# Health Care Associated Pneumonia

**PROF .*HANAN HABIB***

*Department of Pathology, Microbiology Unit ,*

*College Of Medicine*

# 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 .

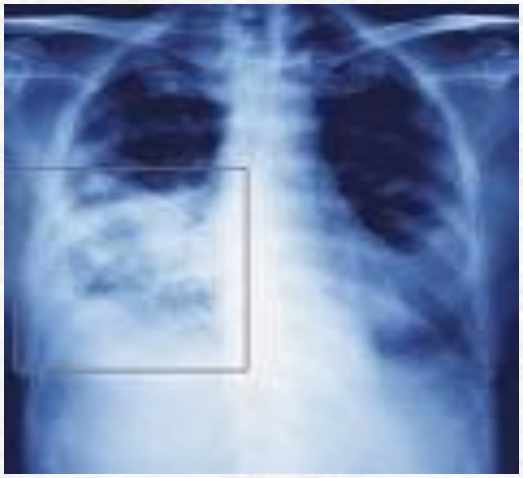
# Objectives

- 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.

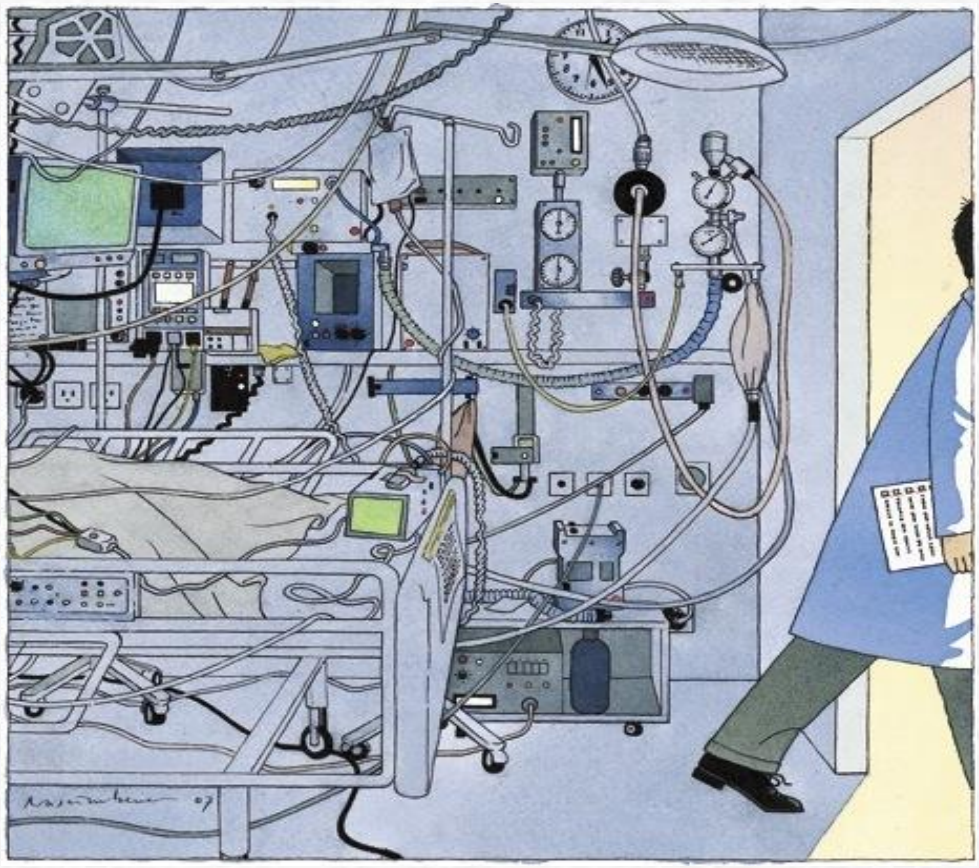
# Health care associated Pneumonia

- **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)

