

*Chronic Obstructive  
Pulmonary Disease*

**BY**

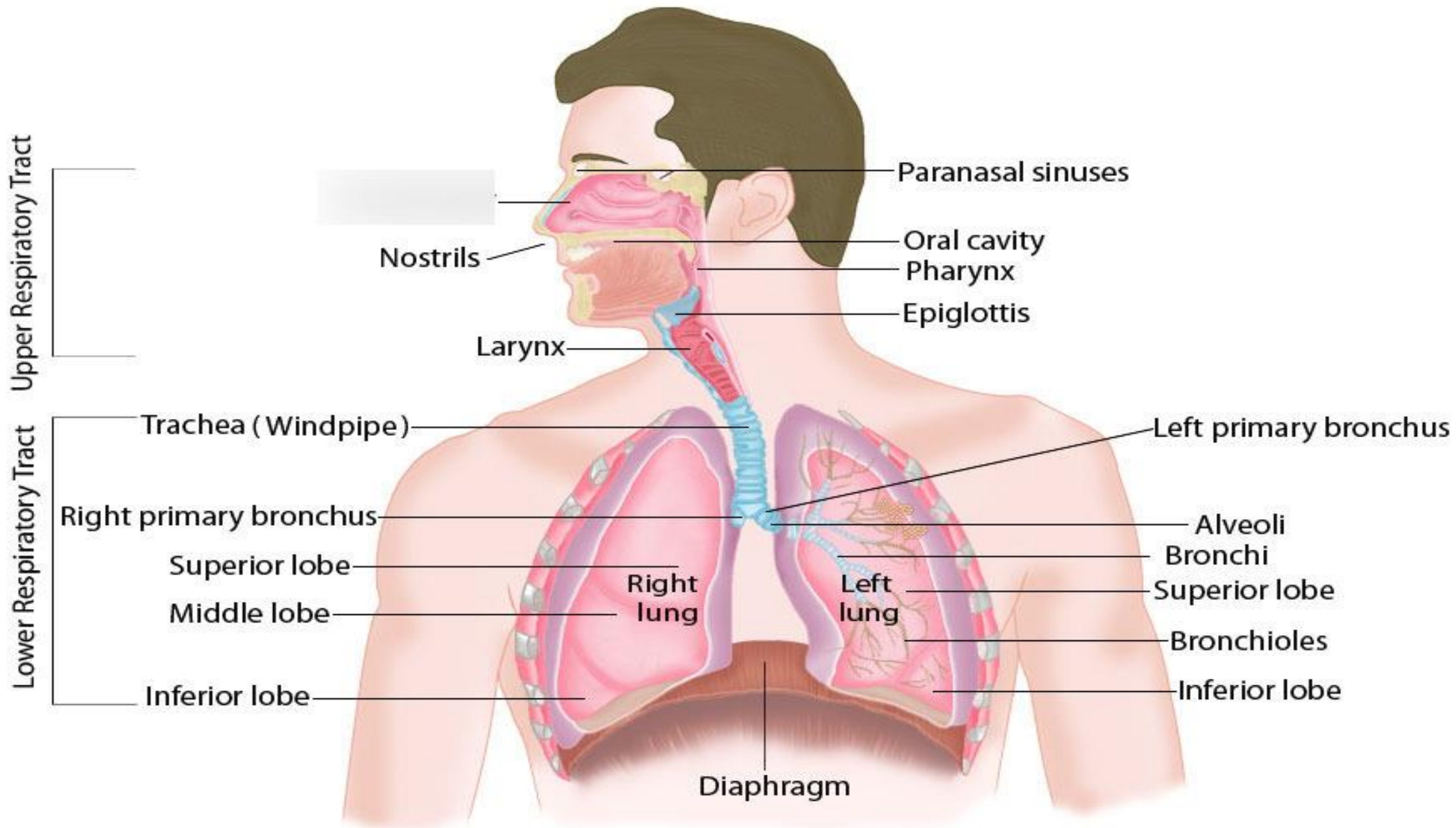
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# INTRODUCTION

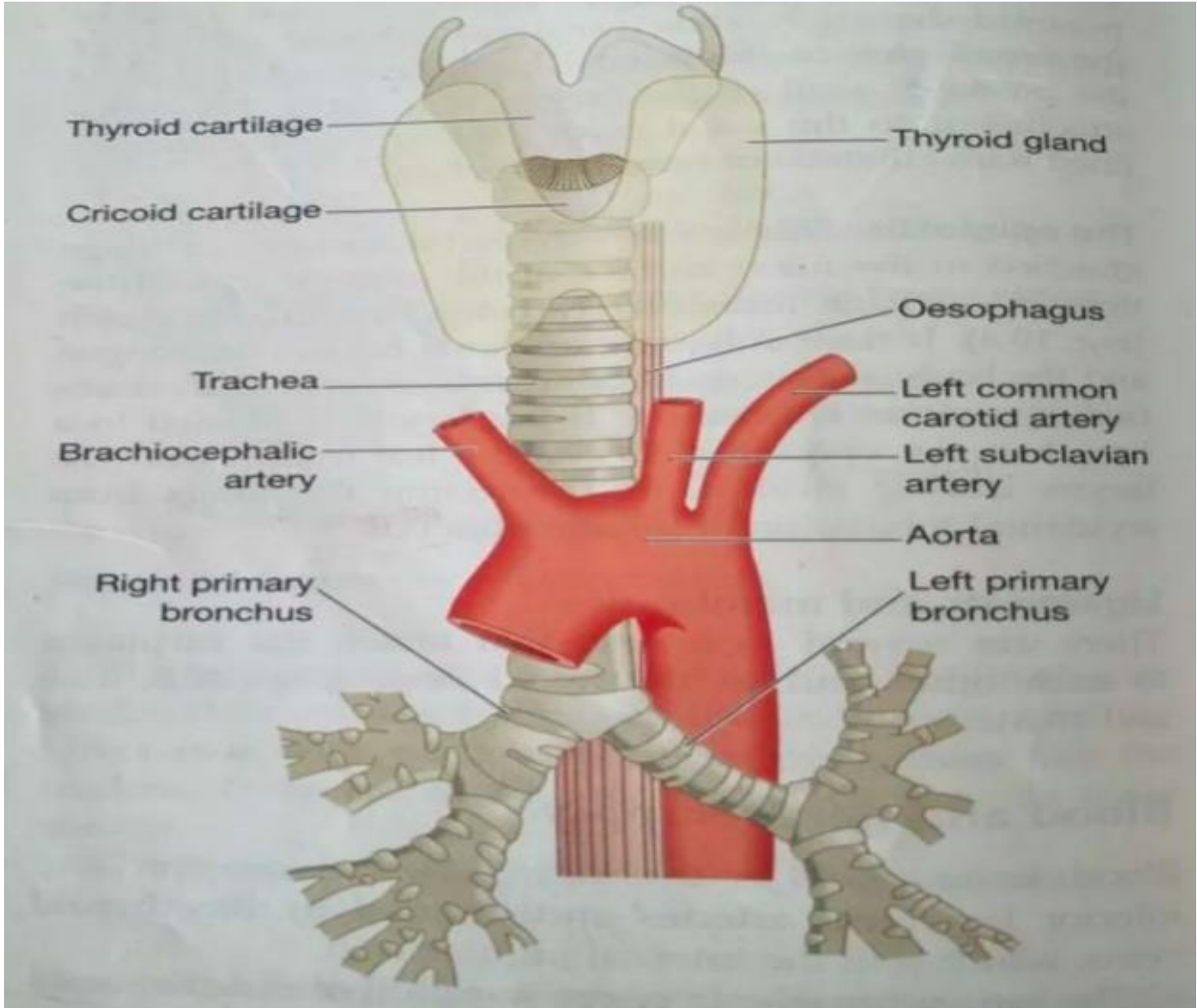
# WHAT IS RESPIRATORY SYSTEM?

