



# *Pneumonia*

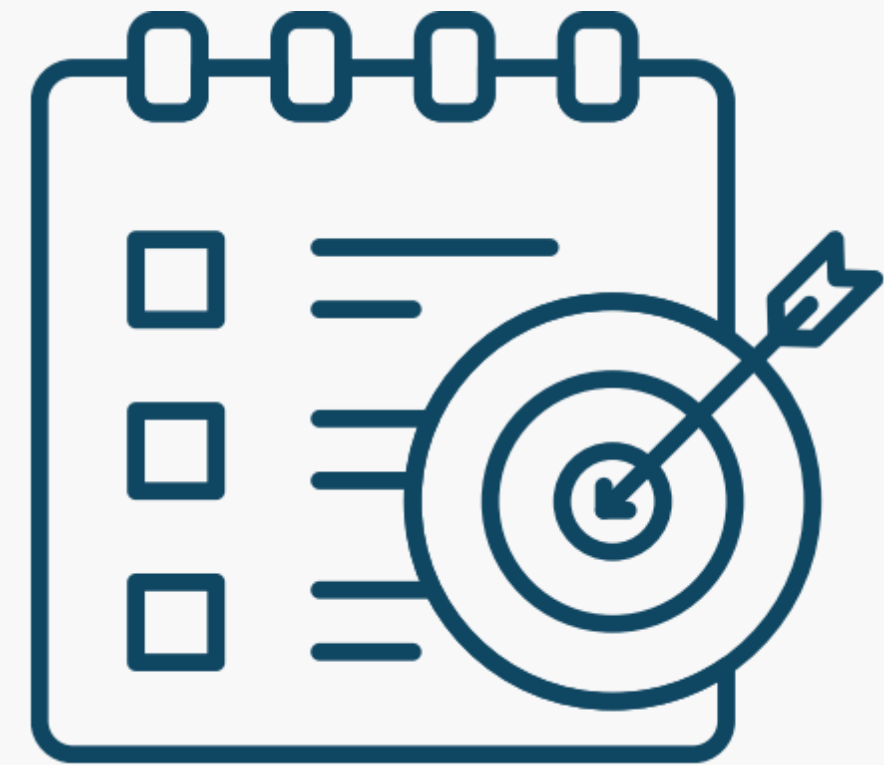
ASEEL OMOSH  
DANIA WALEED  
JEENA OMOR  
MARIA HOOD  
SAJA OMOUSH  
RAMA KHRESAT  
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AYMAN BATAINEH  
MOFEED OBEIDAT  
MOHAMMAD ABUSHAQRA  
MOHAMMAD ABDULLATIF



# ***Pneumonia Objectives ;***

- 1) basic info & ntroduction**
- 2) community acquired**
- 3) nosocomial pneumonia**
- 4) Aspiration pneuemonia**
- 5) Complications**
- 6) Investigations**
- 7) Treatment**



# Pneumonia Anatomy

**Right lung has 2 fissures:** horizontal & oblique dividing lung into 3 lobes : upper, lower & middle lobes.

**right upper lobe :** apical / posterior / anterior

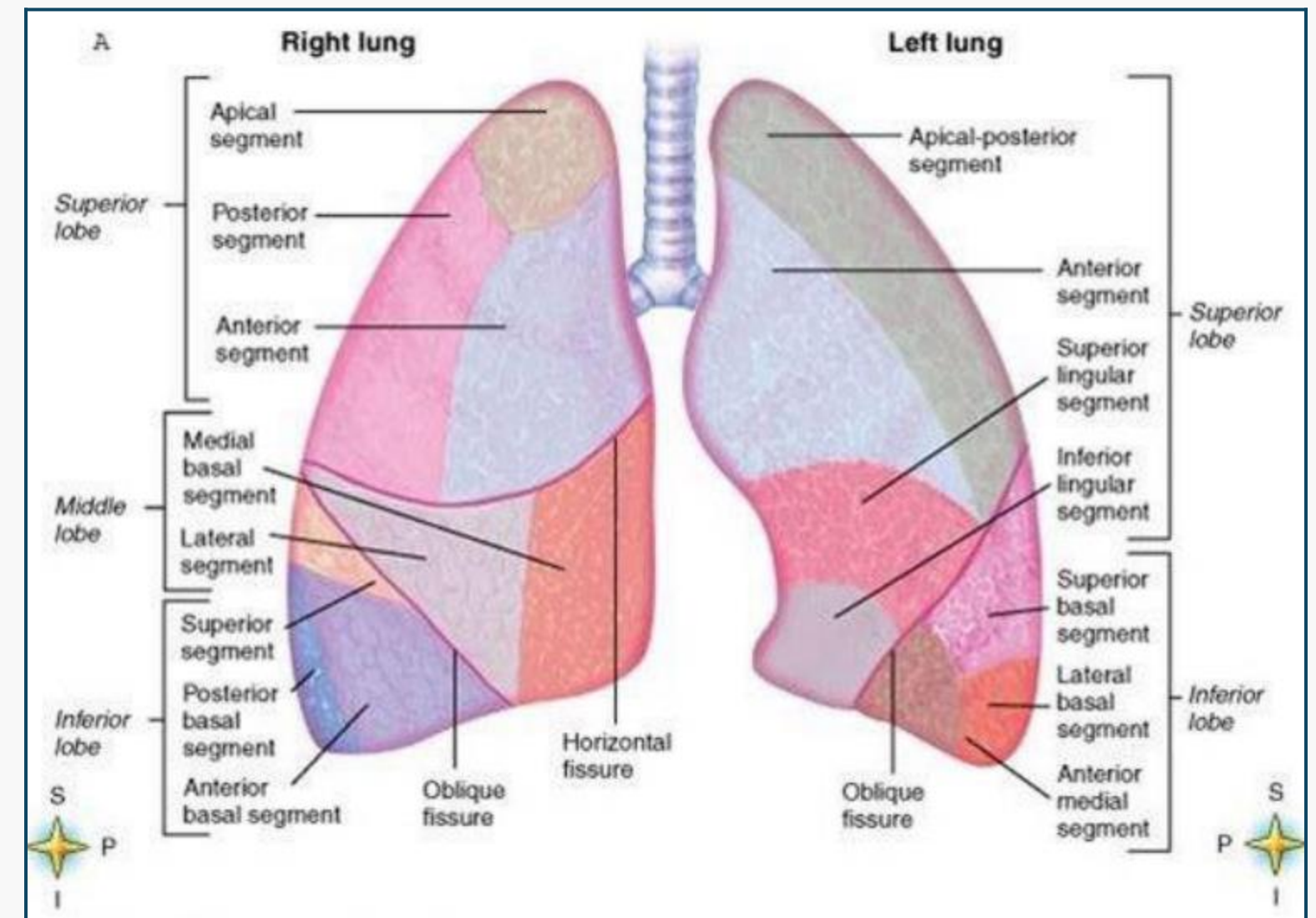
**right middle lobe :** lateral / medial

**right lower lobe :** superior /posterior / medial / lateral / anterior

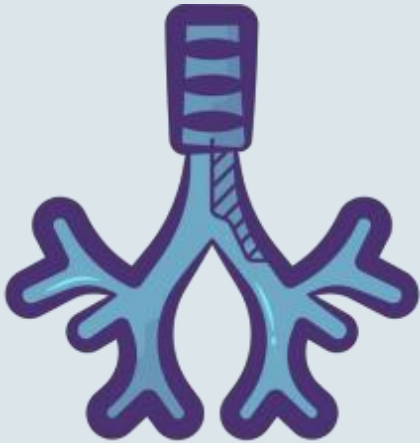
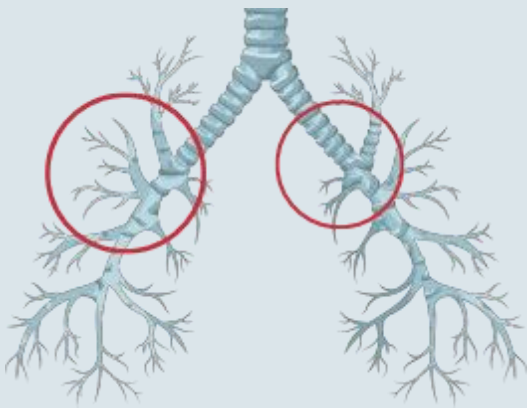
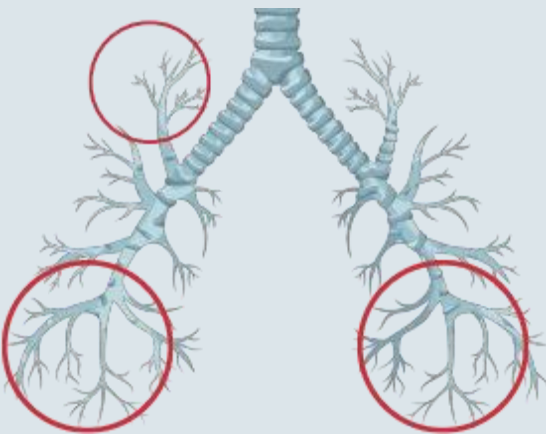
**left lung** has one oblique fissure dividing lung into upper & lower lobes.

**left upper lobe:** apicoposterior segment / anterior / superior lingular / inferior lingular

**left lower lobe :** superior /posterior/ anterior / medial / lateral



# Pneumonia Histology

| TRACHEA                                                                                      | PRIMARY BRONCHI                                                                              | SECONDARY BRONCHI                                                                             | TERTIARY BRONCHI                                                                      | TERMINAL AND RESPIRATORY BRONCHIOLES                                                  | ALVEOLI                                                                                                                                              |
|----------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------|
| <div>RESPIRATORY EPITHELIAL</div> <div>CILIA TED PSEUDO STRATIFIED COLUMNAR EPITHELIUM</div> | <div>RESPIRATORY EPITHELIAL</div> <div>CILIA TED PSEUDO STRATIFIED COLUMNAR EPITHELIUM</div> | <div>RESPIRATORY EPITHELIAL</div> <div>CILIA TED PSEUD - STRATIFIED COLUMNAR EPITHELIUM</div> | <div>CILIA TED CUBOIDAL EPITHELIUM</div>                                              | <div>CILIA TED CUBOIDAL EPITHELIUM</div>                                              | <div>TYPE 1 PNEUMOCYTE<br/>TYPE 2 PNEUMOCYTE<br/>MACHROPHAGES</div>                                                                                  |
| <div>CONNECTIVE TISSUE</div>                                                                 | <div>SMOOTH MUSCLE</div>                                                                     | <div>SMOOTH MUSCLE</div>                                                                      | <div>SMOOTH MUSCLE<br/>IN TERTIARY</div>                                              | <div>SMOOTH MUSCLE<br/>IN TERTIARY</div>                                              |                                                                   |
| <div>GLANDS</div>                                                                            | <div>GLANDS</div>                                                                            | <div>GLANDS</div>                                                                             | <div>-</div>                                                                          | <div>CLARA CELLS</div>                                                                |                                                                                                                                                      |
| <div>C-HYALINE CARTILAGE</div>                                                               | <div>RING -HYALINE CARTILAGE AND PLATES</div>                                                | <div>NO CARTILAGE ONLY C.T.</div>                                                             | <div>-</div>                                                                          | <div>-</div>                                                                          | <div>INTERSTITIAL</div>                                                                                                                              |
|           |           |          |  |  | <div>CONNECTIVE TISSUE SURROUNDS AND SUPPORT THE ALVEOLI</div>  |

# ***introduction :***

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## **Risk factors !!**

Impairment of defense mechanisms leading to pulmonary infections :

- 1- Loss or suppression of cough reflex: coma , general anesthesia, drugs, alcohol.
- 2-Injury to mucociliary apparatus: smoke, alcohol
- 3-Decrease in macrophage function: alcohol, smoking, phagocyte killing defects.
- 4- Impaired immune system:  
chronic diseases like DM, CKD , acquired or congenital immune deficiency, AGING.
- 5-Existing pulmonary disease: atelectasis, edema, COPD,cystic fibrosis
- 6- Unusually virulent infecting organism



# *introduction*

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Pneumonia Infection of the lung parenchyma (bronchioles , alveoli, interstitium ) (acute inflammation)

Occurs when normal defenses are impaired                      \*ccn lead to death

chief complaint : usually acute cough (dry or productive ), fever ,SOB pleuritic chest pain, with gradual onset within days to weeks

It is usually caused by bacteria, but it could be caused by virus and fungi

the term consolidation refers to solidification of the lung due to replacement of the air by exudate in the alveoli

Secondary pneumonia (superimposed ) ..Bacterial pneumonias often follow a viral URTI

