessment

- History
  - Recent weight loss, dyspnea with exertion
  - Cigarette and tobacco usage
  - Lack of cough
- Physical Exam
  - Barrel chest
  - Prolonged expiration and rapid rest phase
  - Thin
  - Pink skin due to extra red cell production
  - Hypertrophy of accessory muscles
  - “Pink Puffers

Chronic Bronchitis

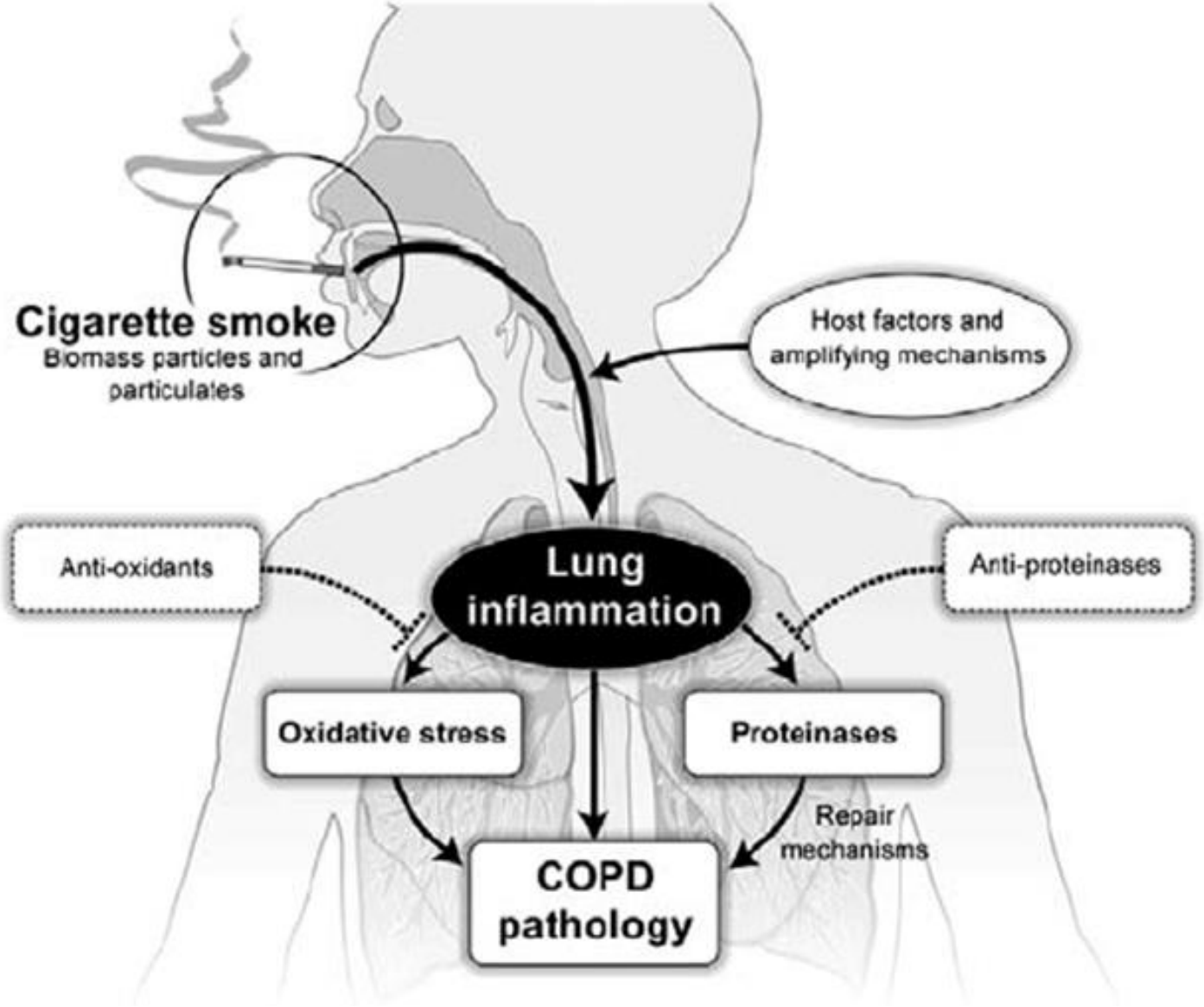
- Increased number of goblet cells in the respiratory tree
- Production of large quantity of sputum
- Often occurs after prolonged exposure to cigarette smoke



Pathophysiology

- Alveoli not severely affected
- Gas exchange is compromised
  - Decreased alveolar ventilation
- Hypoxia
  - Pulmonary vasoconstriction
  - Cor pulmonale
- Vital capacity is decreased

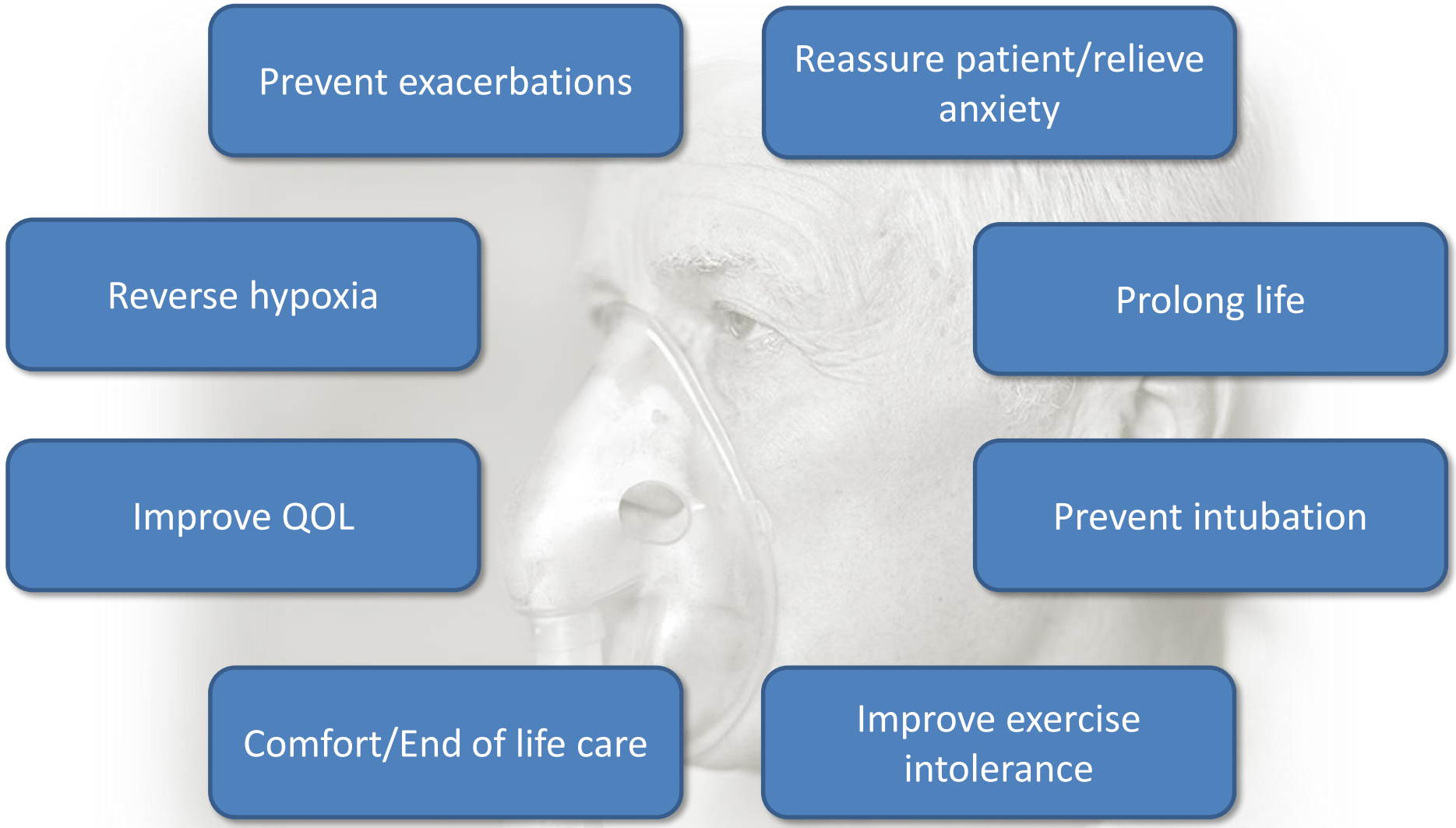
COPD


Cigarette smoke
Biomass particles and particulates
Host factors and amplifying mechanisms
Anti-oxidants
Lung inflammation
Anti-proteinases
Oxidative stress
Proteinases
Repair mechanisms
COPD pathology

COPD

- Incurable
- Progressive
  - The only thing that can slow the course of the disease is?
- Most common cause of exacerbation is viral infection and tracheobronchial bacterial infection
  - Always suspect in the SOB COPD patient

Goals of Therapy



COPD Severity

Table 1: COPD classification by symptoms/disability		
COPD stage‡	**Symptoms**	**Spirometry**
At Risk (not yet COPD)	Asymptomatic smoker or ex-smoker or chronic cough/ sputum	$FEV_1 \geq 80\%$ predicted $FEV_1 / FVC \geq 0.7$
Mild	Shortness of breath from COPD with strenuous exercise or while hurrying on the level or walking up a slight hill	FEV_1 60% - 79% predicted $FEV_1 / FVC < 0.7$
Moderate	Shortness of breath from COPD causing the patient to walk slower than most people of the same age on the level or stop after walking about 100 m on the level	FEV_1 40% - 59% predicted $FEV_1 / FVC < 0.7$
Severe	Shortness of breath from COPD resulting in the patient too breathless to leave the house, or breathless after dressing or undressing or the presence of chronic respiratory failure or clinical signs of right heart failure	FEV_1 30% - 39% predicted $FEV_1 / FVC < 0.7$
Very Severe		$FEV_1 < 30\%$ predicted $FEV_1 / FVC < 0.7$

COPD Diagnosis & S/S

Signs and Symptoms

- Pursed lip breathing
- AMU
- Pulsus paradoxus
- Tacypnea
- Tacyhcardia
- Hypertension

When to consider COPD diagnosis

Dyspnea that is: Progressive (worsens over time). Characteristically worse with exercise. Persistent.
Chronic cough: May be intermittent and may be unproductive.
Chronic sputum production: Any pattern of chronic sputum production may indicate COPD.
History of exposure to risk factors: Tobacco smoke (including popular local preparations). Smoke from home cooking and heating fuels. Occupational dusts and chemicals.
Family history of COPD

Spirometry

AECOPD

Cardinal Symptoms of AECOPD	
Increase in **dyspnea** Increased sputum **volume** Increased sputum **purulence**	Sustained for 48 hours compared to baseline
One cardinal symptom:	Treatment with antibiotics may not be necessary
Two cardinal symptoms:	Treat with antibiotics if one symptom is increased purulence
Three cardinal symptoms:	Always treat with antibiotics

AECOPD Management

Acute

Reverse hypoxia

- Oxygen
- Salbutamol
- Anticholinergic
- Identify cause

Infection

- Exertion
- Irritants
- Heart Failure

Identify severity or risk of decompensating

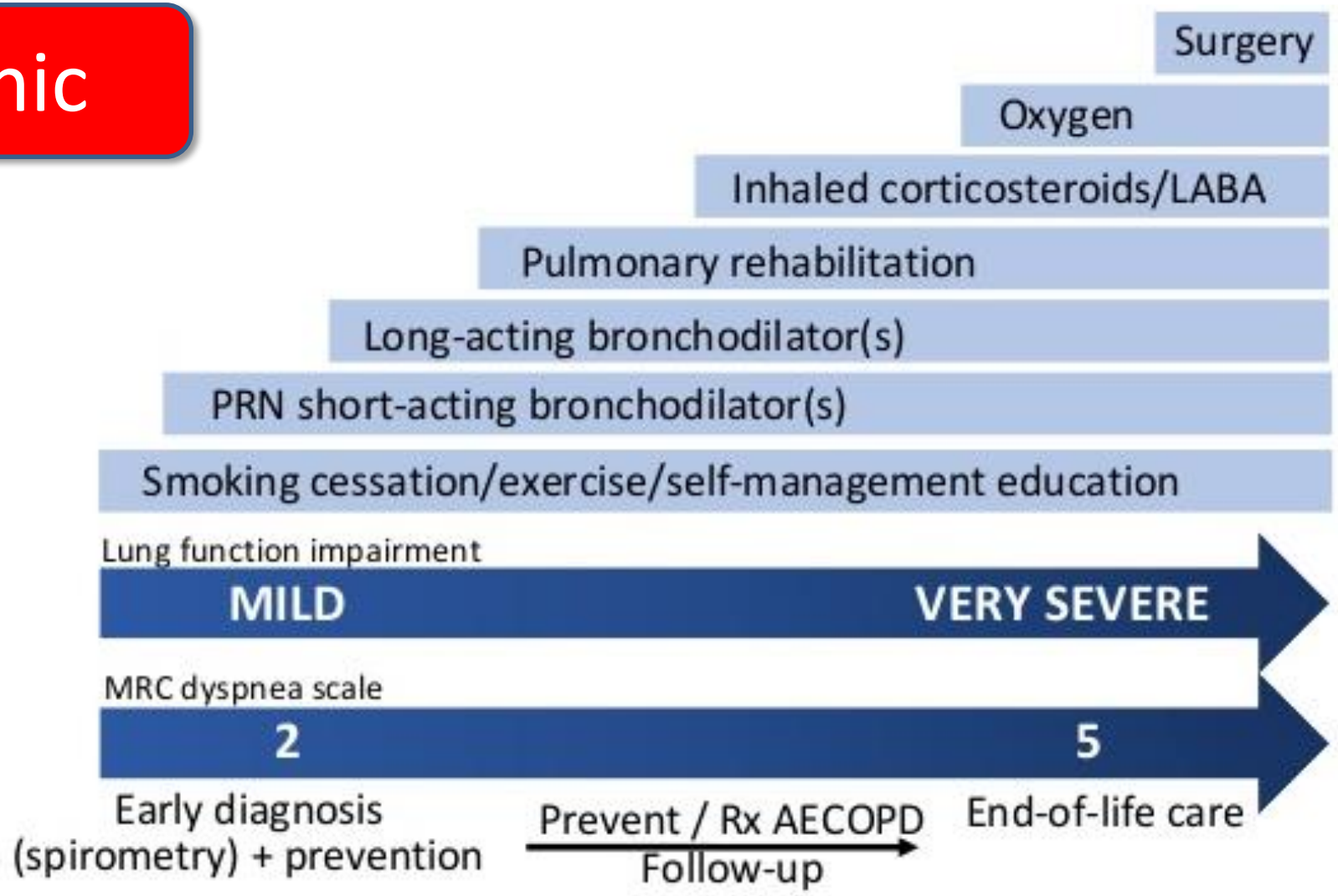
- Objective exam
- Recent intubations
- Prior history of hospitalizations