- The **respiratory system** (also **respiratory apparatus, ventilatory system**) is a biological system, consisting of specific organs and structures used for gas exchange in human.
- Organs of Respiratory System:
  - Nose and nasal cavity.
  - Pharynx
  - Larynx
  - Trachea
  - Two bronchi
  - Bronchioles
  - Two Lungs



# TRACHEA

- **Position**
- The trachea or **windpipe** is a continuation of the larynx & extends downwards to about the level of **T-5** where it divides into right & left primary bronchi.
- **Length-10-11cm**
- **Relation**
  - Superiorly**-the larynx
  - Inferiorly**-the right & left bronchi
  - Anteriorly**-upper part-the thyroid gland.  
lower part-the arch of aorta & the sternum.
  - Posteriorly**-.the oesophagus
  - Laterally**- the lungs



## **STRUCTURE**

- Composed of 3 layers of tissue.
- (i) fibrous & elastic tissue
- (ii) smooth muscle
- (iii) ciliated columnar epithelium
- Held open by between 16-20 incomplete cartilage rings (C-shaped)

## **Blood supply**

Inferior thyroid artery

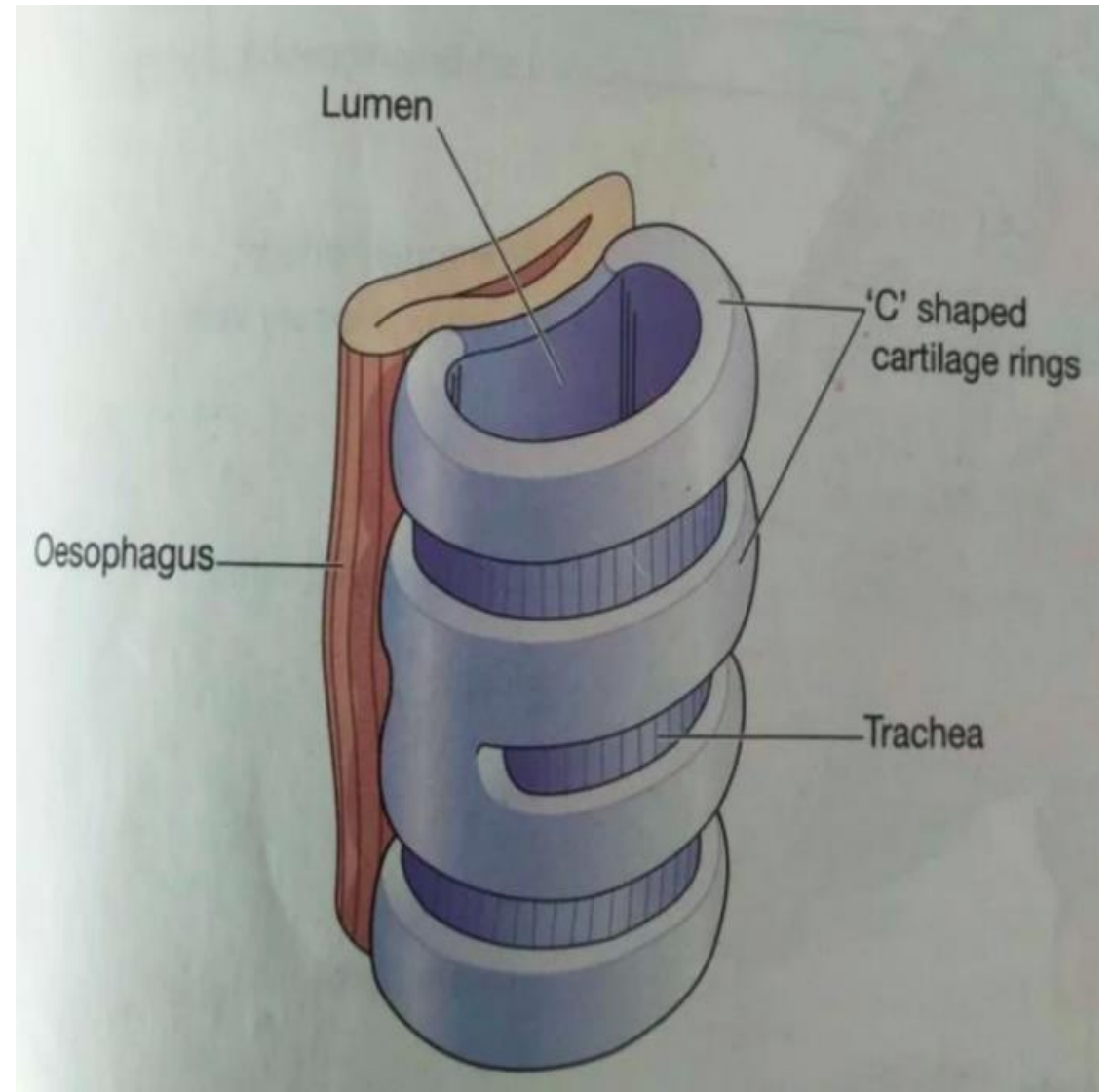
Bronchial artery

## **Venous drainage**

Inferior thyroid veins

## **Nerve supply**

Laryngeal nerve





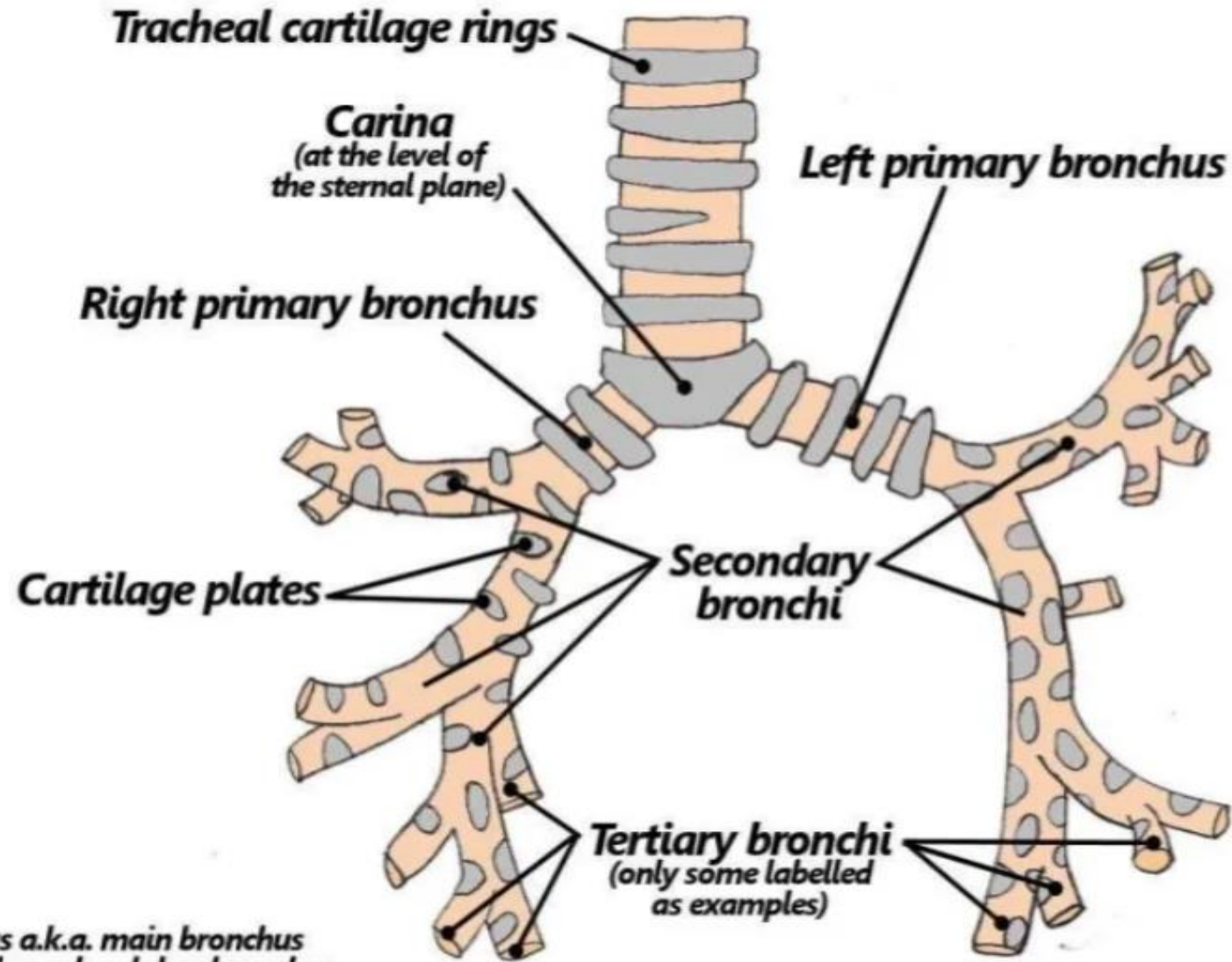
## **FUNCTIONS**

- Support and patency
- Mucociliary escalator
- Cough reflex
- Warming
- Humidifying
- Filtering

