

SOL: COPD Pathology

Chronic Disease (Pulmonary)

= divided into 2 types

→ obstructive

→ Restrictive

Obstructive Lung Disease

Struggle to exhale

Associated → cigarette smoking

progressive loss of lung function

↑ Dyspnoea

Hypoxia

Resp failure

Cor pulmonale - NB

Lung Fr → ↓ FEV₁

Conditions

Chronic Bronchitis

Emphysema

Asthma

Small airway disease (Bronchitis)

Restrictive Lung Disease:

↓ Expansion of parenchyma

↓ TLC

FEV₁ = ↑/normal

→ chest wall disorders

- neuromuscular / obesity / pleural / kyphoscoliosis

→ Chronic interstitial & infiltrative disease

- pneumoconiosis / Interstitial fibrosis

COPD

mainly

Chronic bronchitis

Emphysema

⇒ Many have overlapping features of damage

Emphysema: Damage at alveoli: 1d = over-inflated

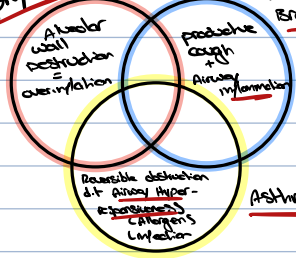
Chronic Bronchitis: Damage at Bronchi = productive cough

Asthma: Reversible bronchospasm

: Bronchial inflammation

: Can have an irreversible component

Emphysema



Emphysema

Irreversible

↑ Size distal terminal bronchiole

(Destruction Alveolar walls)

(without obs. fibrosis)

= massive Alveoli

= ↓ Surface area for Gas x O

Long term Heavy Smoking

(also pollution)

Pathological Classification:

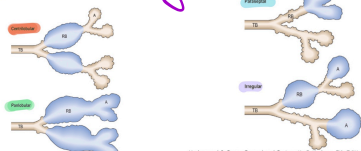
according to location within lobe

1) Centriacinar: middle (Resp Bronchiole)

2) Panacinar: Entire

3) Paraseptal: Peripheral Alveoli

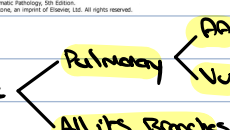
4) Irregular: combo of All 3



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Small airway disease (Bronchitis) Lung lobe?

Terminal Bronchiole



Centriacinar

Proximal part of lobe (R. Bronchiole)

Distal Alveoli = spared (relatively)

usually upper lobes

Heavy smokers

Panacinar

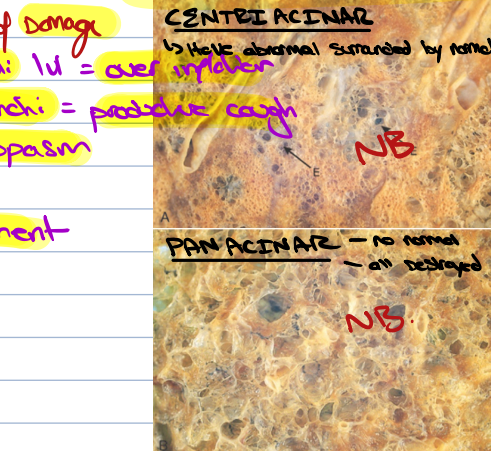
Acini = uniformly enlarged

lower zones of lung

α₁-Antitrypsin deficiency

↳ Enzyme that stops Elastase Action

↳ uninhibited = does more damage than should



Distal (paraseptal)

Proximal = normal

Distal = alveoli affected

↳ closest parts to pleura mostly

↳ multiple enlarged foci

- Esp upper parts of lung

- occurs Adj to areas of fibrosis / scarring / atelectasis

Irregular Emphysema

Acinus irregularly shaped

Scarring

Scar tissue → pulls adj alveoli →

Dilating of some alveoli → larger

Emphysema Pathogenesis

Result of destructive properties of

High protease levels

Disturbance of protease: Anti protease Ratio

Any insult that ↑ no. neutrophils

& macrophages in lung = more

protease production (↑)

Smoking attracts neutrophils (foreign matter)

↳ Smoking also disrupt oxidant: Antioxidant

Ratio (smoking = lots of free radicals)

= Deplete lung Anti-oxidants

→ Additional O's:

Goblet cell metaplasia = mucus plug

Inflammatory infiltration

Thickening of bronchiolar walls

↳ smooth muscle hypertrophy &

peribronchiolar fibrosis.

