NEET SS
PULMONOLOGY
(RESPIATORY)

CONTENT

LUNG ANATOMY AND APPLIED CLINICAL ASPECTS : I

Introduction

00:00:20

Right lung is bigger & heavier than left lung.

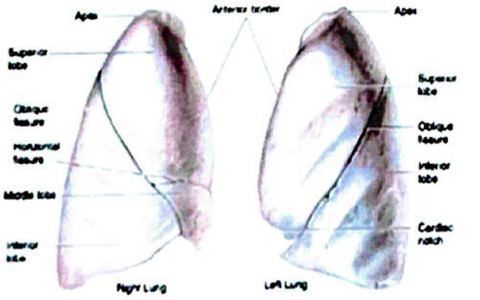
Fissures :

Deep depressions on the lung surface extending to the centre of the lung.

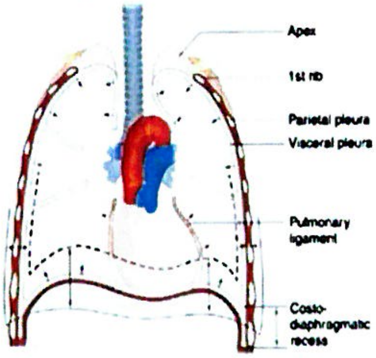
Lined by visceral pleura.

2 fissures on right and 1 fissure on left lung.

- Right horizontal/minor fissure : Between right upper and middle lobes.
- Right oblique fissure/major fissure : Between right middle and lower lobes.
- Left oblique fissure : Between left upper and lower lobes.
- Inferior accessory fissure : Separates median segment of lower lobe from the rest of the lobe.



Fissures of lungs.



Expansile property of lungs.

Applied aspect :

- Auscultation over infrascapular region and back : To examine lower lung lobes/base of the lungs.
- Chest X ray PA view : Portions of lower lobes are hidden.
- To view lower lobes : Lateral view is preferred.

During inspiration, lungs can inflate to 5-6 L or by 4-6 cm.

Pleura

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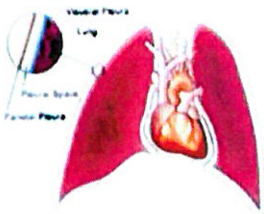
Covering of lungs.

2 layers :

- Outer layer : Parietal pleura.
- Inner layer : Visceral pleura.



Layers of pleura

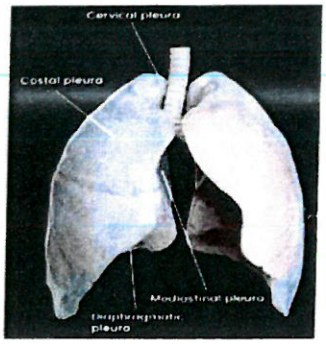
Lined by squamous epithelium (mesothelium).

Pleural cavity : Contains 15-20 mL of clear pleural fluid normally.

Note : mesothelioma is a malignant tumour originating from pleura.

Parts of parietal pleura :

1. Cervical pleura.
2. Costal pleura.
3. mediastinal pleura .
4. Diaphragmatic pleura.



Parts of parietal pleura.

Normal pleural fluid :

- Volume : 15 to 20 mL.
- Total count : 1700 cells/mm^3.
- Differential cell count : 75% macrophages, 23% lymphocytes, 1% mesothelial cells.
- microbiological and cytological analysis : Negative.
- Function of pleural fluid : To reduce friction.

microfiltration from capillaries → Fluid reaches pleural space → Absorbed by stoma/stomata in parietal pleura → Absorbed into lymphatics.

Pleural effusion :

Causes :

1. Increased capillary leak : Inflammation, infection.
2. Poor pleural lymohatic drainage : mediastinal adenopathy, lymphoma, carcinoma lung, pulmonary tuberculosis.

Chest x ray findings :

- Homogenous opacity.
- Blunting of costophrenic, cardiophrenic angles.
- Loss of heart/diaphragmatic borders.
- mediastinal shift opposite side.

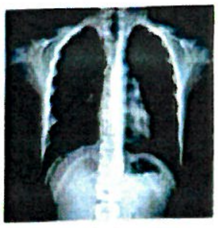

Chest x ray showing left pleural effusion.

Pleural fluid analysis :

- Total count, differential count.
- Cytology.
- Nucleic acid amplification test (NAAT).
- microbiological analysis.
- Adenosine deaminase (ADA).

medical thoracoscopy :

- Direct visualisation of pleura, pleural cavity.
- Biopsy from parietal pleura for histopathology and NAAT.



Pleural based lesion : Pregnant belly sign.

Lateral wall lesions on chest x ray :

Pleural based lesion : Pregnant belly sign.

Obtuse angle with the chest wall.

Lung parenchymal lesion : Acute angle with the chest wall.

Lobes of the lung

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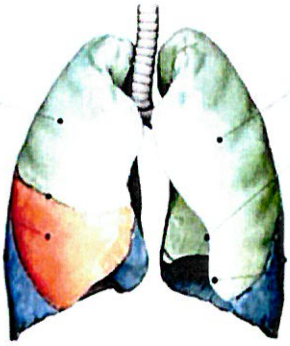
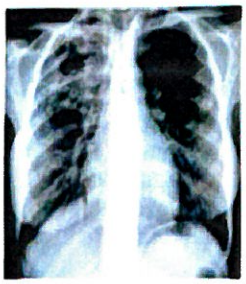
Right lung : 3 lobes (upper, middle, lower).

Left lung : 2 lobes (upper, lower).



Lobes of lungs.

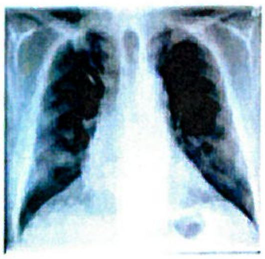
On chest x ray, lung fields are divided into upper, middle and lower zones to decribe lesions.



Right upper lobe cavity with consolidation : Pulmonary tuberculosis.

Idiopathic pulmonary fibrosis : Reticular opacities in lower and upper zones.

Pseudotumour/phantom tumour : Fluid filled within minor fissure.



Azygos fissure (Accessory fissure) : Normal variant in 1-2% population.

Hilum

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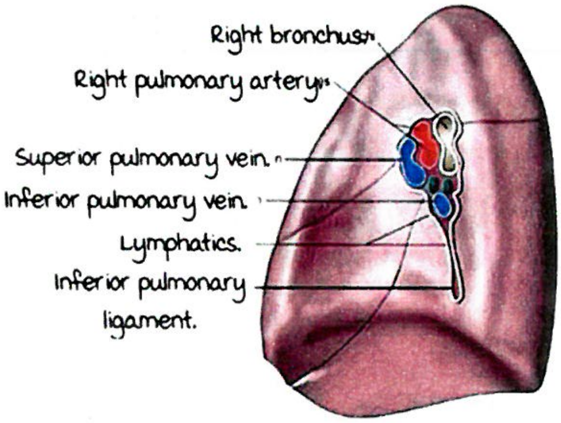
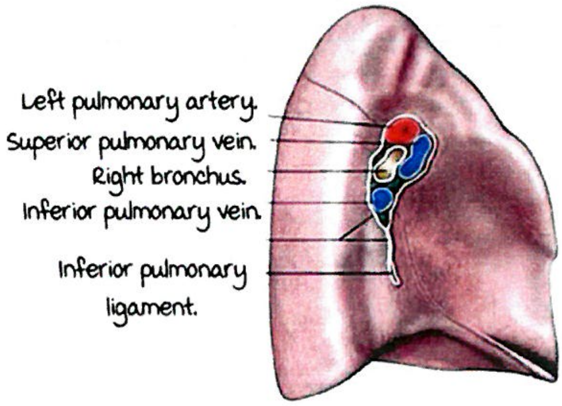
Area through which pulmonary arteries, pulmonary veins, bronchi and lymphatics enter the lungs.
Attaches lungs to the mediastinum.

Right hilum : Pulmonary artery lies in front of the bronchus.
Left hilum : Pulmonary artery lies above bronchus (mnemonic : LAA).



Right hilum.

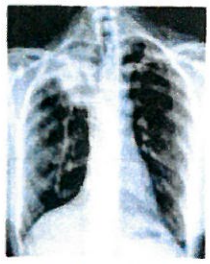


Left hilum.

Left hilum is high than right hilum : Pulmonary artery on left side is above the bronchus.
If right hilum is higher than left : Right upper lobe collapse.

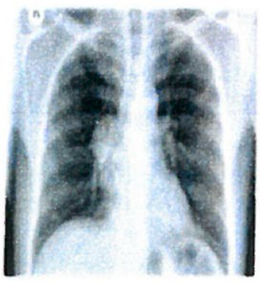
Lesions of hilum :

1. Bronchial lesion : Bronchogenic carcinoma.
2. Dilated pulmonary artery : Pulmonary artery hypertension.
3. Pulmonary vein dilatation : Rare.
4. Hilar lymph node.
5. Mediastinal lesion.



Golden S sign : Right upper lobe collapse.

B/L enlarged pulmonary arteries : Pulmonary artery hypertension.



Opacity in right hilum.

B/L enlarged hilum : B/L hilar lymphadenopathy in sarcoidosis.

Airways

Trachea divides into right and left bronchus at carina.

Left main bronchus is narrower & longer than right main bronchus.

Right main bronchus divides into :

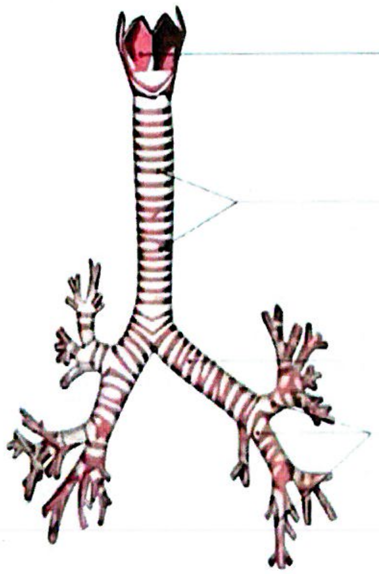
1. Right upper lobe bronchus.
2. Intermediate bronchus : Divides into middle lobe and lower lobe bronchus.