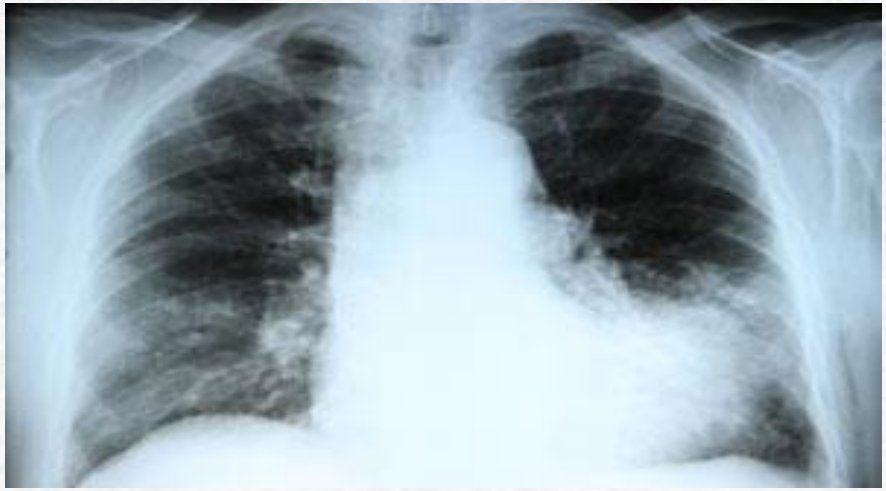



- The incidence of nosocomial pneumonia in ventilated patients is **10-fold higher** than non-ventilated patients

- The reported crude **mortality** for HAP is 30% to greater than 70%.

--- Medical Clinics of North America
**Therapy of Nosocomial pneumonia** 2001 vol.85 1583-94