Manage decompensation

- Mechanical ventilation
- ALS

AECOPD Management

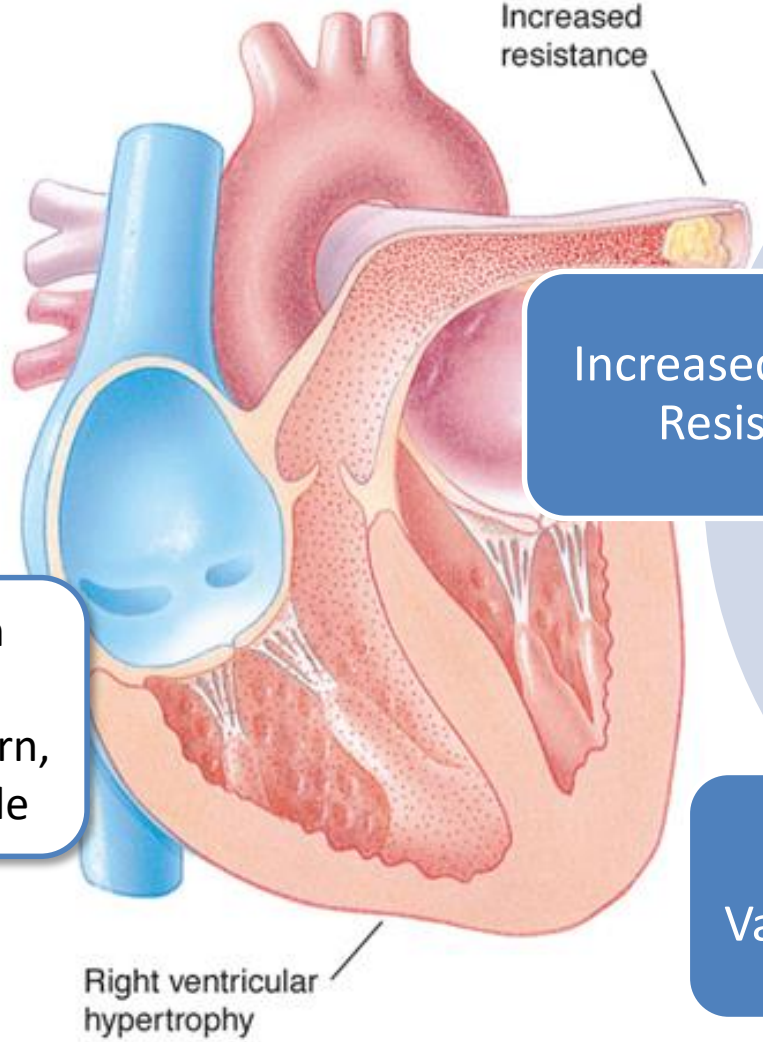
Chronic



Hypoxic Drive

- Normally the amount of pCO_2 stimulates the respiratory drive in the healthy individual
- It is theorized that prolonged exposure to high pCO_2 may result in the patient's normal respiratory drive to change to rely on levels of pO_2
- A universal misnomer is that if you give too much oxygen to patients with COPD they may lose their stimulus to breath.
  - Physicians will cite rising CO_2 levels in patients treated with oxygen as evidence of this.
- The fundamental flaw in this theory:
  - It is the blood oxygen content that is important, not the inspired fraction.
  - Patients, depending on the extent of disease, will have differing extents of V/Q mismatch and diffusion defects
  - the patient needs enough inspired oxygen to return the PO_2 to what is normal for them

Cor Pulmonale



Long standing COPD can cause pulmonary hypertension, which, in turn, may lead to cor pulmonale

Decreased Ventricular Performance

Reduced Cardiac Output

Compensatory Responses:

- Sympathetic stimulation
- RAAS ↑
- ADH ↑
- ANP ↑

↑ Systemic Vascular Resistance

Increased Outflow Resistance

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Pathophysiology and Respiratory Disorders

ASTHMA

Asthma

- In early life, the prevalence of asthma is higher in boys. At puberty, however, the sex ratio shifts, and asthma appears predominantly in women
- A number of long-term prospective studies of children admitted to hospital with documented RSV have shown that approximately 40 percent of these infants will continue to wheeze or have asthma in later childhood
- Atopy is a genetic predisposition for the development of an immunoglobulin E (IgE)-mediated response to common aeroallergens, is the strongest identifiable predisposing factor for developing asthma. (“Hyperallergic”)
  - Usually consists of:
    - Asthma
    - Allergic rhinitis
    - Eczema (dermatitis)

Asthma

Pathophysiology

- Bronchoconstriction
- Hyper-responsiveness
- Remodeling
- Edema

Diagnosis and S/S

Key clinical indicators

The demonstration of variable expiratory airflow compromise, preferably by spirometry

Exclude other potential diagnosis

Triggers
Pet Dander
Pollen
Cockroaches
Mold
Smoke, strong odours
Exercise
Sulfites
Cold air

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Diagnosis and S/S

BOX 3–1. KEY INDICATORS FOR CONSIDERING A DIAGNOSIS OF ASTHMA

Consider a diagnosis of asthma and performing spirometry if any of these indicators is present.* These indicators are not diagnostic by themselves, but the presence of multiple key indicators increases the probability of a diagnosis of asthma. Spirometry is needed to establish a diagnosis of asthma.

- Wheezing—high-pitched whistling sounds when breathing out—especially in children. (Lack of wheezing and a normal chest examination do not exclude asthma.)
- History of any of the following:
  - Cough, worse particularly at night
  - Recurrent wheeze
  - Recurrent difficulty in breathing
  - Recurrent chest tightness
- Symptoms occur or worsen in the presence of:
  - Exercise
  - Viral infection
  - Animals with fur or hair
  - House-dust mites (in mattresses, pillows, upholstered furniture, carpets)
  - Mold
  - Smoke (tobacco, wood)
  - Pollen
  - Changes in weather
  - Strong emotional expression (laughing or crying hard)
  - Airborne chemicals or dusts
  - Menstrual cycles
- Symptoms occur or worsen at night, awakening the patient.

*Eczema, hay fever, or a family history of asthma or atopic diseases are often associated with asthma, but they are not key indicators.

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Asthma Exacerbation

Symptoms	Pattern	Disease History	Risk Factors for Death from Asthma
Cough	Perennial and/or seasonal	Age at onset	Past history of severe exacerbation
Wheezing	Continual or episodic	Present management and medications	≥2 hospitalizations for asthma in the past year
Shortness of breath	Onset	Medication regimen adherence	>3 ED visits for asthma in the past year
Chest tightness	Duration	History of corticosteroid use (chronic and/or intermittent)	>2 canisters per month of inhaled short-acting β_2-agonist
Sputum production	Frequency	Intensive care admissions	Difficulty perceiving airflow obstruction or its severity
Fever	Aggravating factors	History of intubation	Low socioeconomic status or inner-city resident
	Usual pattern of exacerbation and outcome	Best spirometry measures	Illicit drug use
			Psychiatric disease or medical comorbidities

Asthma Emergency Management

Management

- Early recognition, assessment of severity and prompt initiation of treatment
- Triggers
- Rapid reversal of bronchospasm
- Oxygen
- Beta agonists
- Anticholinergic medication
- Epinephrine
- Mechanical ventilation
- CPAP
- BVM
- Additional Treatments (ACP)
  - Reduce inflammation, complications and ICU admission
  - Systemic corticosteroids
  - Magnesium
  - Mechanical ventilation
  - Intubation

Status Asthmaticus

- Asthma exacerbation that do not respond to conventional therapy
- Physical Exam
  - Wheezing is not present in all asthmatics
    - Silent chest
  - Speech may be limited to 1–2 consecutive words
  - Hyperinflation of the chest and accessory muscle use

FIGURE 5–2a. RISK FACTORS FOR DEATH FROM ASTHMA

Asthma history

Previous severe exacerbation (e.g., intubation or ICU admission for asthma)
Two or more hospitalizations for asthma in the past year
Three or more ED visits for asthma in the past year
Hospitalization or ED visit for asthma in the past month
Using >2 canisters of SABA per month
Difficulty perceiving asthma symptoms or severity of exacerbations
Other risk factors: lack of a written asthma action plan, sensitivity to *Alternaria*

Social history

Low socioeconomic status or inner-city residence
Illicit drug use
Major psychosocial problems

Comorbidities

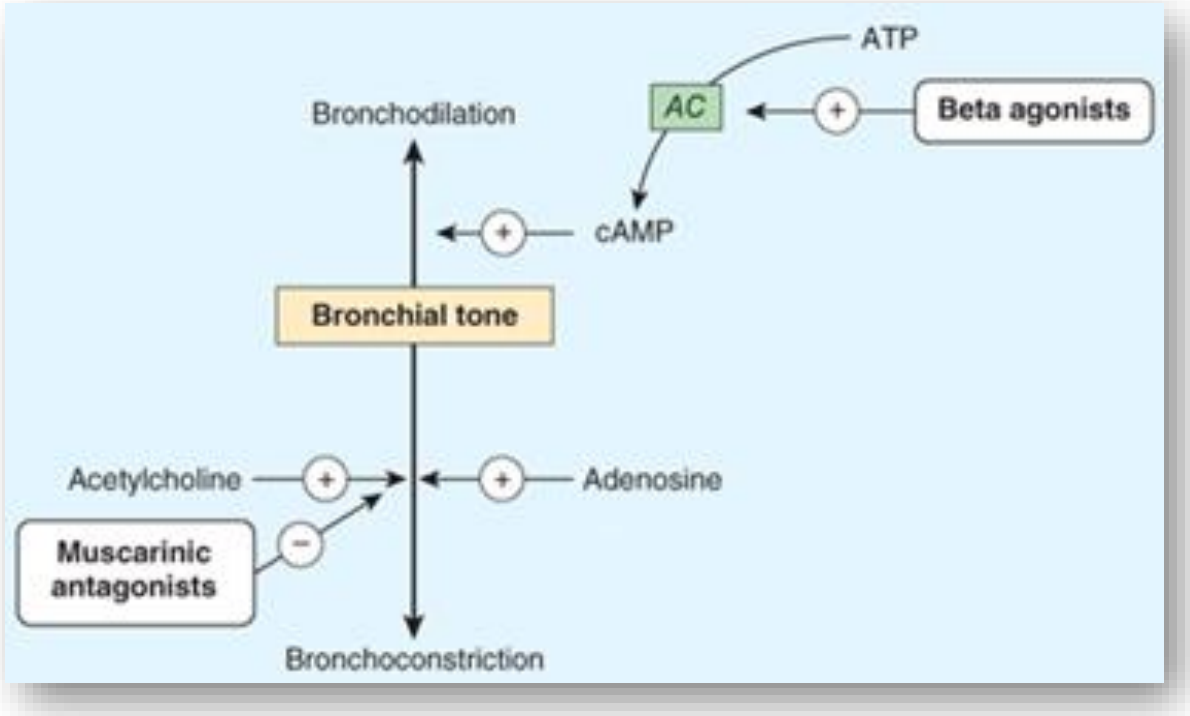
Cardiovascular disease
Other chronic lung disease
Chronic psychiatric disease

Key: ED, emergency department; ICU, intensive care unit; SABA, short-acting beta$_2$-agonist

