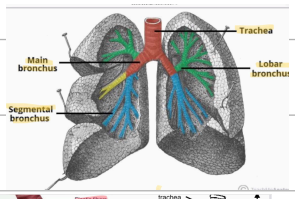
بسم الله الرحمن الرحيم

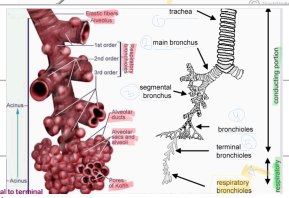
THE RESPIRATORY SYSTEM

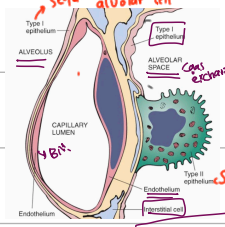
FUNCTION AND ANATOMY:

The major function of the lung is to replenish oxygen and remove carbon dioxide from blood.

(gas exchange)



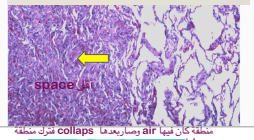




function ① Regenration after truma
② surface tension يقلل ال

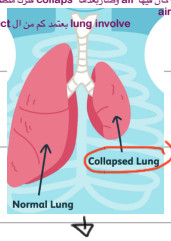
ATELECTASIS (COLLAPSE)

Loss of lung volume caused by inadequate expansion of the airspaces (collapse)



منطقة كان فيها air وصاربعدها collaps فترك منطقة airless

Effect بحسب كم من ال lung involve



① It results in shunting of inadequately oxygenated blood from pulmonary arteries into pulmonary veins aresulting in ventilation perfusion imbalance and hypoxia.

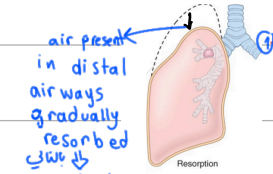
بقل ال oxygenated blood اي رجع → pulmonary veins

2.collapsed airway are at risk of infection

* ACQUIRED ATELECTASIS 3type.

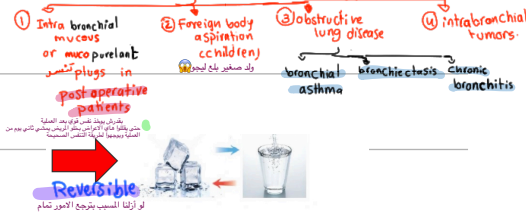
① • Resorption atelectasis

المتصلب



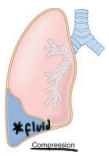
1* total obstruction of airway
bronchi → segmental bronchi → terminal bronchi

2*CAUSED BY: Obstruction of bronchus. (Most common) cause



② • Compression atelectasis

CAUSED BY:

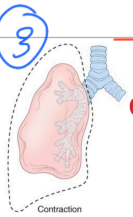


accumulation of significant amount of ① fluid
within plural cavity → Blood, exudate, transudat
② air → pneumothorax
③ tumor

mechanically collaps adjacent lung

eg: pleural effusion , pneumothorax
- pleural effusion → in Congestive Heart failure
- pneumothorax → air in the pleural cavity (RTA) road trophic accident truma أي

③ Contraction atelectasis (cicatrization atelectasis



CAUSED BY: local or generalized fibrosis (scarring) of lung or pleura → بسبب prevent full expansion of lung

irreversible

اللهم صل وسلم على نبينا محمد وعلى آله وصحبه أجمعين

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

Oxygen بيقل
Co2بيزيد عنده

- **ARDS defined as** respiratory failure occurring within 1 week of a known clinical insult with bilateral opacities on chest imaging, NOT fully explained by effusions, atelectasis, cardiac failure, or fluid overload.