# Pathogenesis of HAP



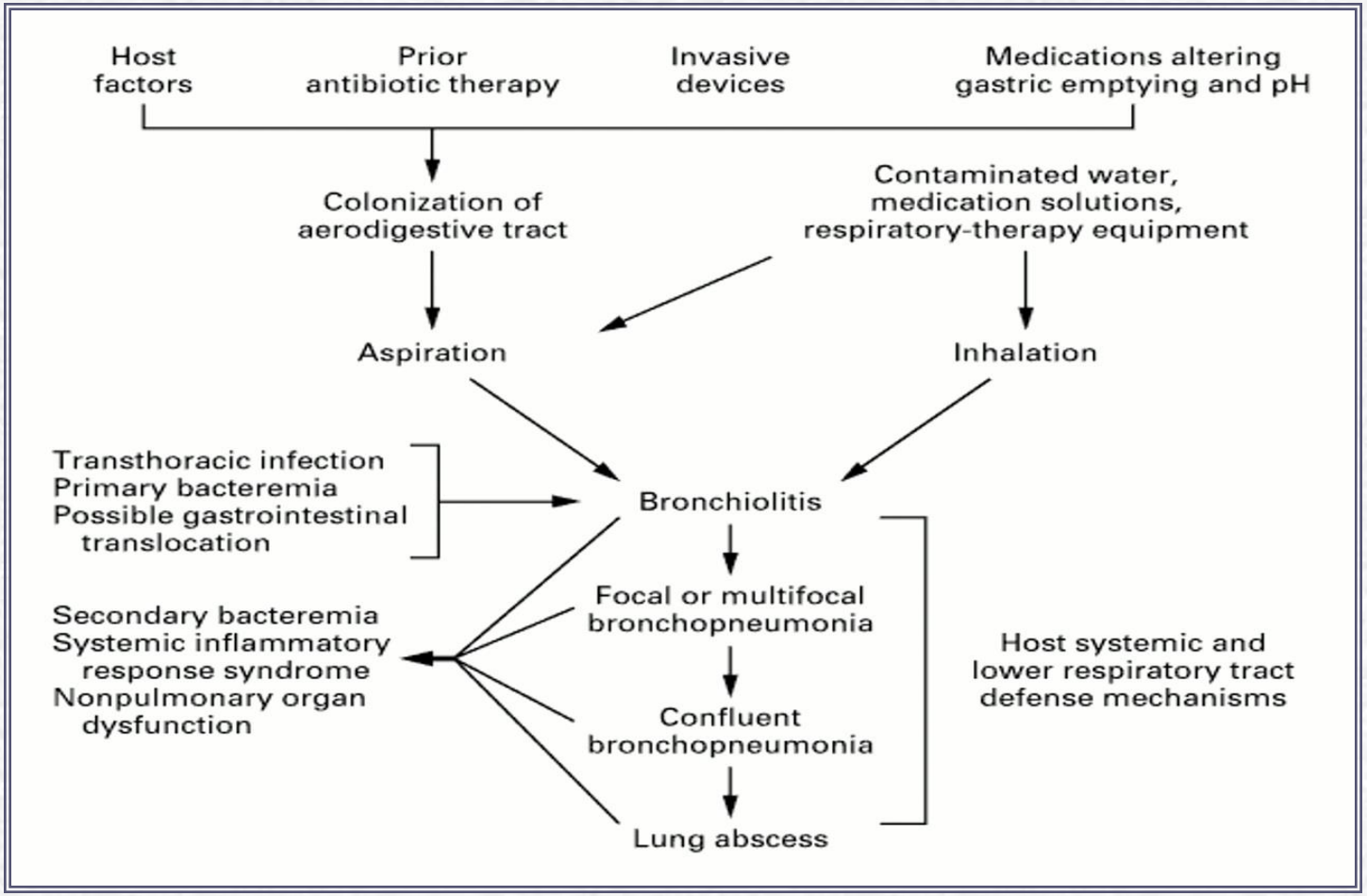
# Pathogenesis

- 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




--- The Prevention of Ventilator-Associated Pneumonia Vol.340 Feb 25, 1999 NEJM

# 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*, or anaerobes.

- Late-onset nosocomial pneumonia:

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

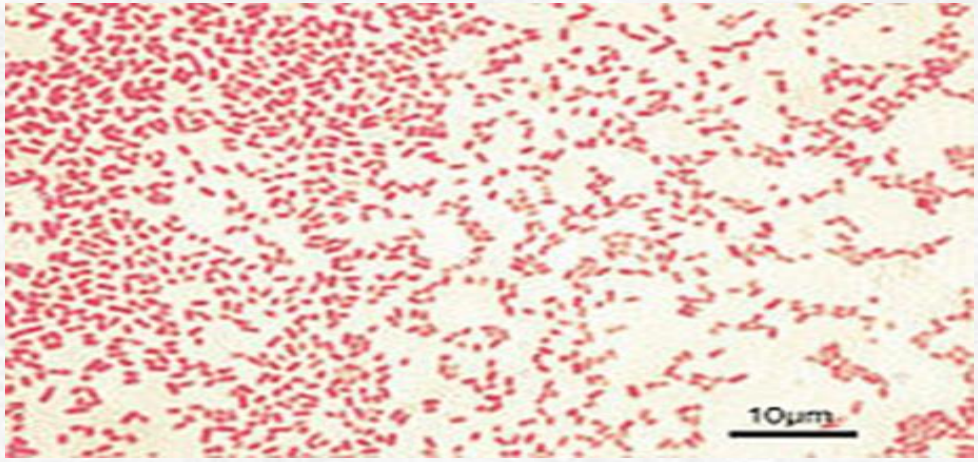
  More commonly by Gram negative organisms, especially: *P. aeruginosa, Acinetobacter, Enterobacteriaceae* (*Klebsiella*, *Enterobacter, Serratia*) or MRSA.