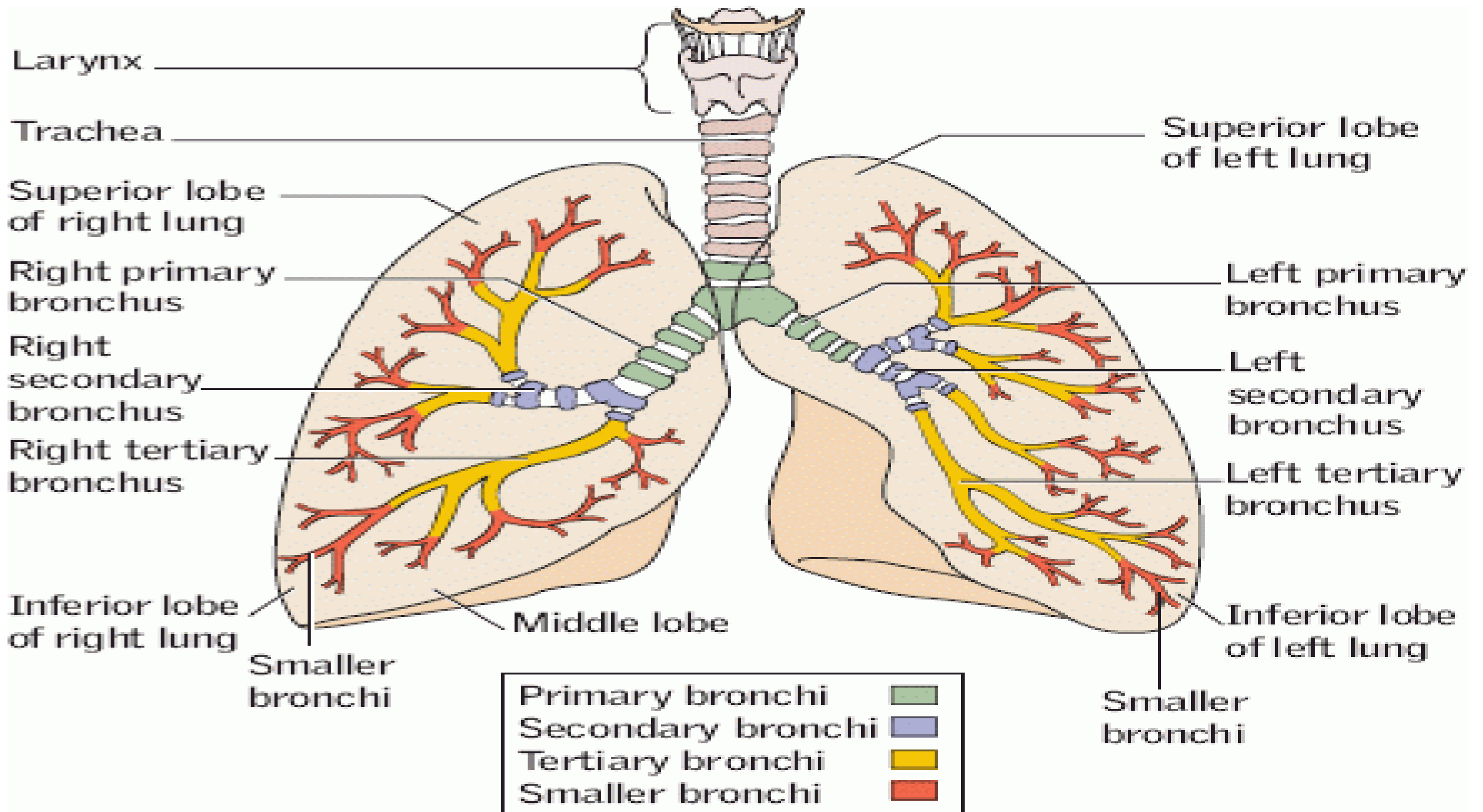


# BRONCHI & BRONCHIOLES

- The two primary bronchi when the trachea divides about the level of T-5.
- **The right bronchus**
- This is wider, shorter and more vertical than the left bronchus.
- Length-2.5cm
- After entering the right lung, it divides into 3 branches, one to each lobe.
- **The left bronchus**
- This is **narrower** than the **right**
- Length-5cm
- After entering the left lung, it divides into 2 branches, one to each lobe.

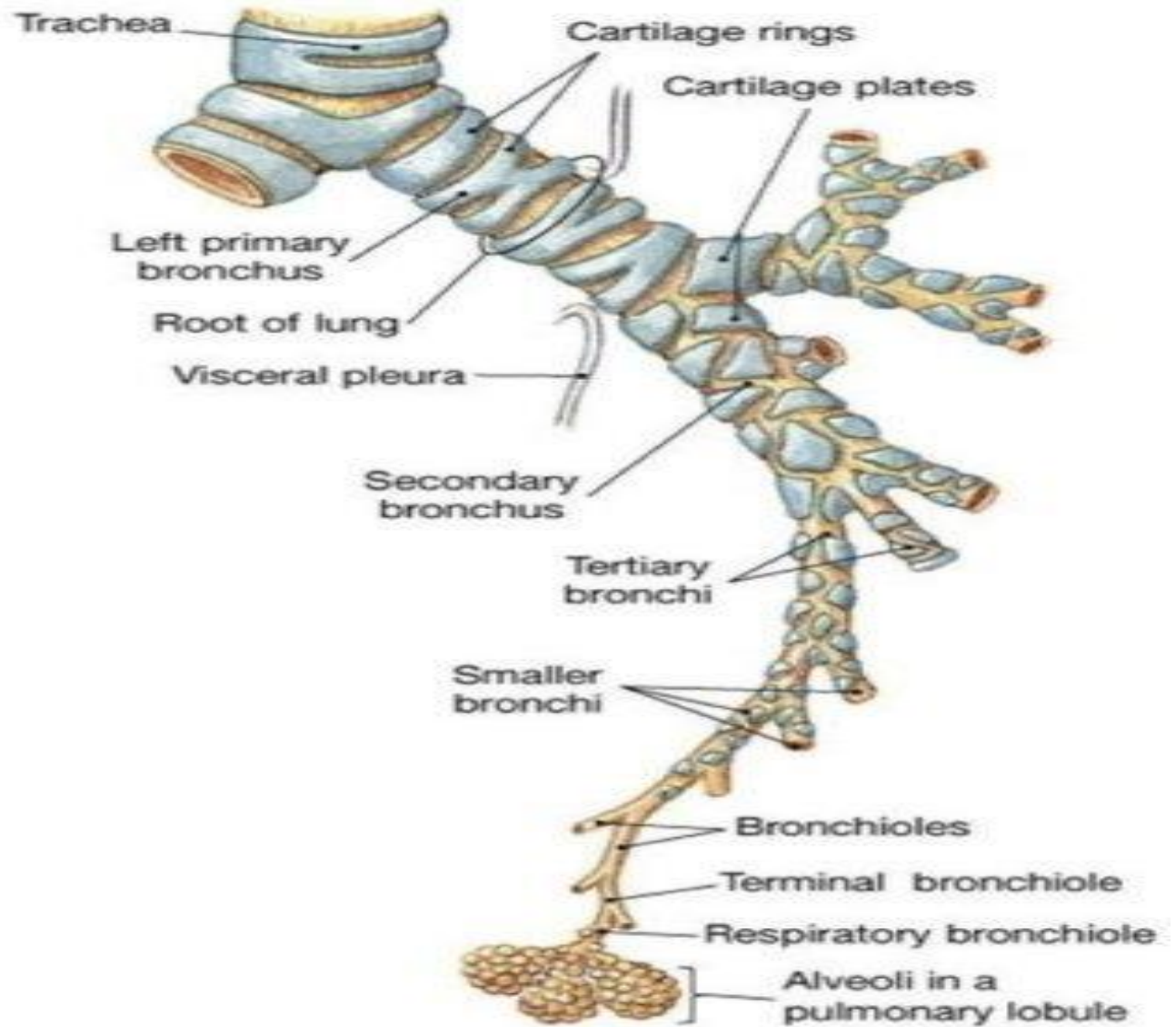
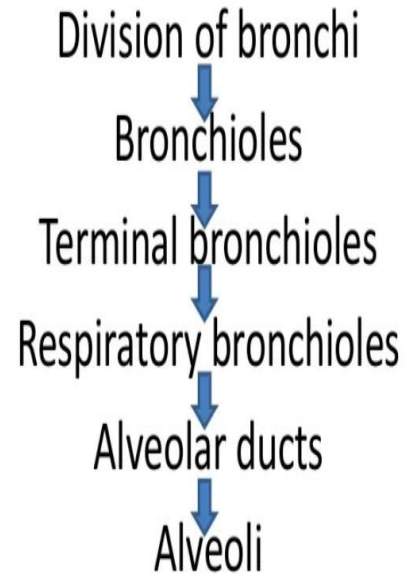


**Note:**  
Primary bronchus a.k.a. main bronchus  
Secondary bronchus a.k.a. lobar bronchus  
Tertiary bronchus a.k.a. segmental bronchus



## STRUCTURE

- The bronchi are composed of the same tissues as the trachea.
- Are lined with **ciliated columnar epithelium**.



