

Health Care Associated Pneumonia

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Objectives

- Define the terms, pneumonia, community acquired pneumonia, health care associated pneumonia (HCAP) and ventilator associated pneumonia (VAP).
- Describe the pathogenesis of the health care associated pneumonia (hospital associated pneumonia) and VAP.
- Classify HCAP according to the time of onset .

Objectives

- Name the different causative bacterial agents .
- Classify and describe types of VAP.
- Recognize the ways by which VAP is prevented.
- Describe the different chemotherapeutic anti microbial agents used for the treatment of health care associated pneumonia.
- Evaluate response to treatment and recognize reasons for failure of treatment.

Health Care Associated Pneumonia

- Definition of Pneumonia:
Infection of the pulmonary Parenchyma



PNEUMONIA can be :

- **A-Community acquired Pneumonia**
acquired in the community, by community acquired organism, eg. *Streptococcus pneumoniae* usually susceptible to antibiotic.
- **B-Health care associated pneumonia**
acquired **48-72 hours** after admission to health care institutions eg. pneumonia caused by organisms in hospital which are usually resistant to antibiotics-eg. *Pseudomonas aeruginosa*

Definition

- **Nosocomial pneumonia:** is defined as hospital associated pneumonia (HAP) or health care associated pneumonia (HCAP).
- Occurring at least **48 hours** after admission and not incubating at the time of hospitalization.

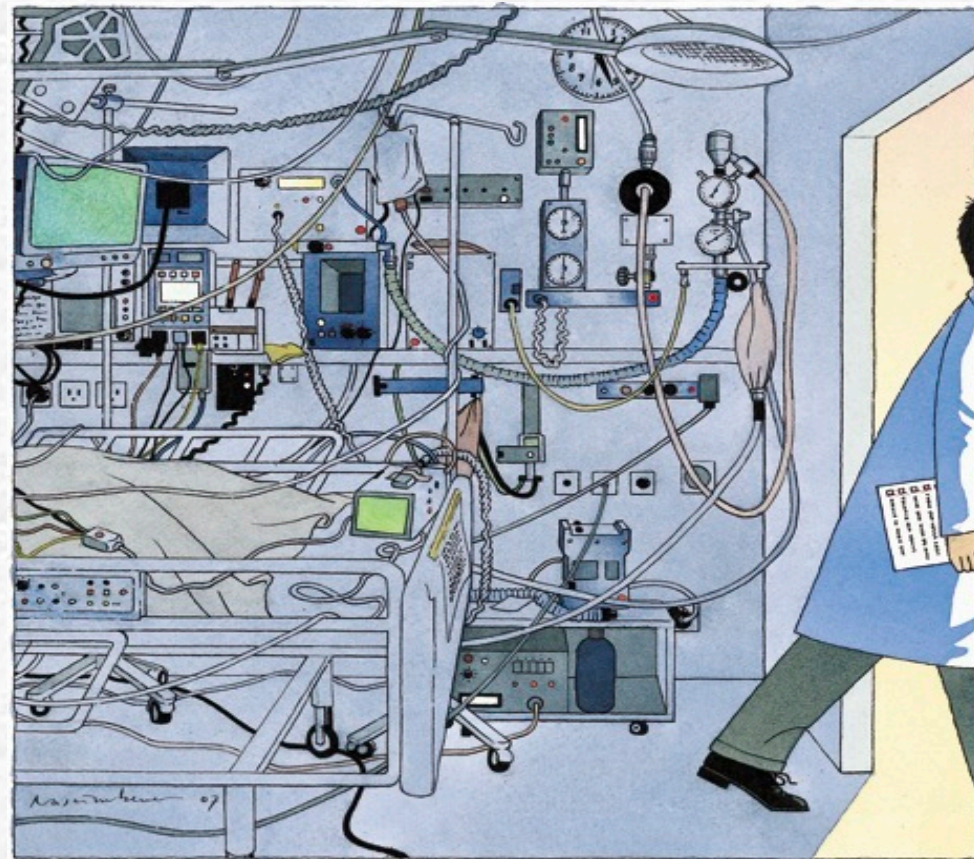
Health care associated Pneumonia

- **A-** Hospital Acquired Pneumonia(**HAP**)
- **B-** Ventilator Associated Pneumonia (**VAP**) in patients with assisted respiration for a period of 48 hours.

