

Pneumonia Is an acute inflammation of the lung parenchyma (essential and distinctive tissue of an organ). Most common cause of death from infectious diseases in US. 7th leading cause of death in the U.S. - Accounts for 66,000 deaths/yr

Risk Factors Associated W/ Pneumonia (CAP & HAP)

Classifications

Community Acquired (CAP)

Nosocomial (HAP)

Fungal pneumonia

Aspiration pneumonia

Opportunistic pneumonia

Immune comp.

Community-Acquired pneumonia

CAP

- Elderly
- LTC
- Lack of pneumococcal vaccination
- Co-existing condition (DM, COPD, alcoholism, HF)
- Exposure to virus or influenza
- Tobacco and or alcohol exposure
- Air pollution
- Bed rest with immobility
- Overuse of antibiotics

Nosocomial pneumonia

HAP

- Onset after 48 hrs w/ no S&S at admission
- Most lethal nosocomial infection
- Most are colonized by multiple MO
- Elderly
- Chronic lung disease
- Altered LOC
- Aspiration
- Endotracheal/trach, nasogastric tube (VAP)
- Poor nutrition
- Immunocompromised (AIDS, steroids, chemo, radiation, sedation - hypoventilation, broad spectrum Abs)
- RX that ↑ gastric ph or alkaline tube feedings
- Thoracoabdominal procedures

ventilation acquired

Pathophysiology (from PPT)

- Congestion: after the organism reaches the alveoli there is an outpouring of fluid into the alveoli. The organisms multiply in the serous fluid, and the infection is spread.
- The pneumococci damages the host by their overwhelming growth and interference with lung function.
- Blood flow decreases, and leukocytes and fibrin consolidate in the affected part of the lung.
- The lung becomes "stiff" and there is a decrease in compliance and a decline of vital capacity.
- Surfactant production decreases further restricting compliance and may lead to atelectasis.
- There may be lung "shunting"-venous blood coming into the lungs passing through underventilated areas. This can cause hypoxia.

Pathophysiology (from smelzer)

Normally, the upper airway prevents potentially infectious particles from reaching the sterile lower respiratory tract.

Pneumonia arises from normal flora (immunocompromised pts), from aspiration, bloodborne organisms

Pts often have acute or chronic underlying disease that impairs host defenses.

Pneumonia affects both ventilation and diffusion.

An inflammatory reaction can occur in the alveoli, producing an exudate that interferes with diffusion of O₂ & CO₂.

WBC migrate into the alveoli and fill the normally air-filled spaces. Poor ventilation results from secretions & mucosal edema that cause partial occlusion of the bronchi or alveoli, with a resultant decrease in alveolar oxygen tension.

Bronchospasm may also occur in patients with reactive airway disease.

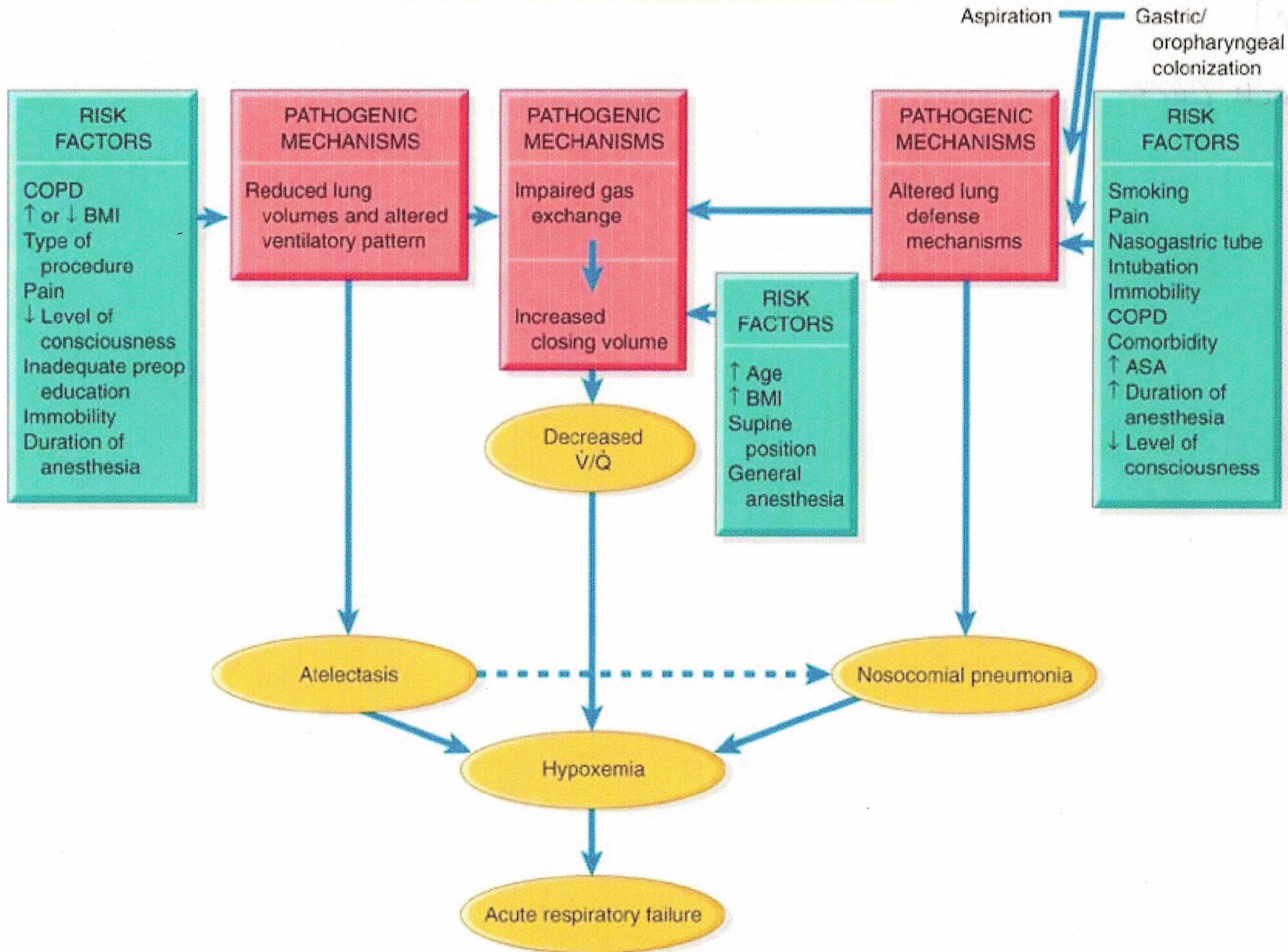
Because of hypoventilation, a ventilation-perfusion mismatch occurs and venous blood entering pulmonary circulation passes through the underventilated area and travels to the left side of the heart poorly oxygenated. The mixing of oxygenated and unoxygenated or poorly oxygenated blood eventually results in arterial hypoxemia.