# 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

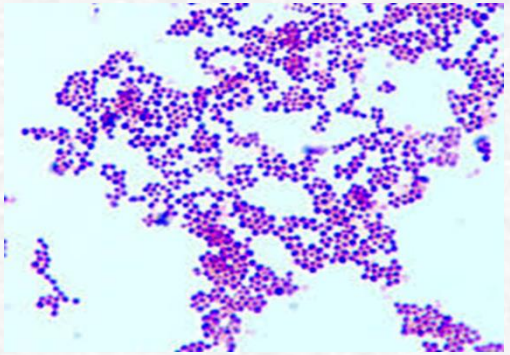
- **MRSA(methicillin resistant *S.aureus*)** is seen more commonly in patients who:

Received corticosteroids

Undergone mechanical ventilation >5 days

Presented with chronic lung disease

Had prior antibiotics therapy

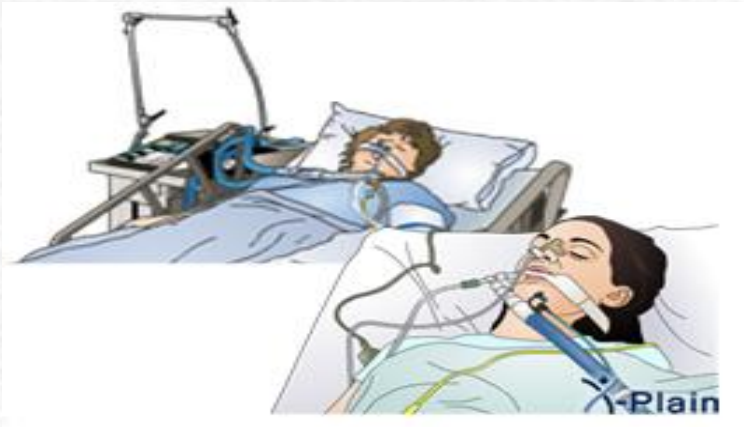


# Causative Agents

- **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 patients receiving mechanical ventilation.

- Classification:

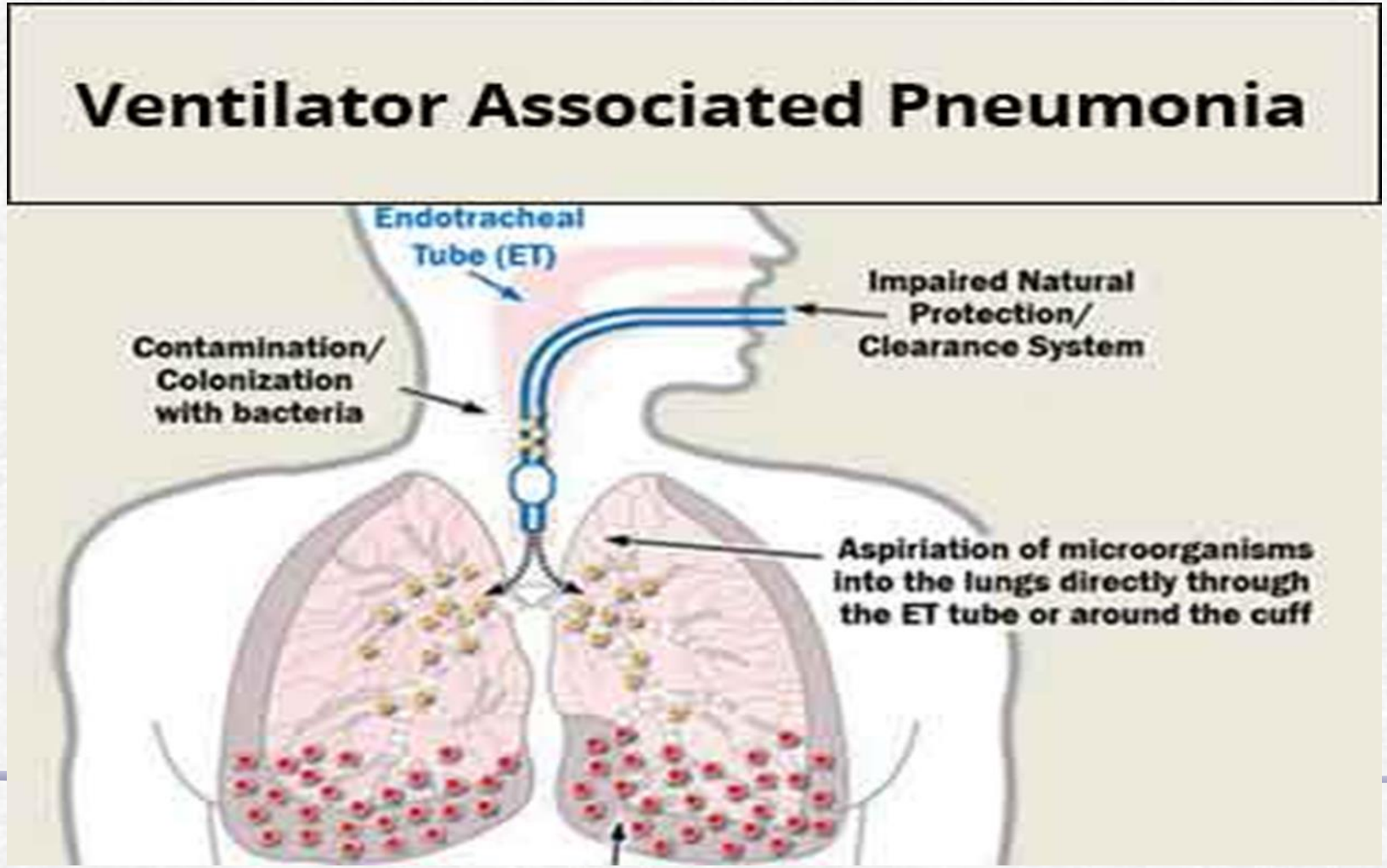
  **Early-onset**: within 48-72 hours after tracheal intubation, which complicates the intubation process