Introduction

- Nosocomial pneumonia is the **2nd most common** hospital-acquired infections after urinary tract infection. Accounting for 31 % of all nosocomial infections
- Nosocomial pneumonia is the **leading cause of death** from hospital-acquired infections.
- The incidence of nosocomial pneumonia is highest in **ICU** (intensive care unit) patients.

Intensive Care Unit (ICU)



Introduction

- The incidence of nosocomial pneumonia in **ventilated patients** is **10-fold higher** than non-ventilated patients
- The reported crude **mortality** for HAP is 30% to greater than 70%.

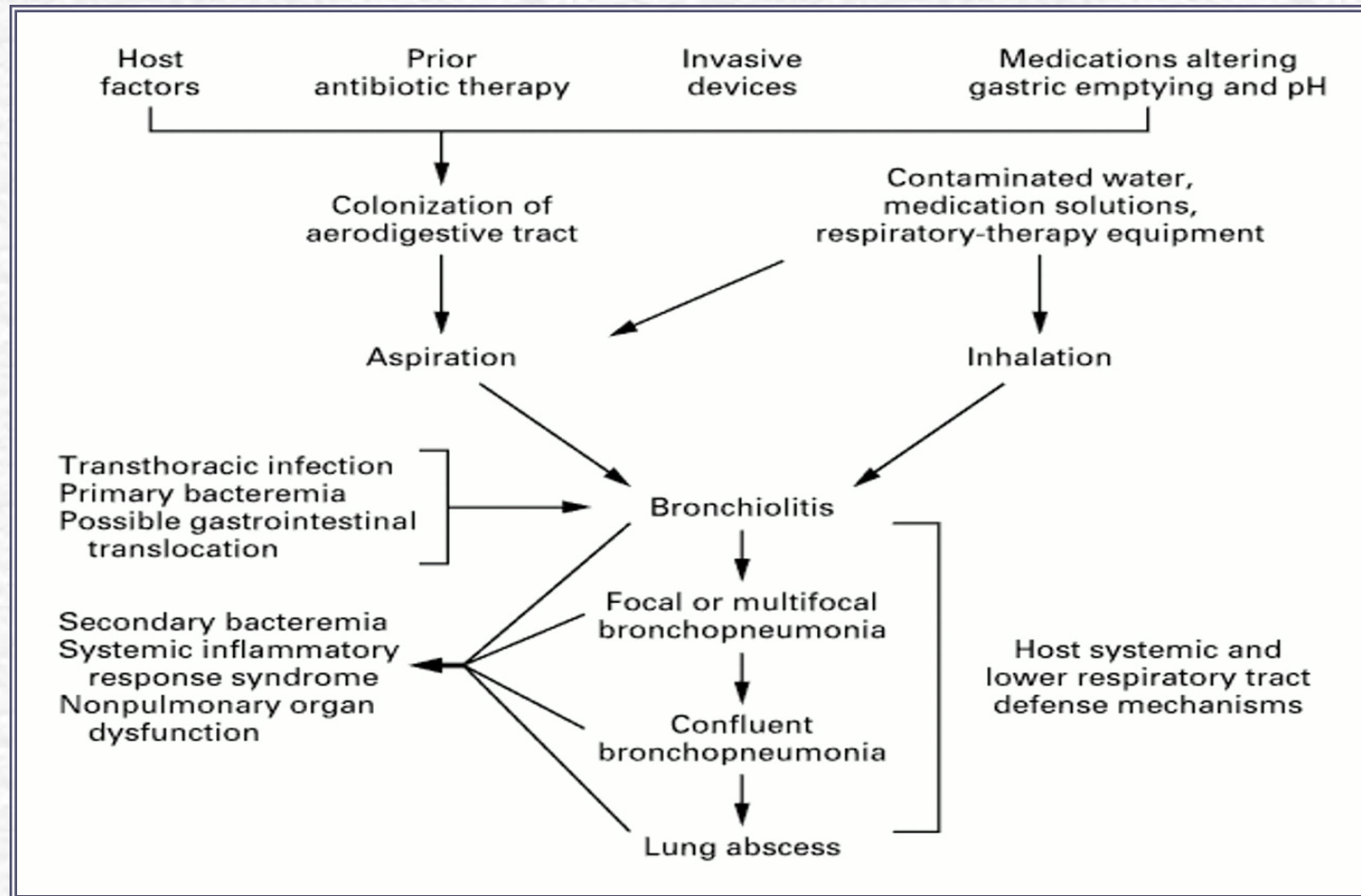
Pathogenesis of HAP



Pathogenesis

- For pneumonia to occur, at least one of the following **three conditions** must occur:
 1. Significant impairment of host defenses
 2. Introduction of a sufficient-size inoculum to overwhelm the host's lower respiratory tract defenses
 3. The introduction of highly virulent organisms into the lower respiratory tract
- Most common is microaspiration of **oropharyngeal secretions** colonized with pathogenic bacteria.

Pathogenesis



Classification

Early-onset nosocomial pneumonia:

Occurs during the **first 4 days** of admission.

Usually is due to *S. pneumoniae*, MSSA (Methicillin sensitive *S.aureus*), *H. Influenza*.

Late-onset nosocomial pneumonia:

Occurs **more than 4 days** of admission.

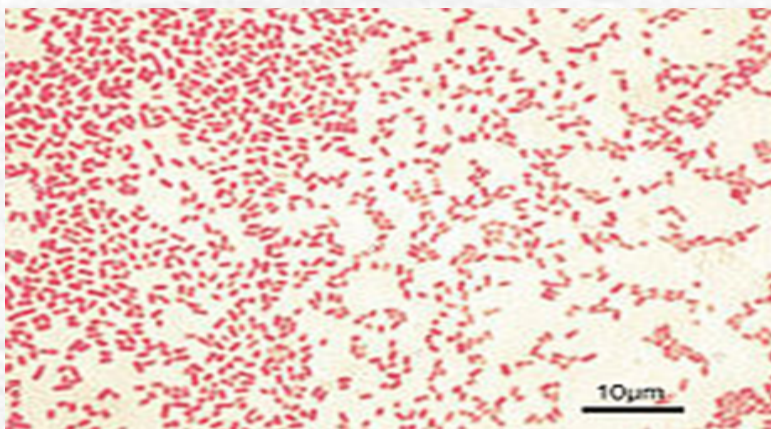