* Sever end of spectrum of acute lung injury

عملت x.ray للchest
لقيت bilateral obesity
يعني اغلب الصورة لونها أبيض للlung2
ولاحظت انه مو cardiac cause ،بالتالي المشكلة
من lung it self

- graded based on the severity of the changes in arterial blood oxygenation into mild, moderate and sever

كل ما قل الo2
بتصير more sever

sever ARDS: Rapid onest life threating
- *respiratory insufficiency
- *cyanosis Blowish discalration mucus membran
- *severe arterial hypoxemia
  - *becomes refractory to oxygen therapy
  - *may progress to multisystem organ failure

بتعطيه o2 وما بستجيب
لانه الtruma موجودة بالtype1,2 pnemocyte
No cell of gas exchange

Sepsis,lung infectio,gastric aspiration,head truma(major trumaj

Table 15.2 Conditions Associated With Development of Acute Respiratory Distress Syndrome

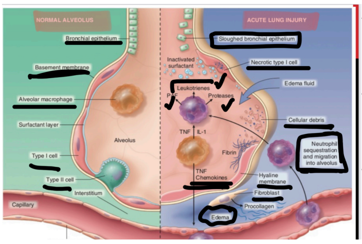
Infection
Sepsis[a]
Diffuse pulmonary infections[a]
Viral, *Mycoplasma*, and *Pneumocystis* pneumonia; miliary tuberculosis
Gastric aspiration[a]
Physical/Injury
Mechanical trauma including head injuries[a]
Pulmonary contusions
Near-drowning
Fractures with fat embolism
Burns
Ionizing radiation
Inhaled Irritants
Oxygen toxicity
Smoke
Irritant gases and chemicals
Chemical Injury
Heroin or methadone overdose
Acetylsalicylic acid
Barbiturate overdose
Paraquat
Hematologic Conditions
Transfusion-associated lung injury (TRALI)
Disseminated intravascular coagulation
Pancreatitis
Uremia
Cardiopulmonary Bypass
Hypersensitivity Reactions
Organic solvents
Drugs

[a]More than 50% of cases of acute respiratory distress syndrome are associated with these four conditions.

ARDS should not be confused with respiratory distress syndrome of the newborn; the latter is caused by a deficiency of surfactant caused by prematurity.

IRDS (familial)

PATHOGENESIS



1. Endothelial and epithelial injuri
   Compromised integrity of alveolar capillary membrane
2. releas IL8 / IL-1 / TNF → 30 minutes after acute insult by pulmonry macro phage

بعد ال30دقيقةمن الinjri ،بعدها الmacrophage
بتصير activated ممكن
Directlyأو indirectly
من خلال الinflamatory mediater

Mac release inflamatory mediator

3. leading to endothelial activation and sequestration.

Inflamatory mediator activate endothelial cell to express adhesion molecule حتى يساعدوا
الmigration neutrophil
Increase vascular permability
وهاد بسمح للprotein rich fluid
ويفوت على الalveoli

4. activated Nutrophils → release ROS, protease } counteracted by endogenous anti proteas, oxidants
   - damage the alveolar epithelium, endothelium
   - ينتج vascular leakiness and loss of surfactant (alveolar unitunable to expand)

Neutrophil عندها dustrctive forces ,
بتواجه المسبب:
Proteases,leukotrienes,PAF

صار في damge لل epithelial cell وال type 2 pneumocyte ,wall
فالfluid enter alveolusوهاد بغسل الsurfactant الي كانوا من قبل موجودين

بتكون thick membrane (hyaline membrane)
Protein,fliud,necrotic depre of epithelial cell بقلل الgas exchange

- **In the end, it is the balance between the destructive and protective factors that determines the degree of tissue injury and clinical severity of the ARDS.**

النتيجة عند المرضى بتحسم
بالblance بين مقاومة الجسم
والinflammatory reaction

CLINICAL FEATURES

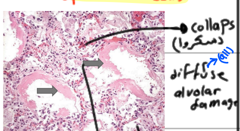
- dyspnea (بتنفس بسرعة بس deep مش breath)
- cyanosis بتيجي بعد tachypnea
- hypoxemia: refractory to oxygen therapy (مش بسبب ventilation perfusion mismatch)
- respiratory failure, respiratory acedosis
- diffuse bilateral infiltrates on radiographic examination