- The wider passages are called **conducting airways**
- Conducting airways, bring air into the lungs & their walls are too thick to permit gas exchange.
- **Blood supply**
- Bronchial arteries
- **Venous drainage**
- Bronchial veins
- **Nerve supply**
- Vagus nerve
- **Lymph drainage**
- The Thoracic duct

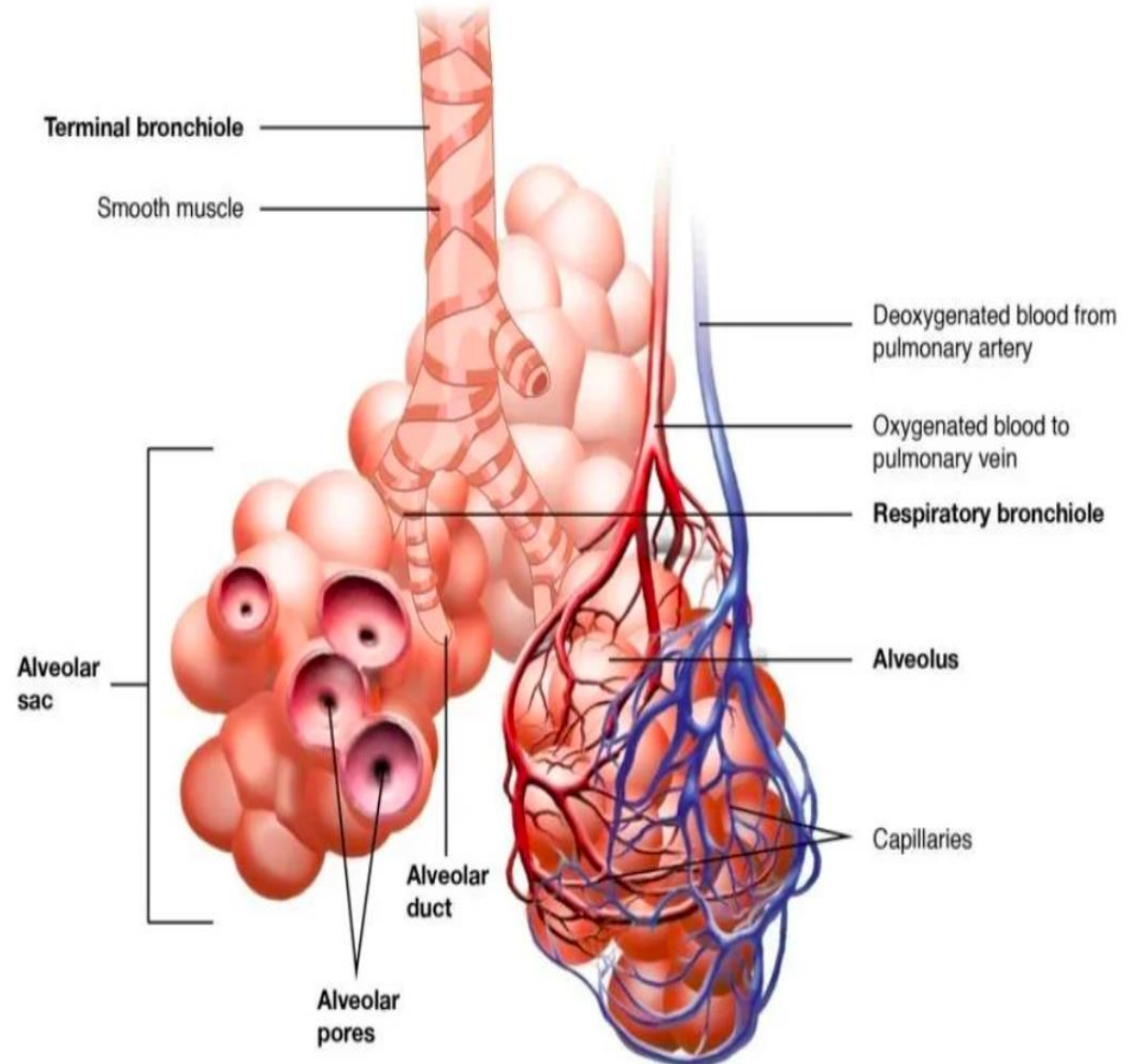
## FUNCTIONS

- Control of air entry
- Warming & humidifying
- Support & patency
- Removal of particulate matter
- Cough reflex



- **RESPIRATORY BRONCHIOLES & ALVEOLI**

- Each lobule is supplied with air by a terminal bronchiole
- Which further subdivides into respiratory bronchioles, **alveolar ducts** and large numbers of **alveoli (air sacs)**
- About **150 million** alveoli in the adult lung
- In these structures that the process of gas exchange occurs.
- As airways progressively divide & become smaller & smaller, their walls gradually become thinner.
- These distal respiratory passages are supported by a loose network of elastic connective tissue.
- Exchange of gases in the lungs takes place in alveoli



- **Nerve supply**

- Vagus nerve

- **FUNCTIONS**

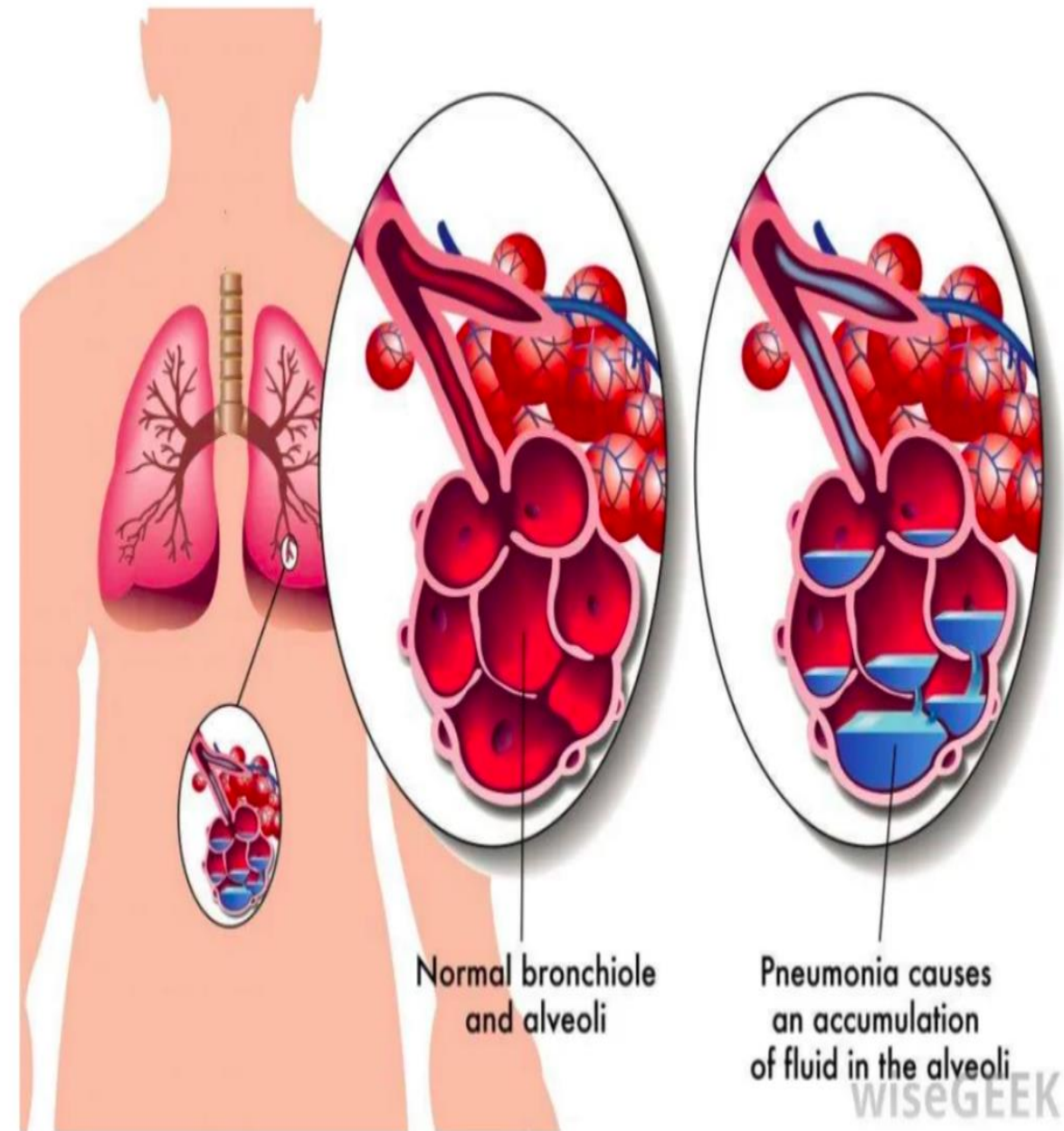
- **External respiration**

This is exchange of gases by diffusion between the alveoli and the blood.

- **Defence against microbes**

Protective cells present within the lung tissue, include lymphocytes & plasma cells, which produce antibodies.

- **Exchange of gases**





# LUNGS

- There are **two lungs**, one lying on each side.
- **Shape**-cone
- **Weight**-600-700gms
- **Length**-20-24cm
- **Colour**-pinkish
- **Lobes**- **three** lobes in the **right** lung  
**two** lobes in the **left** lung
- Lobes are **separate** by **the fissures**
- The area between the lungs is the **mediastinum**.