More commonly by Gram negative organisms, especially: *P. aeruginosa*, *Acinetobacter*, *Enterobacteriaceae* (*Klebsiella*, *Enterobacter*, *Serratia*) or MRSA.

Causative Agents

- **Enteric Gram negative bacilli** are isolated most frequently particularly in patients with late-onset disease and in patients with serious underlying disease often already on broad-spectrum antibiotics.
- Prior use of broad-spectrum antibiotics and an immunocompromised state make resistant Gram-negative organisms more likely.

Causative Agents

- *P. aeruginosa* and *Acinetobacter* are common causes of late-onset pneumonia, particularly in **ventilated patients**.



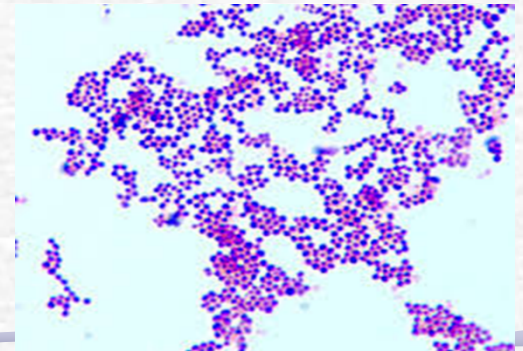
Causative Agents

• ***S. aureus*** is isolated in about 20~40% of cases and is particularly common in :

1. Ventilated patients after head trauma, neurosurgery, and wound infection
2. In patients who had received prior antibiotics or Prolonged care in ICU

• **MRSA(methicillin resistant *S.aureus*)** is seen more commonly in patients who:

- Received corticosteroids
- Undergone mechanical ventilation >5 days
- Presented with chronic lung disease
- Had prior antibiotics therapy



Causative Agents

- **Anaerobes** are common in patients predisposed to aspiration .
- **Ventilator associated pneumonia (VAP)** with anaerobes occurred more often with oropharyngeal intubation than nasopharyngeal intubation.