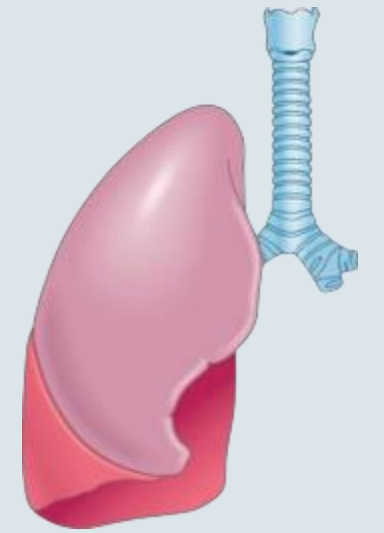


# ***Anatomical classification***

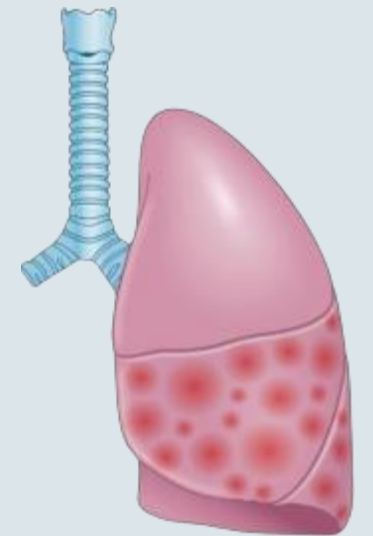
## ***Pneumonia***



**Lobar**



**Bronchopneumonia**

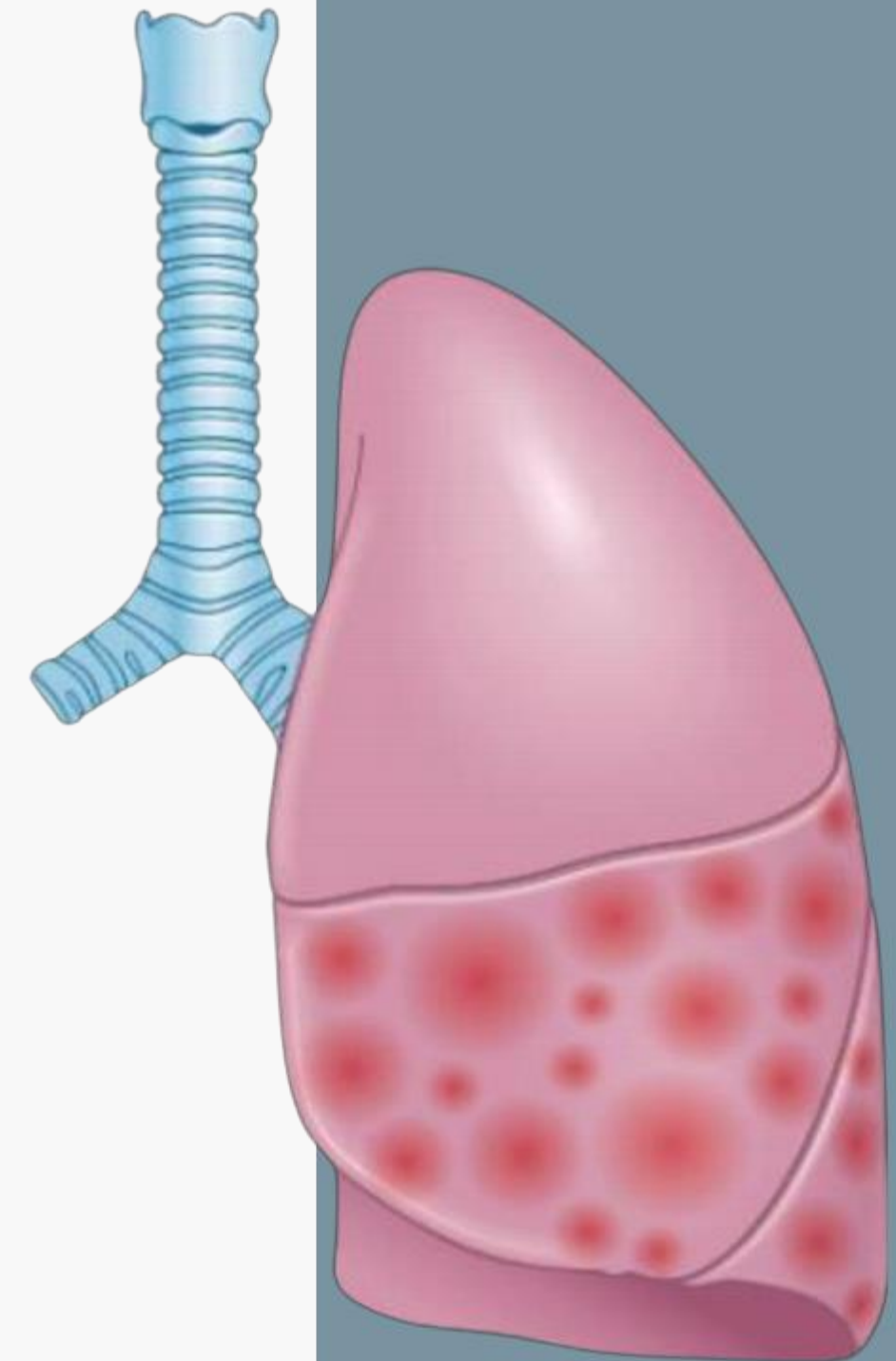


**Interstitial**

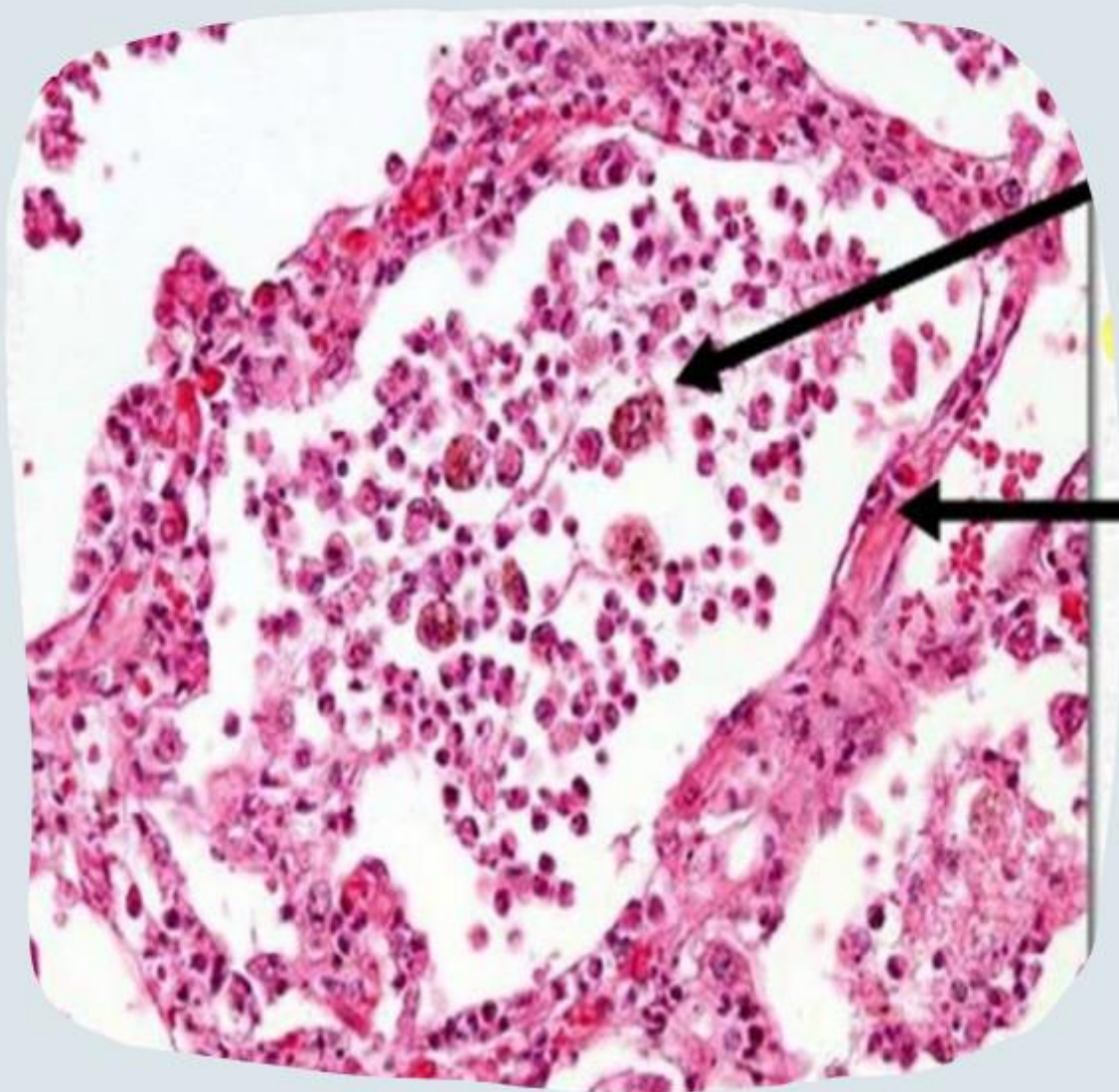


# *Lobar*

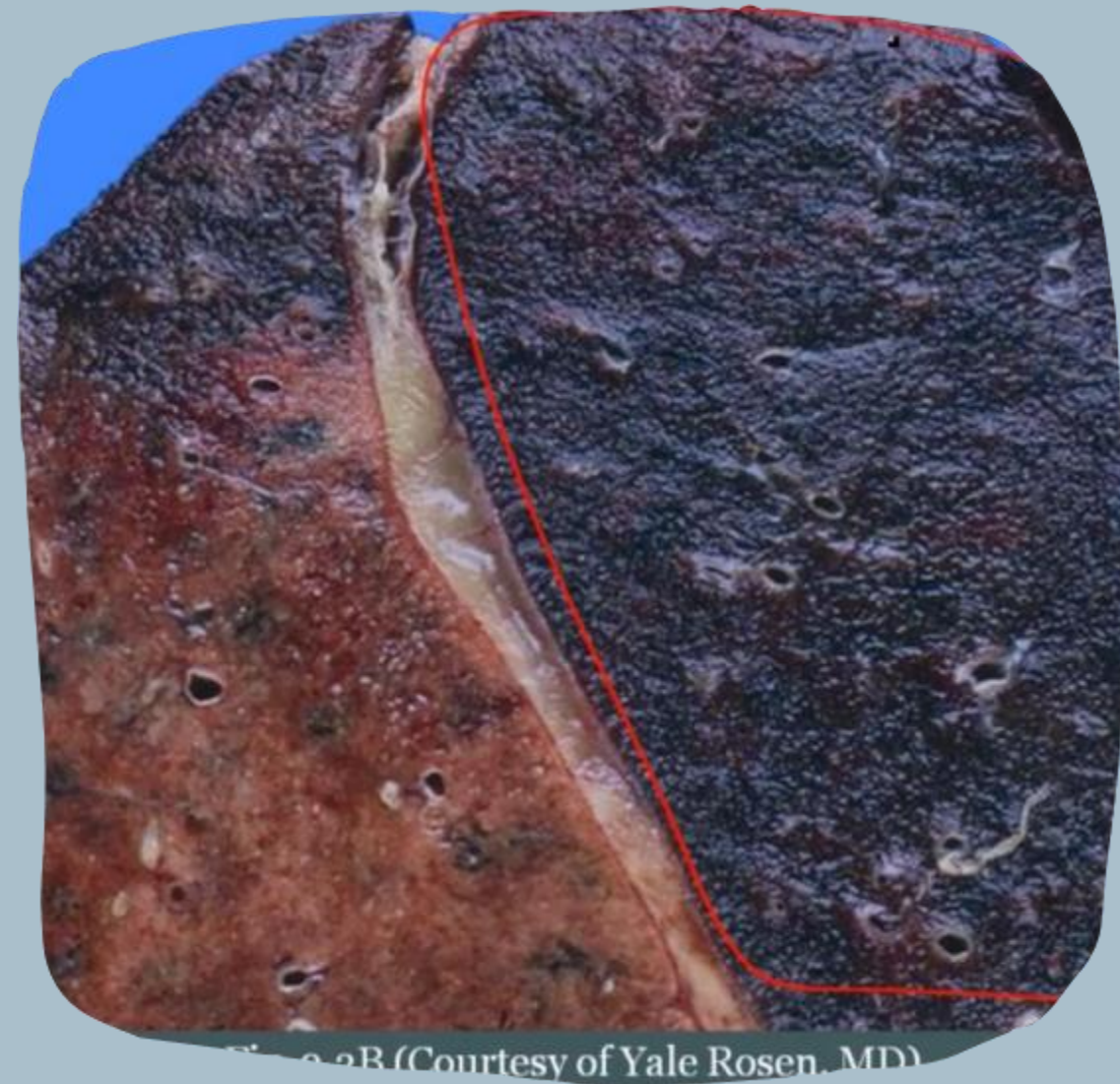
- Intra-alveolar —> exudate / consolidation (REVERSIBLE) ; may involve entire lobe or the whole lung
- Characterized by consolidation of an entire lobe of the lung so adjacent air spaces are homogeneously filled with an exudate.
- Usually bacterial; most common cause are *Streptococcus pneumoniae*