Left main bronchus divides into :

1. Left upper lobe bronchus : Divides into upper lobe and lingular bronchus.
2. Left lower lobe bronchus.(No middle lobe bronchus on left side).

Main bronchus → Lobar bronchi → Divide into segmental bronchus → Subsegmental bronchus.

- Main bronchus : Primary bronchus.
- Lobar bronchus : Secondary bronchus.
- Segmental bronchus : Tertiary bronchus.



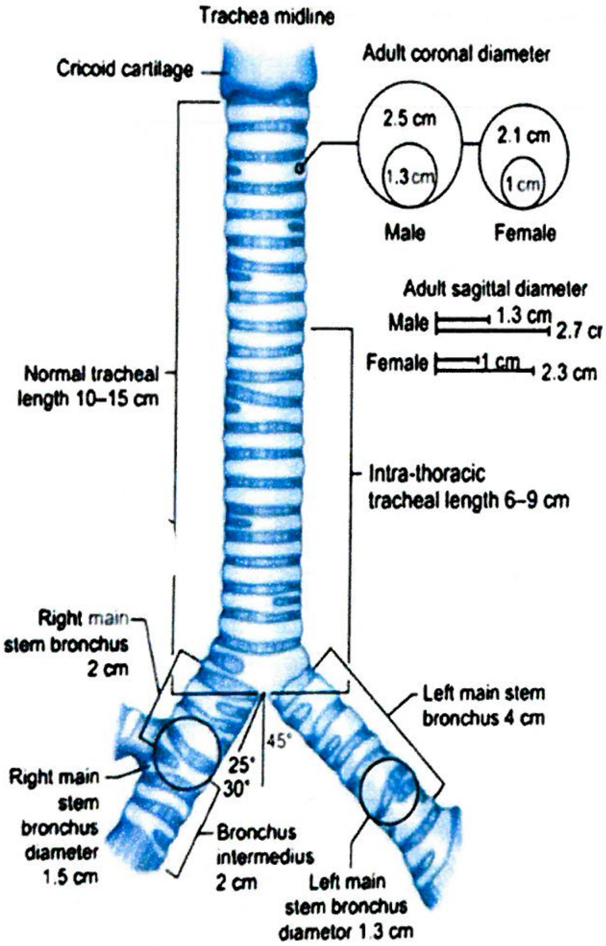
Anatomy of airways.

Dimensions of airways :

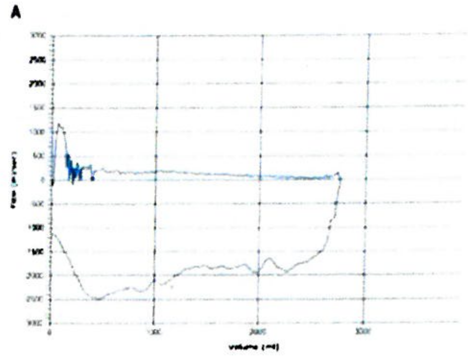
- Total length of trachea : 10-15 cm in adults.
- Intrathoracic portion of trachea : 6-9 cm in adults.
- Coronal diameter of trachea : 1.3-2.5 cm in adult male.
  1-2.1 cm in adult female.
- Sagittal diameter of trachea : 1.3-2.7 cm in adult male
  1-2.3 cm in females.
- Left main bronchus length : 4 cm.
- Right main bronchus length : 2 cm.

- Length of right intermediate bronchus : 2 cm.
- Diameter of right main bronchus : 1.5 cm.
- Diameter of left main bronchus : 1.3 cm.
- Right main bronchus makes an angle of 25-30° with midline.
- Left main bronchus makes an angle of 45° with the midline.

Foreign body goes commonly in right main bronchus : Wider, shorter and more in line with trachea.

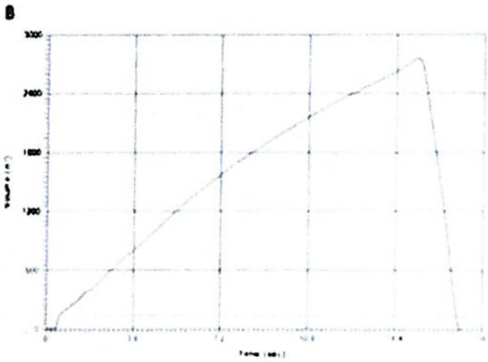


Dimension of airways.



B

Flattening of expiratory loop : Intrathoracic obstruction of trachea.



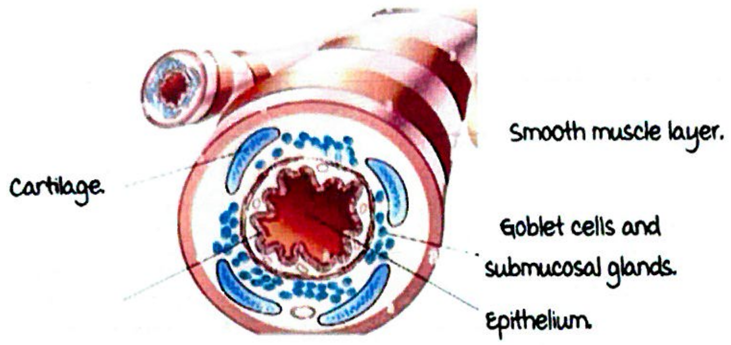
Flattening of inspiratory loop : Extrathoracic obstruction of trachea.

Clinical importance of flow volume curves : Site of obstruction can determine the size of stent required to relieve tracheal obstruction.

Cross sectional anatomy of airways :

Cross sectional anatomy of bronchus :

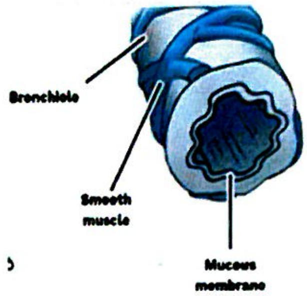
1. Smooth muscle layer : Criss cross pattern, causes bronchoconstriction on contraction.
2. Connective tissue layer : Incomplete cartilage.
   Goblet cells and submucosal glands.
3. Epithelium : Pseudostratified ciliated columnar epithelium.



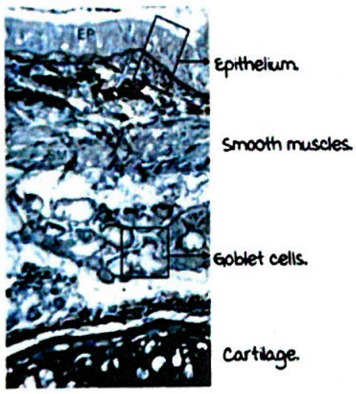
Cross sectional anatomy of bronchus.

Cross sectional anatomy of bronchiole :

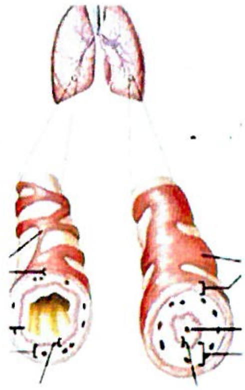
- No cartilage.
- No goblet cells.
- Smooth muscle layer present.



Cross section of bronchiole.



microscopic cross section of airway.



Normal bronchiole vs changes in COPD and asthma

Changes in bronchioles in patients of COPD and asthma exacerbation :

- Smooth muscle hypertrophy.
- Narrow lumen.
- Excessive mucus inside lumen.
- Glandular hypertrophy.

Bronchial thermoplasty :

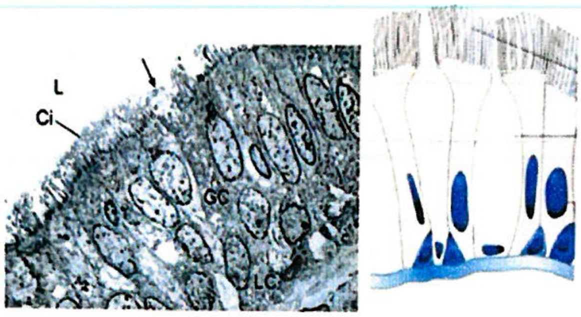
- For treatment of uncontrolled severe asthma.
- Target : Smooth muscle tissue volume is reduced.

Mucociliary escalator :

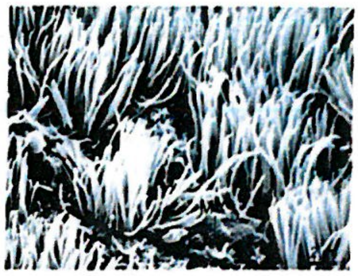
00:53:14

- Protective mechanism of upper airway.
- Epithelium : Pseudostratified ciliated columnar epithelium.
- Goblet cells produce mucus layer over the ciliated epithelium.

- To and fro movement of cilia (mucociliary escalator) moves mucus towards the pharynx.
- Normal frequency of ciliary movement : 12-20 Hz.
- Foreign bodies lodged in mucus layer is pushed out into the pharynx by mucociliary escalator.



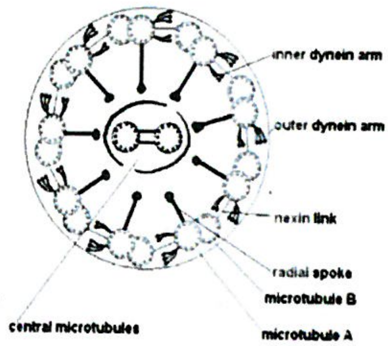
Pseudostraitified ciliated columnar epithelium with goblet cells.



microvilli and cilia on electron microscopy.

Structure of cilia :

- Perpheral microtubules :
  9 doublets consisting of microtubule A and B.
- Central microtubules : 1 pair.
- Radial spokes connect peripheral microtubules to central microtubules.
- Perpheral doublets are are connected by nexin.
- Dyenin : Outer and inner dyenin.
  ATP producing part of the cilia.



Cross section of cilia.

Primary ciliary dyskinesia : AKA Immotile cilia syndrome.