  **Late-onset**: after 72 hours

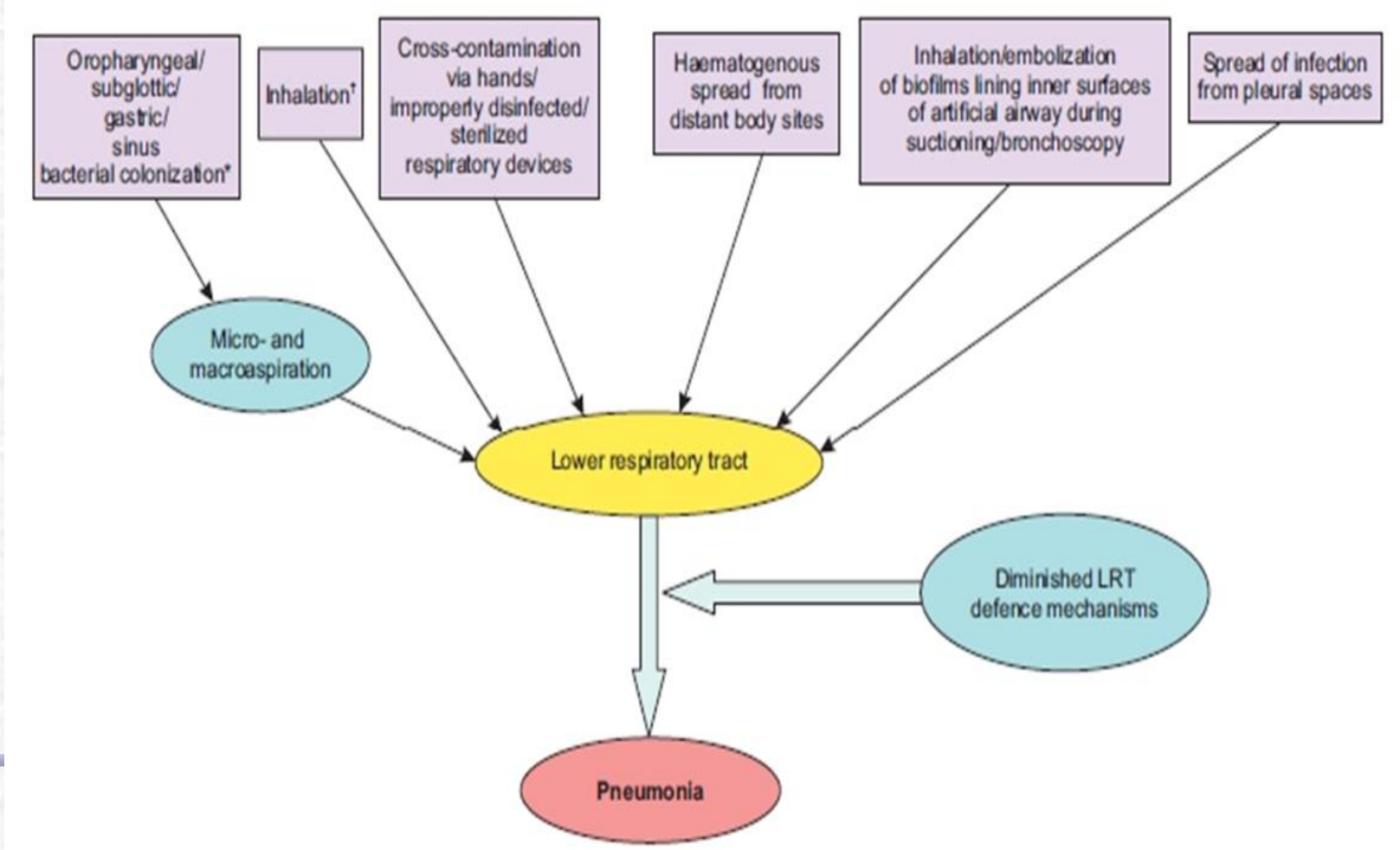
# Pathogenesis

- Two important processes required:
  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