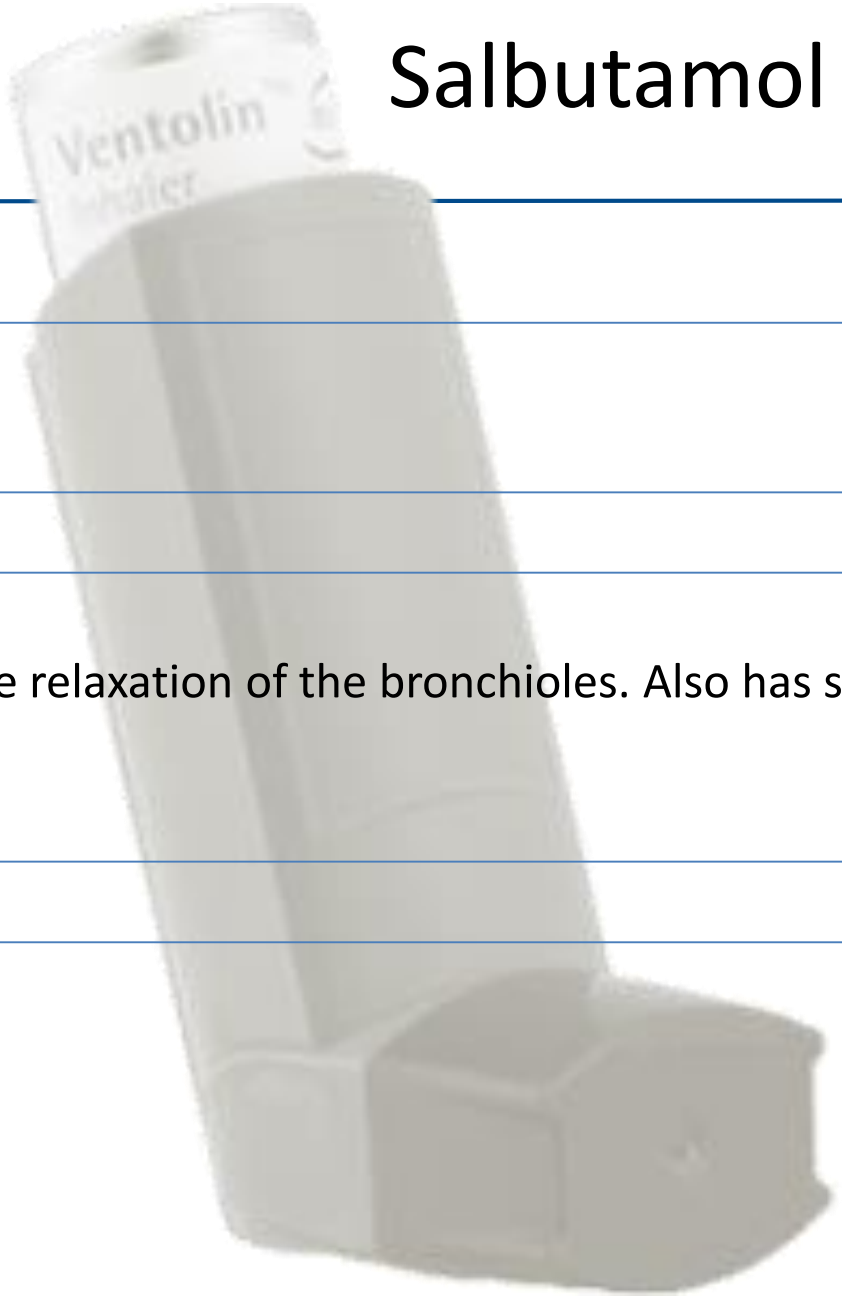
Sources: Abramson et al. 2001; Greenberger et al. 1993; Hardie et al. 2002; Kallenbach et al. 1993; Kikuchi et al. 1994; O'Hollaren et al. 1991; Rodrigo and Rodrigo 1993; Strunk and Mrazek 1986; Suissa et al. 1994

Beta Agonist

- Cornerstone of asthma management
- The most common adverse drug reaction of β-adrenergic drugs is skeletal muscle tremor



Salbutamol (Ventolin)



Classification

- Bronchodilator, sympathomimetic B-2 agonist

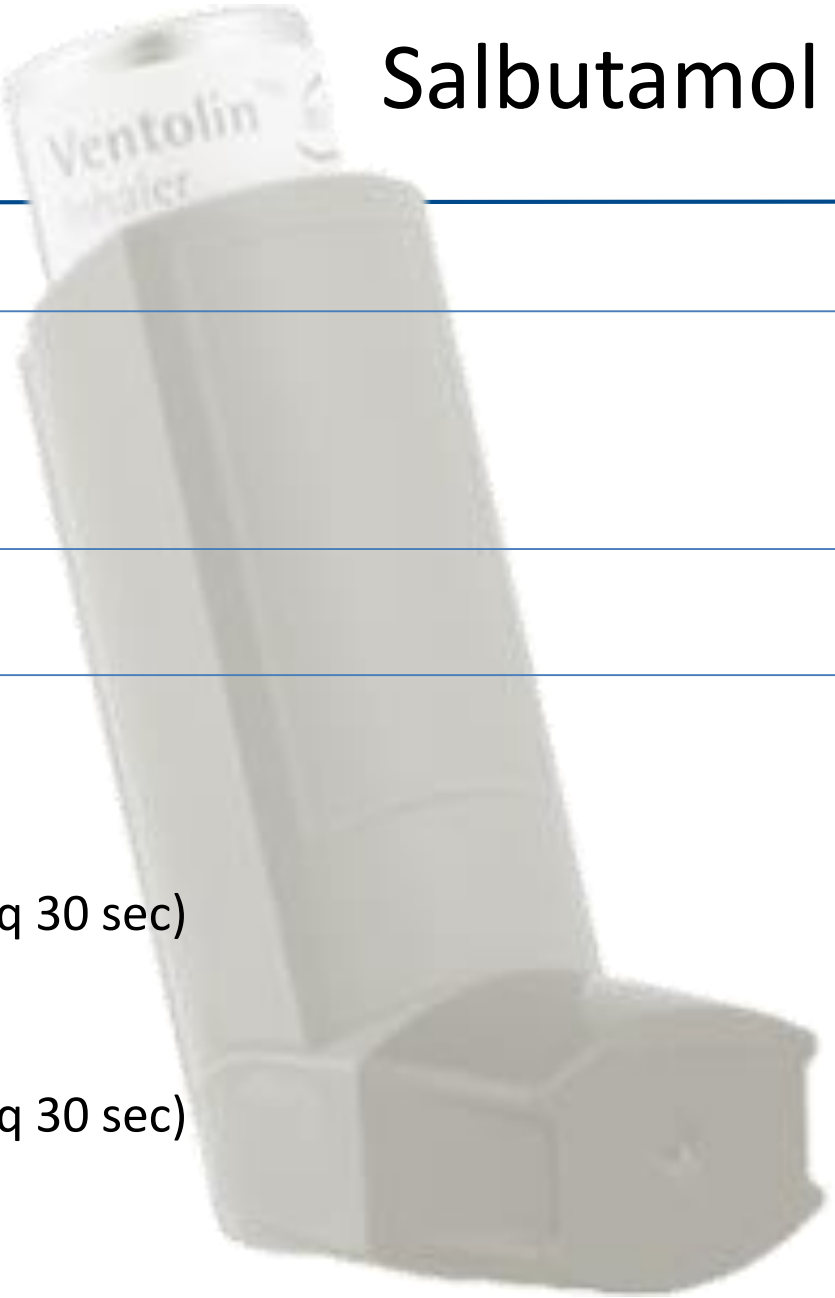
Mechanism of Action

- Selective B-2 stimulation allows for smooth muscle relaxation of the bronchioles. Also has some B-1 affects causing an increase in HR

Indications

- Bronchoconstriction (wheeze) with SOB

Salbutamol (Ventolin)



Contraindications

- Hypersensitivity
- Ischemic Chest Pain (Relative)

Dosage

- Adults
  - 5.0 mg Aerosol
  - 400 – 600 mcg (4 - 6 puffs) via MDI (1 puff q 30 sec)
- Pediatric (10 – 30 kg)
  - 2.5 mg Aerosol
  - 200 – 300 mcg (2 - 3 puffs) via MDI (1 puff q 30 sec)
- Infant (< 10 kg)
  - 1.25 mg via Aerosol

Ipratropium Bromide

- Anticholinergics:
  - Inhibit muscarinic cholinergic receptors and reduce intrinsic vagal tone of the airway.
  - Ipratropium bromide provides additive benefit to SABA in moderate-to-severe asthma exacerbations.
- Chronically - May be used as an alternative bronchodilator for patients who do not tolerate their salbutamol puffer
- Due to poor systemic absorption, ipratropium is ideal for inhalation.

Ipratropium Bromide

Classification

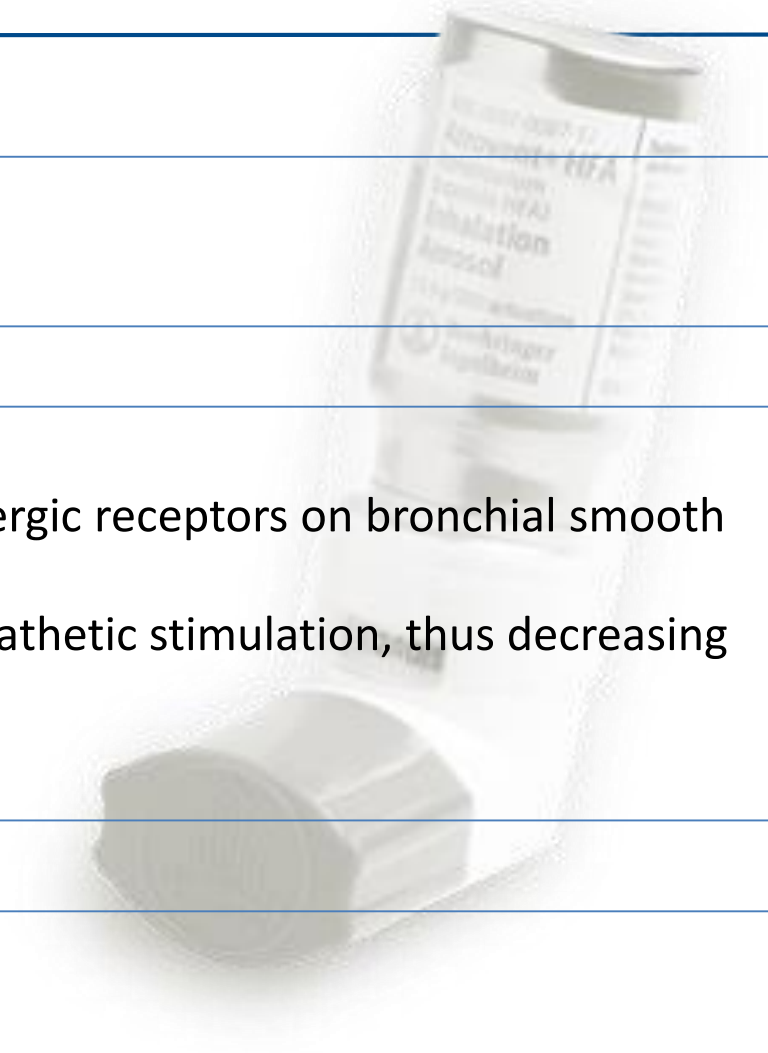
- Anticholinergic, parasympatholytic

Mechanism of Action

- Causes bronchodilation by competitive inhibition of cholinergic receptors on bronchial smooth muscle
- Blocks the action of acetylcholine, which inhibits parasympathetic stimulation, thus decreasing bronchial secretions
- Dries respiratory tract secretions

Indications

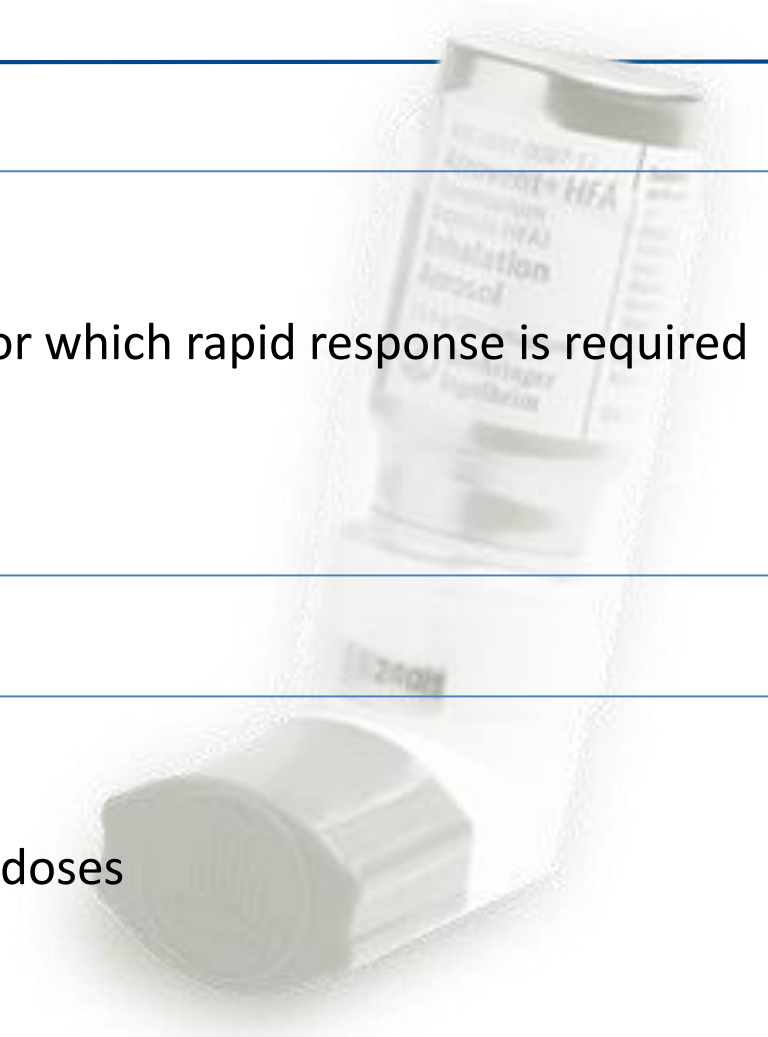
- Bronchial asthma
- Bronchospasm associated with COPD



Ipratropium Bromide

Contraindications

- Known hypersensitivity
- Is not indicated for acute treatment of bronchospasm for which rapid response is required
- Ischemic chest pain
- Acute narrow angle glaucoma



Dosage

- Adult
  - 250-500 mcg mixed with Ventolin q 20 minutes x 3 doses
  - 2-4 puffs q 20 minutes x 3 doses
- Pediatric
  - 125 – 250 mcg mixed with Ventolin and/or NS to a minimum of 2 cc
  - 1-2 puffs

Asthma Medications

- "Relievers" vs "maintainers"

“Dartmouth Spacer”

- Someone did a study on it
  - They are very expensive

Cochrane Library
Cochrane Database of Systematic Reviews

Commercial versus home-made spacers in delivering bronchodilator therapy for acute therapy in children (Review)

Rodriguez-Martinez CE, Sossa M, Lozano JM

Figure 21. Examples of VHCs and spacers

Authors' conclusions

Overall, this review did not identify a statistically significant difference between these two methods for delivering bronchodilator therapy to children with acute asthma or lower airways obstruction attacks. Care should be taken in the interpretation and applicability of our results because of the small number of RCTs along with few events available meeting the criteria for inclusion in the review, absence of the primary outcome of interest and other clinically important outcomes in the majority of included studies. The possible need for a face-mask in younger children using home-made spacers should also be considered in practice.