- mutation in ciliary structures.
- Clogging of airways due to secretions → Infections.
- Sinusitis.
- Recurrent lung infections → Bronchiectasis.
- Infertility.
- 50% cases : Situs invertus.
- Kartagener syndrome :
  1. Triad of sinusitis.
  2. Bronchiectasis.
  3. situs invertus.

Cystic shadows in B/L lower lobe : Bronchiectasis and situs invertus.

- Young syndrome : Kartagener's triad + infertility.

LUNG ANATOMY AND APPLIED CLINICAL ASPECTS II

Segments of the lung

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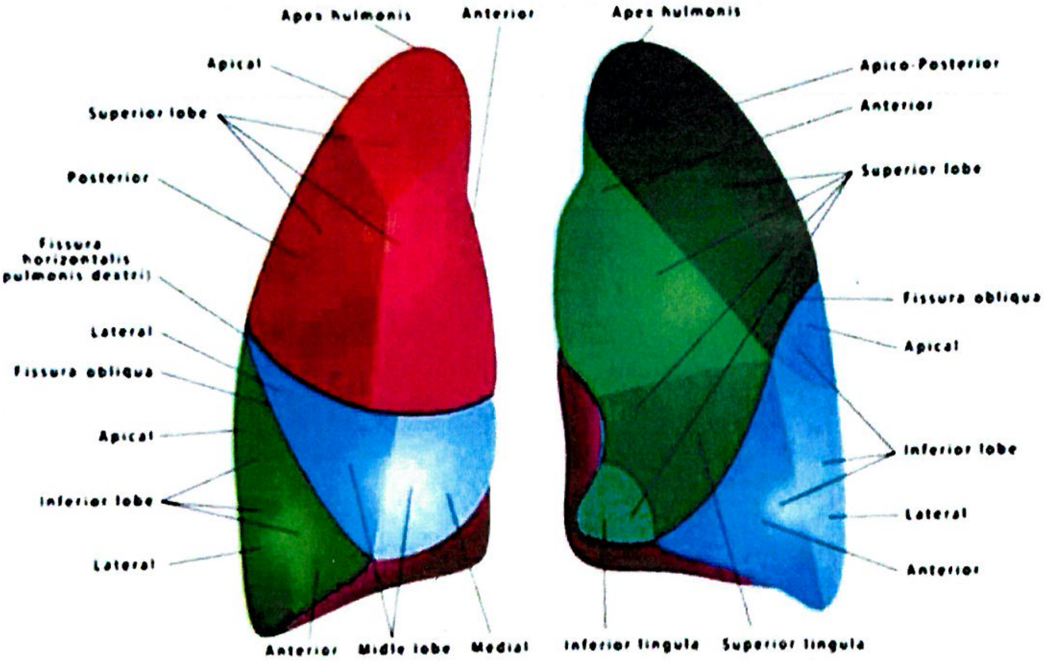
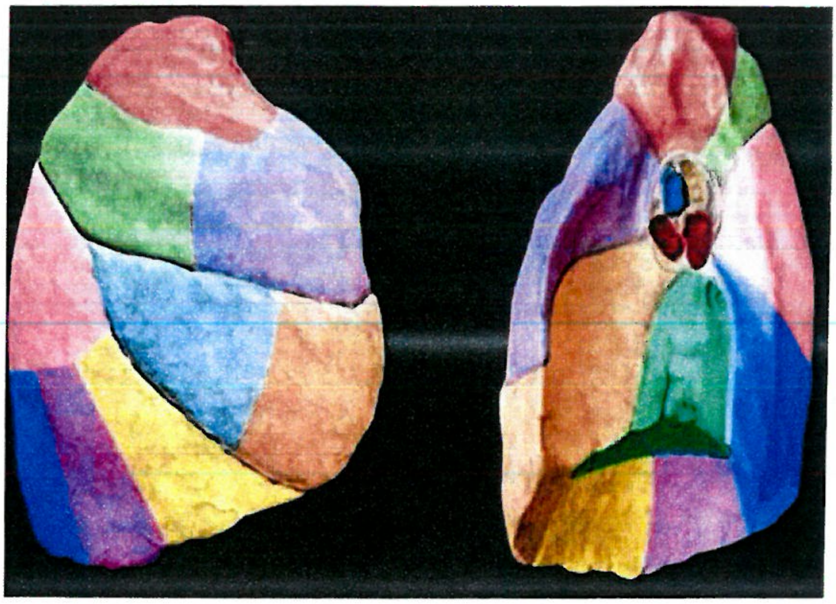


Part of lung	No. of segments	Name of segments
Right upper lobe	3	Anterior, apical and posterior.
Right middle lobe	2	medial and lateral
Right lower lobe	5	Apical, anterior, posterior, medial and lateral.
Left upper lobe	4	Apicoposterior, anterior, superior lingula and inferior lingula.
Left lower lobe	4	Apical, anterior, posteri nd lateral.

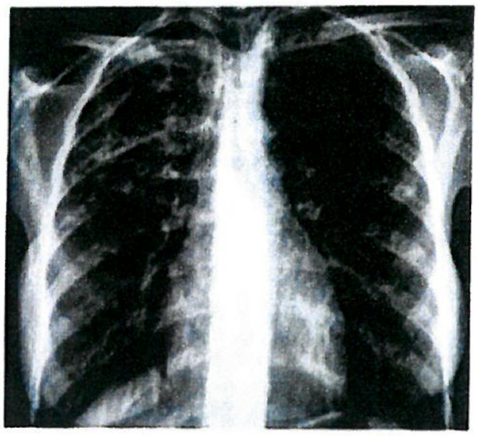
Bronchopulmonary segments :

- Basic functional anatomical unit of lung, with it's own bronchial artery, vein and lymphatic channels.
- Each segment is supplied by a segmental/tertiary bronchus along with a tertiary branch of pulmonary artery.
- Pyramidal in shape, apex directed towards hilum and base is directed towwards pleural surface.

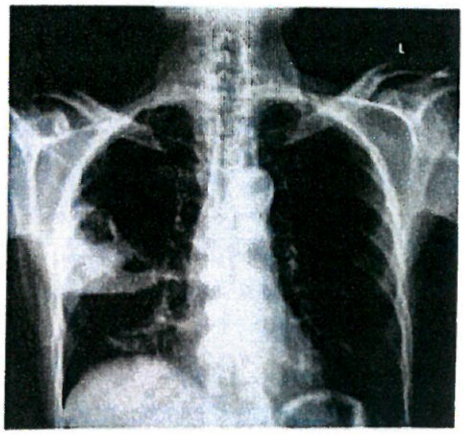


Clinical significance :

- Each segment can be surgically resected without affecting the function of adjacent segments.
- Certain diseases commonly affect specific segments :
    a. Apical and posterior segments of right upper lobe : Tuberculosis.
    b. Anterior segment of right upper lobe : Lung cancer.
    c. Posterior basal segment of left lower lobe : Intra-pulmonary lung sequestration.
    d. Posterior segment of right upper lobe and superior segment of right lower lobe in supine position : Aspiration/lung abscess.
    e. Basal segments of both lower lobe can also be affected by aspiration.



TB : Apical and posterior segments of right upper lobe.



Lung abcess : Posterior segment of right upper lobe.

Divisions of airways

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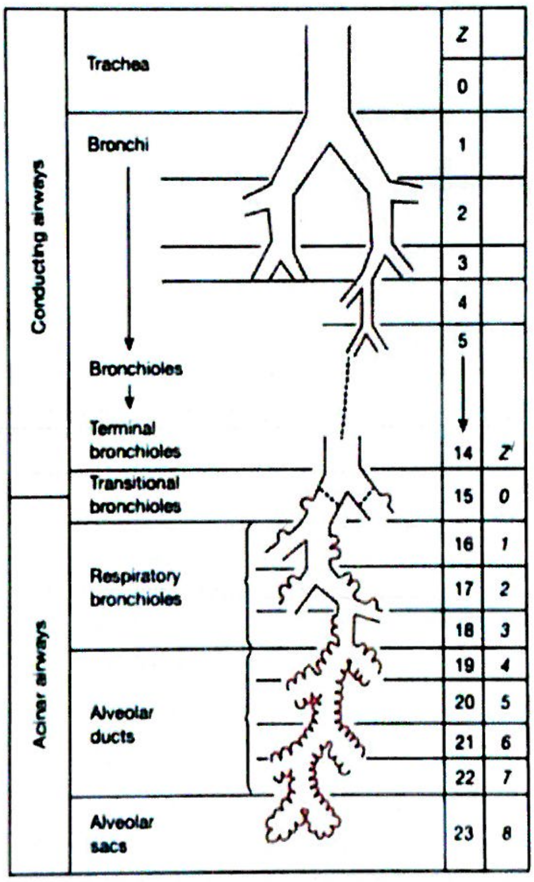
Types :

1. Conduction zones :
    - 1st 14 generations of airways.
    - Only involved in conduction of air alone (Terminal bronchiole included).
    - AKA anatomical deadspace.

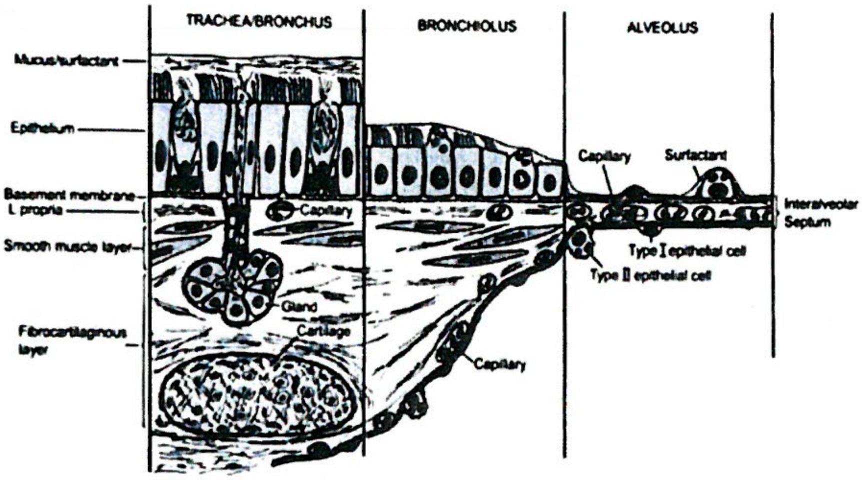
2. Acinar airways/terminal respiratory unit :
    - Last 8 generations of airways.
    - Involved in gas exchange.
    - Acinus : Parenchymal unit in which all airways have alveoli attached to their wall and thus participate in gas exchange.