**Non Smokers  
Lungs**

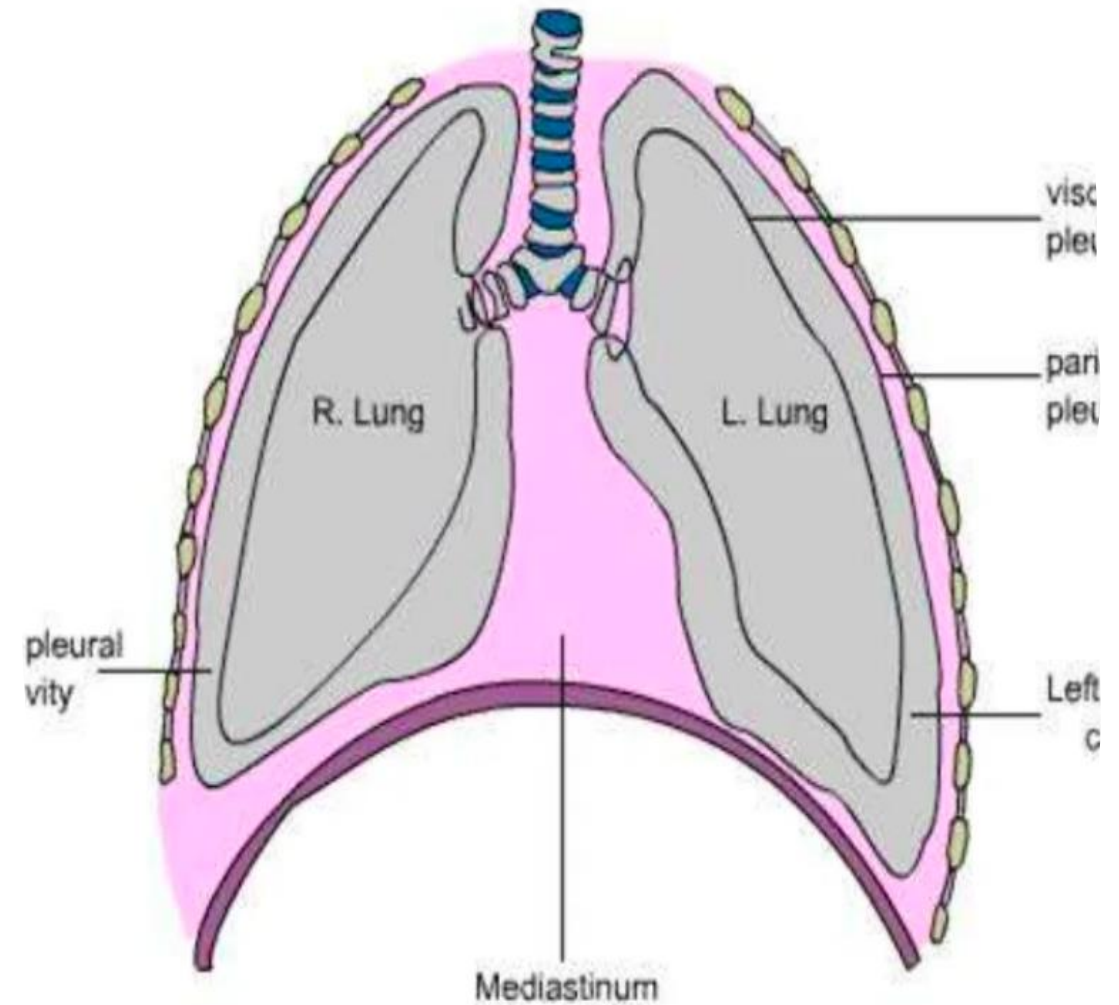
**Rollup Cigarette  
Smokers Lungs**

## Pleura

- The **pleura** consists of a closed **sac** of serous membrane, one for each lung which contains a small amount of **serous fluid**.
- The lung is invaginated or pushed into this sac.
- It forms **two layers**:
  - (i) The **visceral** pleura
  - (ii) The **parietal** pleura
- (i) The **visceral** pleura
  - This is **adherent to the lung**, covering each lobe & passing into the fissures that separate them.
- (ii) The **parietal** pleura
  - This is **adherent to** the inside of the **chest wall & the thoracic surface** of the diaphragm.

## The pleural cavity

- The two layers of pleura are separated by a thin film of serous fluid which allows them to glide over each other.
- **Preventing friction** between them during breathing.
- The **serous fluid** is secreted by the epithelial cells of **the membrane**.



# *DEFINITIONS*

## **Chronic Obstructive Pulmonary Disease (COPD) :**

- Is a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction.

**EMPHYSEMA**

**CHRONIC  
BRONCHITIS**



**COPD**



# *Emphysema:*

- Abnormal **permanent** enlargement of the airspaces distal to the terminal bronchiole, accompanied by destruction of their walls without fibrosis.
- Types:
  - (1) Centrilobular (centriacinar)
  - (2) Panlobular (panacinar)
  - (3) Paraseptal (distal acinar)
  - (4) Irregular



# *Chronic bronchitis:*

- Presence of ***chronic productive cough*** for ***3 months*** in each of ***2 successive years*** in a patient in whom ***other causes of chronic cough*** have been ***excluded.***

# RISK FACTORS

# RISK FACTORS FOR COPD

## Smoking and Nonsmoking Risk Factors

Cigarette smoking is the single most important risk factor for the development of COPD. However, a significant burden of disease occurs in nonsmokers. Alpha-1 antitrypsin deficiency and occupational exposures are implicated as the other major causes of COPD. Other significant causes include outdoor air pollution, secondhand smoke, and biomass smoke.

**SMOKING**— Nearly 80% of all COPD cases can be attributed to smoking. In a retrospective cohort, smokers were more likely than never smokers to develop COPD over a 25-year span (36% vs. 8%).

Risk of COPD and smoking:

- Cigarette smoking: 15–20% of 1 pack per day (PPD) smokers and 25% of 2 PPD smokers
- Pipe and cigars: Elevated risk, but lower than cigarette smokers
- Passive smoking: Suggestive evidence for secondhand smoke as a risk factor

**Table 3-24. Risk of COPD Not Related to Smoking**

<b>Risk</b>	<b>Comment</b>
Occupational exposures	Coal miners, hard-rock miners, tunnel workers, cement workers, cotton workers (Table 3-25)
Genetic susceptibility	$\alpha_1$ -antitrypsin deficiency with > 90% caused by homozygous PiZZ phenotype; cutis laxa (emphysema in children); metalloproteinase-12 gene mutation
Asthma/bronchial hyperreactivity	Chronic asthma and hyperreactivity can lead to FEV <sub>1</sub> decline and fixed obstruction in nonsmokers
Biomass smoke	Indoor burning of wood, animal dung, crop residue, and coal in poorly ventilated dwellings (primarily affects women)
Poverty	Strong risk factor but unclear if related to combination of poor nutrition, air pollution, etc.
Poor lung development	Bronchopulmonary dysplasia (neonatal chronic lung disease), low birth weight
Infections	Childhood infections, tuberculosis, HIV (accelerated emphysema in smokers)
Air pollution	Outdoor pollution has been shown to be an independent risk factor for decline in FEV <sub>1</sub>

FEV<sub>1</sub>, 1-second forced expiratory volume; HIV, human immunodeficiency virus.

# Clinical Indicators for Considering a Diagnosis of COPD

Table 2.1

**Consider the diagnosis of COPD, and perform spirometry, if any of these clinical indicators are present:** (these indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of the presence of COPD; in any case, spirometry is required to establish a diagnosis of COPD)

<b>Dyspnea that is</b>	Progressive over time Worse with exercise Persistent
<b>Recurrent wheeze</b>	
<b>Chronic cough</b>	May be intermittent and may be unproductive
<b>Recurrent lower respiratory tract infections</b>	
<b>History of risk factors</b>	Tobacco smoke (including popular local preparations) Smoke from home cooking and heating fuels Occupational dusts, vapors, fumes, gases and other chemicals Host factors (e.g., genetic factors, developmental abnormalities, low birthweight, prematurity, childhood respiratory infections etc.)

# Modified MRC Dyspnea Scale

Table 2.7

PLEASE TICK IN THE BOX THAT APPLIES TO YOU | ONE BOX ONLY | Grades 0 - 4

mMRC Grade 0	mMRC Grade 1	mMRC Grade 2	mMRC Grade 3	mMRC Grade 4
I only get breathless with strenuous exercise <input data-bbox="359 1053 422 1118" type="checkbox"/>	I get short of breath when hurrying on the level or walking up a slight hill <input data-bbox="698 1053 764 1118" type="checkbox"/>	I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level <input data-bbox="1029 1053 1095 1118" type="checkbox"/>	I stop for breath after walking about 100 meters or after a few minutes on the level <input data-bbox="1383 1053 1449 1118" type="checkbox"/>	I am too breathless to leave the house or I am breathless when dressing or undressing <input data-bbox="1724 1053 1791 1118" type="checkbox"/>

Reference: ATS (1982) Am Rev Respir Dis. Nov;126(5):952-6.