# *Lobar*



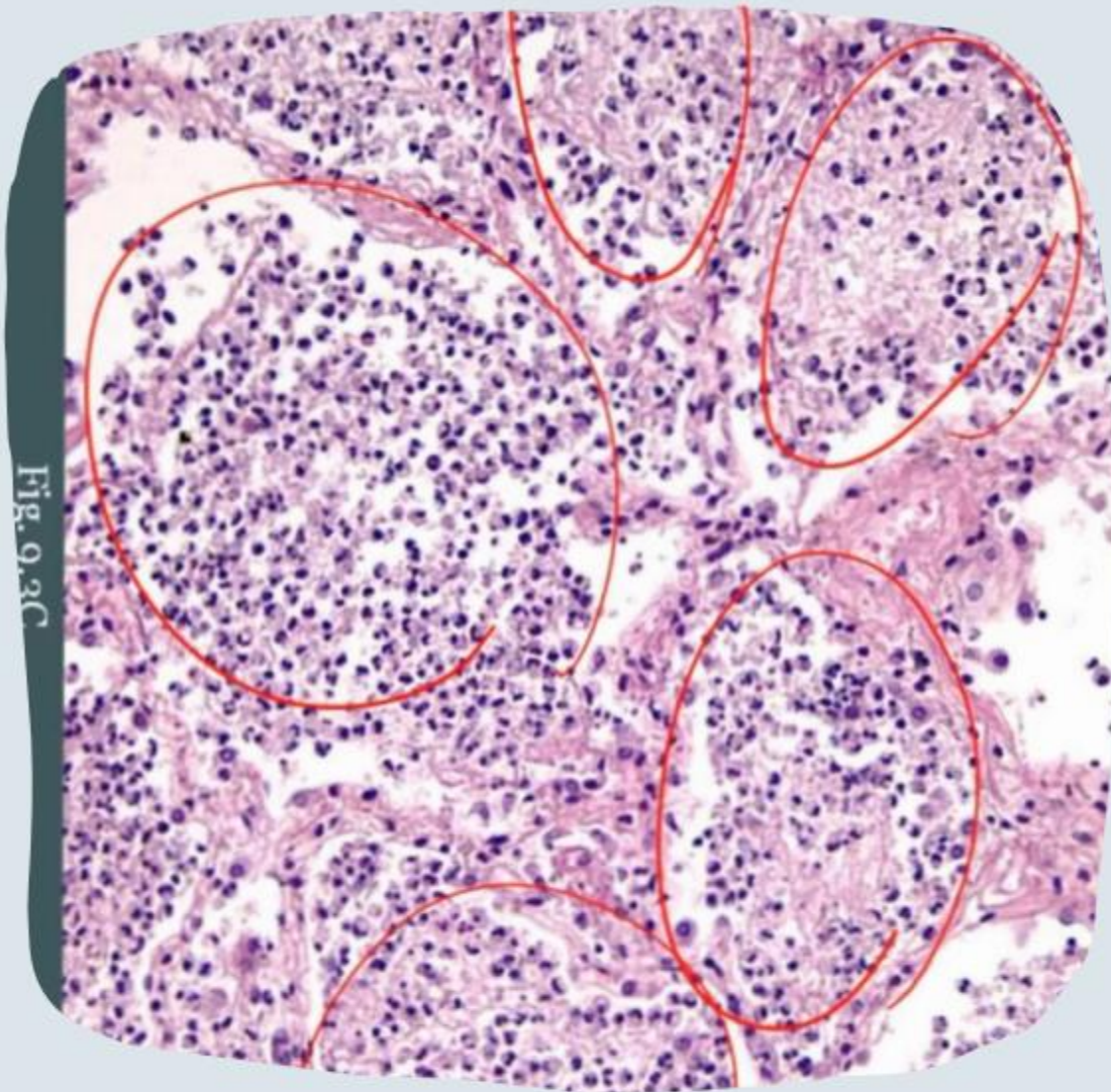
LM



GROSS



# *Lobar*



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GROSS



# ***Classic gross phases of lobar pneumonia***

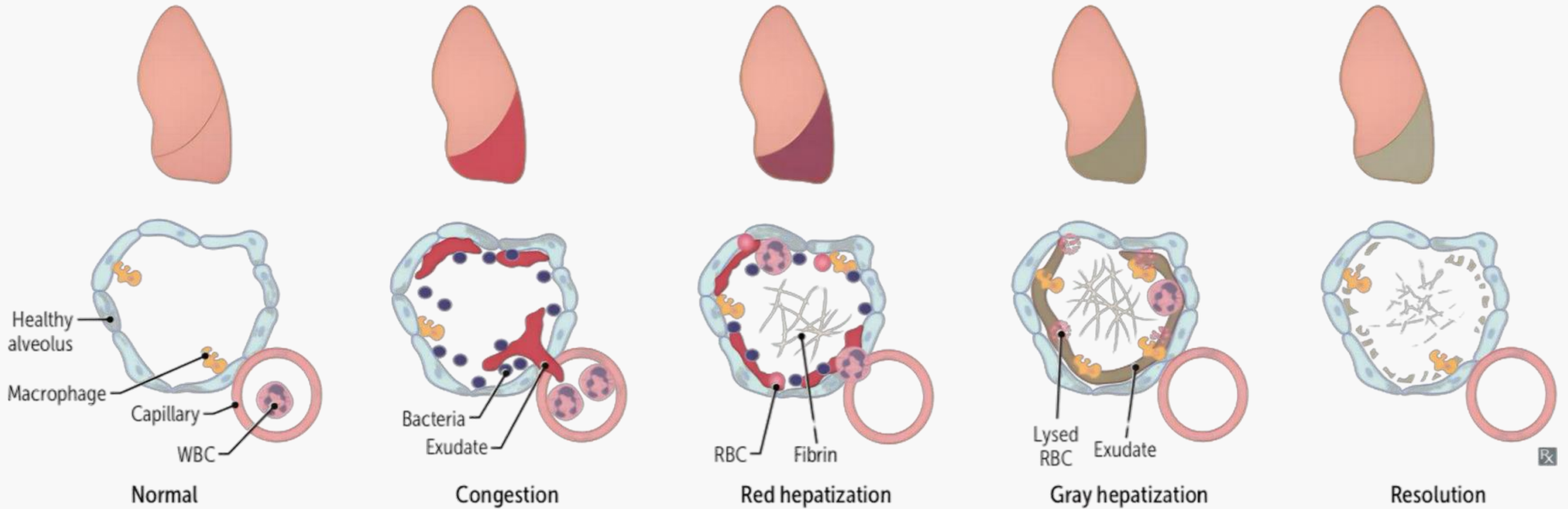
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• • • • •

1. **Congestion**- due to congested vessels and edema, Exudate with mostly bacteria
2. **Red hepatization**-due to exudate, fibrin, bacteria, neutrophils, and hemorrhage RBC filling the alveolar air spaces, giving the normally spongy lung a solid consistency
3. **Gray hepatization**-due to degradation of red cells within the exudate, Exudate full of WBCs, lysed RBCs, and fibrin.
4. **Resolution** : Enzymatic digestion of exudate by macrophages, and regeneration of normal lung tissue by type 2 pneumocytes (stem cells)

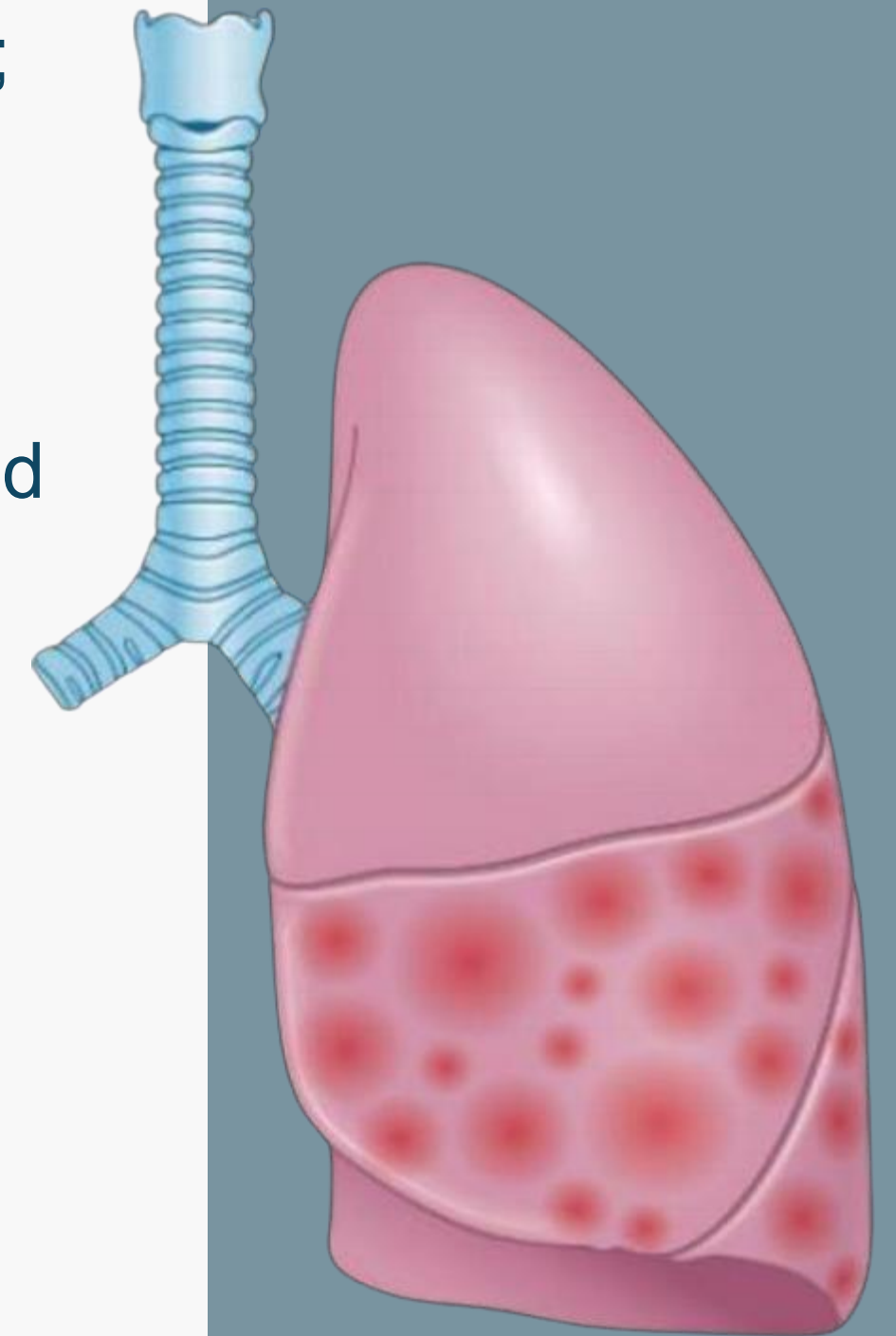
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# Lobar

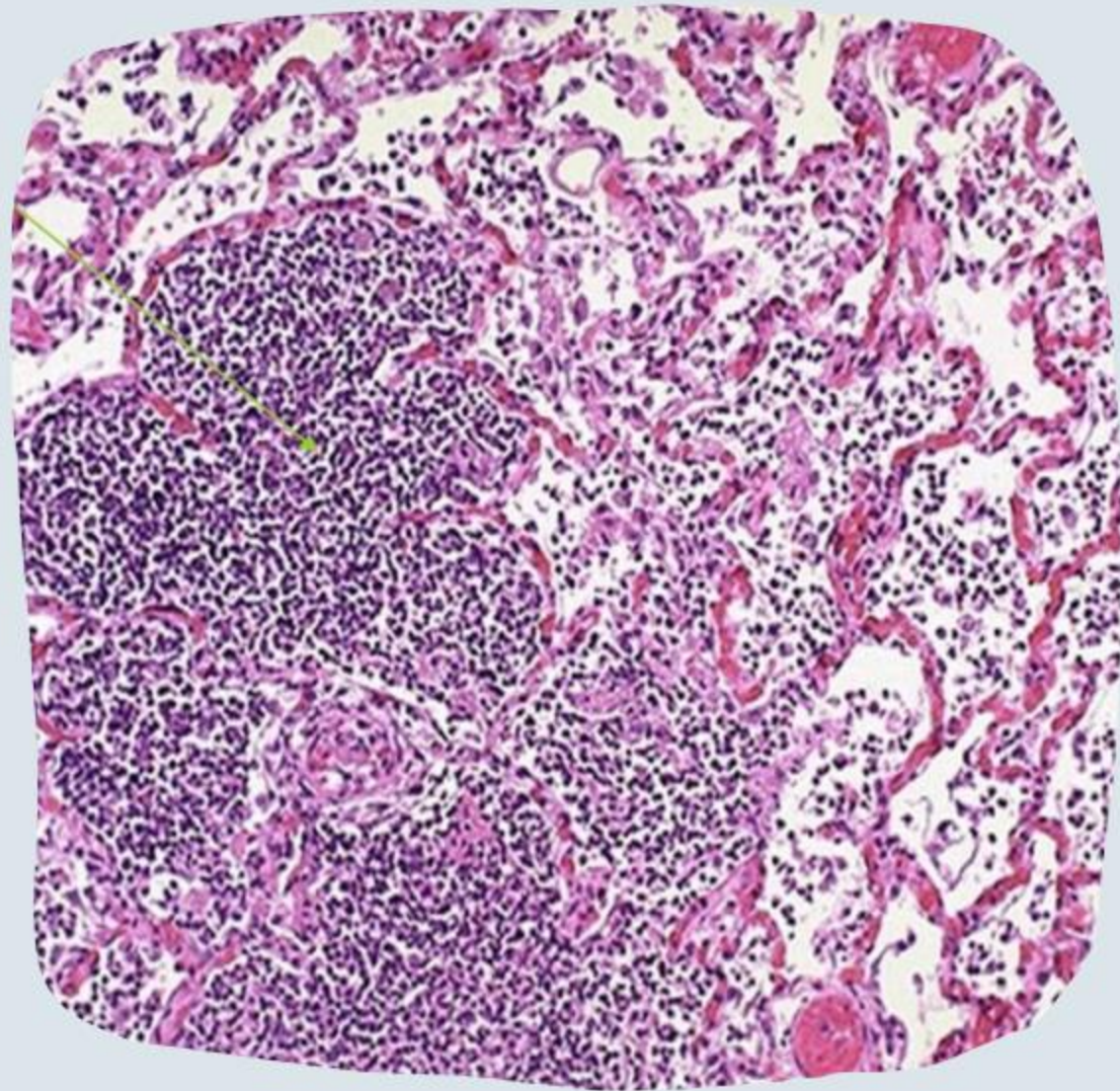


# ***Bronchopneumonia***

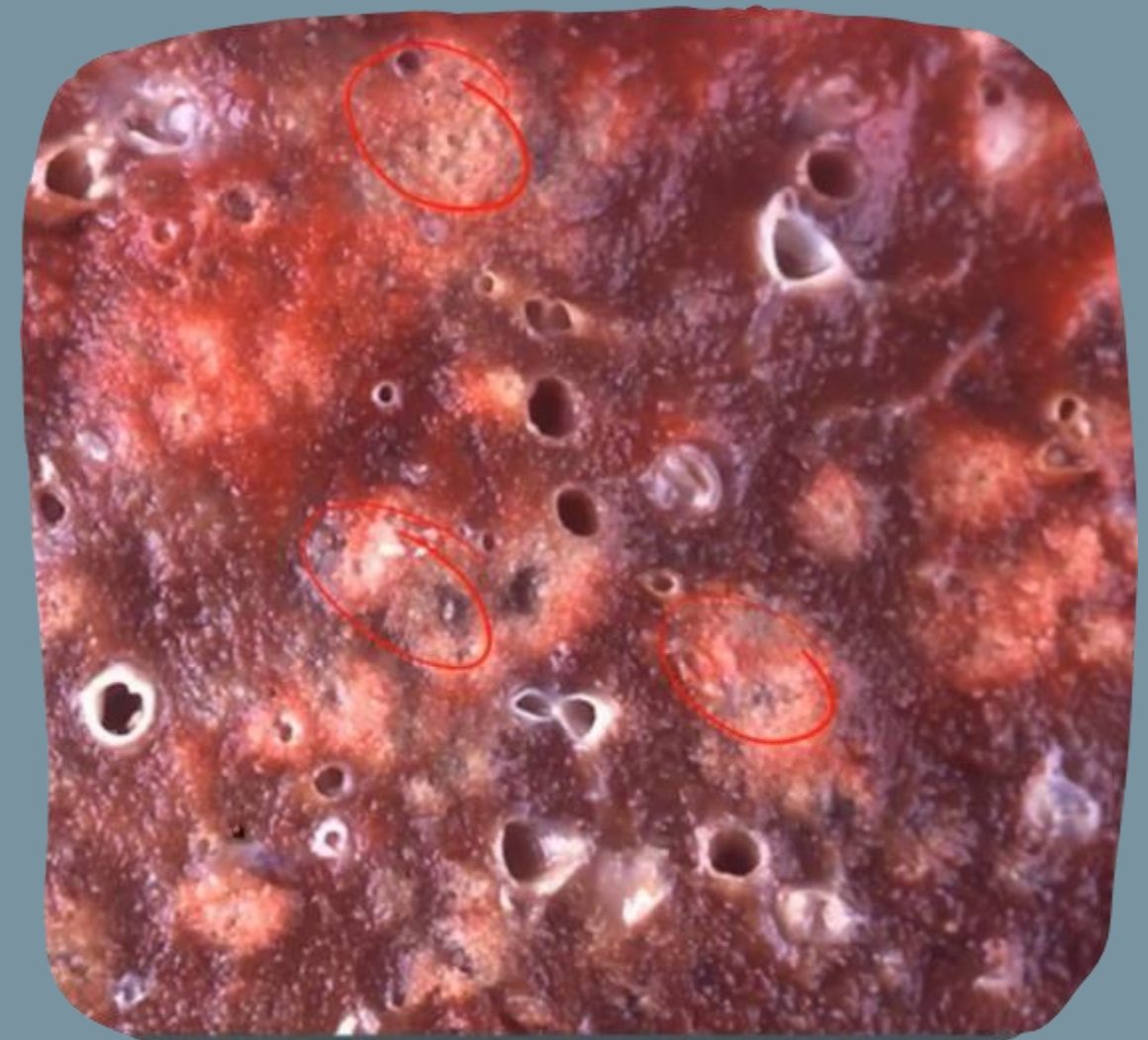
- Acute inflammatory infiltrates from bronchioles into adjacent alveoli; patchy distribution involving  $\geq 1$  lobe .
- So Characterized by scattered patchy consolidation centered around bronchioles neutrophil-rich exudate fills the bronchi, bronchioles, and adjacent alveolar spaces.
- Often multifocal , multilobar and bilateral .
- Caused by a variety of bacterial organisms such as staph aureus