Bronchiole :

- Silent zone of lung.
- Bronchiole is a non cartilaginous airway < 2mm in diameter.
- Terminal bronchiole → Respiratory bronchiole → Alveolar duct and alveoli.
- Upto and including terminal bronchiole is the conducting zone → Involved in conducting air not in gas exchange.
- From the respiratory bronchiole onwards : Respiratory zone starts.

Airway Generation	Generations	Characteristic	Role	TRU
Trachea	0	Cartilaginous	Conducting	
Bronchi	1-3	Cartilaginous	Conducting	
Bronchioles	4-13	Membranous	Conducting	
Terminal bronchioles	14	Membranous	Conducting	
Respiratory bronchioles	16-18	Partially membranous	Partially conducting and gas exchange	Yes
Alveolar ducts	19-22		Gas exchange	Yes
Alveoli	23		Gas exchange	Yes

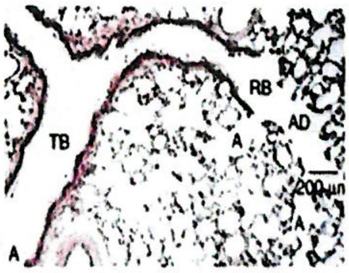


Bronchus vs bronchiole :

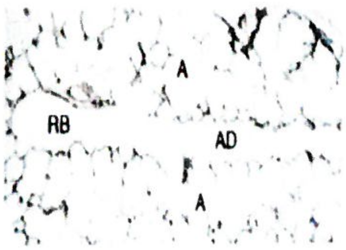
Bronchus	Bronchiole
• Cartilaginous rings present	• No cartilage.
• Ciliated columnar epithelium.	• Ciliated cuboidal epithelium.
• Goblet cells present.	• No goblet cells.
• Submucosal glands present.	• No gland, Instead club cell present.
• Smooth muscle present.	• Smooth muscle present.

Club cell :

- Previously known as clara cell.
- Non-ciliated cell in bronchiole.
- Function :
  a. Produces CCSP (Club cell secretory protein).
  b. Produces surfactant components.
  c. Xenobiotics.



Terminal bronchiole



Respiratory bronchiole

Features :

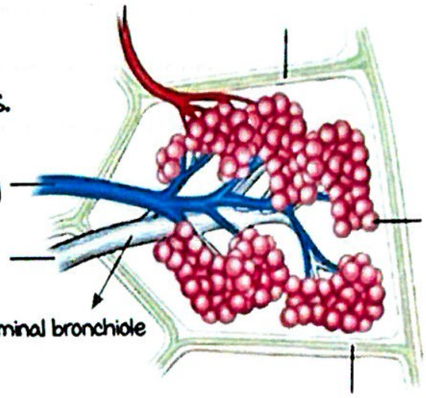
Functional unit of lung.

Enclosed by connective tissue septa on all sides.

Contains 3-5 terminal bronchioles.

Interlobular septum made of :

- Connective tissue septa.
- Lymphatic channels.
- Pulmonary veins.



Broncho-arterial unit :

- Each lobule has its own bronchiole and arterial supply.

Dimensions and geometry :

- Pentagonal in shape.
- Size : 5-2 cm.
- 30 acini present.
- 1500-4000 alveoli present.

Components :

- Septal structures :
  - a. Consists of lymphatic channels and veins.
  - b. 0.1 mm thickness in the periphery.
  - c. Diseases causing its thickening : Pulmonary edema, lymphangitis carcinomatosis.
- Centrilobular structures : Include bronchiole, lymphatics and 3° artery enclosed in connectve tissue sheath called bronchovascular bundle.
- Lobular parenchymal structures :
  - a. Consists of acinus and alveoli.
  - b. Peripheral lymphatics

Centrilobular structures

CT picture

Leads to hypoxemia due to diffusion defect across the alveolar capillary membrane.

Type II alveolar epithelial cells :

- Defensive cell of alveoli.
- Seen at the corners of alveoli, solitary cell.
- more number of organelles : Lamellar bodies (Intracellular storage form of surfactants).
- Functions : Reperative action, produces surfactant (B and C : Protection and stabilization of alveoli, A and D : Immunological and protective function).

Clinical significance of surfactant :

Respiratory distress syndrome (RDS) :

- Deficiency of surfactant in the immature lungs of premature neonate.
- Rx : Exogenous surfactant replacement therapy.

Pulmonary alveolar protenosis : Inefficient catabolism and excess deposition of surfactant.

Interstitium

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Features :

- Collection of supporting tissues within the lung.
- Provide supporting framework for the delicate alveolar sacs.
- Consists of : Elastic fibers and bundle of collagen fibrils in an extracellular matrix.
- Interstitial cells : Fibroblasts and contractile cells.
- Cells that are part of defense system : Interstitial marcrophages and mast cells.

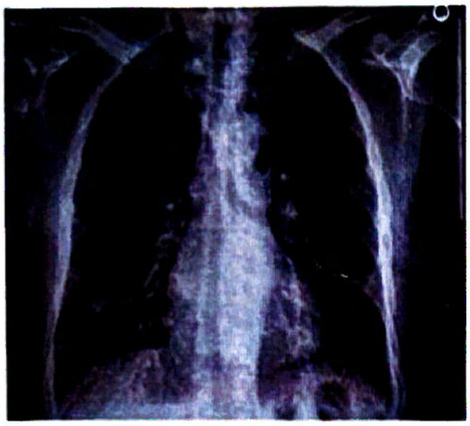
Types :

1. Axial/peribronchovascular interstitium.
2. Paranchymal or intralobular interstitium.
3. Subpleural or peripheral interstitium.

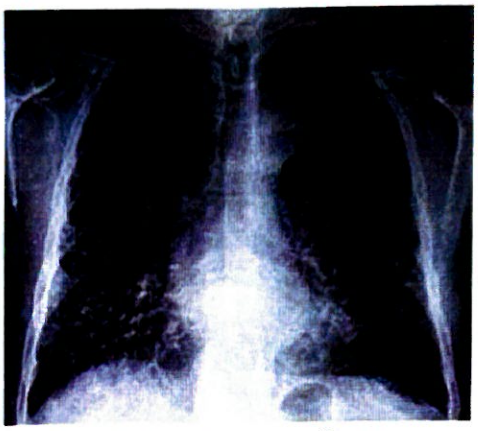
Flluid balance in interstitium :

Fluid formed at higher rate and not cleared adequately → Interstitial edema.

Chest x ray : Kerley B lines.



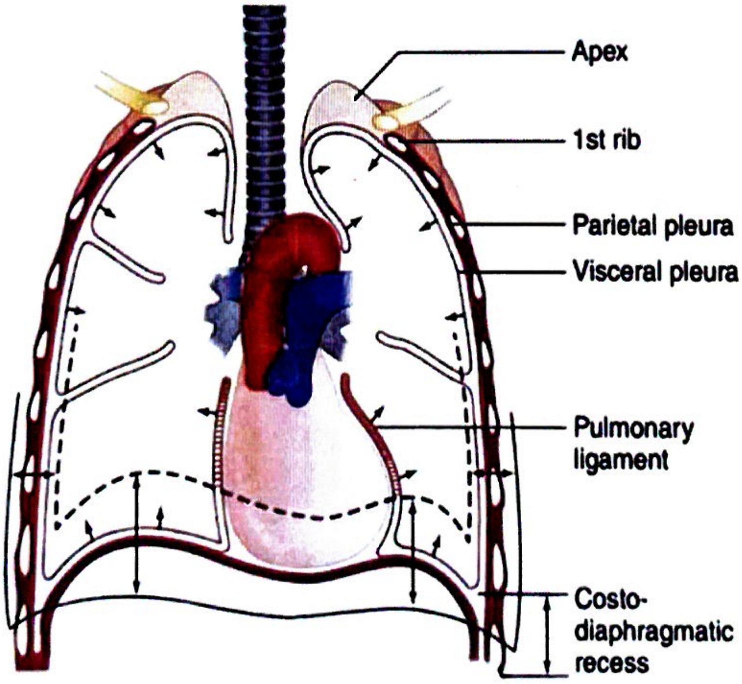
Pulmonary edema



Idiopathic pulmonary fibrosis

Pulmonary ligament:

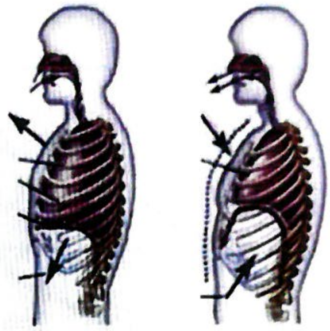
- Connects the visceral pleura of the lung to the mediastinum.
- Extends from hilum to diaphragm.
- Also called triangular ligament.
- Usually not seen in a chest X ray.



PULMONARY MECHANICS I

Introduction 00:01:36

Pulmonary mechanics :
It is the study of movement of gas in and out of respiratory system which depend on physical laws that govern pressure, volume and flow.



Normal breathing.

It helps in understanding :
- Pathophysiology of diseases.
- Severity of diseases.
- management of pulmonary disease :
  ARDS → Lung protective ventilation.

Normal breathing :

Inspiration :
- Diaphragm contracts → Apex-base diameter of lung ↑.
- Respiratory muscles contract → Outward movement of chest wall.
- Lung volume ↑ → Air moves inside lungs.
- Visceral and parietal pleura are in close approximation with each other.

Expiration :
- Passive process.
- Diaphragm relaxes back to normal dome shape position along with chest wall.
- Elastic recoil of lung → Forces the air out.