*Poor prognosis:
- advanced age
- bacterimia (sepsis)
- development of multi organ failure

HISTOLOGY: ARDS

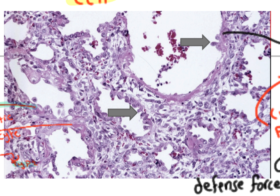
Diffuse alveolar damage (DAD)
لانه بتصير pathogenesis ل Both lung

acut phase
1. hyaline membrane: مكونة Fibrin-rich edema fluid admixed with remenants of necrotic epithelial cells



collaps (مسكرة), diffuse alvolar damage, dilated
Cnot line by: type 1 pnumocyte, Type 2 pnumocyte

organizing stage (healing ما بعد الإصابة)
1. proliferation of type II pnumocytes. ✓ (بدنا نعوض الي فقدناها)
2. intraalveolar fibrosis due to organization of fibrin-rich exudates (edema fluid) → regnration more organized
3. marked thickening of alveolar septa
   - TGFB by macrophage
   - Poliferation interstitial cell
   - Collagen deposition



hyaline membrane
macrophage phago cyte
(healing)
تغلب الdefense force

OUTCOME:

بتعتمد على كمية الFibrosis والScaring in lung

- hospital mortality rate 38.5% ≈ 40%
- Death attributable to (متعلقة):
  - Sepsis
  - multi organ failure
  - sever lung injury

*most people: Recover normal respiratory function (خلال ٦-١٢ شهر)

*Some People: lung damage
- *interstitial fibrosis
- *chronic Pulmonary disease

سبحان الله
والحمد لله
ولا إله إلا الله
والله أكبر

DIFFUSE PULMONARY DISEASES

Two categories

Obstructive airway diseases

characterized by an increase in resistance to airflow caused by partial or complete obstruction at any level

easy to fill (inhale) (get air in) → ↑compliance, ↓recoil

difficult to exhale (get air out)

Restrictive disease

characterized by reduced expansion of lung parenchyma and decreased total lung capacity.

مثل زوج الجواب — الديه عنده elasticity ↓ (difficult inhale)

not easy expand

occur in two general conditions:

- Chest wall disorder (normal lung)
  - severe obesity
  - disease of pleura
  - neuromuscular disorders → affect respiratory muscle
- *acute interstitial lung disease
  - ARDS
- *Chronic interstitial lung disease
  - Chronic restrictive disease:
    - pneumoconioses
    - interstitial fibrosis
    - unknown etiology
    - sarcoidosis

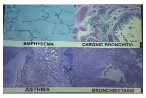
*Question

A 58-year-old man with ischemic heart disease undergoes coronary artery bypass graft surgery under general anesthesia. Two days postoperatively, he experiences increasing respiratory difficulty (→ dyspnea) with decreasing arterial oxygen saturation (→ hypoxia). On physical examination, his heart rate is regular at 78/min, respirations are 25/min, and blood pressure is 135/85 mmHg. The hemoglobin concentration has remained unchanged, at 13.7 g/dL, since surgery. After he coughs up a large amount of mucoid sputum, his condition improves. Which of the following types of atelectasis does he most likely have?