Medication Delivery

- No advantage in terms of relief between nebule, MDI, DPI
  - Advantage of pt not needing to coordinate breath with nebule
  - Paramedic advantage with nebulizer of freeing up hands

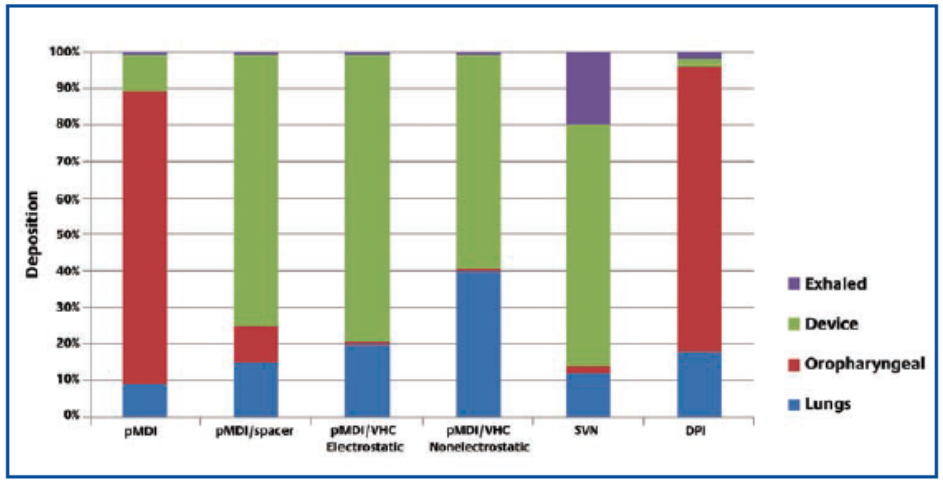


Figure 2. Drug deposition with common aerosol inhaler devices. Shown by color are the varying percentages of drug lung deposition and drug loss in the oropharynx, device, and exhaled breath.
pMDI = pressurized metered-dose inhaler; VHC = valved holding chamber; SVN = small-volume nebulizer; DPI = dry-powder inhaler
(Modified, with permission, from Reference 1 and Reference 7)

J Emerg Med. 2011 Mar;40(3):247-55. doi: 10.1016/j.jemermed.2008.06.029. Epub 2008 Dec 11.

Efficacy and cost comparisons of bronchodilatator administration between metered dose inhalers with disposable spacers and nebulizers for acute asthma treatment.

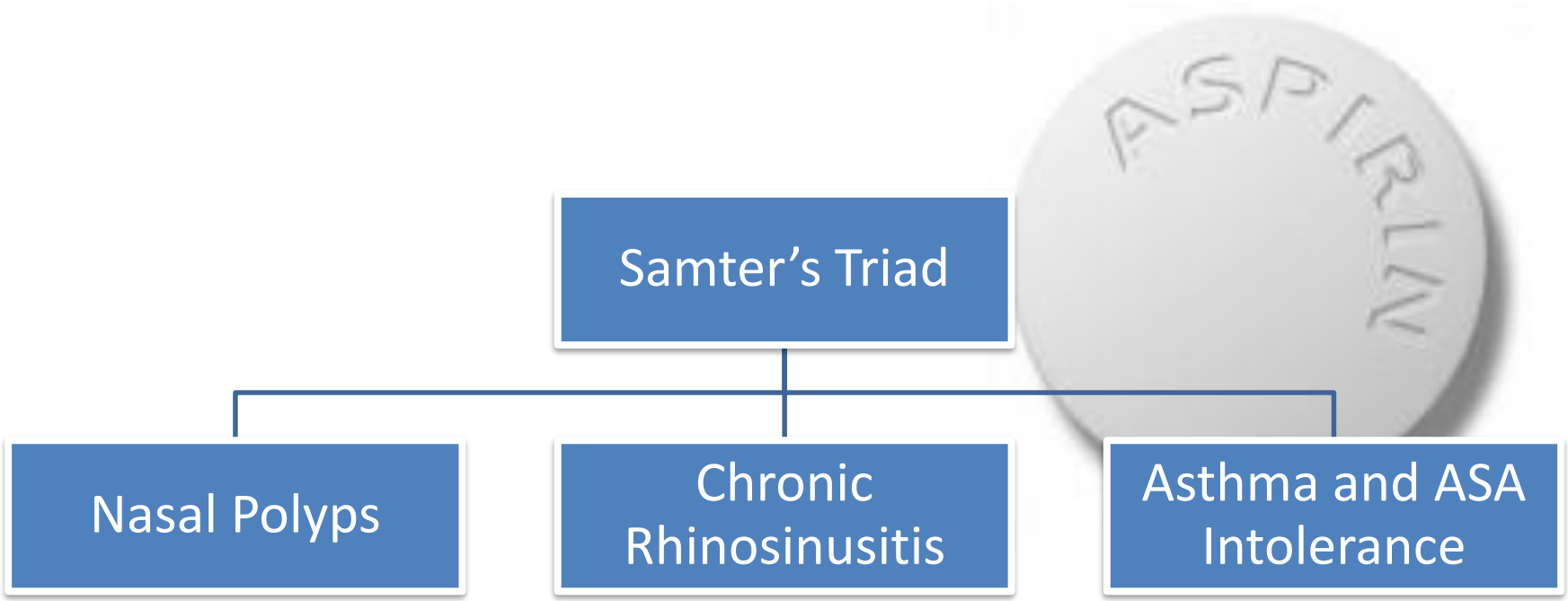
Dhuper S[1], Chandra A, Ahmed A, Bista S, Moghekar A, Verma R, Chong C, Shim C, Cohen H, Choksi S.

Authors' conclusions

There is no evidence of superiority of nebulizer to MDI/spacer beta agonist delivery for emergency management of acute asthma in the inner-city adult population. MDI/spacer may be a more economical alternative to nebulizer delivery."

Samter's Triad (Aspirin Exacerbated Respiratory Disease)

- Typically beings 20 minutes to 3 hours after ingestion
- Can include other NSAIDs



COPD vs Asthma

TABLE 4. A COMPREHENSIVE APPROACH TO THE MANAGEMENT OF COPD		
	Asthma	**COPD**
Age of onset	Usually <40 years	Usually >40 years
Smoking history	Not causal	Usually >10 pack years
Sputum production	Infrequent	Often
Allergies	Often	Infrequent
Disease course	Stable (with exacerbations)	Progressive worsening (with exacerbations)
Spirometry	Often normalizes	May improve but never normalizes
Clinical symptoms	Intermittent and variable	Persistent

COPD = chronic obstructive pulmonary disease

This information was originally published in Can Respir J *2007;14(suppl B):5B-32B.*

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Pathophysiology and Respiratory Disorders

PNEUMONIA

Pneumonia

- Affects the respiratory membrane (alveoli)
- Three portals of entry to lower respiratory tract
  - They may be inhaled as aerosolized particles
    - Impaired mucociliary clearance
  - They may enter the lung via the bloodstream
  - Aspiration of oropharyngeal contents, a common occurrence in both healthy and ill persons during sleep
    - Major mechanism by which pulmonary pathogens gain access to the normally sterile lower airways and alveoli.

Pneumonia

- Community acquired pneumonia (CAP)
  - Not hospitalized in last 14 days
- Hospital acquired pneumonia (HAP)
  - pneumonia > 48 hours after admission, which was not incubating at the time of admission
- HAP is the second most common nosocomial infection with a crude overall rate of 6.1 per 1000 discharges
- Ventilator acquired pneumonia (VAP)
  - pneumonia that arises > 48-72 hours after endotracheal intubation
    - -HAP and VAP together are the second most common cause of hospital-acquired infection and have been associated with a higher mortality than any other
- Different causative pathogens
    - Question on recent abx use
    - Less likely to be susceptible
    - Resistance

Pneumonia Etiology

- Bacterial pneumonia results in an intense inflammatory response which often leads to a productive cough
- HIV patients (CD4 <200)
  - Pneumocystis jiroveci (PJP) pneumonia
    - Fungus
    - Suspect in HIV patients with respiratory symptoms

Etiology (CAP)
Streptococcus pneumoniae
Mycoplasma pneumoniae
Haemophilus influenzae
Chlamydophila pneumoniae
Respiratory Viruses

Pneumonia Assessment

- Hallmark clinical features of CAP include:
  - Cough
  - Fever
  - Pleuritic chest pain
  - Dyspnea
  - Sputum production
    - Mucopurulent sputum production is most frequently found in association with bacterial pneumonia, while scant or watery sputum production is more suggestive of an atypical pathogen
- Not always so easy
  - May be preceded by an upper respiratory tract viral infection
- Other symptoms
  - Weakness, joint pain, rash

Assessment

- Conduct a physical examination
  - Lung sounds
    - A patient with acute pneumonia may demonstrate evidence of alveolar fluid on auscultation as crackles, may demonstrate consolidation as bronchial breath sounds, and may demonstrate pleural effusion (dullness and decreased breath sounds), or bronchial congestion (rhonchi and wheezing)
- The diagnosis of pneumonia is based on the presence of select hallmark features (previous slide) and is supported by imaging of the lung, usually by chest radiography.
- Physical examination to detect rales or bronchial breath sounds is an important component of the evaluation but is less sensitive and specific than chest radiographs

Pneumonia Management

- Goals
  - Maintain the airway
  - Support breathing
  - High-flow oxygen or assisted ventilation as indicated
- Treat symptoms and sequalae of the disease
  - Beta agonists
  - Prevent progression to sepsis
    - Monitor vital signs
    - Establish IV access
    - Avoid fluid overload (Caution in elderly and those with renal failure)
- Definitive therapy
  - Antibiotics if fungal or bacterial

Antibiotic Resistant Strains

TABLE 3
Microbiological causes of hospital-acquired pneumonia and ventilator-associated pneumonia (level A-2)

Microbiological diagnosis	Frequency of isolation (% of patients)
Gram-negative bacilli	35–80
Escherichia coli	
Klebsiella species	
Enterobacter species	
Proteus species	
Serratia marcescens	
Pseudomonas aeruginosa	
Acinetobacter species	
Stenotrophomonas maltophilia	
Gram-positive cocci	9–46
Streptococcus pneumoniae	
Streptococcus species	
Staphylococcus aureus (MSSA and MRSA)	
Polymicrobial	9–80
Anaerobes	0–54
Blood culture positive	0–40
No growth	2–54

MSSA Methicillin-susceptible S aureus, *MRSA Methicillin-resistant* S aureus.
Adapted from references 11,44-91

- "But I would like to sound a note of warning ... It is not difficult to make microbes resistant to penicillin in the laboratory by exposing them to concentrations not sufficient to kill them and the same thing has occasionally happened in the body.“

  Sir Alexander Fleming, 1945

- It is estimated that up to 50% of antimicrobial use in hospitals is unnecessary

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Pathophysiology and Respiratory Disorders

SIRS

SIRS

- Systemic Inflammatory Response Syndrome
  - HR > 90, RR > 20, Temp > 38°C or < 36°C
  - Sepsis if determination that SIRS caused by pathogen
- Severe sepsis
  - Sign of hypoperfusion
    - Confusion
- Septic shock
  - Hypotension
    - Refractory to bolus
- Recognize and treat

Management

- Patient will need antibiotics
  - Common antibiotics you will encounter
    - NS guidelines

Antibiotic	Regimen for CAP in Outpatient Adults	Cost per day
MONOTHERAPY previously healthy, low risk patient and no risk factors for drug-resistant *S pneumoniae*		
Doxycycline	200 mg for 1st dose then 100 mg BID	$1.17
Clarithromycin	500 mg BID	$3.26
DUAL THERAPY presence of comorbidities[a], antimicrobial use within the previous 3 months, or other risk factors for drug-resistant *S. pneumoniae* i.e. exposure to children in day care		
Choose one of the above drugs[b] and add a 2nd drug from below. If an antibiotic has been used in previous 3 months ensure a different class of drug is used.		
Amoxicillin	1.0 g TID	$2.06
Cefuroxime	500 mg BID	$2.90
TREATMENT FAILURE (worsening after 72 hours or no response after completion of therapy) and if there is no fluoroquinolone use in previous 3 months		
Levofloxacin	750 mg OD	$6.55
Moxifloxacin	400 mg OD	$6.45
Duration of therapy is usually 7 to 10 days for all regimens except levofloxacin (5 days).		