Pressure relationship : 00:05:56

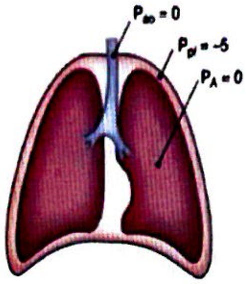
At end-expiration :
- No air flow.
- (Pressure at airway opening) $P_{ao} = 0$, (Pressure at alveoli) $P_A = 0$.
- (Pleural pressure) $P_{Pl} = -5$ cmH_2O (Subatmospheric, opposite tendency of chest wall to expand and elastic recoil of lung).

At inspiration :
- $P_{Pl} = -8$ cmH_2O, contraction of respiratory muscles → Push out chest wall.

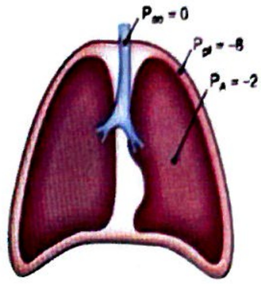
- P_A = -2 cmH_2O, a portion of negative pleural pressure transferred to alveoli.
- Tranpulmonary pressure : +6 cmH_2O.
- Air moves from high pressure to low pressure.

Transpulmonary pressure (TPP) :

- Difference between alveolar (PA) and pleural (Ppl) pressures.
- Net distending pressure applied to the lung.
- By contraction of inspiratory muscles or by positive-pressure ventilation.
- In a normal spontaneously breathing person TPP is always positive → Keeps lung expanded.
- In pneumothorax, TPP is 0 and lung collapses.



At end expiration.



At inspiration.

Expiration :

Passive process.

Diaphragmatic relaxation, elastic recoil of lung.

In cases of airway obstruction → Complete expiration does not happen, contraction of expiratory muscles occurs (Active process).

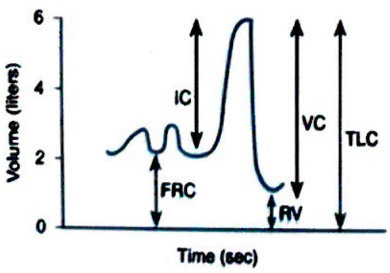
Flow : Volume of gas in unit time.

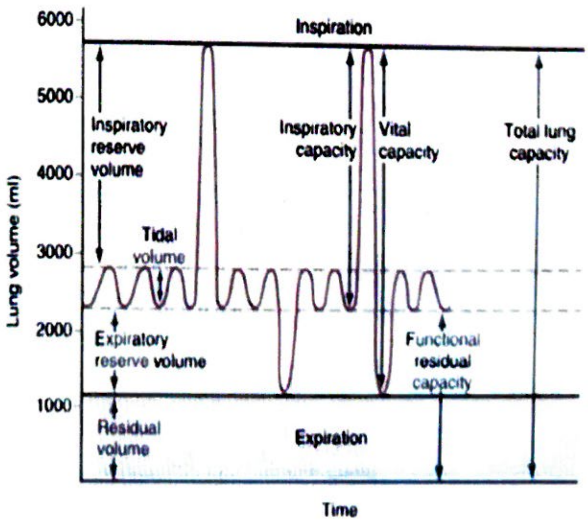
Volume : Space occupied by a gas.

Determined by temperature and pressure.

measured by spirometry.

Lung volumes : 00:16:26





Lung volumes.

Tidal volume : Volume of air drawn into the lungs during inspiration from the end-expiratory position during quiet breathing.
Normal value : 500 mL.

Expiratory reserve volume (ERV) : maximum volume of air that can be forcibly exhaled after a quiet expiration has been completed.
ERV : 0.7-1.1 Litre.

Residual volume (RV) : Volume of air that remain lungs after a maximal expiratory effort.
RV : 1.1-1.2 Litre.
In emphysema, RV ↑ (Obstructive airway defect).

Functional residual capacity (FRC) : Volume of air that remain in lungs at the end of normal expiration.
Elastic recoil of lungs = Expansion of chest wall at FRC.
FRC= ERV + RV.
1.8-2.3 Litres.

Inspiratory reserve volume (IRV) : Extra air that can be taken in addition to tidal volume.
IRV : 1.9-3 Litres.

Inspiratory capacity (IC) : maximum volume of air that can be inhaled from the end-expiratory position.
TV + IRV.
IC : 2.4-3.5 Litres.

Total lung capacity (TLC) : Total volume of air contained in the lungs at the end of a maximum inspiration.
TLC : 4.2-5.8 Litres.

Vital capacity (VC) : Volume of air that is exhaled by a maximum expiration after a maximum inspiration.
VC : 3.1-4.6 Litres.

Elastic property of lungs

00:26:47

Compliance : Distensibility of lung, easiness to inflate the lung.

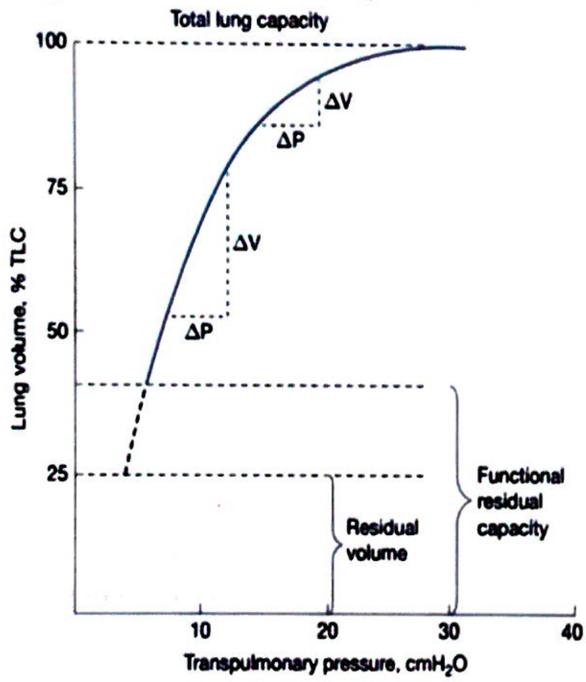
$$\text{Compliance} = \frac{\text{Change in lung volume}}{\text{Change in transpulmonary pressure}}$$

$$C = \Delta V / \Delta(P_A - P_{pl})$$

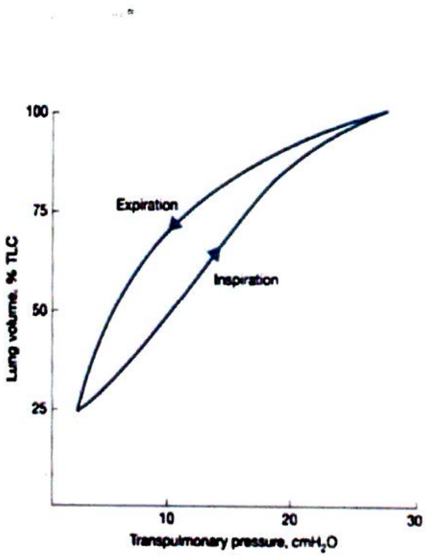
Elastance : Ability to resist transformation or any distorting force.

If lung volume near RV, compliance ↑.

If lung volume near TLC, compliance ↓.



Compliance.



Hysteresis.

Hysteresis : 00:30:49

Difference in pressure-volume relationship during inspiration and expiration.

Depends on elastic recoil nature of lung and surface tension of lung.

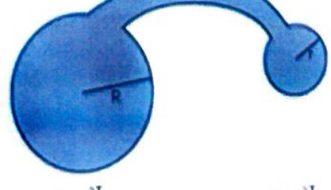
Surfactant :

Alveoli take the shape of sphere and take the minimum possible volume, have tendency to collapse.

Pressure inside a smaller alveoli >large alveoli, leads to inequality of ventilation.

Surfactant reduces surface tension and helps in maintaining equality of ventilation.

Composition : D-palmitoyl phosphatidyl choline.



$P_1 = \frac{2T}{R}$ $P_2 = \frac{2T}{r}$

Role of surfactant.

Functions :

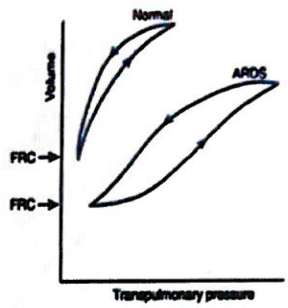
- ↓ Surface tension.
- ↓ Atlectasis of lung.
- ↓ Transpulmonary pressure.

more pressure is required for maintaining lung volume during inspiration than in expiration :

- During the beginning of inspiration, most of the airways and alveoli are completely collapsed.
- Surfactant is less effective in reducing the surface tension during inspiration.

Volume pressure relationship in normal lung vs ARDS lung :

- ARDS lung is less complaint.
- Widening of hysteresis curve : Requires more pressure during inspiration to open up the alveoli.



Normal vs ARDS lung.

Elastic recoil force of lung :

Structures with property of elastic recoil :

- Pleura.
- Interlobular septum.
- Connective tissue.
- Alveoli.

Components of connective tissue :

Elastin : Elastic recoil.

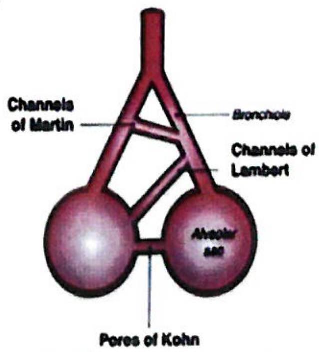
Collagen : Limit the over-expansion of lung (Good tensile strength).

Changes in compliance :

- With ageing : Elastic property decreases, ↑ compliance.
- In emphysema : Elastic fibers destruction → ↓ Elastic recoil → Air trapping → ↑RV and hyperinflation.
- Interstitial fibrosis : ↓ Compliance.

Complete collapse of one alveoli is prevented by :

1. Surfactant.
2. Interdependence of alveoli.
3. Collateral communication.



Collateral communication.

Collateral communication :

Pores of Kohn : Intercommunication between alveoli.

Canals of lambert : Communication between adjacent bronchiole and alveoli.

martins canal : Interbronchiolar communication.

Elastic property of thorax

00:45:01

Chest wall has the tendency to expand at lower lung volumes.