A) Compression B) Contraction

C) Resorption

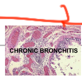
فيا شباب الأمة وأشبالها! هذا كتاب الله ينادي!.. وهذه الأمة تستغيث!..
فمن ذا يبادر لحمل الرسالة؟ من ذا يكون في طليعة السفراء الربانيين، الحاملين
لرسالات هذا الدين، إلىٰ جموع التائهين والمحتارين هنا وهناك؟.. من يفتح
صدره لنور القرآن، فيقدح به أشواقَ العلم بالله والمعرفة به؟ عساه ينال شرفَ
الخدمة في صفوف الإغاثة القرآنية، والإنقاذ لملايين الغرقىٰ في مستنقعات
الشهوات والشبهات؟ من يمد إلىٰ رسول الله ﷺ يدًا غير مرتعشة؛ فيبايعه علىٰ أخذ
الكتاب بقوة؟ ويقبض علىٰ جمر هذا الإرث الدعوي العظيم: رسالات القرآن؟ من
يقول: «أنا لها يا رسول الله!» فيقوم بحقها ويَفِي بعهدها؟ ثم ينخرط في مسلك
بلاغات الوحي، سيرًا علىٰ أثر الأنبياء والصديقين: ﴿ٱلَّذِينَ يُبَلِّغُونَ رِسَٰلَٰتِ ٱللَّهِ
وَيَخۡشَوۡنَهُۥ وَلَا يَخۡشَوۡنَ أَحَدًا إِلَّا ٱللَّهَۗ وَكَفَىٰ بِٱللَّهِ حَسِيبًا﴾ [الأحزاب: ٣٩]. فهل من عبد -حقَّ
عبد لله- يجعل حياتَه وقفًا علىٰ دين الله، يتلقىٰ كلمات الله، ويُبَلِّغُ رسالاتِه! عسىٰ



OBSTRUCTIVE LUNG DISEASES

It's hard to get the air OUT
It's hard to EXHALE
Lungs are hyperinflated

irreversable



① EMPHYSEMA ② CHRONIC BRONCHITIS
③ ASTHMA ④ BRONCHIECTASIS
reversable — irreversable

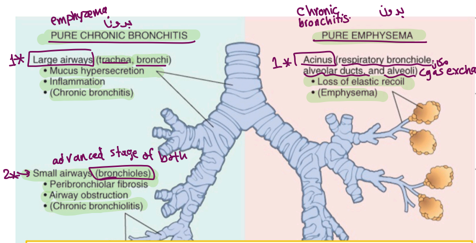
mixed picture — pure picture not dominant — smoking

Note:
* total lung capacity (TLC) → obestruction
↳ is the volume of air in the lungs upon the maximum effort of inspiration.

* lung compliance: lung ability to stretch or expand.

① emphysema
② chronic bronchitis
→ COPD:
- persistent respiratory symptoms
- preventable ✓ treatable ✓ common ✓
- air flow limitation — due to
  - * airway abnormalities
  - * alveolar
  - caused by exposure to ① noxious particles or ② gases.
- 4th leading cause of death in world
- strong associated with cigarette smoking (35% - 50%)
- بيصير عندهم COPD — 90% COPD patient → smokers

A 20-year-old, previously healthy gentleman is jogging one morning when he falls to the ground. He suddenly becomes markedly short of breath. In ER no breath sounds audible over the Rt side of the chest. A CXR shows shift of the mediastinum from right to left. A chest tube is inserted on the right side, and air rushes out. Which of the following underlying diseases is most likely to have produced this complication?
A. Centriacinar emphysema
B. Chronic bronchitis
C. Distal acinar emphysema
D. Panlobular emphysema



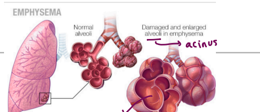
ANATOMIC DISTRIBUTION DEFINITION

Chronic bronchitis	emphysema
(pure): large airways (trachea, bronchi)	pure: Acinus (Respirator bronchiol, alveolar ducts, sac, alveoli) gas exchange
in advance stage: small air way — involve in both	
smoking	smoking
clinical history	need morphology and radiology

diagnose → السبب الأكثر انتشارا

1. EMPHYSEMA

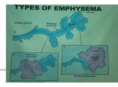
Perminant (irreversable)
* enlargment of air spaces distal to the terminal bronchioles (acinus) and
* destruction of their wall without significant fibrosis.