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Pathophysiology and Respiratory Disorders

BRONCHITIS

Acute Bronchitis

- Acute inflammation of the trachea and bronchi
- Generally Viral and self-limiting
  - Influenza, parainfluenza virus, respiratory syncytial virus, coronavirus, adenovirus, rhinovirus, and human metapneumovirus,
  - Although majority of patients are given abx
- Pneumonia, asthma, other causes ruled out
- Cough 1-3 weeks, can be productive
  - Often afebrile
- Malaise
- Generally do not respond to beta agonists (may reduce cough), although you will see many patients with this condition given puffers

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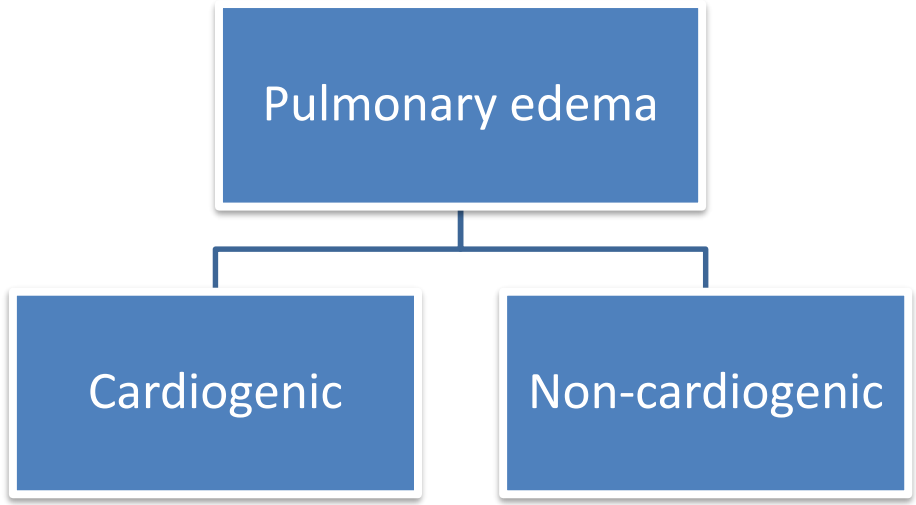
Pathophysiology and Respiratory Disorders

PULMONARY EDEMA

Pulmonary Edema

- Manifestation of another condition
  - Fluid ends up in pulmonary extravascular compartment
- Fluid accumulates in the interstitial space because of imbalance between hydrostatic and osmotic forces
  - Eventually can end up in alveoli
- Acute or Chronic
  - Acute HF vs CHF



Edema Etiology

Cardiogenic

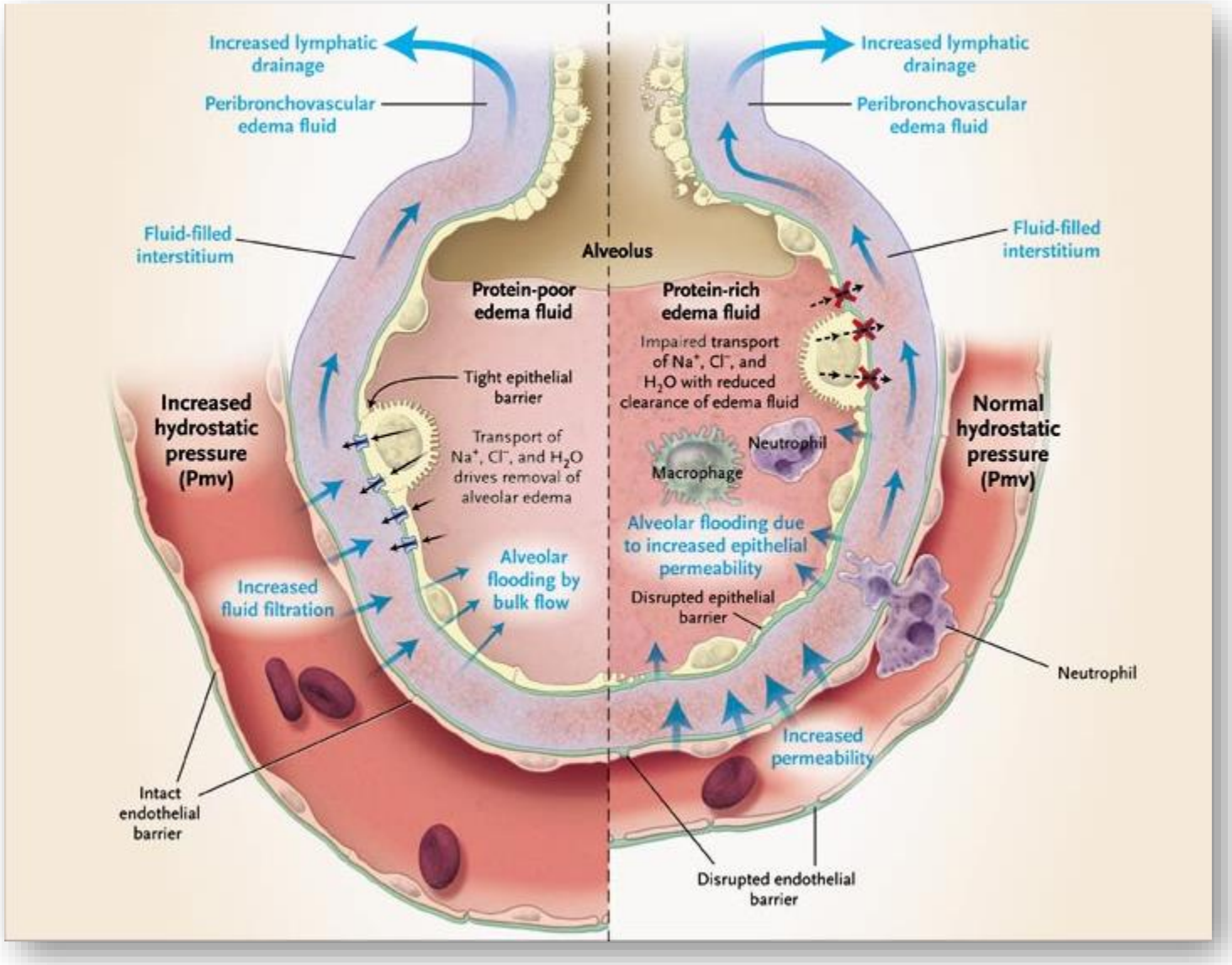
- High pulmonary capillary pressure due to left sided heart failure
- Early increases in pulmonary venous pressure may be asymptomatic
  - Mild cough
  - Non-productive
- Consider patient not tolerating chronic a-fib
  - May need rhythm as opposed to standard rate adjustment
- Consider MI

Non-Cardiogenic

- Damage occurs to the pulmonary capillary lining
  - Subsequent leakage of proteins and other large molecules.
  - Fluid follows the protein as oncotic forces are shifted from the vessel to the surrounding lung tissue.
- Surfactant dysfunction
- Vasoconstriction and intrapulmonary shunt

Edema Etiology

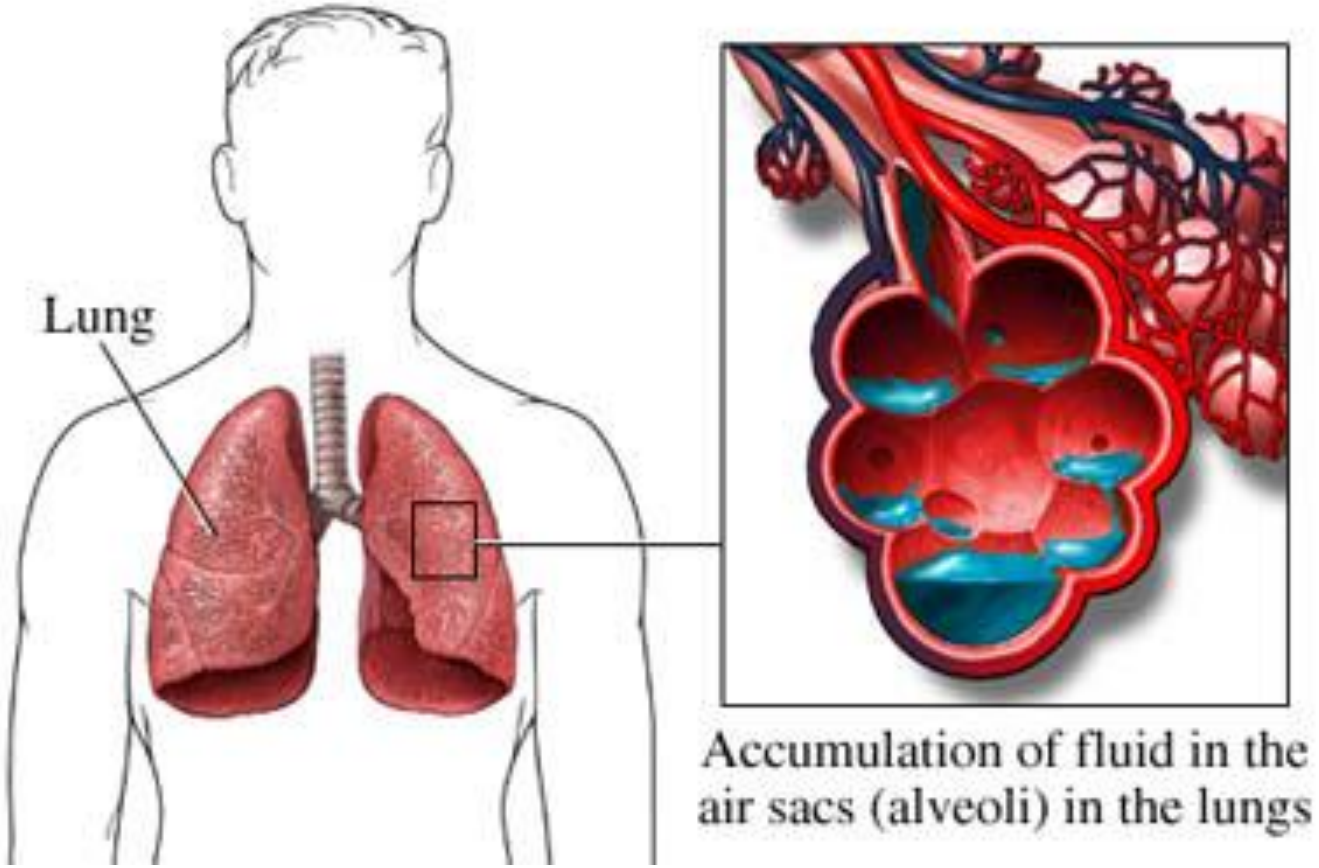
Cardiogenic



Non-Cardiogenic

Pulmonary Edema

- S/S
  - Nocturnal dyspnea
    - "pillow count"
  - Peripheral edema
  - Tachypnea
  - Tachycardia
  - Crackles on auscultation
  - Possible ectopy on 4 lead



Pulmonary Edema Stages

- Stage I: Interstitial pulmonary edema is present. Patients often become tachypneic as pulmonary compliance begins to decrease. “Cuffing” seen on X-ray.
- Stage II: Fluid fills the interstitium and begins to fill the alveoli. Near-normal gas exchange may be preserved. Pt may be asymptomatic.

Pulmonary Edema Stages

- Stage III: Many alveoli become completely flooded and no longer contain atmospheric gas.
  - Flooding is most prominent in dependent areas of the lungs.
  - Blood flow through the capillaries of flooded alveoli results in a large increase in intrapulmonary shunting. Hypoxemia and hypocapnia (the latter due to dyspnea and hyperventilation) are characteristic.
- Stage IV: Marked alveolar flooding spills into the airways as froth. Gas exchange is compromised due to both shunting and airway obstruction, leading to progressive hypercapnia and severe hypoxemia.

Pulmonary Edema Management

Treatments

- Oxygen
- Nitrates
- Mechanical ventilation
  - CPAP
  - BVM

Additional Treatments (ACP)

- Morphine
- Diuretics
- Inotropes
- Mechanical ventilation
  - Intubation
    - PEEP

Nitroglycerine

Classification

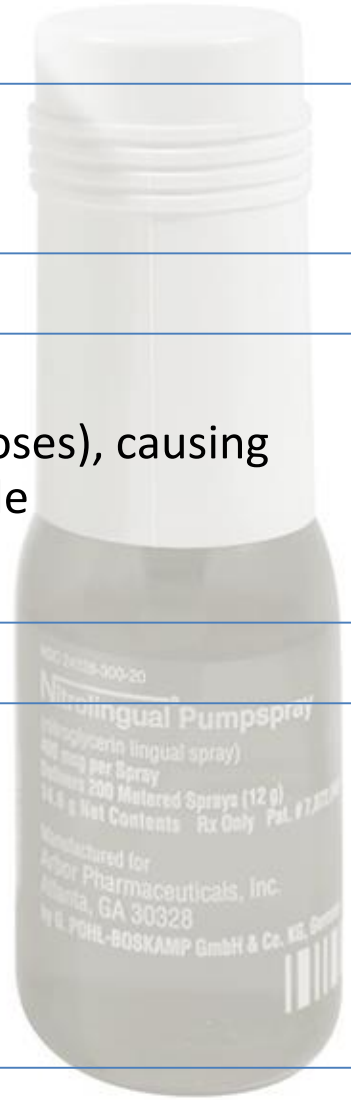
- Anti-angina, vascular smooth muscle relaxer, vasodilator

Mechanism of Action

- Relaxes vascular smooth muscle, there by dilating the veins and arterioles (at higher doses), causing blood pooling, which reduces the preload thus decreasing workload of the heart muscle
- Reduces left ventricular systolic wall tension, which decreases afterload

Indications

- Possible ischemia due to ACS:
  - Unstable angina
  - AMI
- Pulmonary edema/CHF



Nitroglycerine

Contraindications

- Hypotension (< 90 mmHg)
- Severe bradycardia/tachycardia (< 50 or > 150 bpm)
- Increase ICP or intracranial hemorrhage
- Patients taking erectile dysfunction medications
- Viagra within 24 hours
  - Cialis, Levitin, Staxyn within 36 hours

Dosage

- 0.4 mg SL q 3 - 5 min

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Pathophysiology and Respiratory Disorders

ARDS

Acute Respiratory Distress Syndrome (ARDS)

- According to the consensus Berlin Definition
  - ARDS is characterized by acute onset (< 7 days) of bilateral radiographic pulmonary infiltrates and respiratory failure not fully explained by heart failure or volume overload
- America European Consensus Conference
  - Acute onset, bilateral pulmonary infiltrates on chest radiograph consistent with pulmonary edema, poor systemic oxygenation, and the absence of evidence of left atrial hypertension
- “Final common pathway” from previous insult
- Hypoxia resistant to oxygen therapy
- Most patients require ventilator support
- Mortality up to 40%