# ***Bronchopneumonia***



LM



GROSS

# ***Interstitial***

- Characterized by diffuse interstitial infiltrates localized to interstitial areas at alveolar walls without intra alveolar infiltration
- there is no consolidation and its finding
- Presents with relatively mild upper respiratory symptoms ,atypical, presentation (“walking” pneumonia).
- Caused by bacteria or viruse , but the most common cause is m.pneumonia
- usually bilateral multifocal



# *Interstitial*

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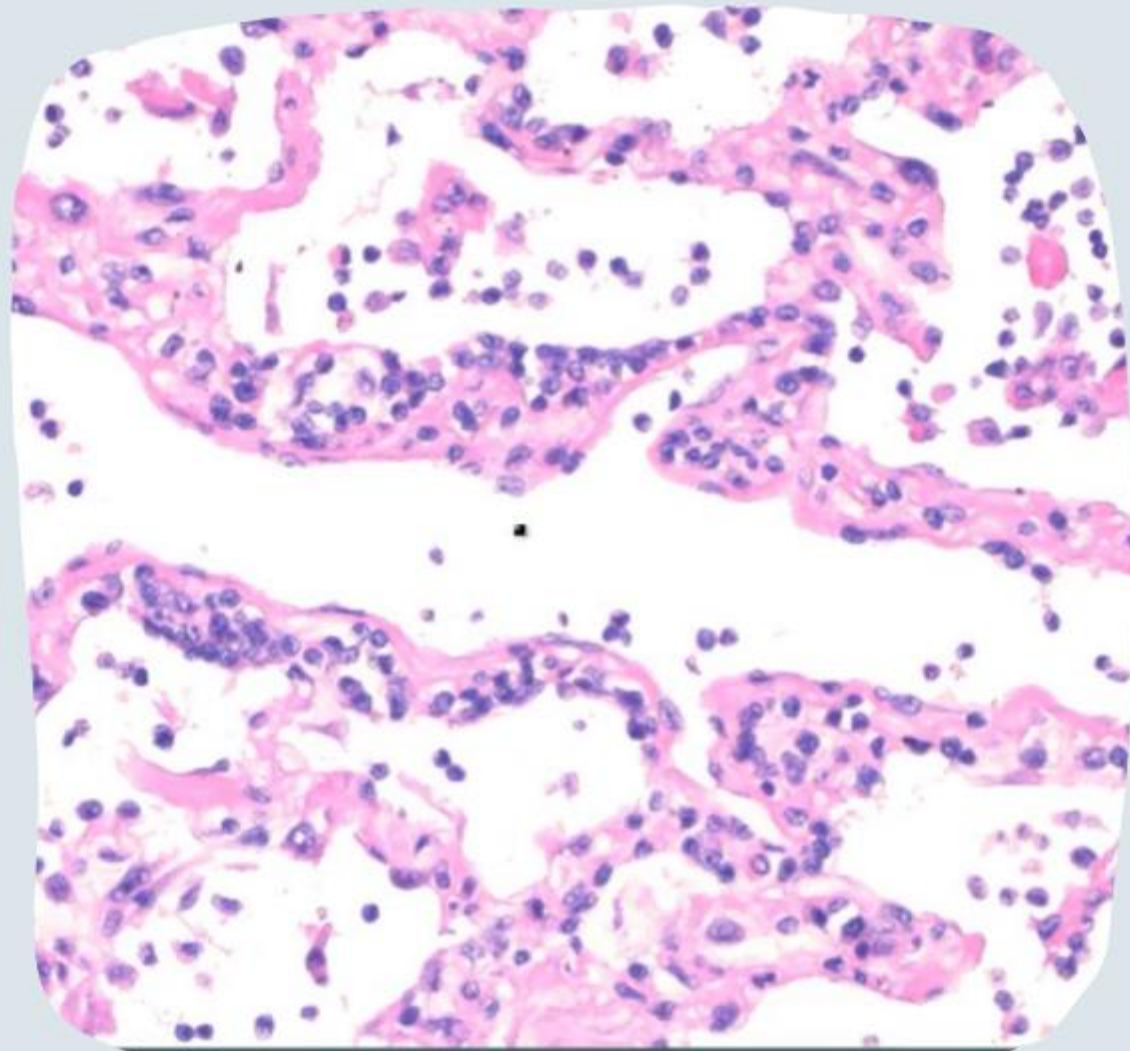
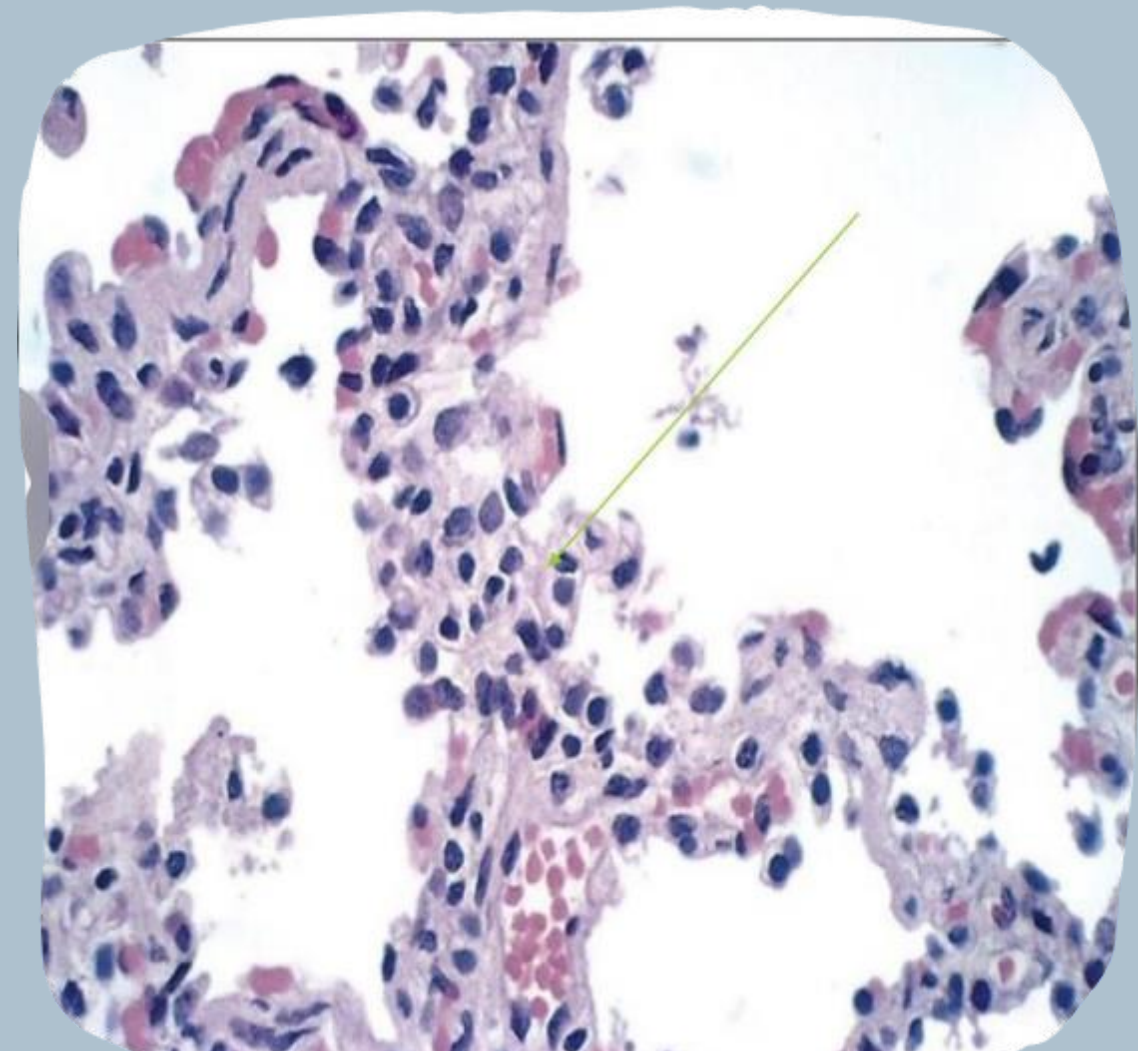


Fig. 9.5B

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GROSS



# ***Clinical classification :***

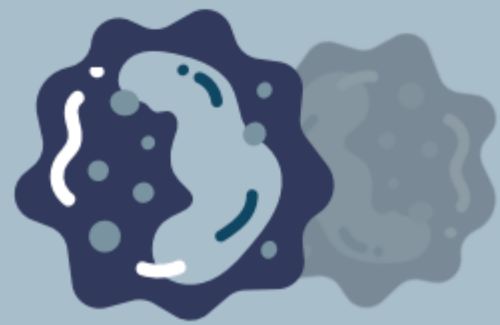
Classification according to clinical setting will considerably narrows the list of suspected pathogens for administering empirical antimicrobial therapy

## COMMUNITY AQCUIRED

TYPICAL

ATYPICAL

ONSET OUTSIDE HOSPITAL  
OR WITHIN 72 HOURS  
AND THE PATIENT IS NOT  
IMMUNOCOMPROMISED



## NOSOCOMIAL

HOSPITAL  
AQCUIRED

VENTILATOR  
ASSOCIATED

ONSET >72  
HOURS AFTER  
HOSPITALIZATI  
ON

ONSET  
>48HOURS  
AFTER  
VENTILATION



## OTHERS

ASPIRATION

IMMUNO  
COMPROMISE  
D

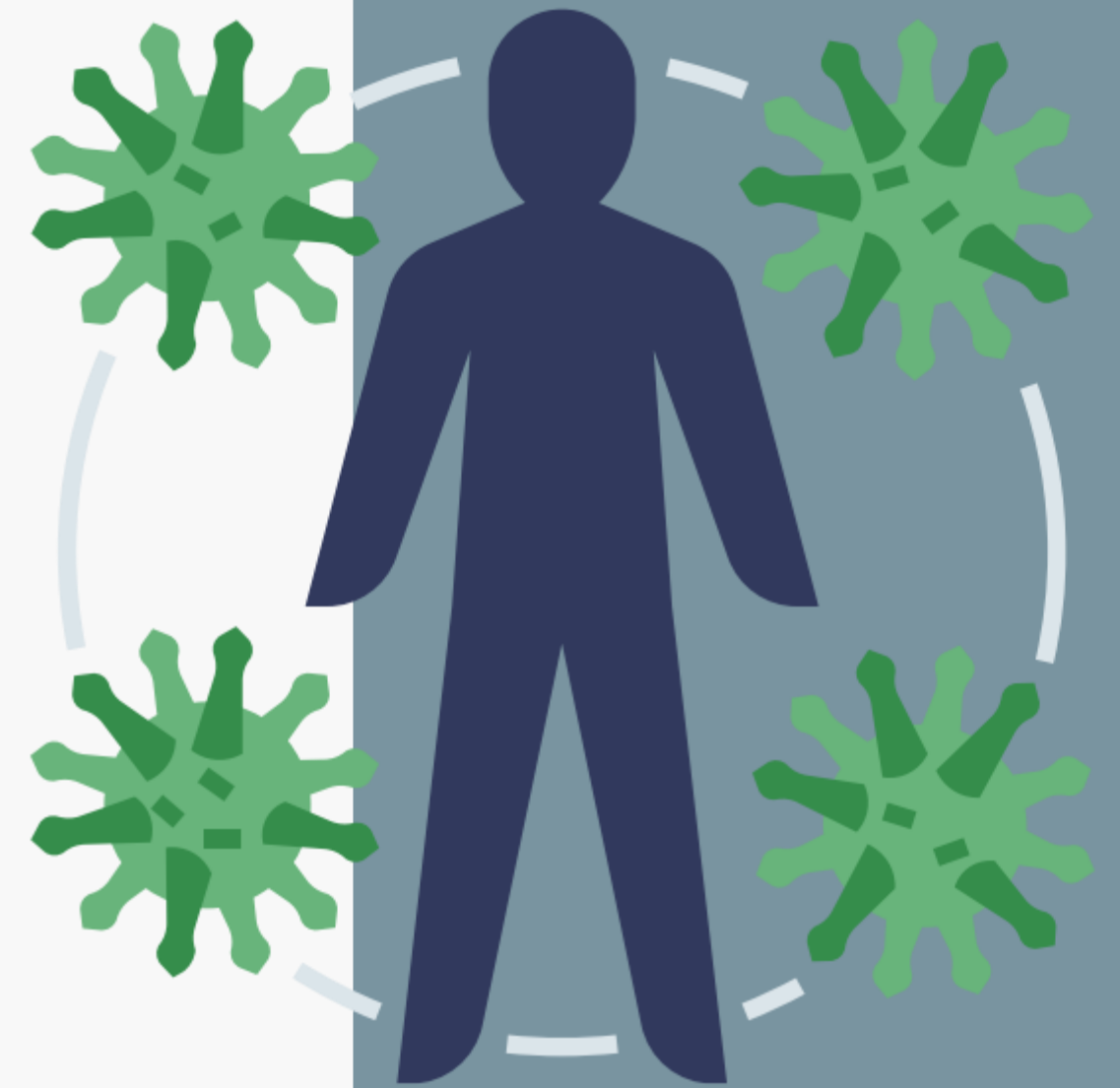
COMING  
SOON

COMING  
SOON



# *Community-acquired*

- community-acquired pneumonia in a person with no underlying immunosuppression or malignancy.
- occurs across all ages but is more common at the extremes of age.
- *Streptococcus pneumoniae* is the most common cause overall; however, in 30-50% of cases no organism is identifiable, while in about 20% more than one organism is present.