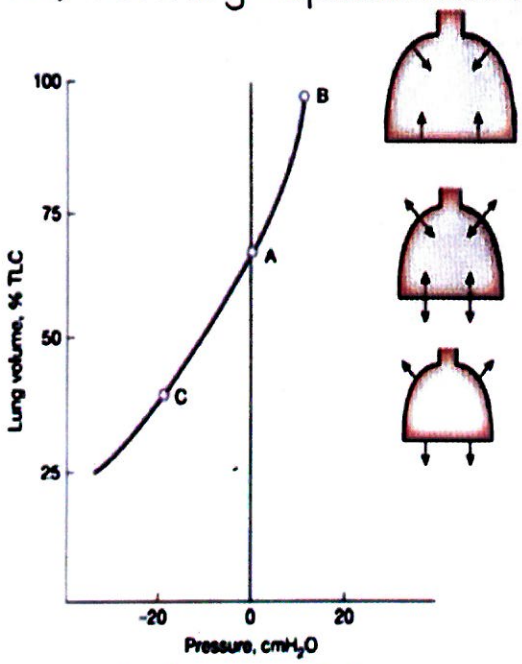
Chest wall has the tendency to recoil when at TLC.

Elastic property ↓ in chest wall diseases :

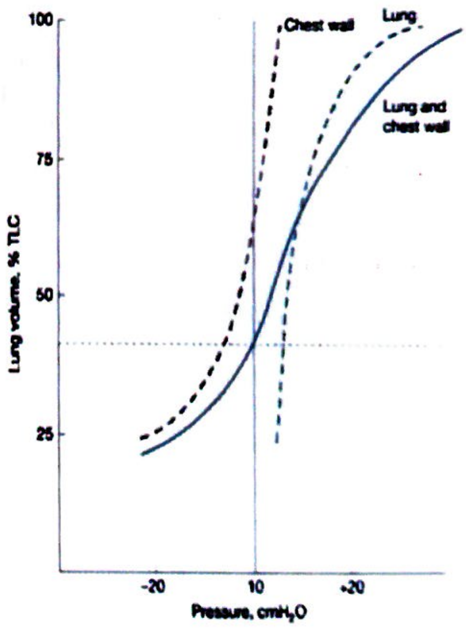
1. Kyphoscoliosis.
2. Ankylosing spondylitis.
3. Obesity.

Relaxation pressure-volume curve :

At FRC, recoil of lung = Expansive nature of chest wall.



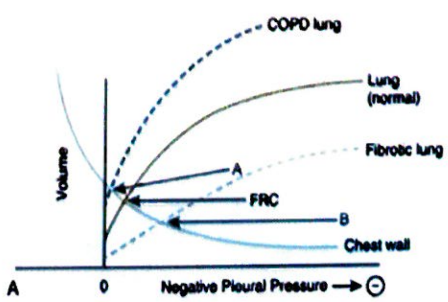
Elastic property of thorax.



Relaxation pressure-volume curve.

Campbell diagram :

- Between negative pleural pressure and volume.
- In COPD : FRC↑.
- In interstitial lung disease (ILD) or lung fibrosis : FRC↓.



Campbell diagram.

Total elastic property of the respiratory system :

$(P_A - P_{pl}) + (P_{pl} - P_{bs}) = P_A - P_{bs}$.

PA-Ppl : Transpulmonary pressure represents elastic property of lungs.

Ppl-Pbs : Elastic property of chest wall.

Pbs : Pressure at body surface (Atmospheric pressure).

Dynamic mechanical properties of respiratory system :

- Resistance : Pressure required to maintain a flow.
- Airway flow resistance : 80% Pressure required to overcome resistance to gas flow through the airways.
- Pulmonary tissue resistance : 20%, in patients with ILD/parenchymal lung disease tissue resistance is high.
- major resistance to airflow : upper respiratory tract, nose constitutes 50 %.
- Remainder of airway resistance : Lobar, segmental and subsegmental bronchi.
- Airway resistance in bronchiole is lesser than in bronchus : more cross sectional area of bronchioles.

Factors affecting airway resistance : 00:56:10

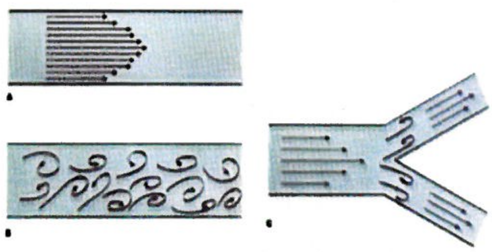
1. Diameter of airway : Inversely proportional.
2. Lung volume : Inversely proportional.
3. Density and viscosity of gas.
4. Flow pattern.
5. mucosal edema, smooth muscle hypertrophy.
6. Autonomic nervous system : β_2 receptors (↓), cholinergic system (↑).
7. Vasoactive intestinal peptide (VIP), nitric oxide : Reduce resistance.

Patterns of airflow :

A : Laminar airflow in small airways.

B : Turbulent airflow in major airways.

C : mixed flow pattern at the point of bifurcation.



Patterns of airflow

Poiseullie's equation : 01:01:32

V : Flow, η : Viscosity of gas.

r : Radius, l : Length of tube.

$$\Delta P = \frac{V 8 \eta l}{\pi r 4}$$

$$\text{Flow} = \frac{\pi \times \text{radius4} \times \Delta l}{8 \times \text{length} \times \text{visc}}$$

Pressure is inversely proportional to 4^{th} power of radius.

If radius is reduced by 1/2, pressure is increased by 16 fold.

Reynolds number (Re) : 01:03:08

Dimensionless number.

Re = $v D \rho / \eta$.

v : Velocity, D : Diameter of tube, ρ : Density of gas, η : Viscosity of gas.

- <2000 : Laminar flow.
- >4000 : Turbulent flow.
- 2000-4000 : mixed flow.

Heliox :

- mixture of 80% helium and 20% oxygen.
- Density is 66 times less dense than air.
- more laminar flow.

PULMONARY MECHANICS II

Strain 00:00:11

Definition :

Change in shape or size due to force acting upon it.

In aspects of lung, strain is change in tidal volume with respect to functional residual capacity (ΔV/FRC).

Clinical application :

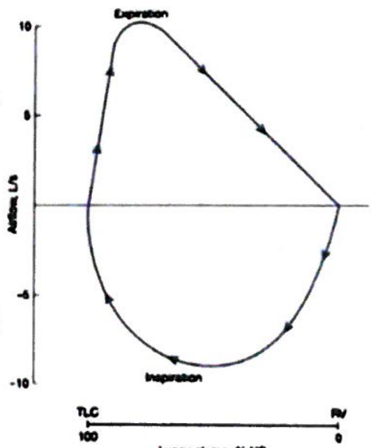
In ARDS : Low tidal ventilation is given.

Low tidal ventilation → ↓Over expansion of lungs → ↓Strain of alveoli → ↓Ventilator induced lung injury.

Airflow 00:02:16

Flow volume relationship :

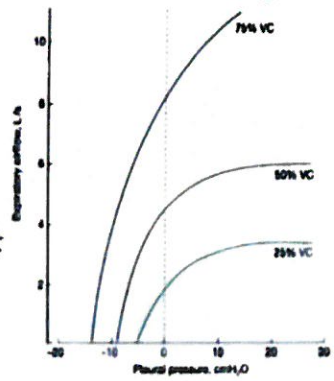
- Airflow in Y axis and lung volume in X axis.
- Normally inspiration is at negative side and expiration at positive side in the flow volume loop.
- In mechanical ventilator, inspiration is at positive side and expiration is at negative side.
- Expiration attains the maximum level called peak expiratory flow.



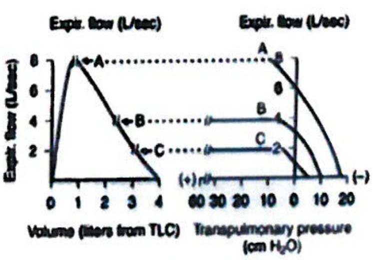
Flow volume loop.

Expiratory airflow vs pleural pressure :

- At high lung volume, airflow is effort dependent.
- At lower lung volume (50%/25% vital capacity) airflow is independent of effort.
- Expiratory airflow does not increase after a particular threshold however high the effort is given.



Airflow vs pleural pressure.



Expiratory flow.

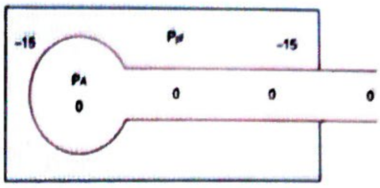
Reason for airflow not increasing beyond a limit with effort :

1. Equal pressure point.
2. Bernoullie principle.

Equal pressure point : 00:06:49

No airflow at the end of expiration :

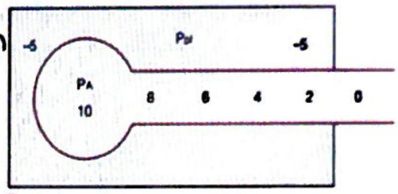
- Pressure within the alveoli and tube is 0.
- Pleural pressure is sub atmospheric pressure(-15).



No airflow at the end of expiration.

During quite expiration :

- Pleural pressure is less sub atmospheric (-5).
- Expiration happens by relaxation of diaphragm and chest wall coming to normal position → Positive airway pressure within the alveoli (10).
- Pressure in the airway is more than pleural pressure.

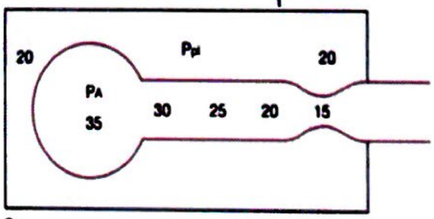


B

Quite expiration.

Forceful expiration :

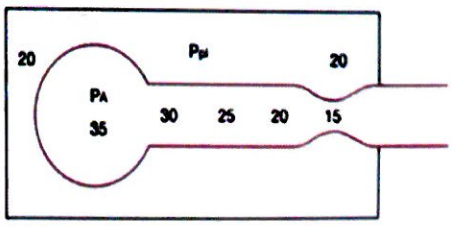
- During forceful expiration expiratory muscles contract→ Positive pleural pressure.
- Point at which the pressure in the airway is equal to pleural pressure is called equal point pressure (EPP).
- Beyond EPP there is dynamic compression of airway.