# Pathogenesis of VAP



## Endogenous sources of micro-organism

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

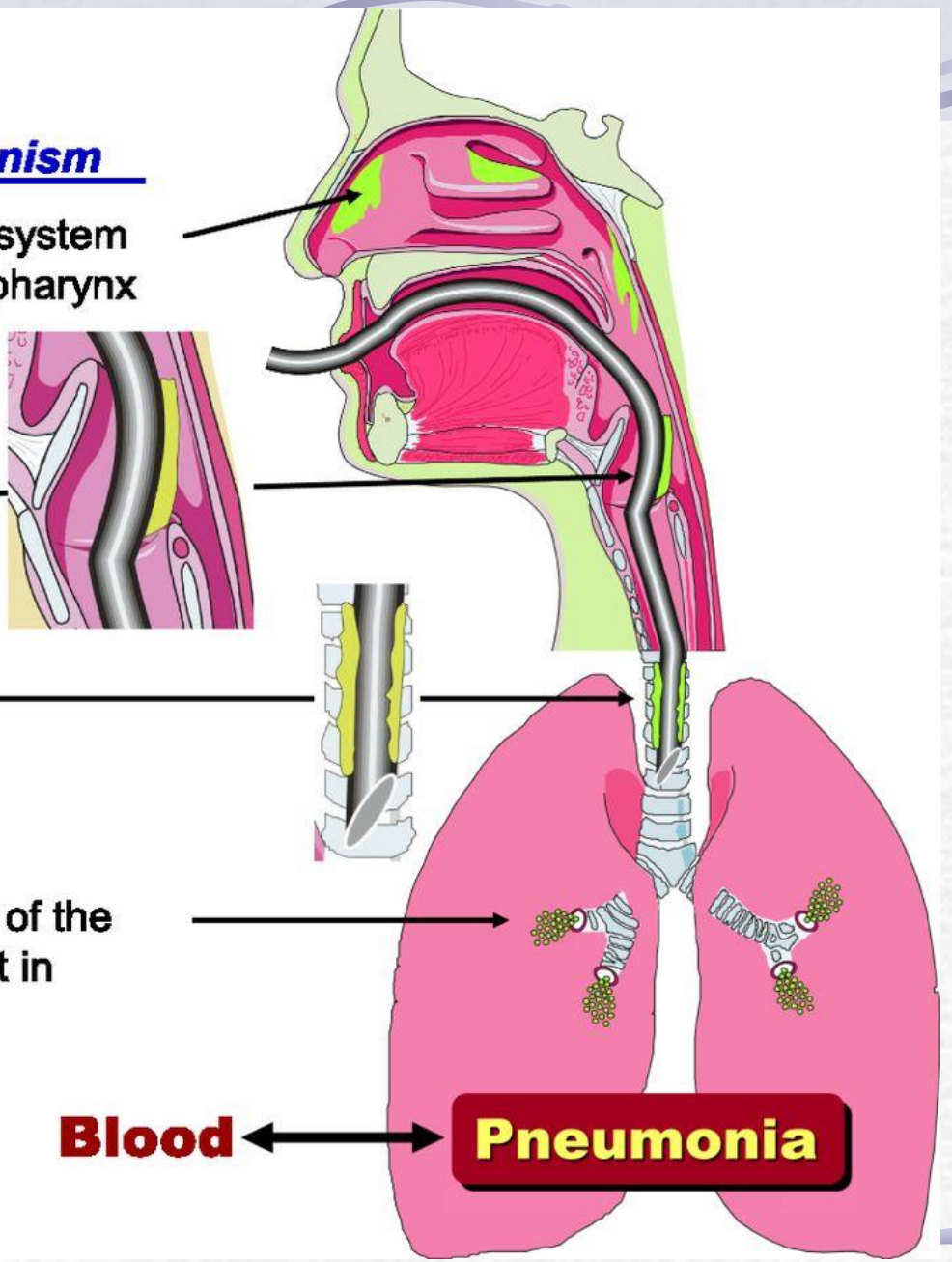
(2) Colonized oropharynx and gastric fluid pool along tube in neonates