Ventilator-associated Pneumonia (VAP)



Ventilator-associated Pneumonia (VAP)

- Definition:

Nosocomial pneumonia that has developed in patient who are receiving mechanical ventilation.

- Classification:

Early-onset: within 48-72 hours after tracheal intubation, which complicates the intubation process

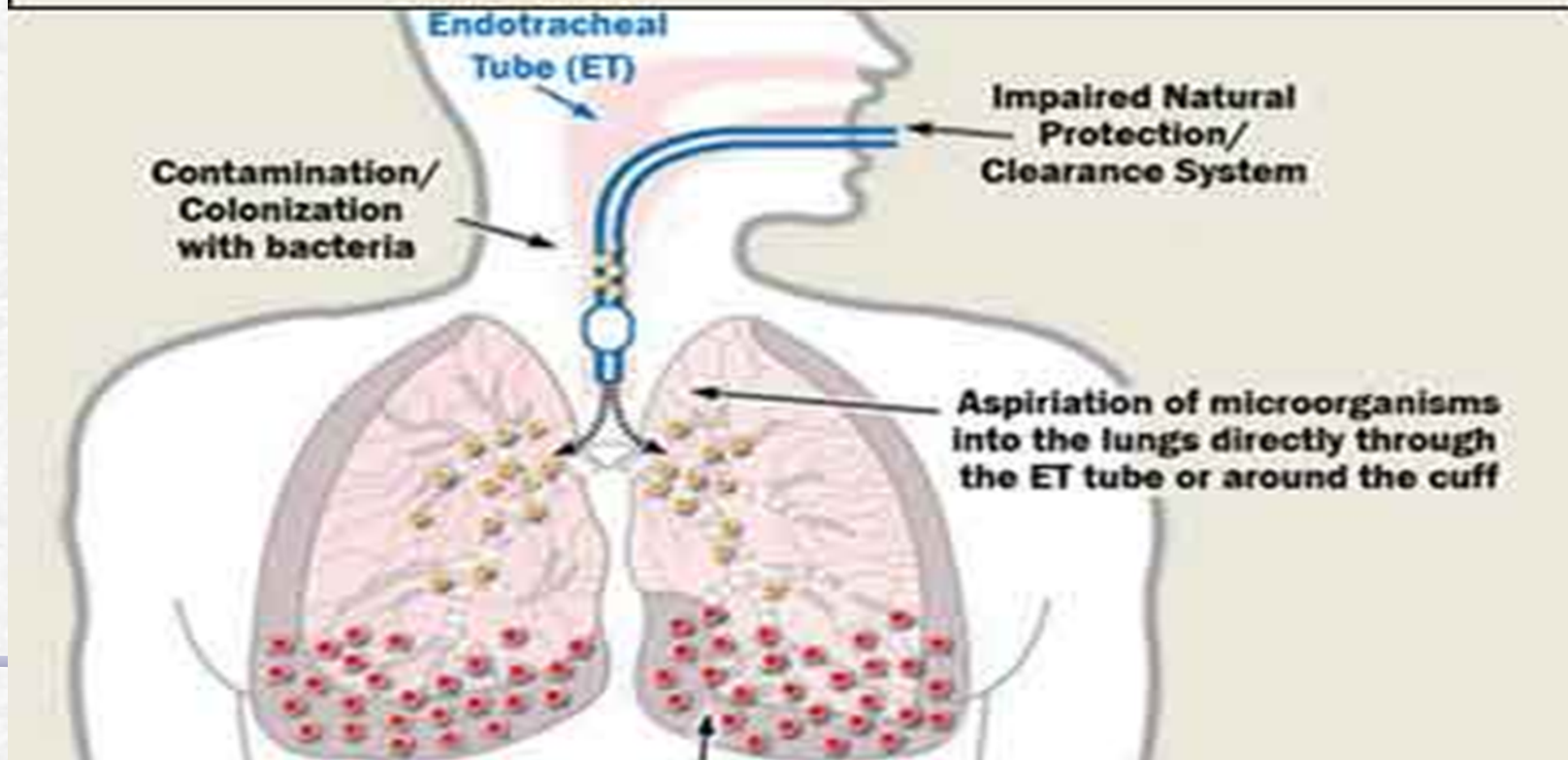
Late-onset: after 72 hours