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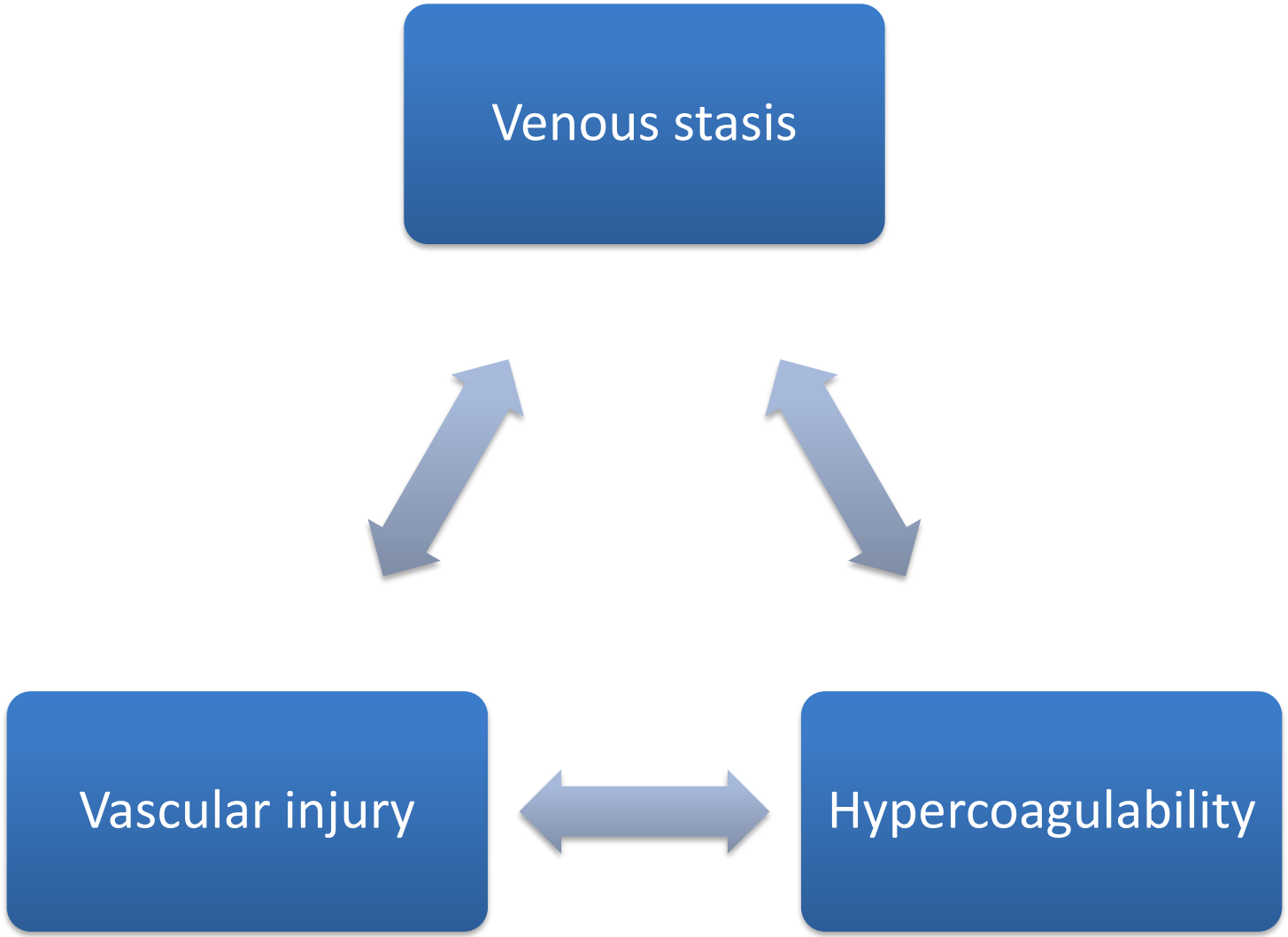
Pathophysiology and Respiratory Disorders

PULMONARY EMBOLISM

PE & DVT

- Pulmonary Embolism and Deep Vein Thrombosis
  - Blood clots that develop in the leg and can travel to the lung
- Most common cause of preventable death in hospitalized patients
  - However many stable DVT patients can be treated as outpatient
- Mortality at 3 months as high as 15%
  - African Canadians at higher risk
- Can be a cause of sudden cardiac arrest
- Underdiagnosed condition
  - Anticoagulation prophylaxis is underused
  - Many diagnosis occur postmortem

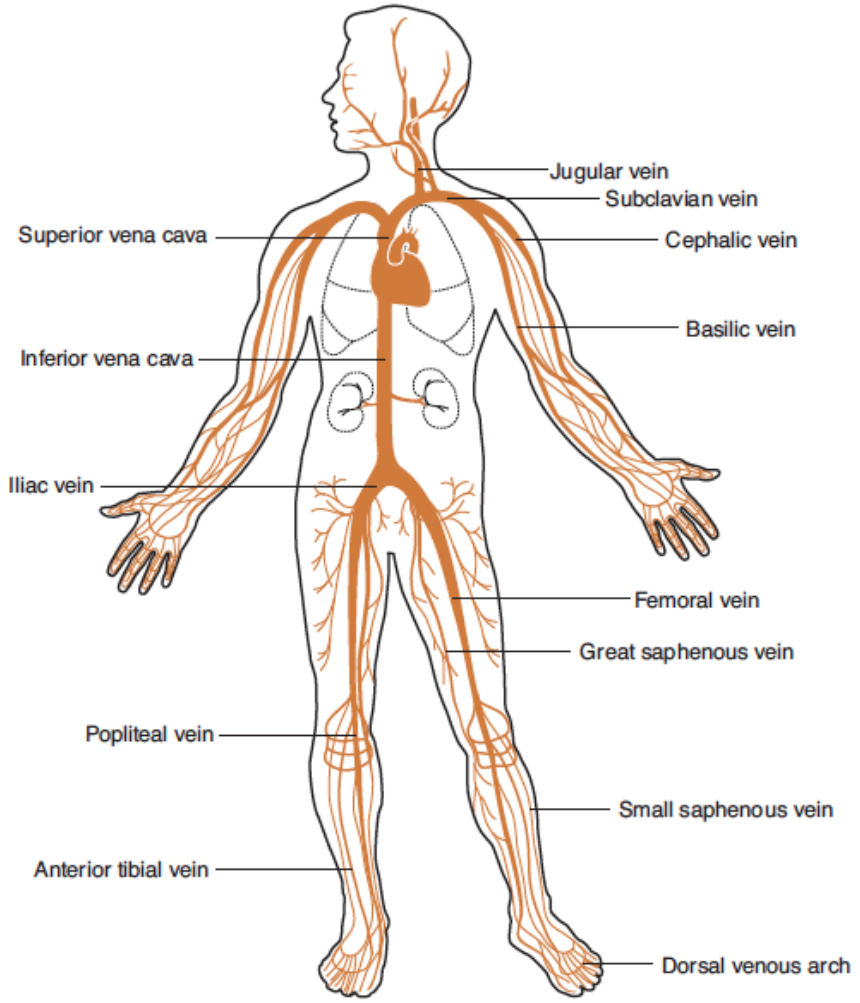
Virchow's Triad



DVT Risks

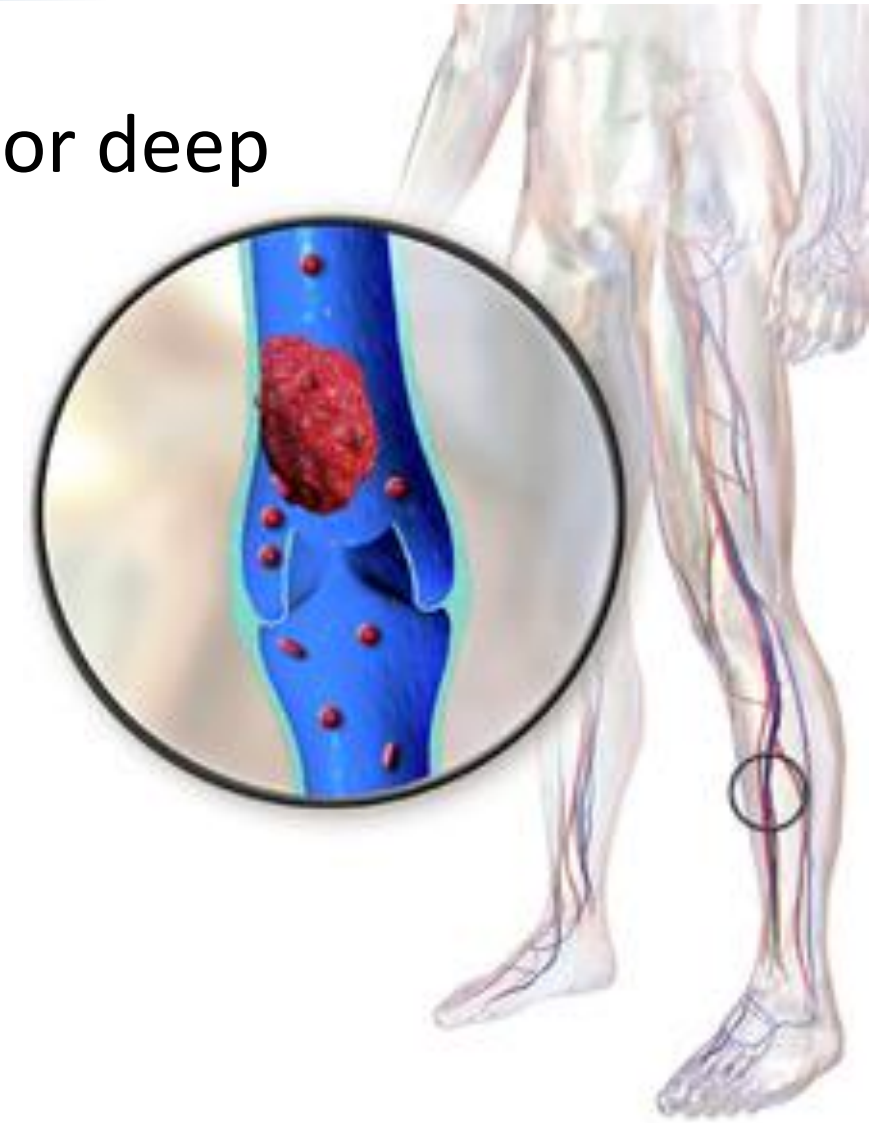
Venostasis	Vascular Injury	Hypercoagulability
Surgery	Vascular catheters	Protein C deficiency
Paralysis	Trauma	Protein S deficiency
Cast	Artificial valves	Pregnancy
Immobility		Oral contraceptives
Varicose veins		Estrogens
Obesity		Malignancy

Venous Circulation

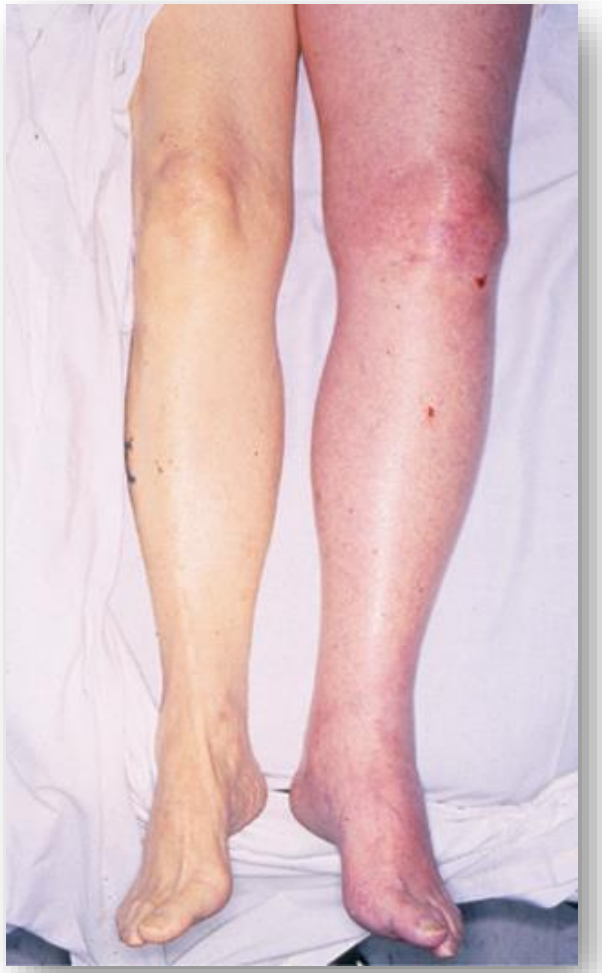


DVT

- Development of blood clot in 1 of the major deep veins in the leg, thigh, pelvis or abdomen
- Definitions
  - Thrombus
  - Embolus
  - Thrombosis
    - More to follow in cardiology



Clinical Suspicion



- Upper and lower leg symptoms not to be taken lightly!
- Potential symptoms
  - Swelling, tenderness, erythema, asymmetric swelling
  - 2005 meta-analysis of cohort studies showed only positive predictor of DVT was calf diameter
- Wells' Score
- Fever and chills suggest cellulitis

Risk Assessment

Clinical Feature	Points*
Active cancer (treatment within 6 mo, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for >3 d because of surgery (within 12 wk)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of >3 cm (below tibial tuberosity)	1
Unilateral pitting edema	1
Collateral superficial veins	1
Alternative diagnosis as likely as or more likely than deep venous thrombosis	−2
Prior history of DVT or PE†	1

DVT Differential

- Muscle strain, tear, or twisting injury to the leg
- Leg swelling in a paralyzed limb
- Lymphangitis or lymph obstruction
- Drug induced edema
- Popliteal (Baker's) cyst
- Cellulitis
- Knee abnormality
- Unknown
  - Many cases are not DVT

Diagnosis of DVT

- D-dimer and clinical risk scores
  - D-dimer evaluates fibrin degradation products
  - Useful in excluding people with low clinical suspicion
- Compression Ultrasound
  - When pressure is applied to the proximal veins with an ultrasound probe, the veins should fully compress
- Venography
  - Gold standard but difficult to obtain
  - Only used when noninvasive tests unreliable or not available
  - Contrast can cause clots

Treatment

- Patients are treated with blood thinners
  - Warfarin and low molecular weight heparins (enoxaparin, dalteparin)
- First DVT results in a minimum of 3 months of treatment
- Caution – bleeding risk