(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

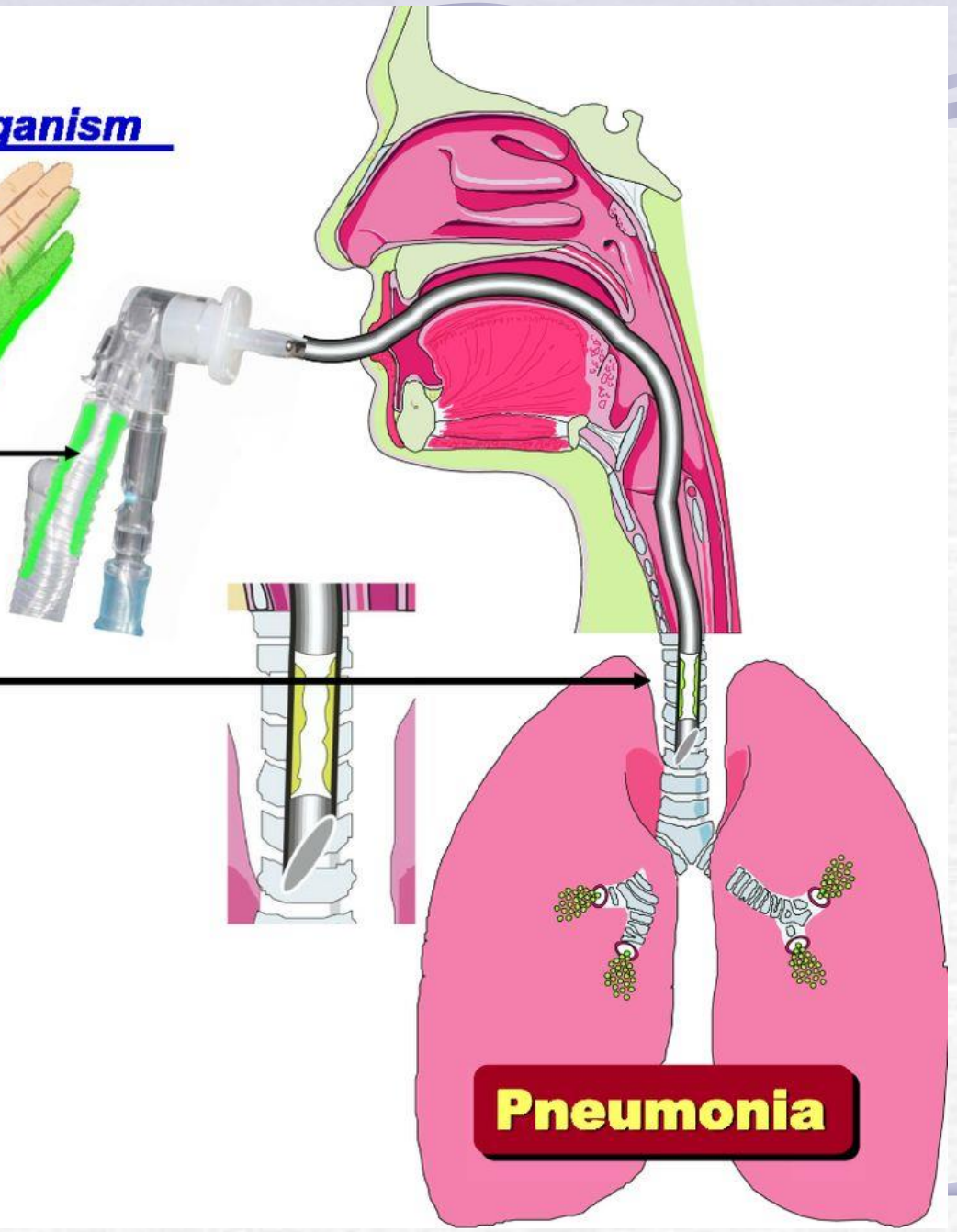


## Exogenous sources of micro-organism

(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

# 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 medicine2001 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

--- The Prevention of Ventilator-Associated Pneumonia Vol.340 Feb 25, 1999 NEJM

# Preventions for VAP

## Pharmacologic strategies

- Stress-ulcer prophylaxis
- Combination antibiotic therapy
- Prophylactic antibiotic therapy
- Chlorhexidine oral rinse
- Prophylactic treatment of neutropenic patients
- Vaccines

--- The Prevention of Ventilator-Associated Pneumonia Vol.340 Feb 25, 1999 NEJM

# 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.

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

---

# Treatment

- Guidelines by American Thoracic Society has divided patients into three groups, each with a set of probable pathogens.

Group 1: mild to moderate HAP with no risk factor

Group 2: mild to moderate HAP with risk factor

Group 3a: severe HAP, early-onset with no risk factor

Group 3b: severe HAP, late-onset or with risk factor

# Treatment

- For **mild-to-moderate** HAP, monotherapy has been shown to be effective.

- For **severe** HAP in which infection with resistant organisms is likely, combination therapy probably should be instituted until culture result are available.

# Treatment

- Patients with *S. aureus* infection, agents against this organism are necessary, including **Vancomycin** if MRSA is suspected.
- **Linezolid** is comparable with Vancomycin.

  The advantage of Linezolid is less possible nephrotoxicity.

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

# Treatment

- Combination of antipseudomonal drugs is controversial:

1. **Traditional:**

   **antipseudomonal Beta-lactam with an Aminoglycoside**.
   Synergy but potential nephrotoxicity.

2. **Another approach**:

   **antipseudomonal Beta-lactam with a Fluoroquinolone.**
   No benefit of synergy but reduce concern of nephrotoxicity, and quinolone gets into the lungs at higher concentrations.

# 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, Atelectesis.