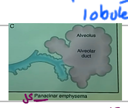
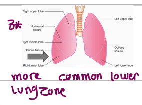
enlargment acinus air space due to distruction wall without significant fibrosis.

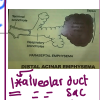
* small air ways fibrosis (subtle, functionally) ↳ significant contributor to airflow obstruction

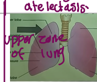
Classified:

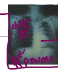
① centriacinar


① * affect central part of acinus (Respiratory bronchiole)
② * alveolar duct, sac not involve
③ * smoking — most common type of emphezema
④ associated with chronic bronchitis
⑤ * more sever + common in upper part lung (apical sigment).

② panacinar
entire pulmonary lobule

1 * uniformly (* Respiratory bronchiol, alveolar duct, sac)
2 * EMPHYSEMA α1-antitrypsin deficiency (genetic disease)

3 * more common lower lung zone

③ distal acinar (paraseptal)


1 * alveolar duct — sac
proximal part spared — Respiratory bronchiol
2 * unknown etiology
3 * happen (CT-septa), at margins of lobules
adj. area of fibrosis scarring atelectasis



trachea heart shift on left side

④ irregular
* part of acinus can be involve
* associated with scar
* common
* No significant disease.

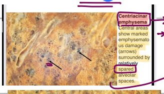
Pathogensis
- * infection — acute emphyzema
- air pollutant or smoking — genetic predisposition
- Congenital α-1 antitrypsin deficiency

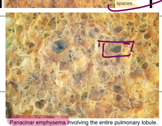
different mechanism:
① oxidative stress (ROS)
② ↑ apoptosis
③ ↑ senescence
- inflamatory cells & inflammatory mediators (protease, ROS) — damage of alveolar wall
- proteas (anti) — proteas imbalance — protective mechanism

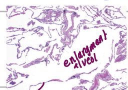
→ alveolar wall destruction

smoking → inflamatory mediator → alveolar wall damage ↳ ROS →

emphyzema → relevant defency for anti protases (α1-antitrypsin) defency → elastase (proteas) → damage elastic fiber → alveolar wall damage

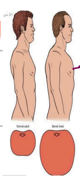
Morphology
- gross apearnce
- microscope
* Macroscopic: advanced emphysema ↳ voluminos lung


→ central part: dilated
→ alveolar duct, sac: not enlarge

Panacinar emphysema involving the entire pulmonary lobule.
enlarg. all

* microscopic examination
- abnormally large alveoli are spreaded by thin septa with only focal centriacinar fibrosis

wall not thick (no significant fibrosis).
gaps in walls (distriction)

Out come (destruction alvelor spaces)
① decreased capillary bed area due to
- * Destruction of alveolar wall
- * enlarged air spaces (bulla and blebs)
→ hypoxia induced → Pulmonary vascular spasm → secondary Pulmonary hypertension (20-30%) Right CHF (cor pulmonale)
↳ in advanced disease causing compression of the respiratory bronchioles and lung vasculature

present ation:
- symptoms do not appear until at least 1/3 of the functioning pulmonary parenchyma is damaged
- * Dyspnea: appears first beginning insidiously but progressing steadily
- * weight loss, common
- * barrel-chested (trap air) over inflation


anterior posterior diameter increase
- * prolonged expiration
- sitting forward in a hunched over position

- breathes through pursed lips
- * ↑ Respiratory rate → hyperventilation → adequate oxygenation of hemoglobin (at rest)
- * cough, wheezing
- pink puffer

لا إله إلا أنت سبحانك
إني كنت من الظالمين.

II. CHRONIC BRONCHITIS

* (affect large air ways) → trachea, main bronchi
* diagnose by clinical symptomes.

Defined clinically as Persistent productive cough for AT LEAST 3 consecutive months in AT LEAST 2 consecutive years in the absence of any other identifiable cause.

cough → 3 month / 2 year (at least)
→ No cause identifiable. بدون أي سبب آخر

- 90% cigarette smokers; air pollutants also contribute.