# ***Factors that increase the risk of developing CAP: . . . .***

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- Age: <16 or >65 years
- Co-morbidities:

HIV infection, diabetes mellitus, chronic kidney disease, malnutrition, recent viral respiratory infection

- Other respiratory conditions:

cystic fibrosis, bronchiectasis, chronic obstructive pulmonary disease, obstructing lesion (endoluminal cancer inhaled foreign body)

- Lifestyle: cigarette smoking, excess alcohol, intravenous drug use
- Iatrogenic: immunosuppressant therapy (including prolonged corticosteroids)



## CAP classification according type of agent:

1- typical pneumonia

2-atypical pneumonia

Typical pneumonia:

**\*\*common agents**

*S pneumoniae* (60%)

*Haemophilus influenzae* (15%)

Aerobic gram-negative rods

(6% to 10%) - *Klebsiella* (and

other *Enterobacteriaceae*)

*S. aureus* (2% to 10%)

| SYMPTOMS                                         | SIGNS                                                                                                              |
|--------------------------------------------------|--------------------------------------------------------------------------------------------------------------------|
| 1 -Acute onset of fever and shaking chills       | 1 -Tachycardia                                                                                                     |
| 2-cough productive of thick, purulent sputum     | 2-tachypnea                                                                                                        |
| 3-pleuritic chest pain suggests pleural effusion | 3-Late inspiratory crackles, bronchial breath sounds, increased tactile and vocal fremitus, dullness on percussion |
| 4-Dyspnea                                        | 4-pleural friction rub (associated with pleural effusion)                                                          |

## CAP classification according type of agent:

1 - typical pneumonia

2-atypical pneumonia

### ATYPICAL PNEUMONIA:

**\*\*common agents:**

Mycoplasma pneumoniae (most common)

chlamydia pneumoniae

chlamydia psittaci

coxiella burnetii (Q fever)

Legionella spp

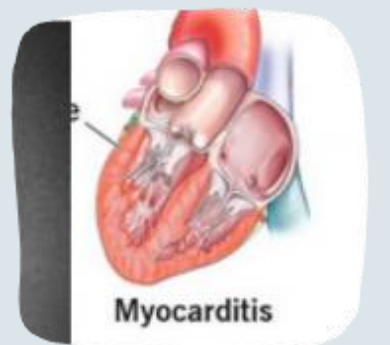
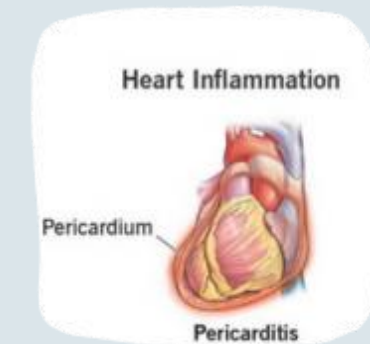
viruses: influenzavirus (ASB),

adenoviruses, parainfluenzavirus, RSV.

| SYMPTOMS                                                           | SIGNS                                                                                                                  |
|--------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------|
| 1 -Insidious onset –<br>headache,sore throat,<br>fatigue, myalgias | 1 -pulse-temperature<br>dissociation – normal<br>pulse in the sitting of<br>high fever is suggestive<br>of atypicalcAP |
| 2-Dry cough ( No sputum<br>production)                             | 2-wheezing,crackles,<br>rhonchi                                                                                        |
| 3-Fever ( chills are<br>uncommon )                                 |                                                                                                                        |

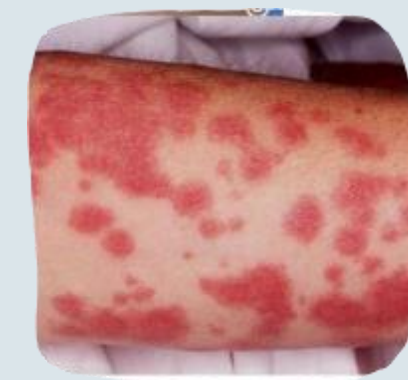
Extrapulmonary features of community-acquired pneumonia:

- 1 ► Myalgia, arthralgia and malaise are common, particularly in infection caused by Legionella and Mycoplasma
- 2 ► Myocarditis and pericarditis are cardiac manifestations of infection, most commonly in Mycoplasma pneumonia
- 3 ► Headache is common in Legionella pneumonia.  
(Meningoencephalitis and other neurological abnormalities also occur but are much less common)



## Extrapulmonary features of community-acquired pneumonia

- 4 ▶ Abdominal pain, diarrhea and vomiting are common.  
(Hepatitis can be a feature of Legionella pneumonia)
- 5 ▶ Labial herpes simplex reactivation is relatively common in pneumococcal pneumonia.
- 6 ▶ Other skin rashes, such as erythema multiforme and erythema nodosum, are found in Mycoplasma pneumonia.
- 7 ▶ (Stevens-Johnson syndrome is a rare and potentially life-threatening complication of pneumonia)



# *Nosocomial Pneumonia*

## ► Hospital-acquired (HAP):

- onset > 72 hours after hospitalization

## ► ventilator-acquired (VAP):

- onset > 48 hours after ventilation

More SERIOUS !

- 1 - sick patients have weakened immune systems
- 2 - microbe in hospital are more resistant to antibiotics





► **HAP is the second most common form of hospital-acquired infection after urinary tract and carries a significant mortality risk, particularly in the elderly or those with co-morbidities such as stroke, respiratory disease or diabetes.**

► **This is defined as new onset of cough with purulent sputum along with a compatible x-ray demonstrating consolidation, in patients who are beyond 2 days of their initial admission to hospital or who have been in a healthcare setting within the last 3 months (including nursing/residential homes as well as acute care facilities such as hospitals).**





► In HAP, the causative organisms differ from those causing CAP . viral or fungal pathogens are not responsible in immunocompetent hosts.



#### Box 15.10 Organisms implicated in hospital-acquired pneumonia

- Gram-negative bacteria (*Pseudomonas* spp., *Escherichia* spp., *Klebsiella* spp.)
- Anaerobic bacteria (*Enterobacter* spp.)
- *Staphylococcus aureus* (including MRSA)
- *Acinetobacter* spp.



- 
- 
- ▶ Aerobic Gram-negative bacilli are commonly involved (e.g. *P. caeruginosa*, *E. coli*, *klebsiella pneumoniae* and *Acinetobacter* species)
  - ▶ **Staphylococcus aureus is increasingly recognized in HAP, particularly MRSA** . HAP due to *S. aureus* is more common in patients with diabetes mellitus, head trauma, and patients in intensive care units.
  - ▶ **Other conditions should be excluded, including aspiration of gastric contents due to impaired swallowing or bulbar weakness**





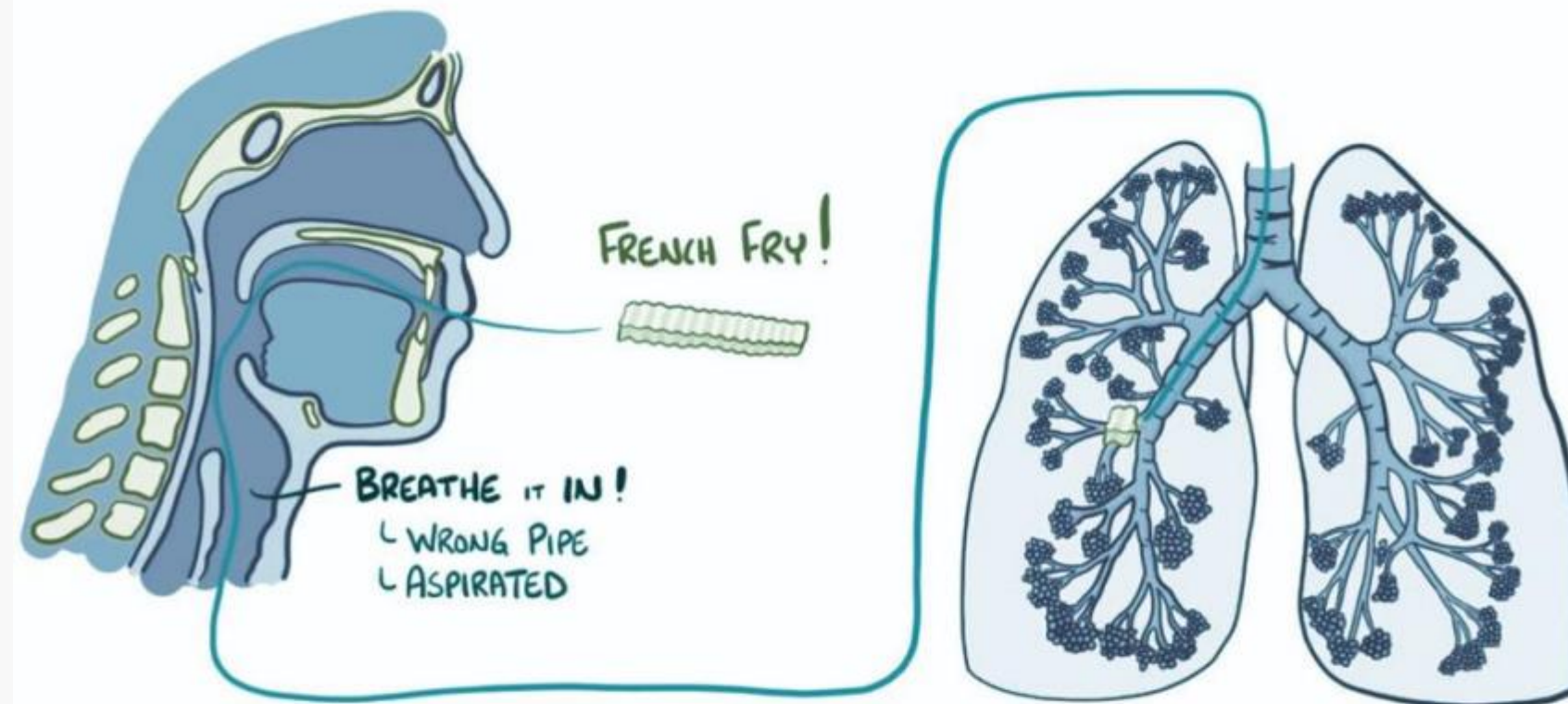
► **VAP** is not much different from **HAP**, except that patients are on a ventilator. The organisms are much the same, except **MDR** organisms are more frequently involved, and patients are more sick.



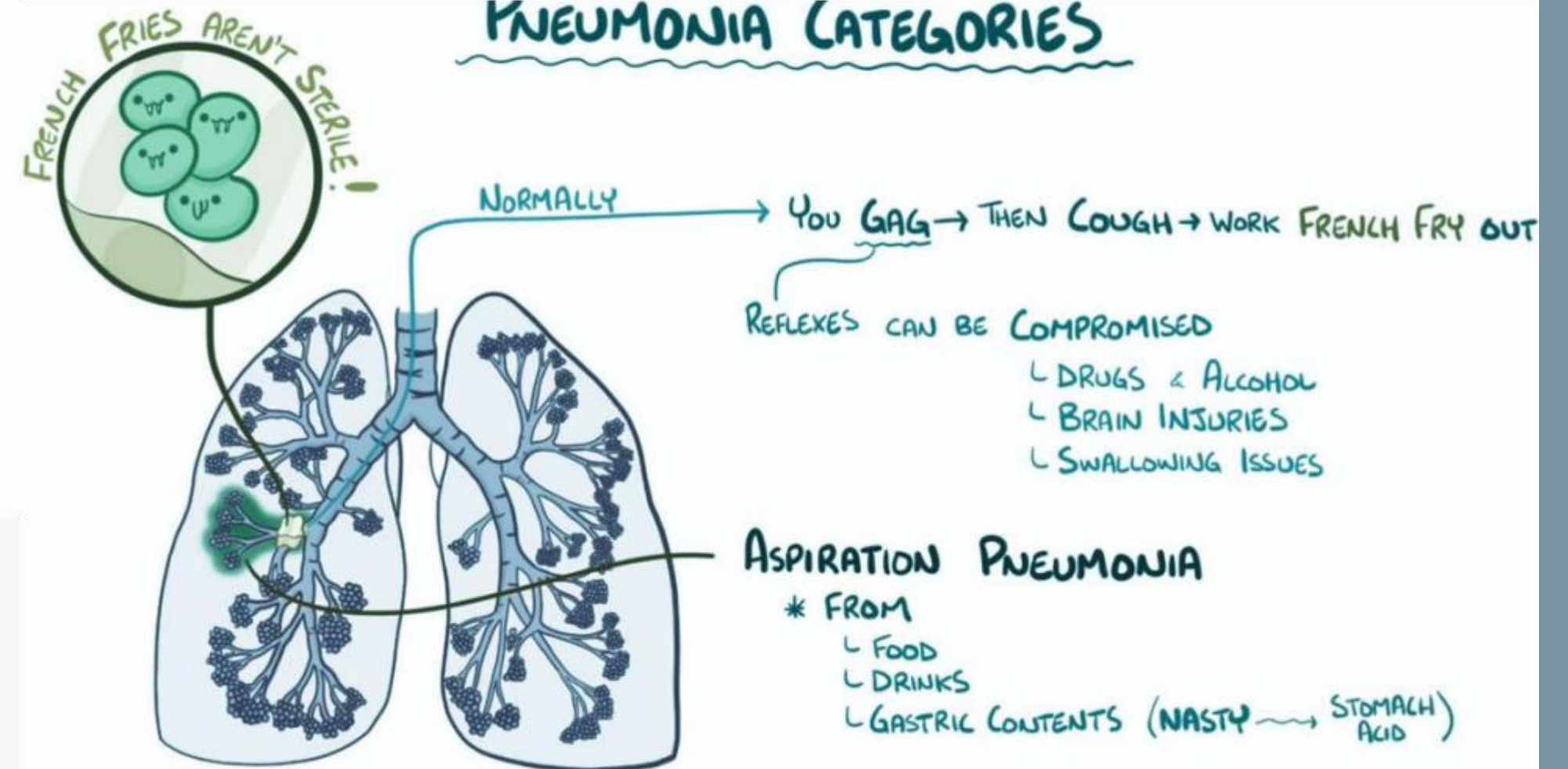
# Aspiration pneumonia



## PNEUMONIA CATEGORIES



## PNEUMONIA CATEGORIES



# Aspiration pneumonia

It is due to inhalation of oropharyngeal secretions colonized by pathogenic bacteria