Blood Thinners

- Warfarin
  - PO medication
  - Inhibits vitamin K dependent clotting factors
    - 1, 2, 9, 7 of 13 clotting factors
  - Very narrow therapeutic index
  - INR determines therapy
  - Older but still relevant due to no CI in pts with renal compromise
  - Vitamin K is the antidote
- Low Molecular Weight Heparins (LMWH)
  - injectable
  - Inactivate factor 10
  - CI in patients with creatinine clearance < 30ml/min
  - Weak antidote (protamine)

Pulmonary Embolism

- Pathophysiology
  - Obstruction of a pulmonary artery
    - Emboli may be of air, thrombus, fat, or amniotic fluid
    - Foreign bodies may also cause an embolus
  - DVT most common cause
- Diagnosis
  - Massive PE
    - Beside echocardiography
  - Stable patients
    - D-Dimer
    - Spiral CT
    - V/Q scan
    - Pulmonary arteriogram

PE assessment

- Clinical suspicion and detection are critical
  - You do not have the tools to diagnose but your thorough knowledge and assessment skills should lead you to strong hypothesis
- Can range from nothing to sudden cardiac arrest
  - Dyspnea without adventitious lung sounds, abnormal ECG
  - Pleuritic chest pain
- Definitive therapy is use of anticoagulants in DVT

Assessment

- Focused history and physical exam
- SAMPLE and OPQRST History
  - Presence of risk factors
  - Sudden onset of unexplained dyspnea
  - Cough
  - Chest pain
  - Hemoptysis (coughing blood up) is a very late and rare sign
- Circulatory collapse: hypotension, syncope, coma
- Physical Exam
  - Signs of heart failure, including JVD and hypotension
  - Warm, swollen extremities (DVT)

Management

- Maintain the airway
- Support breathing
  - High-flow oxygen or assist ventilations as indicated
  - Intubation may be indicated
- Establish IV access
- Monitor vital signs closely
- Transport to appropriate facility
  - Use of thrombolytics in PE is controversial in hospital

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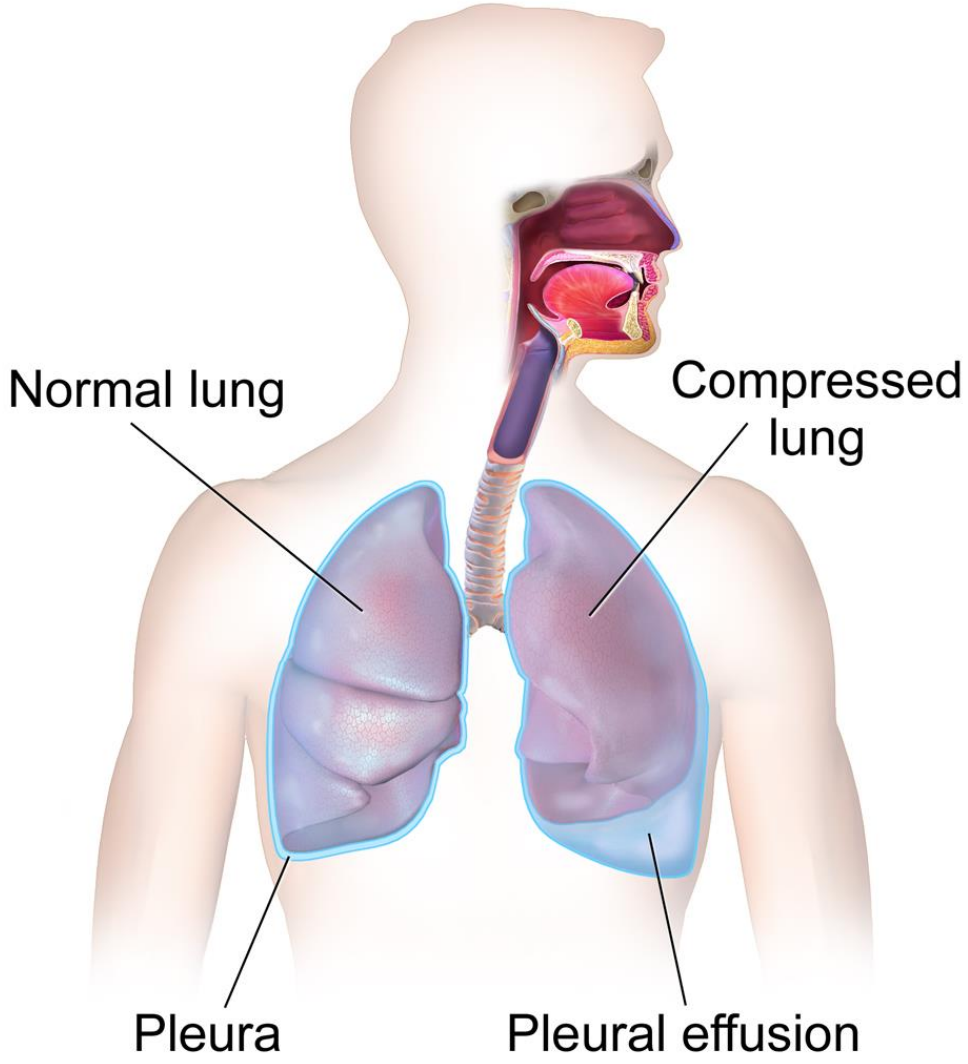
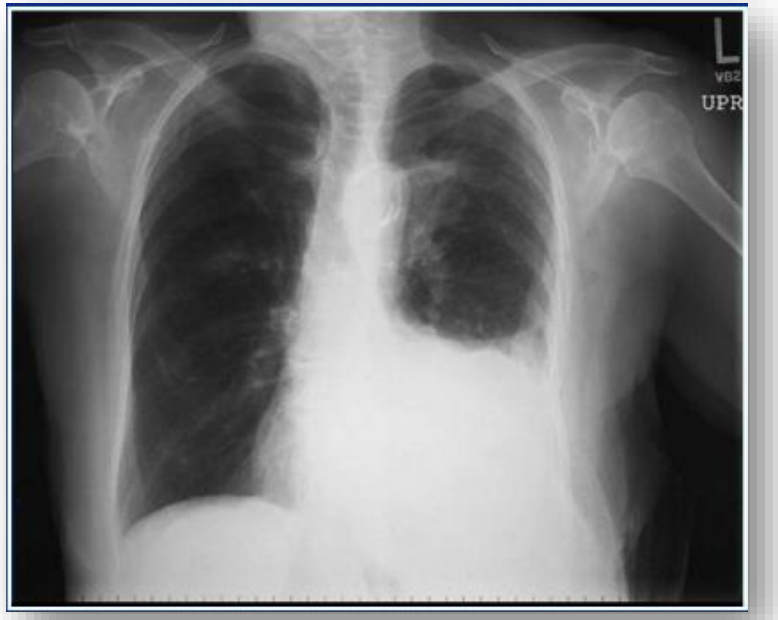
Pathophysiology and Respiratory Disorders

PLEURAL EFFUSION

Pleural Effusion



- Results from fluid accumulating in the potential space between the visceral and parietal pleura (influx of fluid exceeds the efflux of fluid)

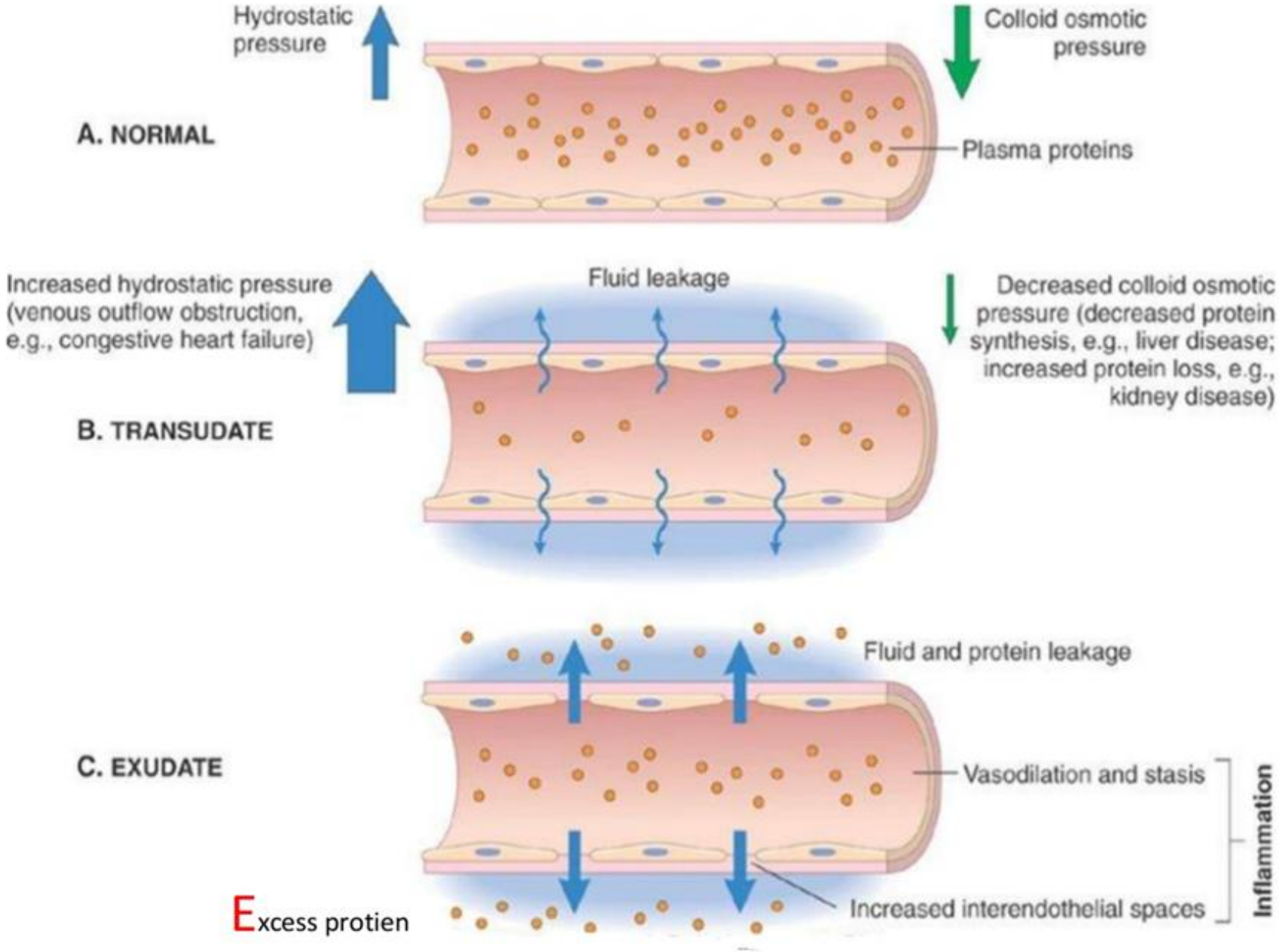


Pathophysiology

- Altered pleural membrane permeability
- Decreased intravascular oncotic pressure
- Increased capillary hydrostatic pressure
- Lymphatic obstruction
- Abnormal sites of entry

Causes

- CHF
- Bacterial infection (pneumonia)
- Pulmonary embolism
- Malignancy
- TB


Hydrostatic pressure
Colloid osmotic pressure
A. NORMAL
Plasma proteins
Increased hydrostatic pressure (venous outflow obstruction, e.g., congestive heart failure)
Fluid leakage
Decreased colloid osmotic pressure (decreased protein synthesis, e.g., liver disease; increased protein loss, e.g., kidney disease)
B. TRANSUDATE
Fluid and protein leakage
C. EXUDATE
Vasodilation and stasis
Inflammation
Increased interendothelial spaces
Excess protien

Assessment and Treatment

Assessment

- Often asymptomatic initially
- Dyspnea
  - Deep inspiration may cause cough and increase in pain
  - Degree of SOB does not always correlate to size of effusion
- Chest pain
  - Due to inflammation
  - Most often shooting or stabbing
- Fever may be present
- Hemoptysis

Treatment

- Treat the hypoxia
- Most treatments require hospital
  - Thoracentesis
  - Chest tube insertion
  - Treatment of underlying conditions

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Pathophysiology and Respiratory Disorders

SARS

Severe Acute Respiratory Syndrome (SARS)

- Caused by a novel coronavirus and was first detected in 2003
  - Spread by droplets, however there are cases where respiratory transmission was likely
  - The virus is shed in stools but the role of fecal–oral transmission is unknown.
  - Take ABSOLUTE full precautions for airborne transmission
  - Inform receiving facility
- Symptoms generally begin a week after contact

Severe Acute Respiratory Syndrome (SARS)

Signs and Symptoms

- Fever
- Chills
- Rigors
- Malaise
- Nausea
- Shortness of breath
- Respiratory crackles

Important Points

- Pneumonia begins in 8-10 days
- 20% of patients develop ARDS
  - Anticipate mechanical ventilation
- Many treatments attempted
  - Steroids, antivirals, interferon
  - Efficacy questionable