Pathogenesis

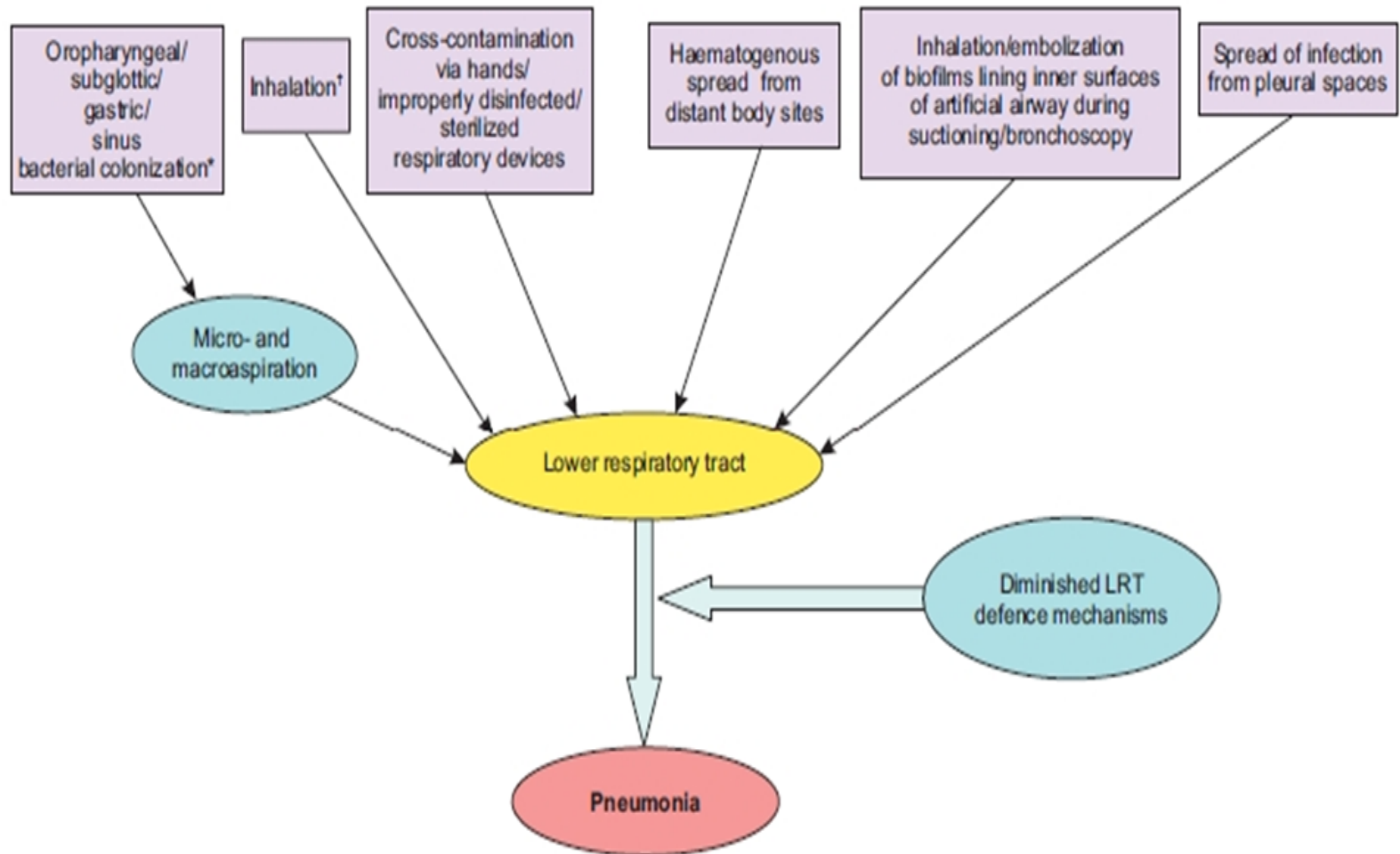
- Requires 2 important processes:
 1. Bacterial colonization of the aerodigestive tract
 2. Aspiration of contaminated secretion into the Lower airway
- Mechanical ventilation prevents mechanical clearance by cough and the mucociliary escalator.
- Sources of infection:** endogenous or exogenous.