# Differential Diagnosis of COPD

Table 2.3

Diagnosis	Suggestive Features
<b>COPD</b>	Symptoms slowly progressive History of tobacco smoking or other risk factors
<b>Asthma</b>	Variable airflow obstruction Symptoms vary widely from day to day Symptoms worse at night/early morning Allergy, rhinitis, and/or eczema also present Often occurs in children Family history of asthma
<b>Congestive heart failure</b>	Chest X-ray shows dilated heart, pulmonary edema Pulmonary function tests indicate volume restriction, not airflow obstruction
<b>Bronchiectasis</b>	Large volumes of purulent sputum Commonly associated with bacterial infection Chest X-ray/HRCT shows bronchial dilation
<b>Tuberculosis</b>	Onset all ages Chest X-ray shows lung infiltrate Microbiological confirmation High local prevalence of tuberculosis
<b>Obliterative bronchiolitis</b>	Can occur in children Seen after lung or bone marrow transplantation HRCT on expiration shows hypodense areas
<b>Diffuse panbronchiolitis</b>	Predominantly seen in patients of Asian descent Most patients are male and nonsmokers Almost all have chronic sinusitis Chest X-ray & HRCT show diffuse small centrilobular nodular opacities & hyperinflation

*These features tend to be characteristic of the respective diseases, but are not mandatory. For example, a person who has never smoked may develop COPD (especially in LMICs where other risk factors may be more important than cigarette smoking).*





## GOLD Grades and Severity of Airflow Obstruction in COPD (based on post-bronchodilator FEV1)

Table 2.6

In COPD patients (FEV1/FVC < 0.7):

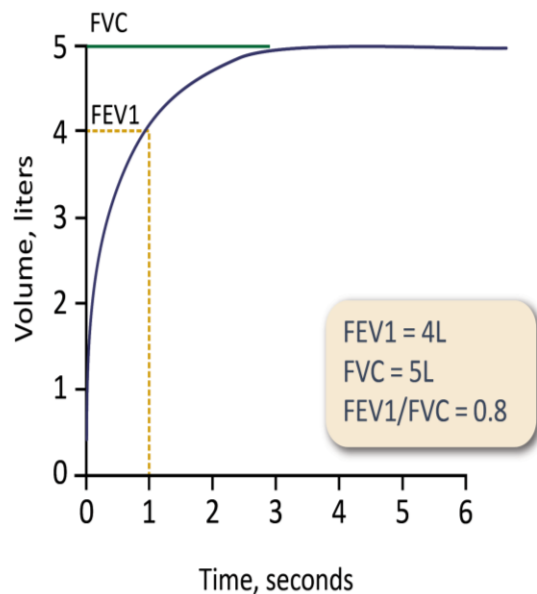
<b>GOLD 1:</b>	Mild	FEV1 ≥ 80% predicted
<b>GOLD 2:</b>	Moderate	50% ≤ FEV1 < 80% predicted
<b>GOLD 3:</b>	Severe	30% ≤ FEV1 < 50% predicted
<b>GOLD 4:</b>	Very Severe	FEV1 < 30% predicted

### A. Spirometry - Normal Trace

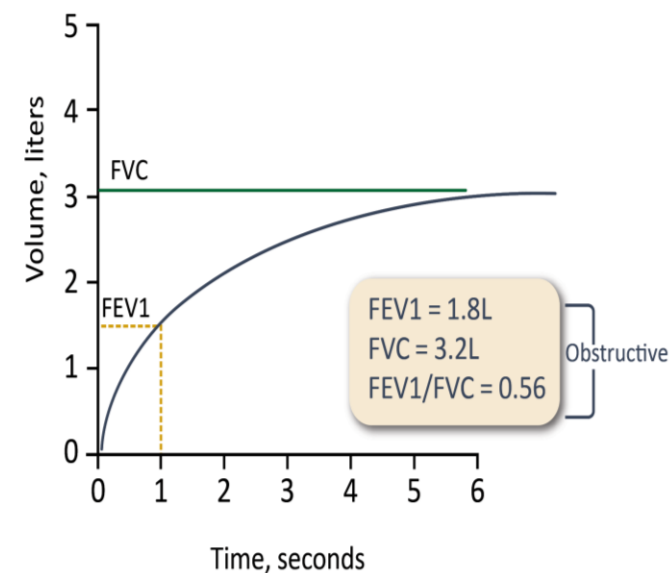
### B. Spirometry - Airflow Obstruction

Figure 2.1

A



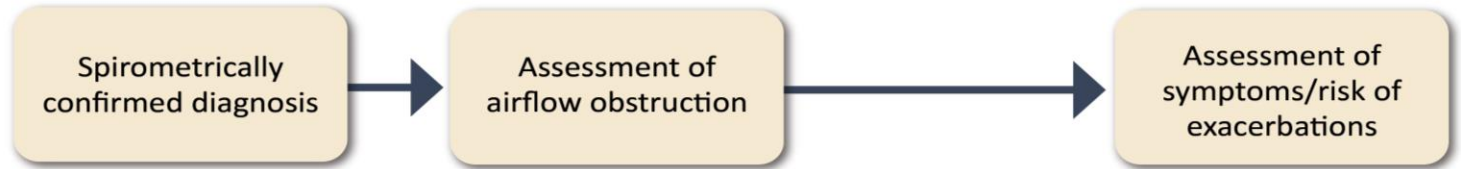
B



FVC = —————  
FEV1 = - - - - -

# GOLD ABE Assessment Tool

Figure 2.3



Post-bronchodilator FEV1/FVC < 0.7

GRADE	FEV1 (% predicted)	EXACERBATION HISTORY (PER YEAR)	SYMPTOMS	
GOLD 1	≥ 80	≥ 2 moderate exacerbations or ≥ 1 leading to hospitalization	E	
GOLD 2	50-79	0 or 1 moderate exacerbations (not leading to hospitalization)	A	B
GOLD 3	30-49			
GOLD 4	< 30			

mMRC 0-1 CAT < 10      mMRC ≥ 2 CAT ≥ 10



## Goals for Treatment of Stable COPD

Table 4.1

- Relieve Symptoms
- Improve Exercise Tolerance
- Improve Health Status



**REDUCE SYMPTOMS**

*AND*

- Prevent Disease Progression
- Prevent and Treat Exacerbations
- Reduce Mortality



**REDUCE RISK**

## Identify & Reduce Risk Factor Exposure

Table 4.3

- Smoking cessation interventions should be actively pursued in all people with COPD (**Evidence A**)
- Efficient ventilation, non-polluting cooking stoves and similar interventions should be recommended (**Evidence B**)
- Clinicians should advise patients to avoid continued exposures to potential irritants, if possible (**Evidence D**)

## Bronchodilators in Stable COPD

Table 3.4

- Inhaled bronchodilators in COPD are central to symptom management and commonly given on a regular basis to prevent or reduce symptoms **(Evidence A)**
- Regular and as-needed use of SABA or SAMA improves FEV1 and symptoms **(Evidence A)**
- Combinations of SABA and SAMA are superior compared to either medication alone in improving FEV1 and symptoms **(Evidence A)**
- LABAs and LAMAs significantly improve lung function, dyspnea, health status, and reduce exacerbation rates **(Evidence A)**
- LAMAs have a greater effect on exacerbation reduction compared with LABAs **(Evidence A)** and decrease hospitalizations **(Evidence B)**
- Combination treatment with a LABA and a LAMA increases FEV1 and reduces symptoms compared to monotherapy **(Evidence A)**
- Combination treatment with a LABA+LAMA reduces exacerbations compared to monotherapy **(Evidence B)**
- Tiotropium improves the effectiveness of pulmonary rehabilitation in increasing exercise performance **(Evidence B)**
- Theophylline exerts a small bronchodilator effect in stable COPD **(Evidence A)** and that is associated with modest symptomatic benefits **(Evidence B)**
- Single inhaler therapy may be more convenient and effective than multiple inhalers

# Key Points for Inhalation of Drugs

Table 4.4

- When a treatment is given by the inhaled route, the importance of education and training in inhaler device technique cannot be over-emphasized
- The choice of inhaler device has to be individually tailored and will depend on access, cost, prescriber, and most importantly, patient's ability and preference
- It is essential to provide instructions and to demonstrate the proper inhalation technique when prescribing a device, to ensure that inhaler technique is adequate and re-check at each visit that patients continue to use their inhaler correctly
- Inhaler technique (and adherence to therapy) should be assessed before concluding that the current therapy is insufficient



## Inhaled Corticosteroids

- An ICS combined with a LABA is more effective than the individual components in improving lung function and health status and reducing exacerbations in patients with exacerbations and moderate to very severe COPD (**Evidence A**)
- Regular treatment with ICS increases the risk of pneumonia especially in those with severe disease (**Evidence A**)
- Lower blood and sputum eosinophils are associated with greater presence of proteobacteria, notably *Haemophilus*, increased bacterial infections & pneumonia
- Independent of ICS use, there is evidence that a blood eosinophil count < 2% increases the risk of pneumonia (**Evidence C**)
- Triple inhaled therapy of LABA+LAMA+ICS improves lung function, symptoms and health status, and reduces exacerbations, compared to LABA+ICS, LABA+LAMA or LAMA monotherapy (**Evidence A**). Recent data suggest a beneficial effect of triple inhaled therapy versus fixed-dose LABA+LAMA combinations on mortality in symptomatic COPD patients with a history of frequent and/or severe exacerbations
- Single inhaler therapy may be more convenient and effective than multiple inhalers