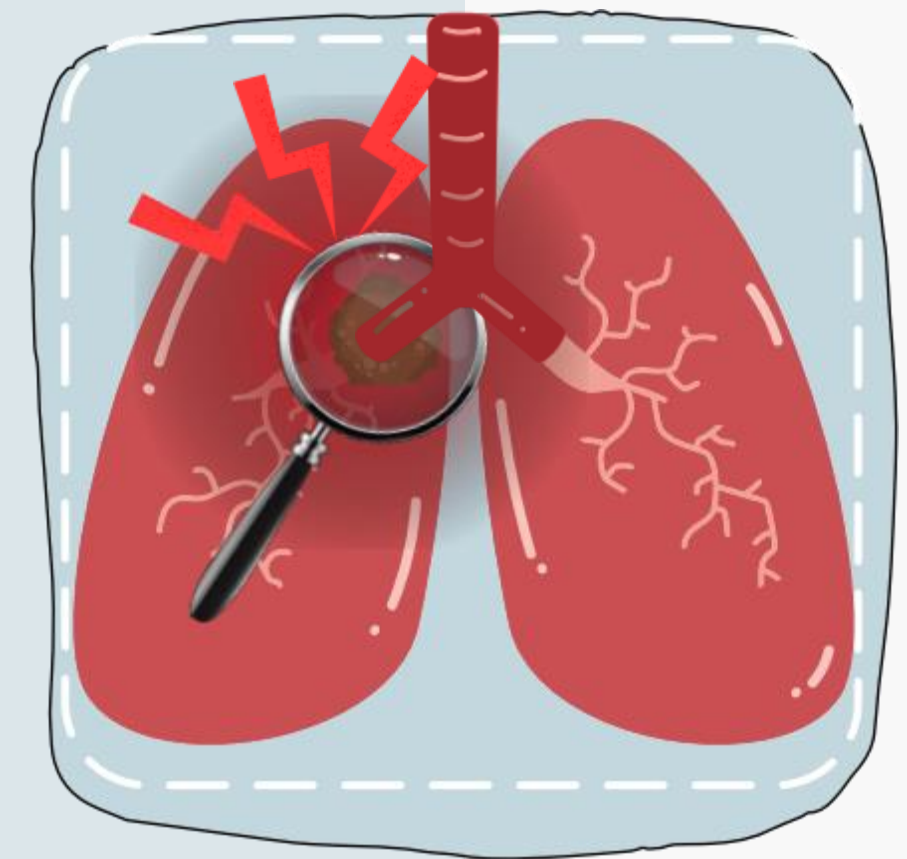
patients usually present with indolent symptoms (days to weeks), foul-smelling sputum, and concurrent periodontal disease.

## Risk factors:

Reduced consciousness (anesthesia)  
seizures

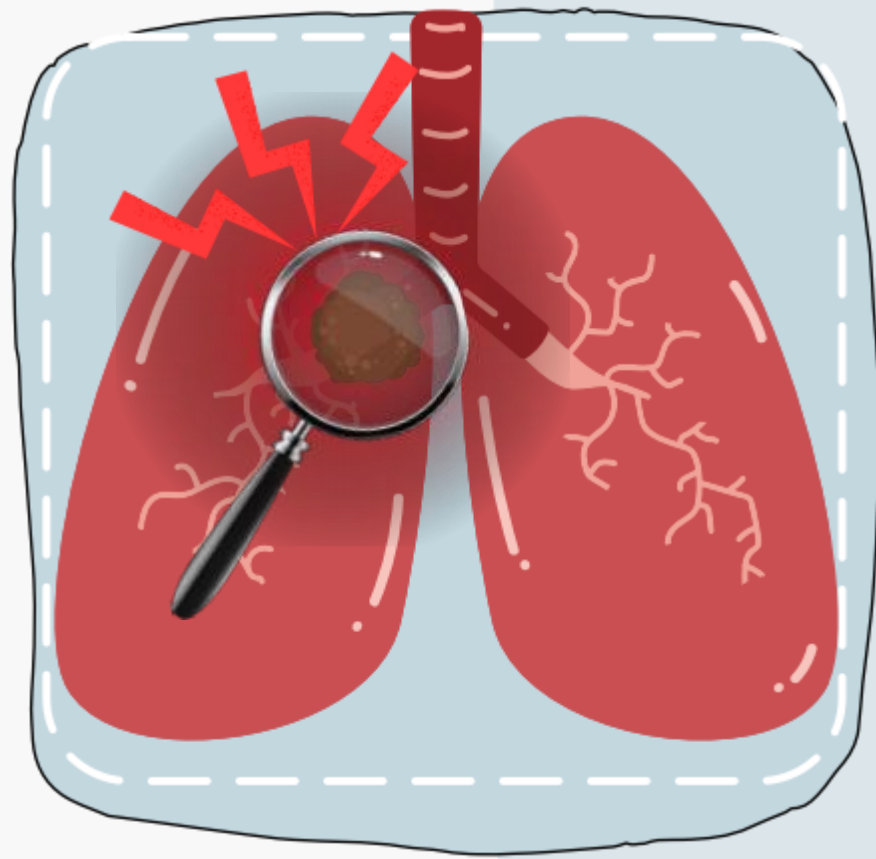
Heavy alcohol use

Dysphagia from neuromuscular weakness



Acute aspiration of gastric contents into the lungs can produce an extremely severe and sometimes fatal illness; owing to the intense destructiveness of gastric acid.

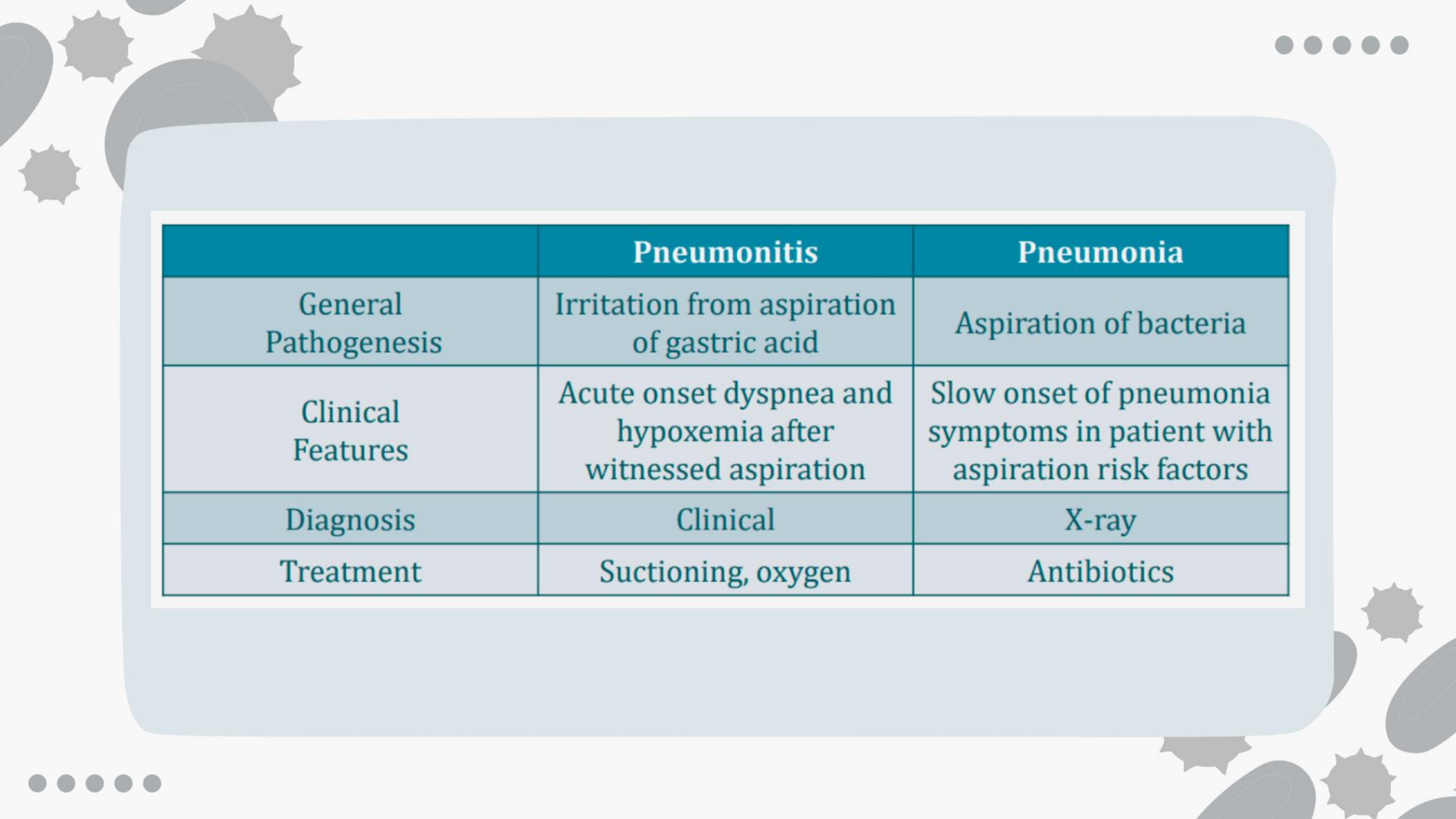
Because of the bronchial anatomy, the most usual sites for aspirated material to end up are the right middle lobe and apical or posterior segments of the right lower lobe



► It is vital to identify any underlying problem, since without appropriate corrective measures aspiration will recur.

► The infectious organisms are frequently oral flora (mixed aerobic and anaerobic).

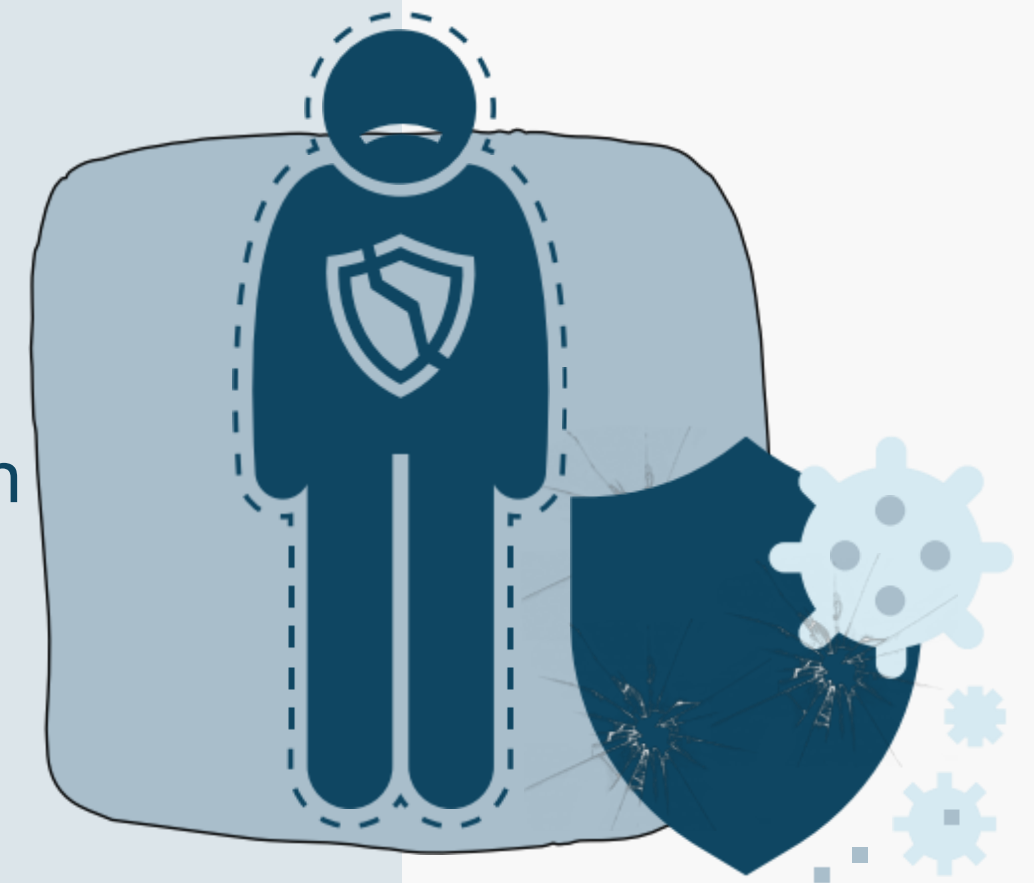
EX: . klebsiella · staph Aureus o  
preyotella . Bacteroides



|                      | <b>Pneumonitis</b>                                           | <b>Pneumonia</b>                                                         |
|----------------------|--------------------------------------------------------------|--------------------------------------------------------------------------|
| General Pathogenesis | Irritation from aspiration of gastric acid                   | Aspiration of bacteria                                                   |
| Clinical Features    | Acute onset dyspnea and hypoxemia after witnessed aspiration | Slow onset of pneumonia symptoms in patient with aspiration risk factors |
| Diagnosis            | Clinical                                                     | X-ray                                                                    |
| Treatment            | Suctioning, oxygen                                           | Antibiotics                                                              |

# *Pneumonia in Immunocompromised*

- ▶ Immunosuppressed people are either iatrogenically or due to a defect in host defenses,
- ▶ EX: those on immunosuppressant therapy such as long-term corticosteroids, monoclonal antibody therapy or methotrexate for autoimmune disease, those on anti-rejection medication post-solid organ transplantation or following stem cell transplantation, and patients infected with HIV.
- ▶ They are at risk not only from all the usual organisms which can cause pneumonia, but also opportunistic pathogens which would not be expected to cause disease.
- ▶ The symptom pattern may resemble cAP Or be more nonspecific.