Pathogenesis of VAP

Ventilator Associated Pneumonia



Pathogenesis of VAP

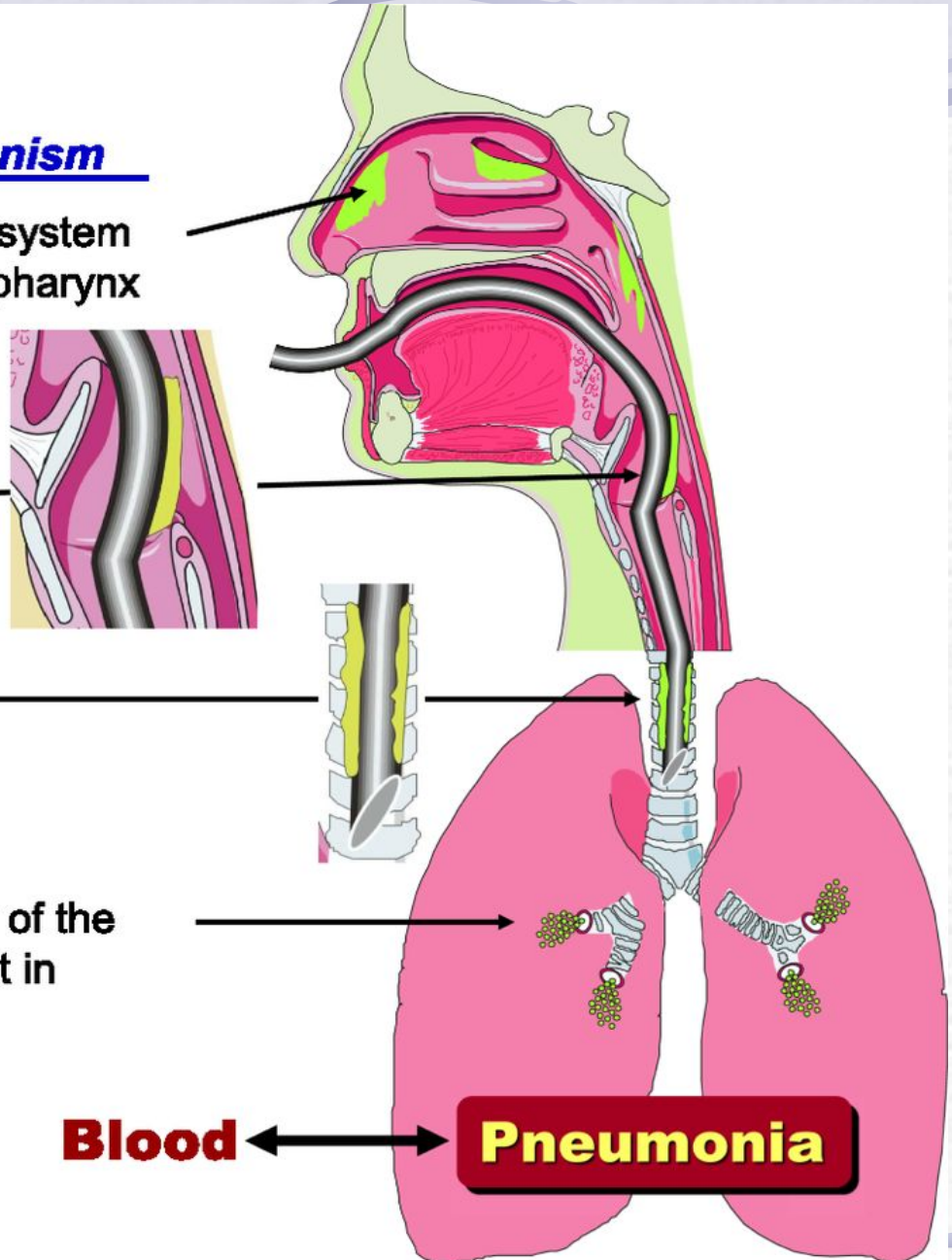


Endogenous sources of micro-organism

(1) Impaired natural protection/clearance system allows increased colonization of nasopharynx

(2) Colonized oropharynx and gastric fluid pool along tube in neonates

(3) Colonized tracheal secretions



Mechanism for pneumonia

(1) Aspiration of colonized fluids from any of the above sources into the lungs can result in pneumonia