- chronic bronchitis is one end of the spectrum of COPD, with emphysema being the other.

emphysema ↔ chronic bronchitis → mixture of both (cigratte smokers)

Table 15-4 Emphysema and Chronic Bronchitis

	Predominant Bronchitis	Predominant Emphysema
Age (yr)	40-45	50-75
Dyspnea	Mild; late	Severe; early
Cough	Early; copious sputum	Late; scanty sputum
Infections	Common	Occasional
Respiratory insufficiency	Repeated	Terminal
Cor pulmonale	Common	Rare; terminal
Airway resistance	Increased	Normal or slightly increased
Elastic recoil	Normal	Low
Chest radiograph	Prominent vessels; large heart	Hyperinflation; small heart
Appearance	Blue bloater	Pink puffer

*PATHOGENESIS

The primary factor in the genesis is exposure to irritating inhaled substances such as tobacco smoke (90%of pt) and dust from grain, cotton, and silica.

1. hyper secretion of mucus.
   - earliest feature of chronic bronchitis
   - beginning in the large air ways.
2. acquired cystic fibrosis transmembran conductance regulator (CFTR) dysfunction
   - Smoking → acquired CFTR dysfunction → secretion of abnormal dehydrated mucus (sticky) → ↑ severity of chronic bronchitis
3. inflammation
   - due to Inhalants; No esinophil (lymphocyte mainly)
   - long standing inflamation and fibrosis involving small airways (small bronchi bronchioles) less than 2-3 mm in diameter
   - = Chronic air way obstruction
4. infection
   - 1* doesn't initiate chronic bronchitis
   - 2* produce acute exacerbations تفاقم حاد

in early stage air flow is not obstructed

airflow obstruction in chronic bronchitis results from:

1. Small airway disease

chronic bronchiolitis: results in mild airflow obstruction. Induced by mucus plugging of the bronchiolar lumen, inflammation, and bronchiolar wall fibrosis (mild airflow obstruction)

2. Coexistent emphysema: The cause of significant airflow obstruction.

When chronic bronchitis persists for years:

- decline in lung function, leading to cor pulmonale
- cause atypical metaplasia and dysplasia of the respiratory epithelium, providing a rich soil for cancerous transformation.

May coexist with hyper-responsive airways with intermittent bronchospasm and wheezing→ asthmatic bronchitis

morphology

* Macroscopic
  - mucosal lyning is hyperemic and swollen
  - layer of mucinous or mucopurulent secretions.
* microscopic
  1. mild chronic inflammation of airways (predominantly lymphocyte)
  2. hyperplasia of the mucus-secreting glands of trachea, bronchi
  3. squamous metaplasia and dysplasia of the bronchial epithelium
  4. changes of emphysema (often co-exist)

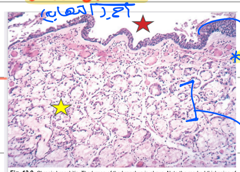


Fig. 13.9 Chronic bronchitis. The lumen of the bronchus is above. Note the marked thickening of the mucous gland layer (approximately twice-normal) and squamous metaplasia of lung epithelium. (From the Teaching Collection of the Department of Pathology, University of Texas, Southwestern Medical School, Dallas, Texas.)

* clinical features
  - persistent cough with production of sparse sputum
  - chronic bronchitis and COPD patients show frequent exacerbations, rapid disease progression, poorer outcomes than emphysema
  - Respiratory functional impairment → develop dyspnea on exertion

*out come:
* progressive disease
  1. pulmonary hyper tension
  2. cardiac failure
  3. recurrent infection and respiratory failure → Death
  - less dyspnea; absence of increased respiratory drive → lung retain CO_2 → hypoxic cyanosis
  - obese → for ulcer reasons designation "blue bloaters" (cyanosis, CO_2 retension, hypoxia)

الحمد لله