## Oral Glucocorticoids

- Long-term use of oral glucocorticoids has numerous side effects (**Evidence A**) with no evidence of benefits (**Evidence C**)

## PDE4 Inhibitors

- In patients with chronic bronchitis, severe to very severe COPD and a history of exacerbations:
  - A PDE4 inhibitor improves lung function and reduces moderate and severe exacerbations (**Evidence A**)
  - A PDE4 inhibitor improves lung function and decreases exacerbations in patients who are on fixed-dose LABA+ICS combinations (**Evidence A**)

## Antibiotics

- Long-term azithromycin and erythromycin therapy reduces exacerbations over one year (**Evidence A**)
- Treatment with azithromycin is associated with an increased incidence of bacterial resistance (**Evidence A**) and hearing test impairments (**Evidence B**)

## Mucoregulators and Antioxidant Agents

- Regular treatment with mucolytics such as erdosteine, carbocysteine and NAC reduces the risk of exacerbations in select populations (**Evidence B**)

## Other Anti-Inflammatory Agents

- Simvastatin does not prevent exacerbations in COPD patients at increased risk of exacerbations and without indications for statin therapy (**Evidence A**). However, observational studies suggest that statins may have positive effects on some outcomes in patients with COPD who receive them for cardiovascular and metabolic indications (**Evidence C**)
- Leukotriene modifiers have not been tested adequately in COPD patients

# Key Points for the Use of Anti-Inflammatory Agents

Table 4.7

- Long-term monotherapy with ICS is not recommended (**Evidence A**)
- We do not encourage the use of a LABA+ICS combination in COPD. If there is an indication for an ICS the combination LABA+LAMA+ICS has been shown to be superior to LABA+ICS and is therefore the preferred choice. This combination can be given as single or multiple inhaler therapy.
- If patients with COPD have features of asthma, treatment should always contain an ICS
- In patients with severe to very severe airflow limitation, chronic bronchitis and exacerbations the addition of a PDE4 inhibitor to a treatment with long acting bronchodilators with/without ICS can be considered (**Evidence B**)
- Preferentially, but not only in former smokers with exacerbations despite appropriate therapy, macrolides, in particular azithromycin, can be considered (**Evidence B**)
- Statin therapy and/or beta-blockers are not recommended for prevention of exacerbations (**Evidence A**)



- Influenza vaccination is recommended in people with COPD (**Evidence B**)
- The WHO and CDC recommends SARS-CoV-2 (COVID-19) vaccination for people with COPD (**Evidence B**)
- The CDC recommends one dose of 20-valent pneumococcal conjugate vaccine (PCV20); or one dose of 15-valent pneumococcal conjugate vaccine (PCV15) followed by 23-valent pneumococcal polysaccharide vaccine (PPSV23) in people with COPD (**Evidence B**)
- Pneumococcal vaccination has been shown to reduce the incidence of community-acquired pneumonia and exacerbations in people with COPD (**Evidence B**)
- The CDC recommends Tdap (dTaP/dTPa) vaccination to protect against pertussis (whooping cough) for people with COPD that were not vaccinated in adolescence (**Evidence B**), and Zoster vaccine to protect against shingles for people with COPD over 50 years (**Evidence B**)

# Pulmonary Rehabilitation, Self-Management and Integrative Care in COPD

Table 3.8

## Pulmonary Rehabilitation

- Pulmonary rehabilitation improves dyspnea, health status and exercise tolerance in stable patients (**Evidence A**)
- Pulmonary rehabilitation reduces hospitalization among patients who have had a recent exacerbation ( $\leq 4$  weeks from prior hospitalization) (**Evidence B**)
- Pulmonary rehabilitation leads to a reduction in symptoms of anxiety and depression (**Evidence A**)

## Education and Self-Management

- Education alone has not been shown to be effective (**Evidence C**)
- Self-management intervention with communication with a health care professional improves health status and decreases hospitalizations and emergency department visits (**Evidence B**)

## Integrated Care Programs

- Integrative care and telehealth have no demonstrated benefit at this time (**Evidence B**)

# Oxygen Therapy and Ventilatory Support in Stable COPD

Table 3.10

## Oxygen Therapy

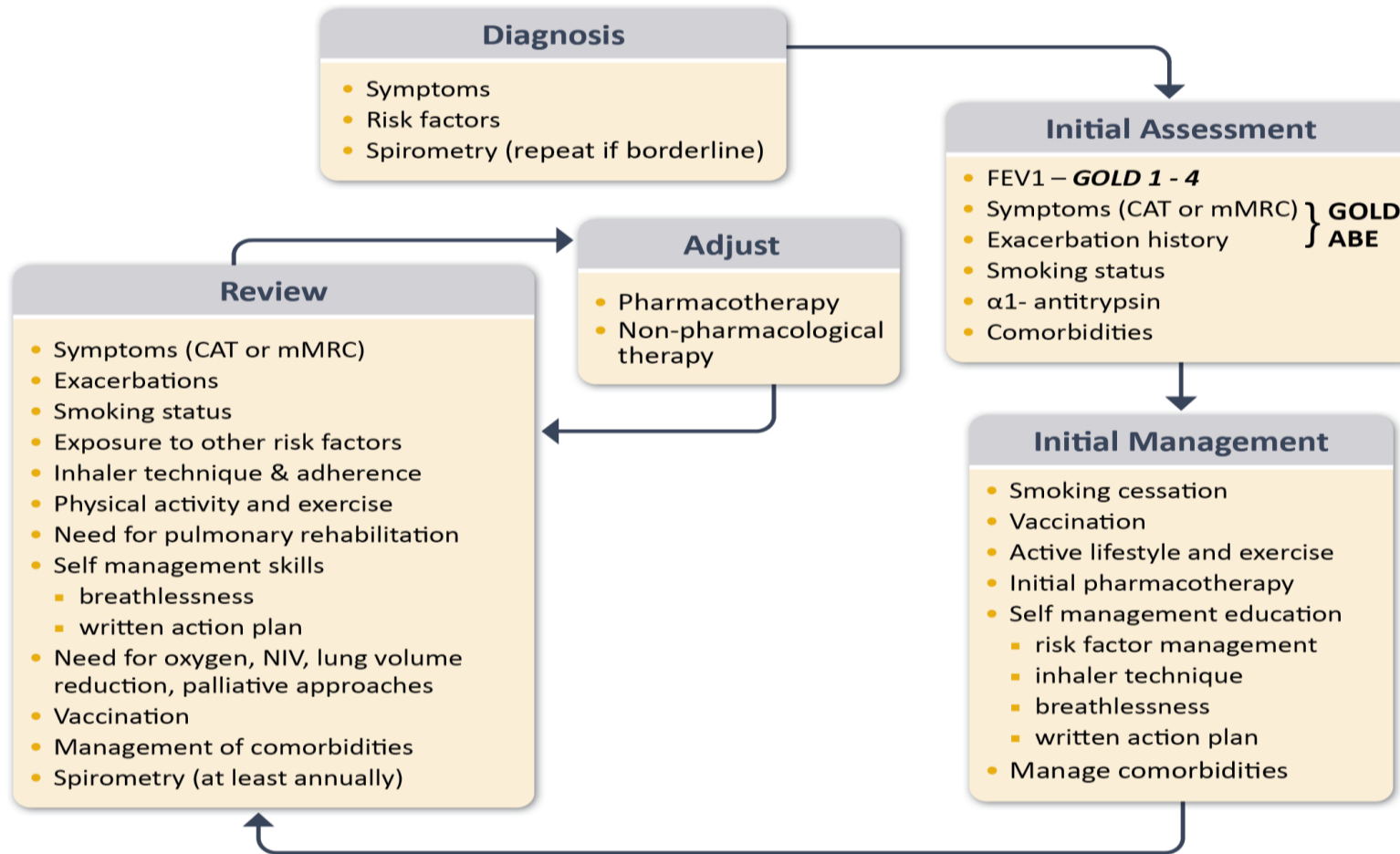
- The long-term administration of oxygen increases survival in patients with severe chronic resting arterial hypoxemia (**Evidence A**)
- In patients with stable COPD and moderate resting or exercise-induced arterial desaturation, prescription of long-term oxygen does not lengthen time to death or first hospitalization or provide sustained benefit in health status, lung function and 6-minute walk distance (**Evidence A**)
- Resting oxygenation at sea level does not exclude the development of severe hypoxemia when traveling by air (**Evidence C**)