(2) A hematogenous source seeding the lungs may rarely cause pneumonia

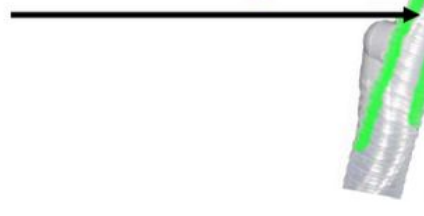
Blood ↔ **Pneumonia**

Exogenous sources of micro-organism

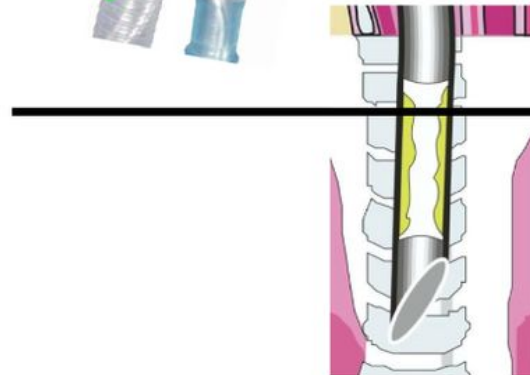
(1) Hands of health-care worker



(2) Ventilator circuit



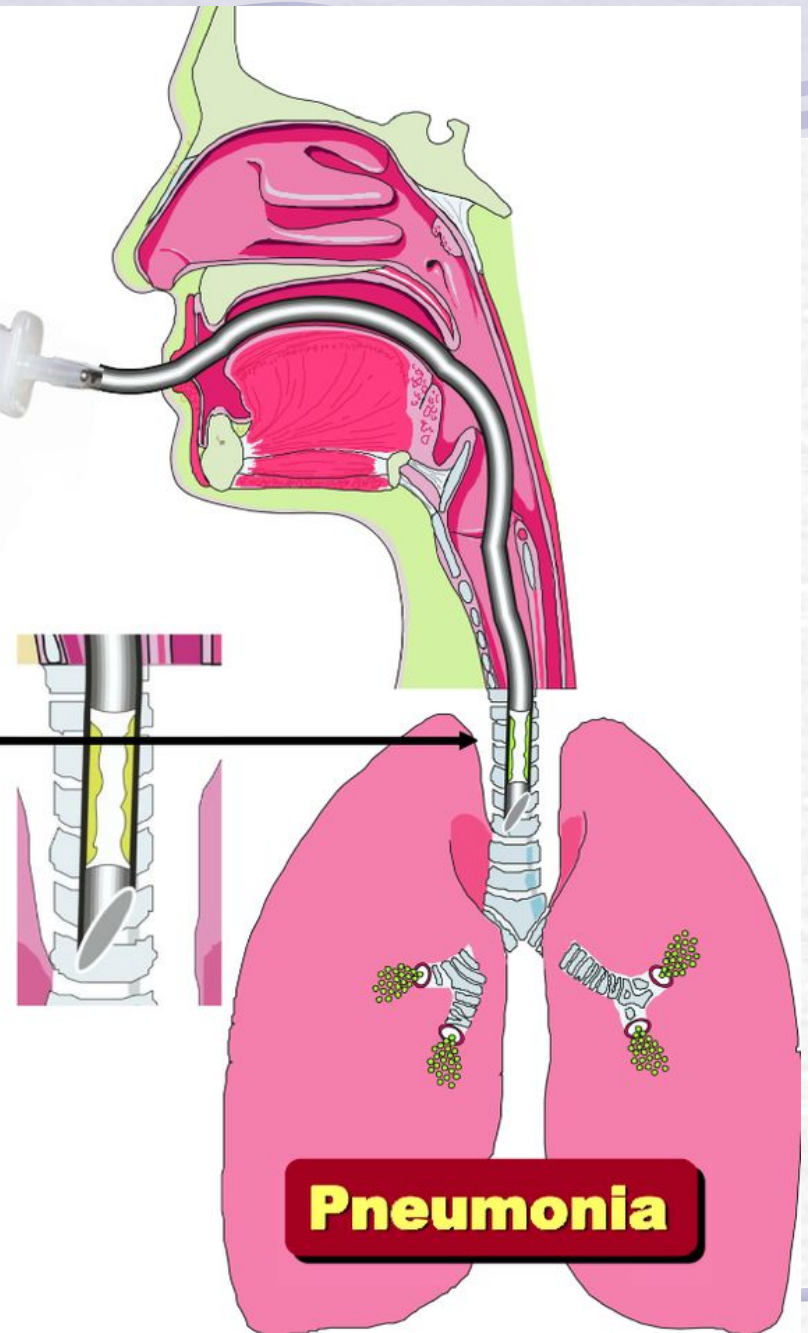
(3) Biofilm of endotracheal tube



Mechanism for pneumonia

Pneumonia occurs when colonized secretions are inhaled into the lungs through the endotracheal tube

Pneumonia



Prevention for VAP

- The oral regimen (*topical Gentamicin, Colistin, Vancomycin cream given every 6h for 3 weeks*) treating oropharyngeal colonization could prevent VAP.

--- **Prevention of VAP by oral decontamination**

American journal of respiratory critical care medicine 2001 164:382-8

Preventions for VAP

Non-pharmacologic strategies

- ✓ Effective hand washing and use of protective gowns and gloves
- ✓ Semirecumbent positioning
- ✓ Avoidance of large gastric volume
- ✓ Oral (non-nasal) intubation
- ✓ Continuous subglottic suctioning
- ✓ Humidification with heat and moisture exchanger
- ✓ Posture change

Preventions for VAP

Pharmacologic strategies

- Avoiding stress-ulcer prophylaxis
- Prophylactic antibiotic therapy
- Chlorhexidine oral rinse
- Prophylactic treatment of neutropenic patients
- Vaccines

Treatment

- Most initial therapy is empiric because no pathogen is identified or results are not available when antimicrobial decisions are made in most patients.