C

Forceful expiration.

Equal pressure point divides the alveoli into upstream and down stream segments. Upstream is more towards alveoli and downstream is more towards the mouth.



C

Equal pressure point.

Bernoulli effect :

When gas flows through the tube, velocity is inversely proportional to pressure. In high velocity flow, the pressure will be low → Perpendicular forces act more in collapsable airway.

Practical application of equal pressure point :

1. In normal patients equal pressure point is at the lobar/segmental bronchi which has cartilagenous support.

2. COPD :

- D/t narrowing of airways → Pressure dissipation is fast and equal pressure point is seen at terminal bronchiole.
- COPD patients should not have fast expiration.
- Pursed lip breathing : Patient exhales very slowly without any extra expiratory effort → No equal pressure point formation in peripheral airways.

Mechanical determinants of regional ventilation 00:18:02

Ventilation is inhomogenous in normal individuals.

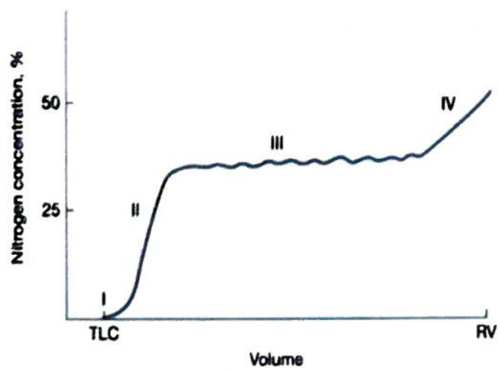
Pleural pressure :

- Pleural pressure is more negative in the apex when compared to the base due to gravity and weight of the lung.
- There is change of 0.25 cm H_2O pressure for every 1 cm distance from the apex of lung to the base.

Clinical implication : D/t more negative pleural pressure at the apex, alveoli are more distended at the apex than base → more chance of developing blebs/bullae at the apex

N_2 washout method : 00:21:34

- During a breath taken from residual volume, air is preferentially distributed to the apex because most of the alveoli are collapsed so the air is distributed to apex → This forms the basis for N_2 washout method.
- Patient is asked to inspire pure O_2 from residual volume and expire in a meter. The meter measures the N_2 content in the exhaled air



N_2 washout method

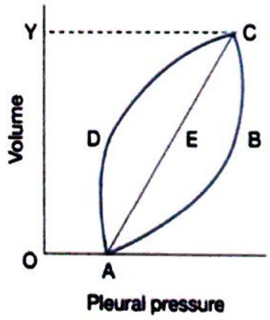
- Phase I → No significant N_2 comes out during initial part of expiration (Air from upper airway).
- Phase II → Rising N_2 concentration.
- Phase III → Plateau phase.
- Closing volume : Point at which the N_2 start rising → Volume that indicates the closure of the airways of the base of the lung.
- After the closing volume, N_2 comes from apex of the lung.

Significance of closing volume :

- In normal individuals, closing volume is 10% of vital capacity.
- At 65 years of age, closing volume is 40% of vital capacity.
- Disease of smaller airways → Closing volume increases.
- Closing capacity : Closing volume + Residual volume.

Dynamic compliance of lungs : 00:25:11

- Slope of the curve AEC represents the dynamic compliance of the lung.
- At normal breathing frequencies, dynamic compliance = Static compliance in normal lungs.
- In diseased state, dynamic compliance may be lower than static compliance, particularly at high breathing frequencies.



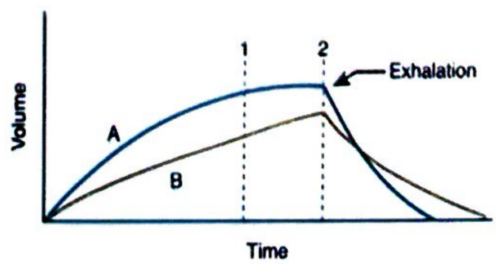
Dynamic compliance of lung.

Time constant :

- Rate of filling and emptying of a lung unit depends on its time constant.
- Time constant = Resistance x Compliance.
- Lung unit with high time constant will have high resistance and high compliance.

Clinical implications :

- In ARDS, time constant of different lung units will be different.
- When respiratory rate is high, the lung module with higher time constant will not have adequate inspiration time to be filled up → Ventilation perfusion mismatch.
- Lung unit with higher time constant will not have adequate time to expire



Time constant.

air → Air trapping present → Intrinsic PEEP.

Intrinsic PEEP : 00:30:50

Positive end expiratory pressure.

Significance : It is due to incomplete emptying of lung → Air trapping → Increased work of breathing.

COPD patients :

- Chances of developing intrinsic PEEP are high .
- Air trapping → Dome shaped lungs become flat → Reduced contractility of lung → Exertional dyspnea.

mechanically ventilated patient :

- Significant intrinsic PEEP present → Triggering asynchrony.
- Difficulty in weaning.

Intrinsic PEEP can be avoided :

- In normal individuals : Bronchodilators can be given to reduce air trapping.
- In mechanically ventilated patients : Increase expiratory time/decrease tidal volume/decrease rate of breathing and treat the underlying bronchospasm.

Dynamic hyperinflation :

Patient with expiratory airflow limitation while exercising → Air trapping will be more significant → more flattening of diaphragm → ↓Exercise capacity and increased work of breathing.

Clinical importance :

- Dyspnea on exertion in COPD patients.
- This can be treated by endobronchial valve which will reduce air trapping by uploading air into the lung unit that has air trapping.

PEEP :

- Positive end expiratory pressure.
- Two types : Intrinsic and extrinsic PEEP.
- Intrinsic PEEP : Developed within the patient.
- Extrinsic PEEP : PEEP set in ventilator.

Plateau pressure :

- Pressure in lungs when there is no airflow at the end of inspiration.

- According to ARDS guidelines : Target plateau pressure <30 cm H_2O is required to avoid ventilator induced lung injury.

Pressure required to ventilate the lungs :
Pressure to distend respiratory system + Pressure to maintain gas flow
= (Δvolume/compliance) + (Flow x resistance).
In case of ARDS with increased resistance and COPD with decreased compliance : Increased pressure is required to ventilate .

Driving pressure :
- Actual distending pressure of lungs.
- Driving Pressure = Tidal volume/compliance.
- Plateau pressure - PEEP = Tidal volume/compliance.
- Driving pressure <14 cm H_2O will reduce the mortality.

Mechanics in ARDS : 00:39:23
Changes in ARDS :
- Decrease in FRC.
- Reduced compliance.
- Damage to surfactant.
- Ventilation perfusion mismatch (Atelectasis).
- Significant shunting.

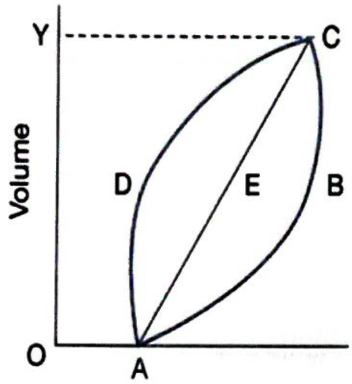
Lung protective ventilation :
- Tidal volume of 6mL/kg and plateau pressure of <30 cm H_2O : Reduce ventilator induced lung injury.
- Lung protective ventilation reduces the strain in ARDS.
- Time constant in ARDS is variable : Inhomogenous lung → Higher tidal volume → Over distension of lungs → Rupture of alveoli/pneumothorax/ pneumomediastinum → Ventilator induced lung injury.
- Lung protective ventilation prevents ventilator induced lung injury.

Work of breathing 00:43:40

Two types of work of breathing : Elastic work and resistive work.
Elastic work : Work done to overcome the elastic recoil of the lung.
Resistive work : Work done to overcome the resistance of airways.

Pattern of breathing :

- It depends on tidal volume and respiratory rate.
- Elastic work of breathing depends upon tidal volume.
- Resistive work of breathing depends on rate of respiration.
- ILD patients : Elastic work of breathing ↑ → ↓ Tidal volume (Shallow and rapid breathing).
- COPD patient : Resistive work of breathing ↑ → ↓ Rate of breathing (Deep and slow breathing).



In the above graph : Line connecting OAECY represents elastic work of breathing and line connecting AECB represents resistive work of breathing.

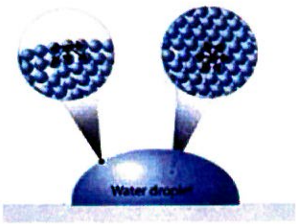
Oxygen cost of breathing :

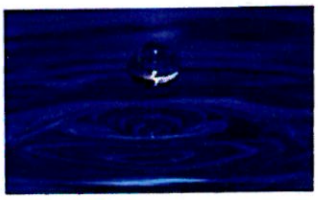
- Reflects the energy requirements of respiratory muscles.
- Indirect measure of work of breathing.
- 1 mL/L of ventilation (Fishman), 0.25 –0.5mL/L (murray).
- Constitutes <5% of total O_2 consumption.
- Increase with rate and depth of ventilation and in respiratory diseases.

SURFACTANTS

Introduction 00:01:00

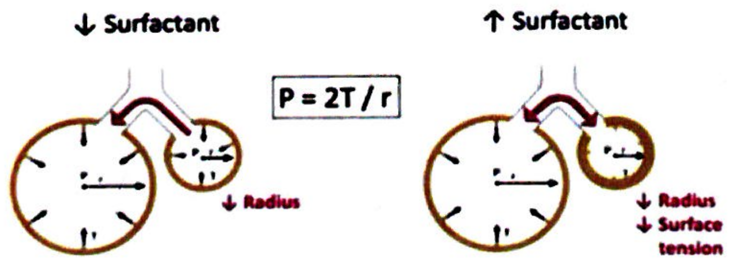
Surface tension : Force experienced over the surface of a liquid due to attractive property of the molecules in the liquid, such that it's surface area is reduced to the minimum.