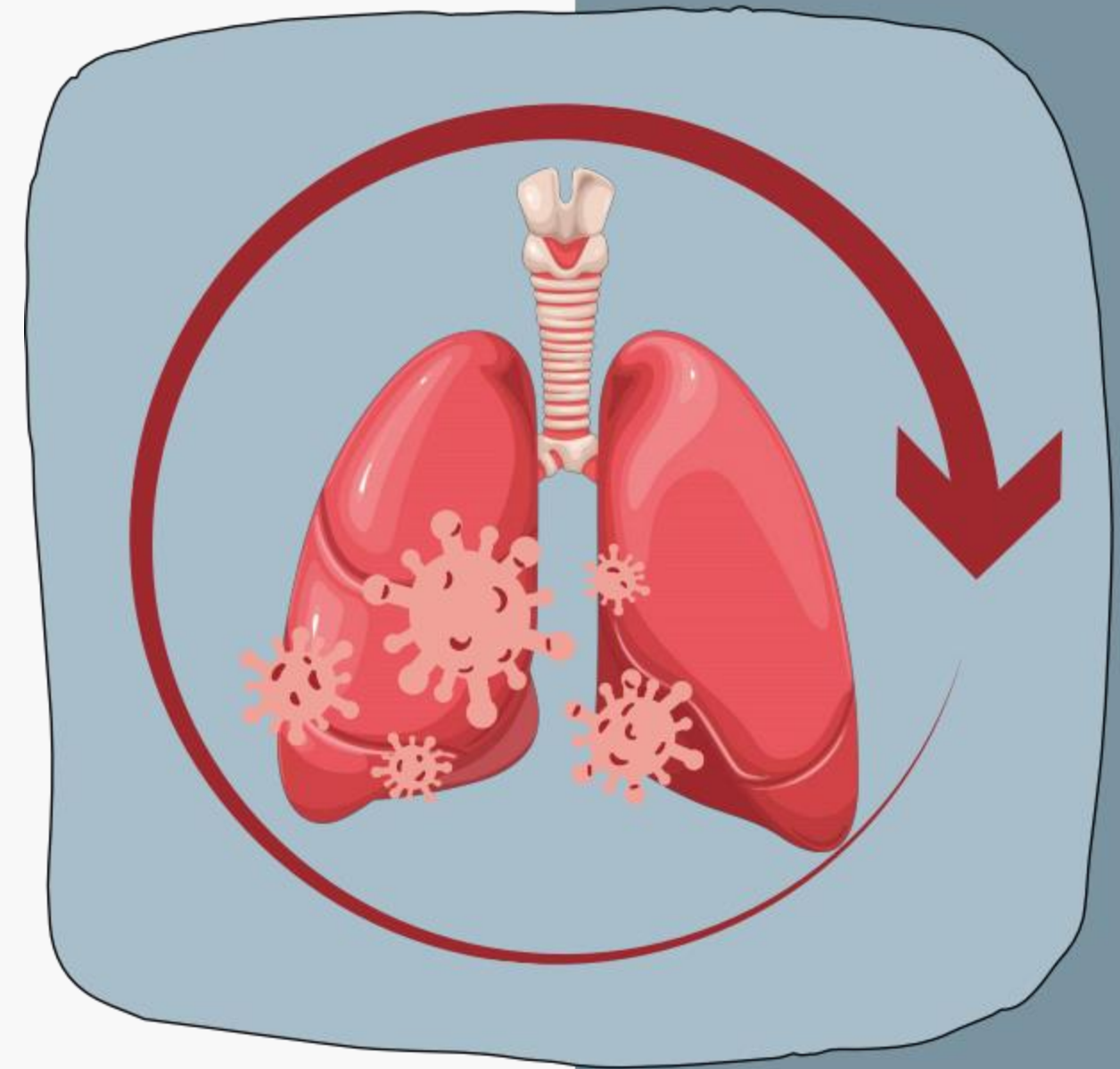
# *Pneumocystis jiroveci*

- ▶ Pneumocystis pneumonia (PCP), caused by the fungal organism now called *Pneumocystis jiroveci*, is an AIDS-defining illness.
- ▶ usually occurs when the CD4 count is  $<200/\text{mm}^3$ .
- ▶ The diagnosis should be considered in any patient with HIV who has dry cough, exertional dyspnea, and fever.
- ▶ Chest x-ray usually shows bilateral interstitial infiltrates.
- ▶ The diagnosis is confirmed by demonstration of the organism in sputum or bronchoalveolar lavage.
- ▶ Trimethoprim-sulfamethoxazole (TMP-SMX) is the initial drug of choice.



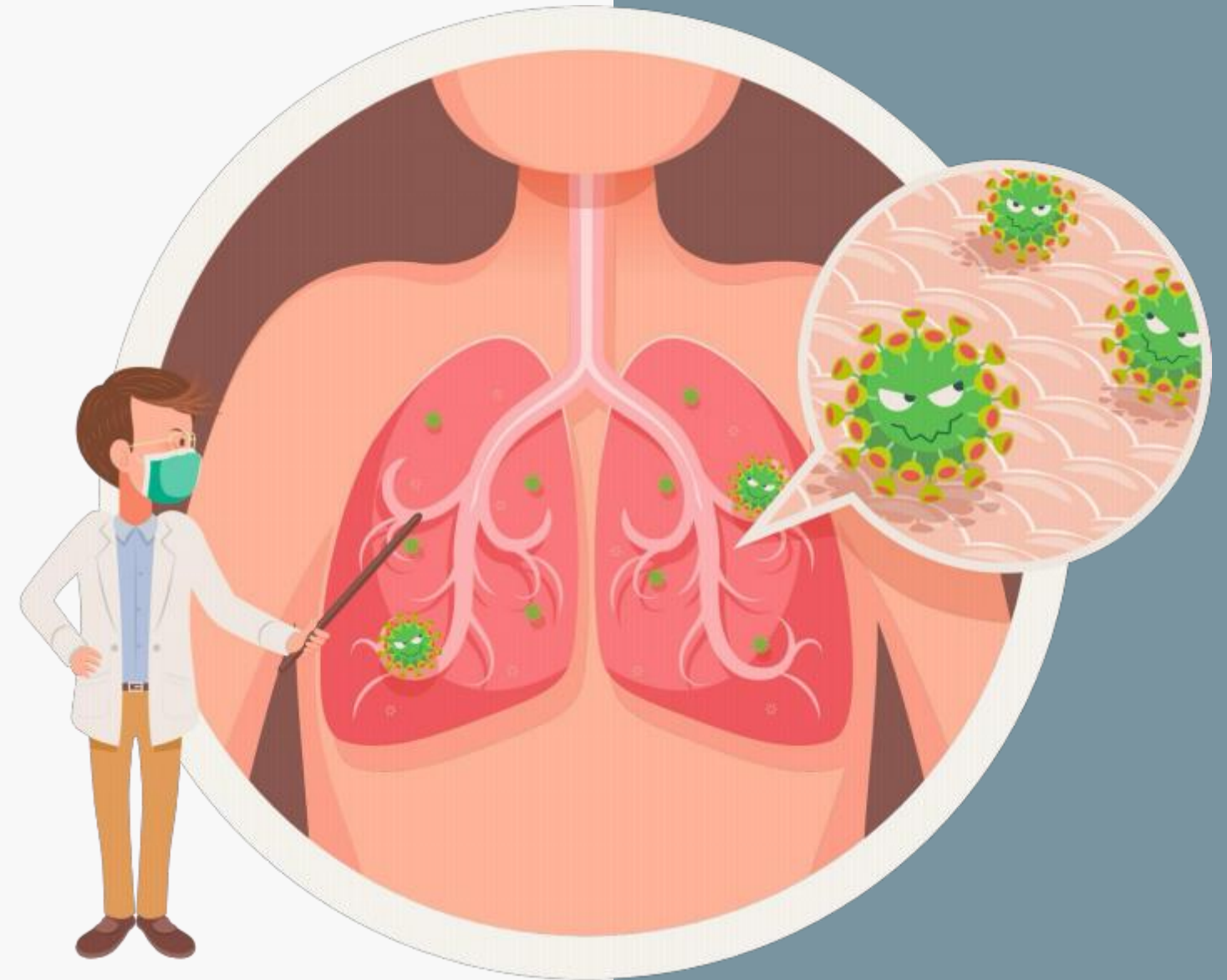
# *Recurrent Pneumonia*

- ▶ Different lung regions:  
Immunodeficiency (HIV, cystic fibrosis)
- ▶ Same lung region:  
Airway obstruction (e.g., malignancy)  
Bronchiectasis
- ▶ Recurrent right middle/lower lobe  
Aspiration



# *Acute bronchitis*

- Viral infection
- Often follows a URI (nasal congestion, sore throat)
- Persistent cough > 5 days
- Blood-tinged sputum
- Normal chest x-ray
- Self-limited
- Supportive treatment
- No antibiotics



# **initial assessment**

When patient comes to hospital we start with 3 step

## 1. History

- patient has classical sign and symptoms of pneumonia like productive cough, pleuritic chest pain, fever, and Dyspnea

## 2. Physical examination:

sign of respiratory distress

dullness on percussion due to consolidation

Bronchial breathing sound , late inspiration crackles

Brochophony and Egophony

## 3. Investigation



# *initial assessment*

.....

*The type and extent of investigation depends on the severity of the illness, which also guides where the patient should be managed and predicts their outcome..*

Severity is commonly assessed by CURB-65 score

C: confusion present (abbreviated mental test score  $<8/10$ )

U: (plasma) urea level  $>7$  mmol/L

R: respiratory rate  $>30/\text{min}$

B: systolic BP  $<90$  mmHg; diastolic BP  $<60$  mmH

65: age  $>65$



.....

# *initial assessment*



1 point for each of the above:

Score 0-1: Treat as outpatient

Score 2: Admit to hospital

3+: Often require ICU care

Mortality rates increase with increasing score.



The CRB-65 score is used in the community where the serum urea level is not usually available



# *investigation*

## **1.Chest x-rays**

*PA and lateral CXR required to confirm the diagnosis*

In Typical CAP:

X ray >> Lobar consolidation

\*Multilobar consolidation indicates very serious illness

In Atypical CAP :

Changes include interstitial infiltrates ,lobar consolidation , and/or cavitation.

in Atypical CAP Because the inflammation is often limited to the pulmonary interstitium and the interlobular septa, atypical pneumonia has the radiographic features of patchy reticular or reticulonodular opacities



Atypical CAP:



Typical CAP:

# ***initial assessment***

• •

• •

**In HAP >> we see Areas of unilateral or bilateral consolidation may be seen depending on severity.**



• • • • •

# *initial assessment*

. . . .



▪

## ▶ **2. Blood tests:**

### ▶ Full blood count

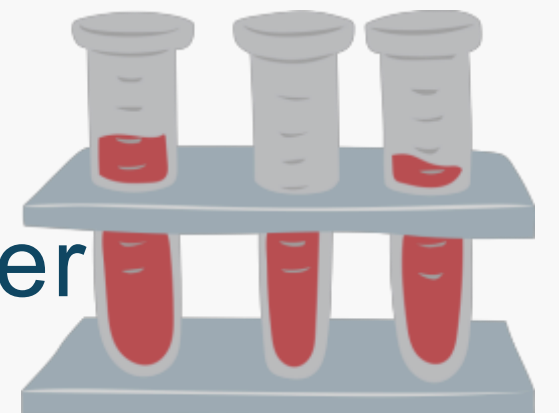
urea and electrolytes

biochemistry and C-reactive protein.

**Strep. pneumonia** : White cell count is  $>15 \times 10^9/L$  (90% polymorphonuclear leucocytosis) , inflammatory markers significantly elevated: ESR  $>100$  mm/hour; CRP  $>100$  mg/L.

**Mycoplasma**. White cell count is usually normal , Haemolytic anaemia: occasional complication of Mycoplasma

**Legionella**. There is lymphopenia without marked leucocytosis, hyponatraemia, hypoalbuminaemia and high serum levels of liver aminotransferases.



# *initial assessment*

...

## 3. Other tests:

- to identify the causative organism Sputum culture and Gram-stain are required

*S. pneumoniae*: Gram-positive diplococci

*S. aureus*: Gram-positive organisms commonly in clusters like a bunch of grapes

- Pulse oximetry and arterial blood gas analysis is necessary if oxygen saturation is below 94%.

- HIV testing: since pneumonia is a common initial presenting illness in patients with previously undiagnosed HIV infection



.....

# Pneumonia



## 1. PLEURAL EFFUSION & RESPIRATORY FAILURE *Complications*

### *Parapneumonic effusion*

*simple exudative effusion*

remember : Exudative: high protein | high LDH | low glucose

incidence among pneumonia patients?      Around **1/3** ...

Early indications of empyema are :  
**fever**, elevated **inflammatory markers**. , despite appropriate antibiotic therapy..

In severe scenarios,  
this serious complication could put you at risk of acute respiratory distress (ARDS),  
respiratory failure, and hospitalization( it can also occur if there is a diffuse damage to  
alveoli by spreading of bacteria )

### *Empyema*

*Exudative Filled with*

***pus***

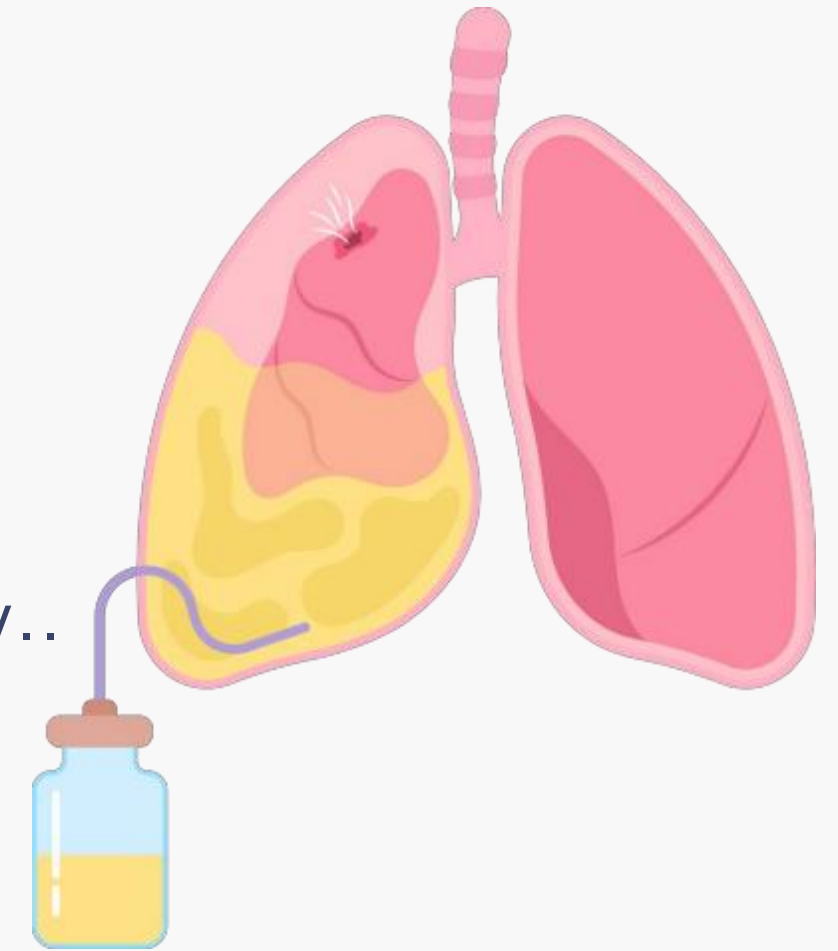




Figure 1: Left Empyema. Case courtesy of Dr Ian Bickle, Radiopaedia.org, riD: 74921



Figure 2: R Pleural Effusion with meniscus sign. Case courtesy of Dr Ian Bickle, Radiopaedia.org, riD: 50327



## 2. LUNG ABSCESS

WHEN PUS FORMS IN A LUNG CAVITY, AN ABSCESS CAN OCCUR , OCCUR MAINLY IN **ASPIRATION PNEUMONIA** CAUSING AGENTS ( **ANAEROBES**).