Fr: 1) voluminous lungs = over-inflated

2) mostly upper 2/3 affected

= Barrel chest

3) Apical Blebs / Bullae

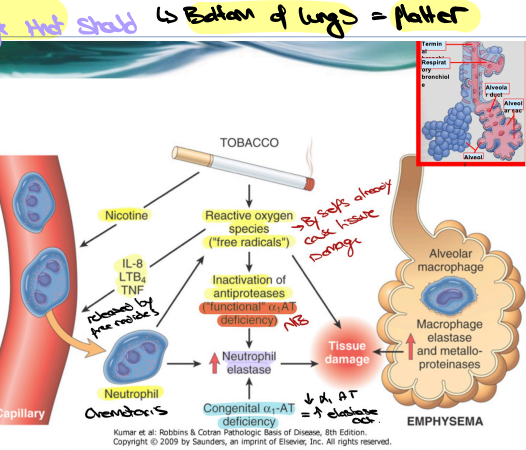
4) Abnormally large Alveoli

(loss of Alveolar septae)

5) ↓ capillary bed

CXR: ↓ shadow

↳ Bottom of lungs = flatter



Emphysema Presentation:

Dyspnoea cough wheezing

Prolonged Expiration

↓ RR (Deep Breathing)

Death d.t: ↑ pCO₂

- Resp Acidosis & coma

- Cor pulmonale

more elastic work

Bullae rupture

- Pneumothorax

Chronic Bronchitis

NBS Definition
Persistent cough
sputum production
3 months
2 consecutive years
Absence of ID cause

common in: smokers
 : urban smog areas

Complications:
 progress to **COPD**
car pulmonale

Metaplasia → Dysplasia → Malignancy

↳ Mostly smoking induced
 RR → Squamous
 (↓ mucociliary?)

Pathogenesis

- Irritant
- Hypersecretion of mucus in large Airways
- = Hypertrophy of submucosal glands in Trachea & Bronchi
- Chronic Bronchitis (with inflammation) develops
- overall = Airway obstruction (small bronchioles)
- = Parts of Acute Bronchitis (superinfection)
- => Airway obstruction / mucus oversecretion / chronic inflammation & fibrosis

Clinical

- Initially Asymptomatic
- Chronic cough
- Dyspnoea of exertion
- Dyspnoea or mild exertion
- COPD features: Hypercapnia (↑ CO₂)
- Hyperoxia (cyanosis) (↓ O₂)
- car pulmonale with Rt failure
- Spontaneous infections / pneumonia
- Death

Asthma

Reversible small airway obstruction

- Bronchospasm
- Inflammation
- oedema

Paroxysmal attacks
 over distended lungs

Enlarged Bronchial mucous glands with
 mucus plugs in Bronchi
 (Hyperplasia/hypertrophy)

Hypersensitivity mediated:

Chronic inflammation with variable
 bronchoconstriction & mucus secretion
 Night / Early morning (circadian rhythm)

NBS

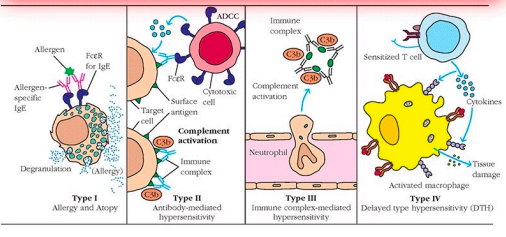
know
 Def.
 future
 Rares!

Clinical Categories of Asthma

- 1) Atopic (TI Hypersensitivity)
- 2) non-Atopic (Resp infections)
- 3) Aspirin-induced
- 4) occupational Asthma
- 5) Allergic Bronchopulmonary Aspergillosis
 - ↳ Aspergillus spores inhalation
 - = mucus plugs with hyper



Type I vs Type II vs Type III vs Type IV



Asthma → Early (Histamine)

→ Late (Eosinophils & lymphocytes)

Asthma Pathology

- over distended lungs
- Bronchi + Bronchioles = occluded mucus inflammation
- Airway walls = thick oedema
- (Smooth muscle hypertrophy)
- ↳ BSM thickened
- ↳ submucosal gland hypertrophy

Obstructive airway Disease Summary

- ↑ Difficulty to exhale
- Partial / complete obstruction of any level
- ↓ FEV₁ / FVC Ratio
- ↳ Chronic Bronchitis
- ↳ Asthma
- ↳ Emphysema
- ↳ Bronchiectasis
- ↳ small airway diseases: Bronchitis