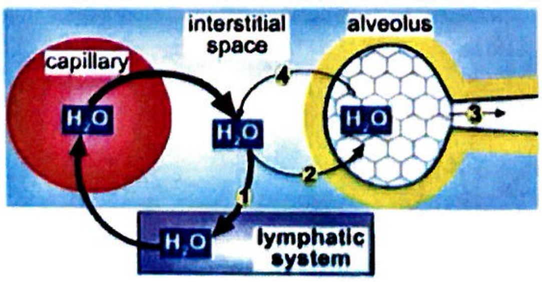
Surfactant :

- Reduces the surface tension in alveoli → ↑ size of alveoli.
- Complex mixture of phospholipids and proteins.
- Create interface between alveolar gas and fluids.
- Prevent end expiratory collapse in alveoli.
- Produced by type II alveolar epithelial cells.



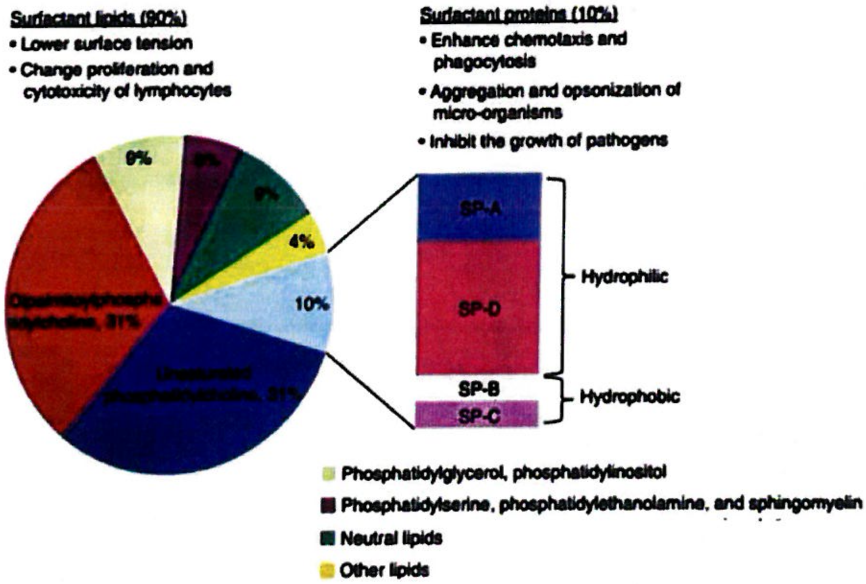
- In lungs : Equalizes the pressure difference among alveoli → maintains the uniformity in size among alveoli.

approximate fluid volume in the lung		
capillaries	interstitial space	alveoli
~ 65 ml	< 500 ml	~ 36 ml



Composition

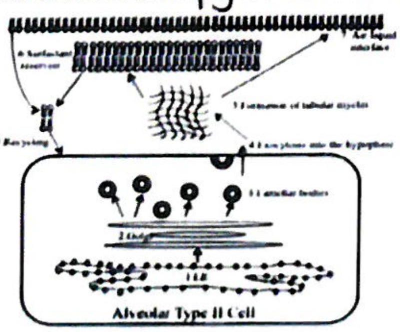
- PL (Phospholipids) + protein.
- PL: 80–90 %, PC (phosphatidyl choline) : 70–80 % and PG (phosphatidyl glycerol) : 5–10 %.
- DPPC (dipalmitoylphosphatidylcholine) : most abundant PC.
- POPG (phosphatidyl oleyl phosphoglycerol) : most abundant PG → Host defence.
- Proteins : 5–15 % (surfactant protein) SP-A, SP-B, SP-C, SP-D.
- Lipids and proteins are synthesized in ER.

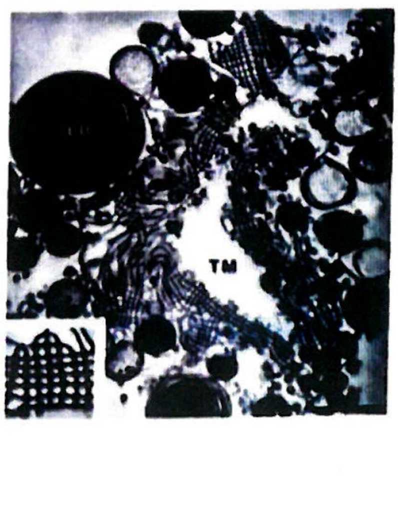


Forms of surfactant :

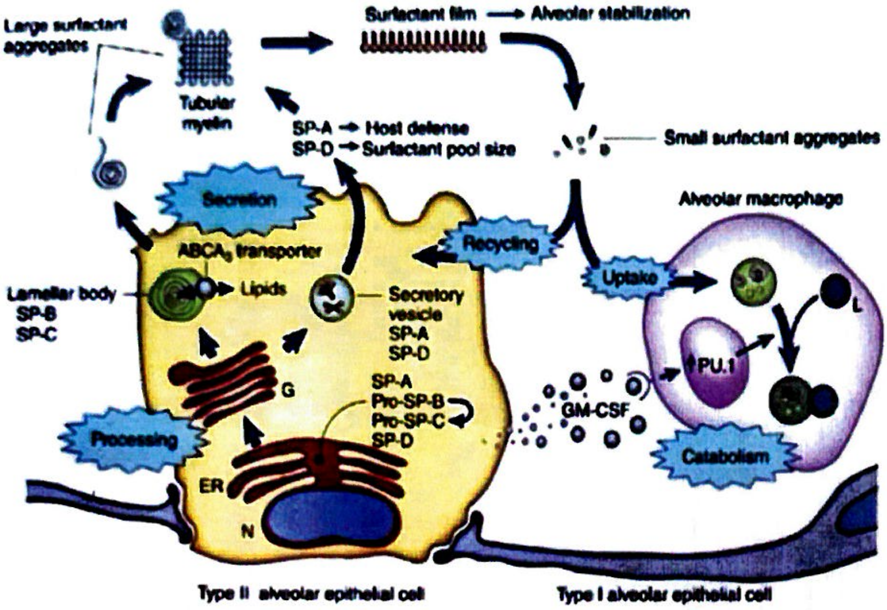
- Tubular myelin : most abundant form, extracellular in alveoli.
- Lamellar body : Intracellular storage form.
- Small aggregates.

Electronmicroscopy of surfactant forms :





Production of surfactant by type II alveolar epithelial cells :



Types of surfactant proteins 00:12:40

SP-A and SP-D
Hydrophilic.
• Innate host defense of lung. • Opsonins and activate alveolar macrophage. • Influence surfactant metabolism.

SP-B and SP-C
Hydrophobic.
Stability of alveoli.

1. SP-B :
   - Chromosome 2.
   - Produced by type 2 alveolar cells and non ciliated bronchiolar cells.
   - Shape : Amphipathic alfa helix.
   - Function : Alveolar stability.
   - AR mutation in Exon4 → deficiency of SP-B → Respiratory failure.
   - Refractory to surfactant replacement : Early death.
   - Pro-SP-C accumulation → Alveolar proteinosis.

2. SP-C :

- Chromosome-8.
- Produced by : Type 2 alveolar cells only.
- Shape : Alfa helix.
- Function : Alveolar stability.
- AD mutation → Respiratory failure → Infantile ILD, IPF.
- No effective treatment.

3. ABCA 3 :

- membrane protein in lamellar body : Involved in migration of lamellar body outside the cell.
- AR mutation.
- mutation causes : Respiratory distress in infants and ILD.
- Not responsive to therapy.

4. NKX2-1 gene :

- Transcriptional regulator of TTF-1.
- TTF-1 : Expressed in lung, CNS, and thyroid.
- Regulate embryonic lung development and surfactant.
- mutation causes : Respiratory failure, ILD, congenital hypothyroidism, and CNS defects .
- Brain-lung-thyroid syndrome.

5. SP-A :

- Chromosome 10.
- Produced by : Alveolar cells and non-ciliated bronchiolar cells.
- Function : Lung defense.
- AD mutation.
- Susceptible to infections, develop ILD by 5-7 th decade and high risk of lung adenocarcinoma.
- Adult onset IPF.

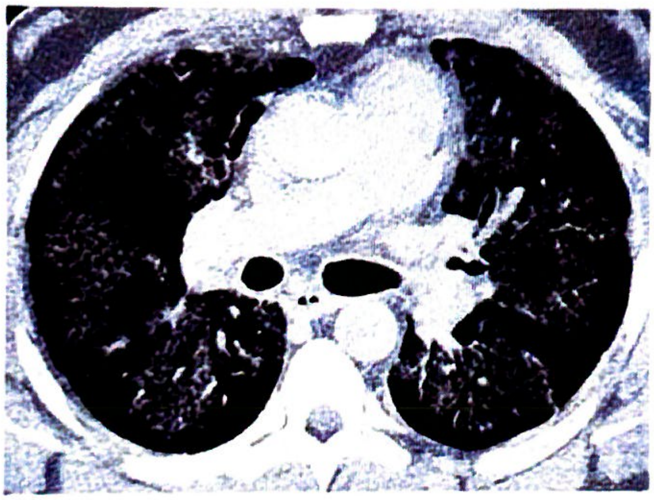
6. SP-D :

- Chromosome 10.
- Produced by : Alveolar cells and non-ciliated bronchiolar cells .
- Function : Lung defense.

- mutation : Susceptible to infections.
- No association with ILD, IRDS.

Production and recycling 00:22:44

- Increase markedly in late gestation.
- Enhanced by glucocorticoids, EGF, cAMP.
- Inhibited by TNFα, TGFβ, insulin.
- Catabolized by alveolar macrophages under regulation of GM-CSF.
- mutation of GM-CSF : Pulmonary alveolar proteinosis (PAP).



PAP :

- Excessive deposition of surfactant in alveoli, presents with dyspnea on exertion and non-productive cough.
- CT : Crazy pavement pattern (also in pneumocystis pneumonia).

IRDS 00:24:17

- Infantile respiratory distress syndrome.
- Associated with prematurity, low surfactant in lung.
- Risk reduces with gestational age.
- Causes alveolar-capillary leak.
- Atelectasis → hemorrhage → hypoxemia.
- Fetal lung maturity assessment : L/S ratio and lamellar body count.