Lobar pneumonia: substantial portion of one or more lobes is involved Bronchopneumonia is used to describe pneumonia that is distributed in a patchy fashion, having originated in one or more localized areas within the bronchi and extending to the adjacent surrounding lung parenchyma. Bronchopneumonia is more common than lobar pneumonia

Resolution

Healing may occur if there are no complications, the exudate becomes lysed and is processed by the macrophages. Normal lung tissue is restored.

Relationship of risk factors, pathogenic mechanisms, and consequences of acute atelectasis in the postoperative patient. ASA, acetylsalicylic acid; COPD, chronic obstructive pulmonary disease; BMI, body mass index; (V./Q.), ventilation-perfusion ratio.



Prevention
 Pneumococcal vaccine
 Influenza vaccine
 Avoid sick people
 Wash hands
 TCBD
 Incentive spirometer
 Ck gag reflex post op/proc
 Watch for pts that are immobile, dysphagic, tubed, sedated

Risk
 CAP, HAP, VAP
 Comorbid: COPD, HF, Alcoholism, DM
 Elderly, LTC, Immunocompromised, etc

MOs
 S. pneumonia
 H. influenza
 Mycoplasm pneumonia
 PCP (fungal & protozoan)
 Legionella
 Chlamydia
 Viral
 Fungal

Treatment
 O2 – NC, ventura mask, non-rebreather
 Pulsox at least QID + PRN
 Nebs w/ albuterol & atrovent, Abs
 3L/d fluid at least (unless fluid restrict.
 1500 kcal/d at least – sm., freq. meals
 Bedrest w/ BSC, HOB at least 30°
 Pulmonary Toilet
 Suction PRN
 CBC – QD
 CXR – Q2-3d

Pneumonia
 Thickened, irritated aveolar walls w/ accum of mucous, inflammatory cells, serous fluid, causing : consolidation, impaired gas exchange, shunting.
 Lobar = 1 lobe w/ consolidation, lobular = (bronchopneumonia) diffuse, patchy consolidation

Physical
 Consolidation-dullness on percussion
 Up Fremitus
 Crackles
 Diminished
 Rhonci

S&S
 Sudden fever (or gradual)
 Fatigue
 Myalgia
 Dyspnea
 Chills
 Prod. or dry cough
 Maybe up HR, RR, BP

Complications
 Pleurisy – give NSAIDS
 Pleural effusion – thorocentesis
 Atelectasis
 Empyema – chest tube, sx drain
 Pericarditis
 Meningitis
 Endocarditis
 Resp failure/shock

Meds
 Abs are mainstay
 Steroids: PO prednisone, IV- Methylprednisone
 Antipyretics/analgesics: ASA, Motrin, codeine, hydrocodene
 Expectorant - mucinex

Diagnostics
 CXR – consolidation, infiltrate
 Sputum gram stain
 ABGs – r/o hypoxemia
 CBC – up leuks, blood culture (r/o sepsis)
 CT – for coexisting infxn, resistant MOs, r/o CA

Acute Treatment
 Pulseox maintained 93-94%
 VS q2h – monitoring for sepsis, shock
 I/O q2h
 Observe for resp. fatigue/failure i.e. Δ LOC, anxiety, restlessness, anxiety