Treatment

- Initially be treated with a broad-spectrum antibiotic regimen aimed at covering all likely bacterial pathogens
- This regimen should subsequently be narrowed, according to the result of culture.**

Treatment

- The pathogen may be influenced by coexisting illnesses, prior treatment, and length of hospitalization.
- The frequency of ICU-acquired *P. aeruginosa* carriage or colonization/infection was 23.4% at 7 days and 57.8% at 14 days.

---- Current opinion in infectious disease 2002, 15:387-94, copyright LWW

Treatment

- The mortality can be reduced with early appropriate empiric therapy.

(From 30 % with appropriate therapy to more than 90 % with inappropriate therapy) .

Treatment

- Cefepime or
 - Piperacillin-tazobactam or
 - Meropenem or
 - Levofloxacin or
-
- If risk for MRSA or more severe disease add vancomycin

Response to Therapy

• If no clinical response is noted or deterioration occurs, we need to consider:

1. **Infectious causes:**

Resistant pathogen

Superinfection

Unusual pathogens

Lung abscess

Extrapulmonary infection

2. **Noninfectious events:**

Heart: congestive heart failure (CHF)

Lung: fibroproliferative acute respiratory distress syndrome (ARDS), pulmonary emboli, Atelectasis.

Reference book

Ryan, Kenneth J. Sherris Medical Microbiology.
Letest edition.

Mc Graw –Hill eduction