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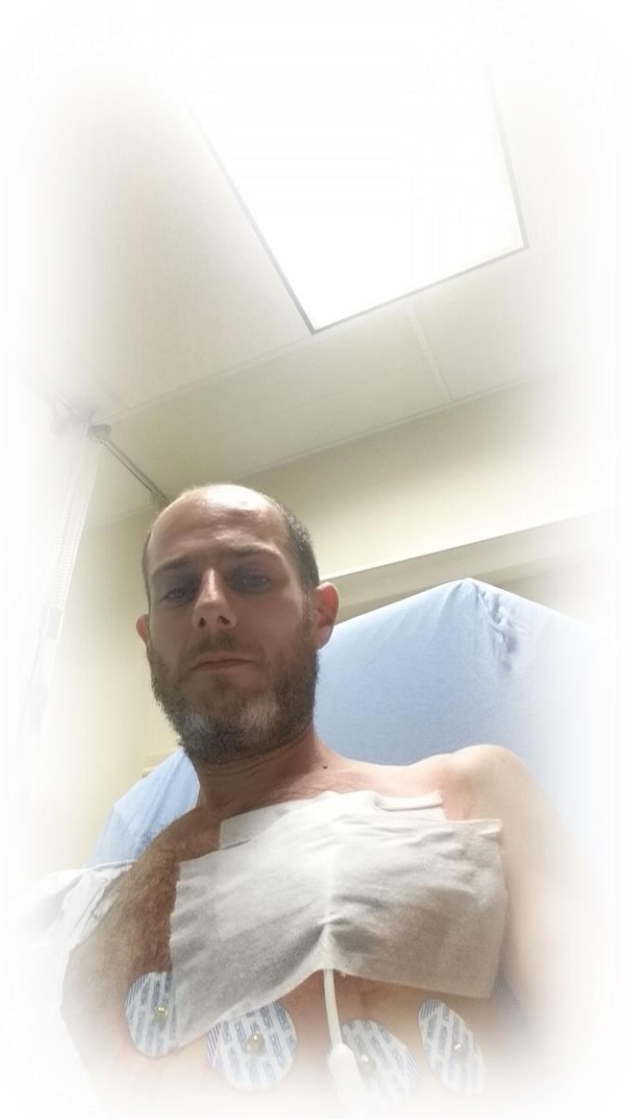
Pathophysiology and Respiratory Disorders

PNEUMOTHORAX

Spontaneous Pneumothorax

- Occurs in absence of trauma
- Risk factors
  - Rare but high recurrence rate
  - More males than females (5:1)
  - Tall, thin stature
  - Between 20 and 40 years
  - COPD (ruptured bleb)



Spontaneous Pneumothorax

- Pathophysiology
  - Disease of ventilation
  - Pneumothorax occupying 15 - 20% of chest cavity generally well tolerated
- Assessment
  - Presence of risk factors
  - Rapid onset of symptoms
  - Sharp, pleuritic chest or shoulder pain
  - Often precipitated by coughing or lifting
  - JVD

Spontaneous Pneumothorax

Management

- Maintain the airway
- Support breathing
- Monitor for tension pneumothorax
  - Tracheal deviation away from the affected side
- Definitive therapy
  - Needle decompression (ACP)
  - Chest tube (CCP or hospital)

Pathophysiology and Respiratory Disorders

OTHER CAUSES

CNS Dysfunction

Pathophysiology

- Traumatic/atraumatic brain injury
- Tumours
- Drugs
- Degenerative motor neuron disease
  - Amyotrophic lateral sclerosis (ALS)
    - Lou Gehrig's

Assessment

- Evaluate potentially treatable causes
  - Narcotic drug overdose
  - CNS trauma.
- Carefully evaluate breathing pattern.

Dysfunction of the Spinal Cord, Nerves, or Respiratory Muscles

- Pathophysiology
  - PNS problems affecting respiratory function
    - Trauma
    - Polio
    - Myasthenia gravis
    - Guillain Barre Syndrome
    - Viral infections
    - Tumours
    - ALS

Dysfunction of the Spinal Cord, Nerves, or Respiratory Muscles

- Assessment
  - Rule out traumatic injury, and assess for numbness, pain, or signs of PNS dysfunction.
- Management
  - Assess ABC
  - Determine pt and families wishes
  - Manage AW as appropriate, including suction

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Pathophysiology and Respiratory Disorders

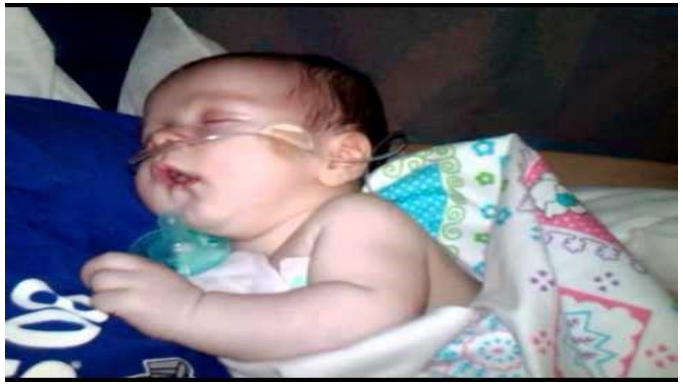
PEDIATRIC CONSIDERATIONS

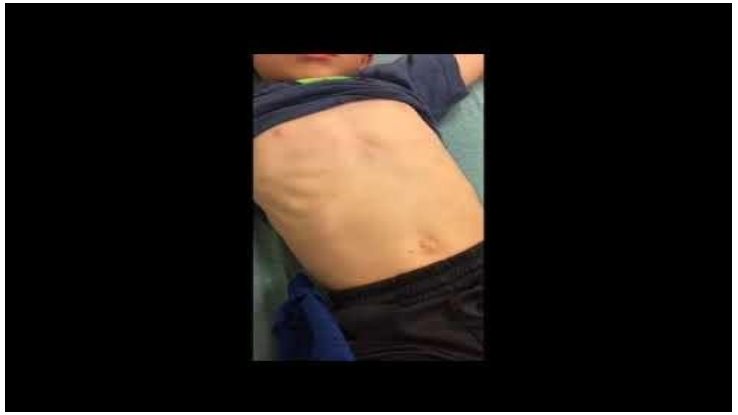
Respiratory Emergencies

- Stages of Respiratory Compromise
  - Respiratory distress
  - Respiratory failure
  - Respiratory arrest

Respiratory Distress

- Normal mental status
  - Deteriorating to irritability or anxiety
- Cyanosis
  - that improves with supplemental oxygen
- Good muscle tone
- Weak cry
- Grunting
- Tachypnea
- Tachycardia
- Retractions
- Head bobbing
- Nasal flaring (in infants)





Respiratory Failure

Definition

- Develops when the rate of gas exchange between the atmosphere and the blood is unable to match the body's metabolic demands

Classifications

- Type 1 (Hypoxemic)
  - PO_2 < 50 mmHg on room air. Usually seen in patients with acute pulmonary edema or acute lung injury.
- Type 2 (Hypercapnic/ Ventilatory)
  - PCO_2 > 50 mmHg (if not a chronic CO_2 retainer). This is usually seen in patients with an increased work of breathing due to airflow obstruction or decreased respiratory system compliance, with decreased respiratory muscle power due to neuromuscular disease, or with central respiratory failure and decreased respiratory drive.
- Type 3 (Peri-operative)
  - Atelectasis being the most common cause post anesthesia
- Type 4 (Shock)
  - Secondary to cardiovascular instability.

Respiratory Failure

- Presentation
  - Tachypnea later deteriorating to bradypnea
  - Retractions later deteriorating to agonal respirations
  - Poor muscle tone
  - Tachycardia later deteriorating to bradycardia
  - Central cyanosis

Respiratory Arrest

- Unresponsiveness deteriorating to coma
- Bradypnea deteriorating to apnea
- Absent chest wall motion
- Bradycardia deteriorating to asystole
- Profound cyanosis

Management

- Establish an airway
- Oxygen therapy
- Ventilation with BVM
- Airway adjunct
- Consider gastric decompression
- Consider needle decompression

Specific Respiratory Emergencies

- Upper airway distress
  - Croup
  - Epiglottitis
  - Foreign body aspiration
- Lower airway distress
  - Asthma
  - Bronchiolitis
  - Pneumonia
  - Foreign body lower airway obstruction

Croup

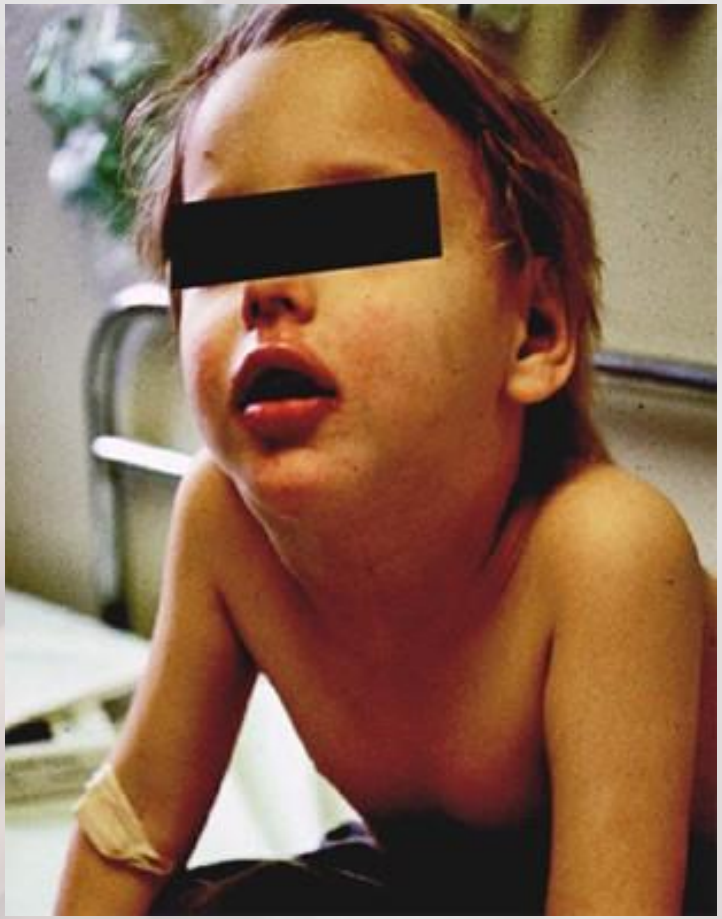
Signs and Symptoms

- Slow onset (12 – 72 hrs)
- Low grade fever (38°C - 39°C)
- Inflammation in the nasal cavity and around the eyes
- Narrowing of the larynx
- Stridor (mild)
- Tachypnea
- Retractions
- Barking cough
- Hoarse voice

Epiglottitis

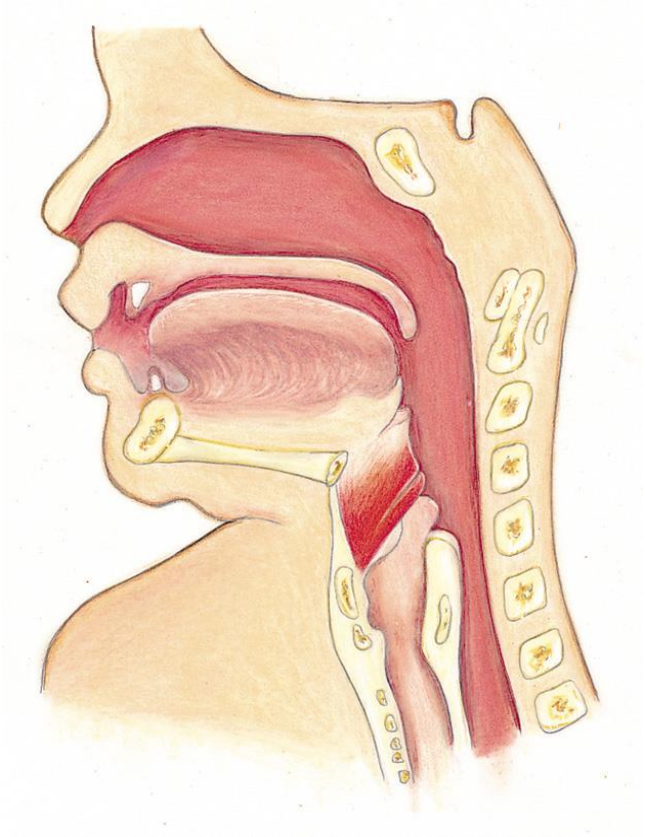
Signs and Symptoms

- Rapid onset
- Fever (>38.5°C)
- Stridor (moderate to severe)
- Tachypnea
- Retractions
- No Barking cough
- **Drooling** (difficulty swallowing)
- Difficulty speaking

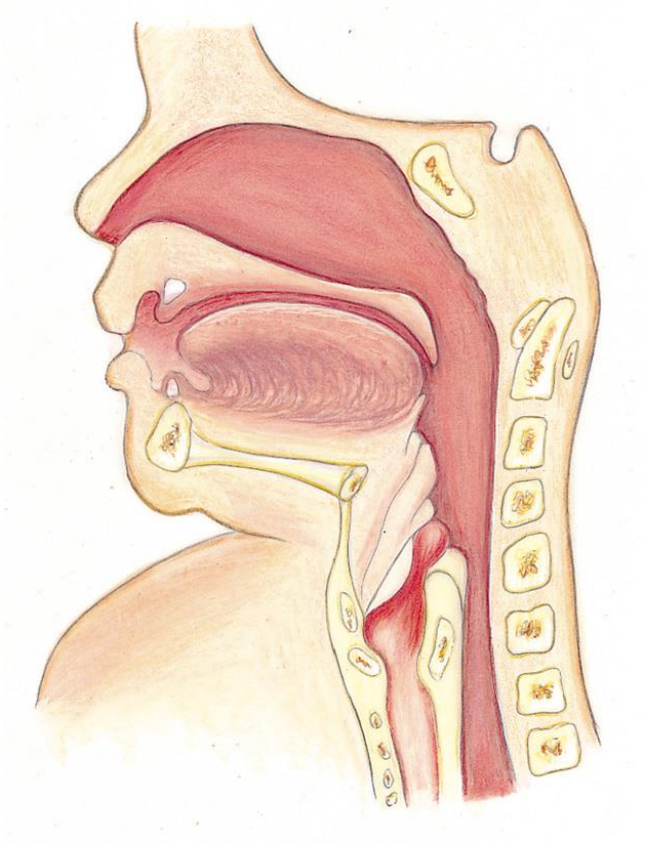


Upper airway distress

- Epiglottitis



- Croup



- Positioning of the child with epiglottitis, often there will be excessive drooling

A child with epiglottitis should be administered humidified oxygen and transported in a comfortable position