THIS IS A COMMON AND SERIOUS PNEUMONIA COMPLICATION THAT CAN SIGNIFICANTLY IMPACT OLDER ADULTS IF NOT TREATED.

A LUNG ABSCESS CAN ALSO LEAD TO THE RARER PNEUMONIA COMPLICATION OF LUNG NECROSIS (NECROTIZING **PNEUMONIA**),WHICH IS A CONSEQUENCE OF SEVERE LUNG INFLAMMATION THAT RESULTS IN **CRITICAL LUNG TISSUE DAMAGE**

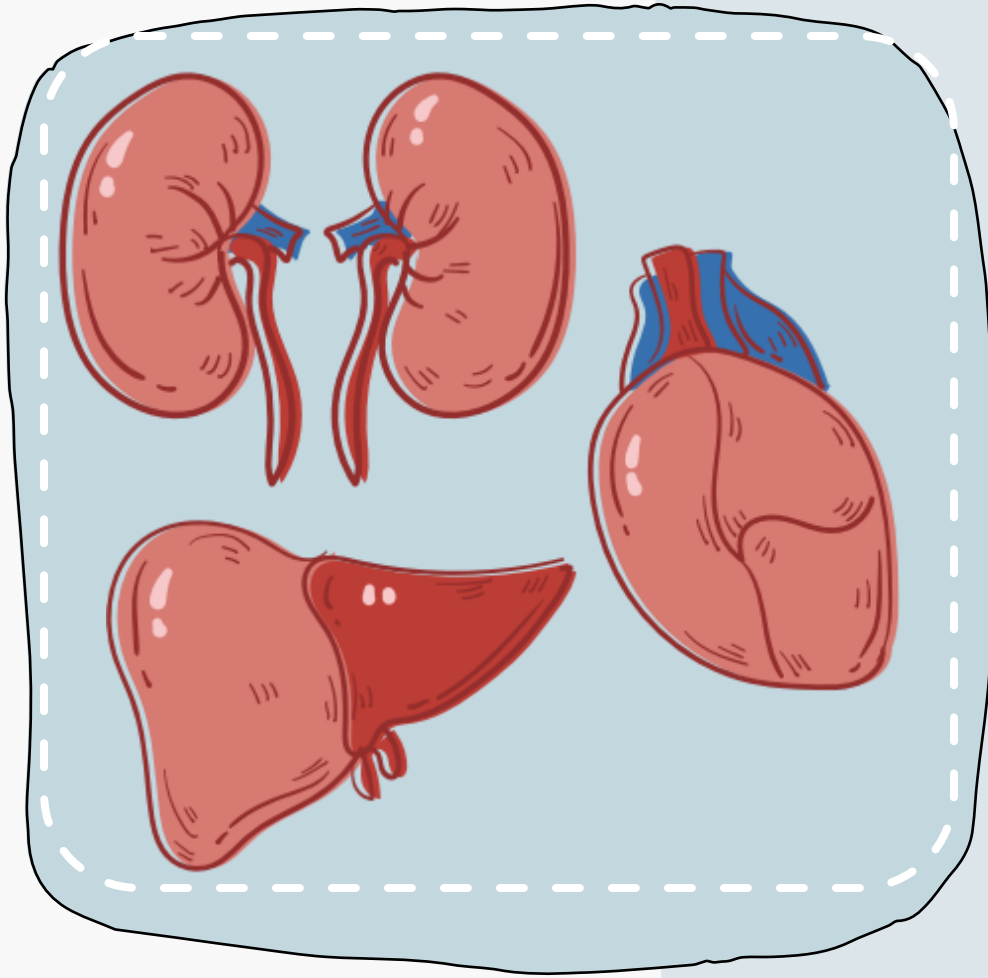
## 2. LUNG ABSCESS

The most common is **anaerobic bacteria** :



### Box 15.9 Common causative organisms in lung abscess

- *Klebsiella pneumonia*
- *Staphylococcus aureus*
- Gram-negative enteric bacilli
- *Mycobacterium tuberculosis*
- *Streptococcus milleri*
- Anaerobic bacteria (post aspiration)
- *Haemophilus influenzae*

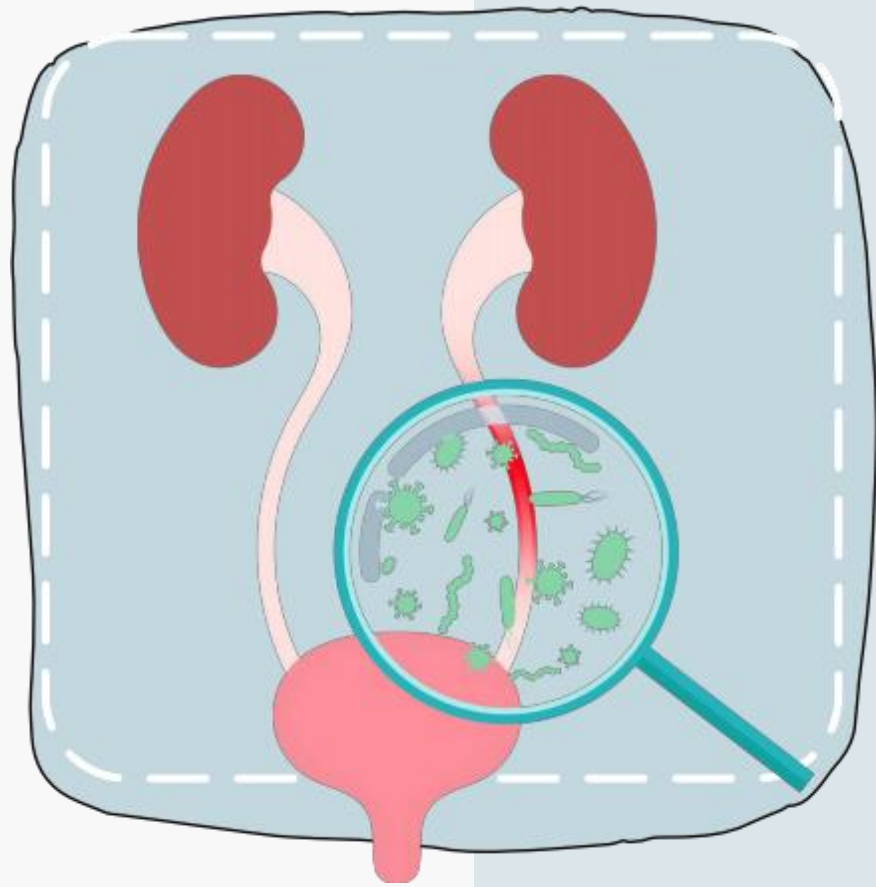


### 3.KIDNEY,LIVER & HEART DAMAGE

YOUR BODY NEEDS OXYGEN TO FUNCTION. FOR OLDER ADULTS, MORE COMMONLY THOSE WITH UNDERLYING CHRONIC HEALTH CONDITIONS, PNEUMONIA CAN CAUSE SEVERE SHORTNESS OF BREATH AND OTHER RESPIRATORY COMPLICATIONS THAT RESTRICT OXYGEN TO THE LUNGS. THIS COMPLICATION CAN CAUSE ORGAN DAMAGE TO THE KIDNEYS, LIVER, AND HEART, WHICH IS WHY IT'S SO IMPORTANT FOR SENIORS TO GET PROPERLY DIAGNOSED AND TREATED FOR PNEUMONIA AS SOON AS POSSIBLE.

#### 4.urinarytract infections

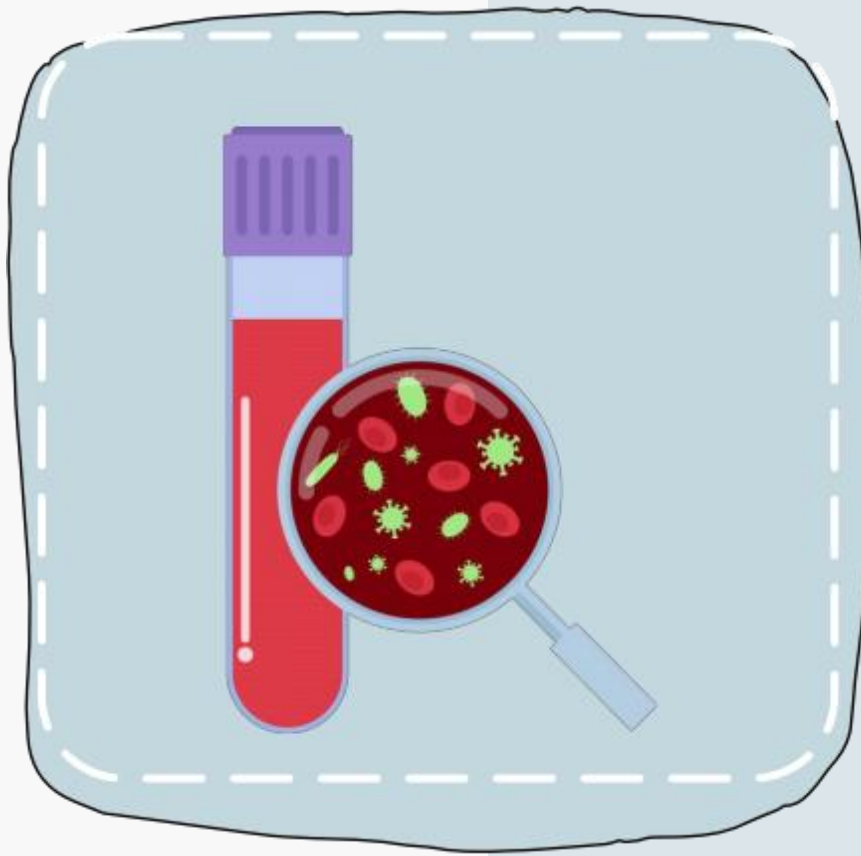
A UTI typically occurs when harmful bacteria invade the kidneys, ureter, bladder, or urethra. since our immune systems weaken with age, most older adults cre already at increased risk for urinary tract infections. A UTI can also be a pneumonia complication, since some types of bacteria that induce the lung infection can spre cad to other areas of the body, like the urinary tract.



## 5.sepsis

sepsis is a particularly dangerous pneumonia complication.

A life-threatening condition, it can lead to tissue damage, organ failure, and death if left untreated. Those with chronic health problems or compromised immune systems are at higher risk of developing sepsis from an infection like pneumonia. Adults over 65, however, are 13 times more likely to be hospitalized with sepsis than any other group.



# *General management*

1. **Oxygen. Supplemental** oxygen should be administered to maintain saturations between **94% and 98%** provided the patient is not at risk of carbon dioxide retention.

\*(due to loss of hypoxic drive in **COPD**. In patients with known COPD, oxygen saturations should be maintained between **88% and 92%**; normally with controlled oxygen via fixed percentage delivery mask)

2. **Intravenous fluids.** Required in hypotensive patients showing any evidence of volume depletion.

3. **Thromboprophylaxis.** If admitted for >12 hours subcutaneous low molecular weight **heparin** should be prescribed unless contraindications exist and **TED** (thromboembolus deterrent) stockings fitted.

4. **Physiotherapy.** Chest physiotherapy is not needed unless sputum retention is an issue



5. Nutritional supplementation. The need for this is assessed, particularly in severe disease, by a dietician.

**6. Analgesia.** Simple analgesia such as paracetamol or non-steroidal anti-inflammatory medication helps treat pleuritic pain, thereby reducing the risk of further complications due to restricted breathing because of pain (e.g. sputum retention, atelectasis or secondary infection).

**7. Antibiotics :** The first dose of antibiotic should be administered within 4 hours of presentation in hospital and treatment **should not be delayed while investigations** are awaited.

- Parenteral antibiotics should be switched to oral once the temperature has settled for a period of 24 hours, provided there is no contraindication to oral therapy.



# Treatment of CAP



## 1. CRP<65=(0–1)

**Oral amoxicillin 500mg ×3 daily**  
**OR oral clarithromycin 500mg ×2 daily**  
**OR doxycycline 100mg daily**  
**(200mg initial loading dose)**  
**IF penicillin allergic**



## 2. CRP<65=2 (admitted to hospital)

**Amoxicillin 500–1000mg ×3 daily**  
**PLUS clarithromycin 500mg ×2 daily**  
**orally (IV if oral not possible)**

- **Penicillin allergy:**

**doxycycline 100mg daily**  
**(200mg initial loading dose)**  
**OR levofloxacin 500mg ×2 daily orally**  
**OR moxifloxacin 400mg daily orally**

# Treatment of CAP

3.  $CRP < 65 = (3-5)$  (admitted to hospital)

- **Co-amoxiclav** 1.2g ×3 daily intravenously **PLUS** **clarithromycin** 500mg ×2 daily intravenously

- **Alternative:**

- **Penicillin allergy:**

**IV cephalosporin**(e.g. ceftriaxone 2g daily) **PLUS** **clarithromycin** 500mg ×2 daily

- **Benzylpenicillin** 1.2g ×4 daily **PLUS** **a fluoroquinolone** (levofloxacin or moxifloxacin)



## ***Treatment of other:***

- a. Cephalosporins with pseudomonal coverage: ceftazidime or cefepime
- b. Carbapenems: imipenem
- c. Piperacillin/tazobactam

### ***treatment of aspiration pneumonia***

Broad spectrum antibiotics clindamycin  
,amoxicillin\_ clavulanic acid

***Treatment of *Pneumocystis jiroveci* :***  
First-line treatment of PCP is with high-dose co-trimoxazole  
(Trimethoprim/sulfamethoxazole)



# Prevention



*Cigarette smoking is an independent risk factor for CAP; if the patient still smokes, cessation advice and support should*

*Vaccination against influenza is recommended for at-risk groups. All patients over the age of 65 who have not previously*

*been vaccinated and are admitted with CAP should have the pneumococcal vaccine before discharge from hospital.*





***Thankyou***