## Ventilatory Support

- NPPV may improve hospitalization-free survival in selected patients after recent hospitalization, particularly in those with pronounced daytime persistent hypercapnia ( $\text{PaCO}_2 > 53 \text{ mmHg}$ ) (**Evidence B**)

# Management of COPD

Figure 4.1



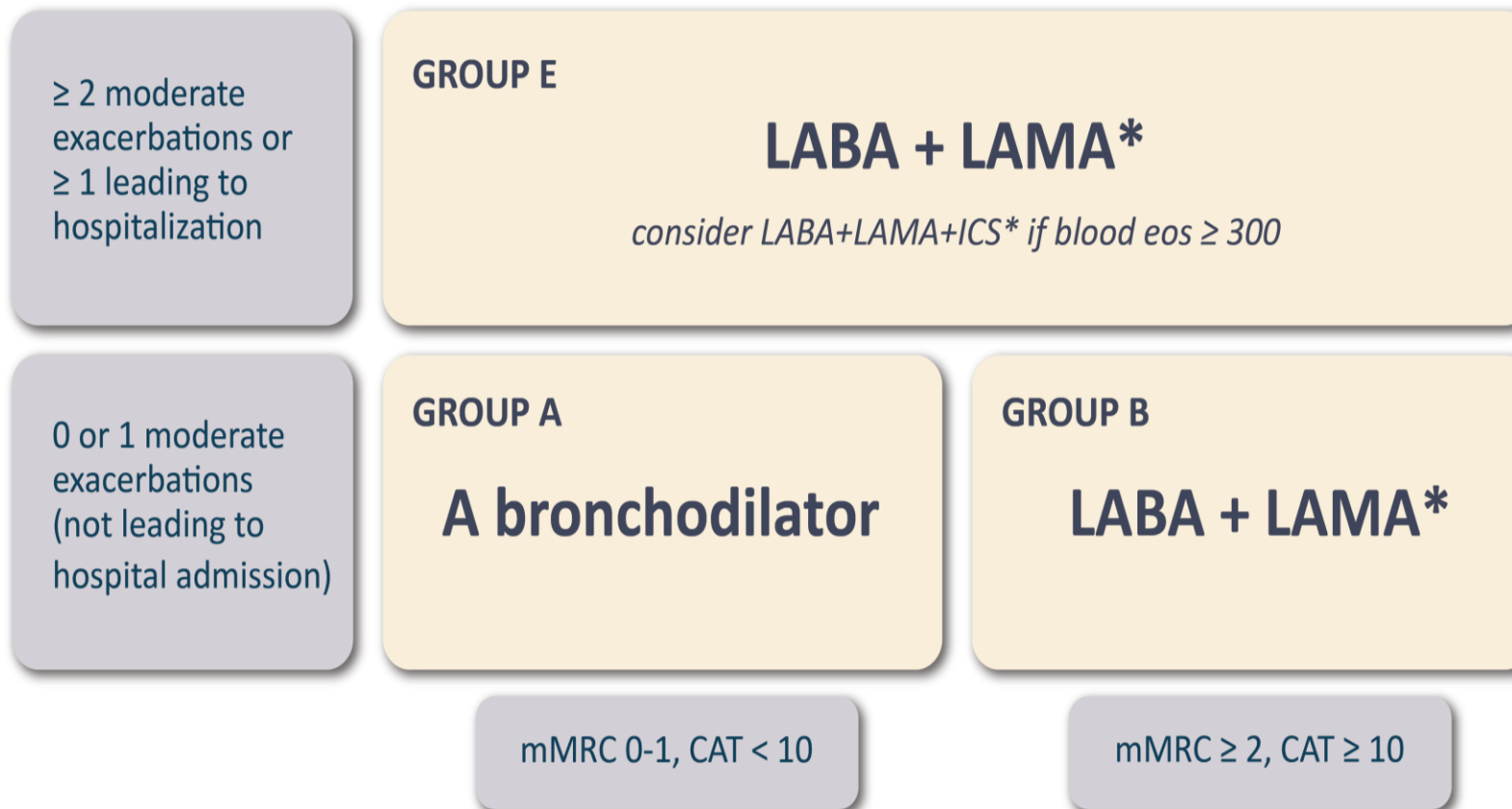
# Key Points for the Use of Other Pharmacological Treatments

Table 4.8

- Patients with severe hereditary alpha-1 antitrypsin deficiency and established emphysema may be candidates for alpha-1 antitrypsin augmentation therapy (**Evidence B**)
- Antitussives cannot be recommended (**Evidence C**)
- Drugs approved for primary pulmonary hypertension are not recommended for patients with a pulmonary hypertension secondary to COPD (**Evidence B**)
- Low-dose long acting oral and parenteral opioids may be considered for treating dyspnea in COPD patients with severe disease (**Evidence B**)

# Initial Pharmacological Treatment

Figure 4.2



\*single inhaler therapy may be more convenient and effective than multiple inhalers  
Exacerbations refers to the number of exacerbations per year



# Non-Pharmacologic Management of COPD\*

Table 4.9

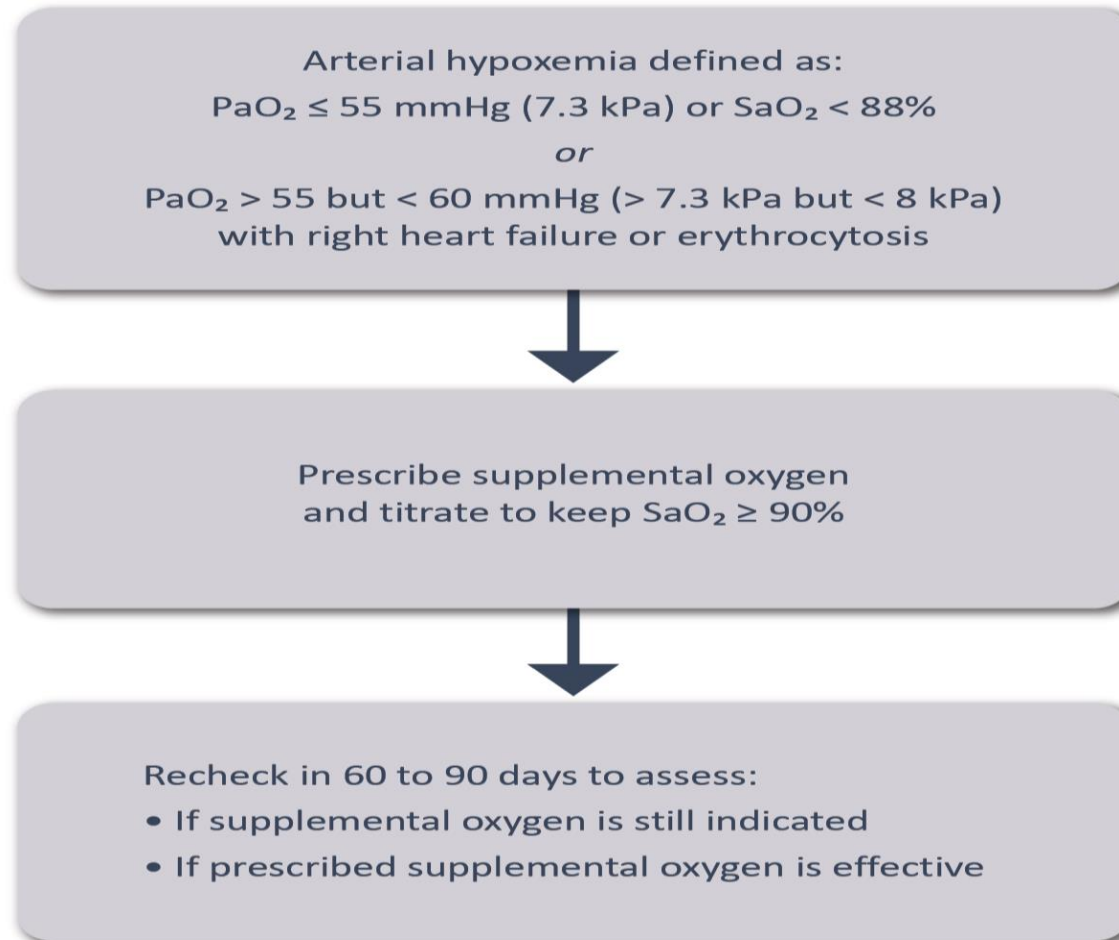
Patient Group	Essential	Recommended	Depending on Local Guidelines
<b>A</b>	Smoking Cessation (can include pharmacological treatment)	Physical Activity	Flu Vaccination Pneumococcal Vaccination Pertussis Vaccination COVID-19 Vaccinations Shingles Vaccination
<b>B and E</b>	Smoking Cessation (can include pharmacological treatment)  Pulmonary Rehabilitation	Physical Activity	Flu Vaccination Pneumococcal Vaccination Pertussis Vaccination COVID-19 Vaccinations Shingles Vaccination

\*Can include pharmacologic treatment



# Prescription of Supplemental Oxygen to COPD Patients

Figure 4.5



**Thank you!!  
Any Questions??**