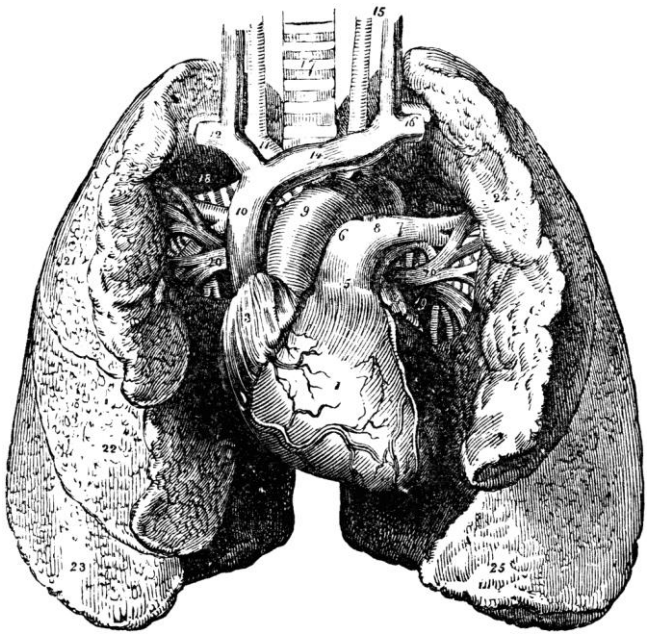


Pathology

teamwork

Lecture (1) :

Pathology of bronchial asthma

Color Index :-

- **VERY IMPORTANT**
- Extra explanation
- **Examples**
- **Diseases names: Underlined**
- **Definitions**

* كيف تعلُك الدُنيا بشيء ..وأنت لعلةِ الدُنيا طبيبُ ؟..

Objectives :

- Define asthma (BA)
- Know the two types of asthma
  1. Extrinsic or atopic allergic
  2. Intrinsic asthma.
- Understand the pathogenesis of BA
- Understanding the morphological changes (gross and microscopic) seen in the lungs in asthmatic patient.
- Know the manifestation and clinical coarse of BA
- List the complications of BA
- Define status asthmaticus
- Know the prognosis and prevention of BA

OVERVIEW : BY DR.RIKABI

before we start we need to revise the anatomical structure and histological of respiratory tract :

- Firstly we have to remember that the lining cells are Pseudostratified columnar ciliated epithelium with goblet cell
- there is main difference between **Bronchi** and **bronchioles** which is the bronchus has cartilage while bronchioles has NO cartilage.
- we find the **clara cells** in the **terminal bronchioles**

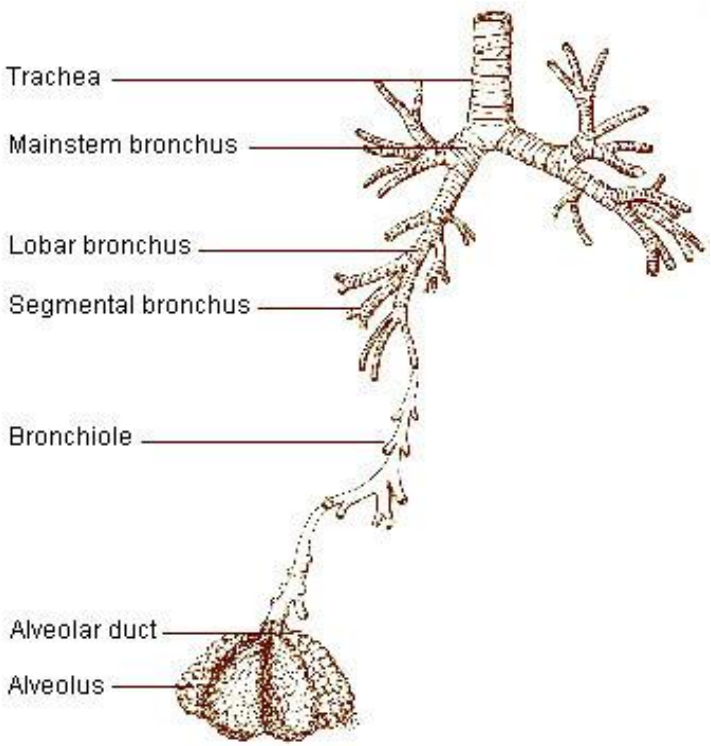
- alveolus have 3 types of cells:

1) **type 1 pneumocytes** :that line in capillary where gas exchange takes place.

2) **Type 2 pneumocytes** : secrete surfactant that keeps alveoli open by decreasing the surface tension.

3- **alveolar macrophage** other name is **carbon lading macrophage** or also called **anthracotic** : Which leads to anthracosis. **What is anthracosis?** accumulation of carbon pigment from breathing in dirty air. that's why it happens more in people who live in city .

- **what is the benefit to know the alveolar macrophages ?** to differentiate
- between **sputum** and **saliva** that has been received from the patient who have cough. So if you find macrophages you will know it's sputum not *saliva*.

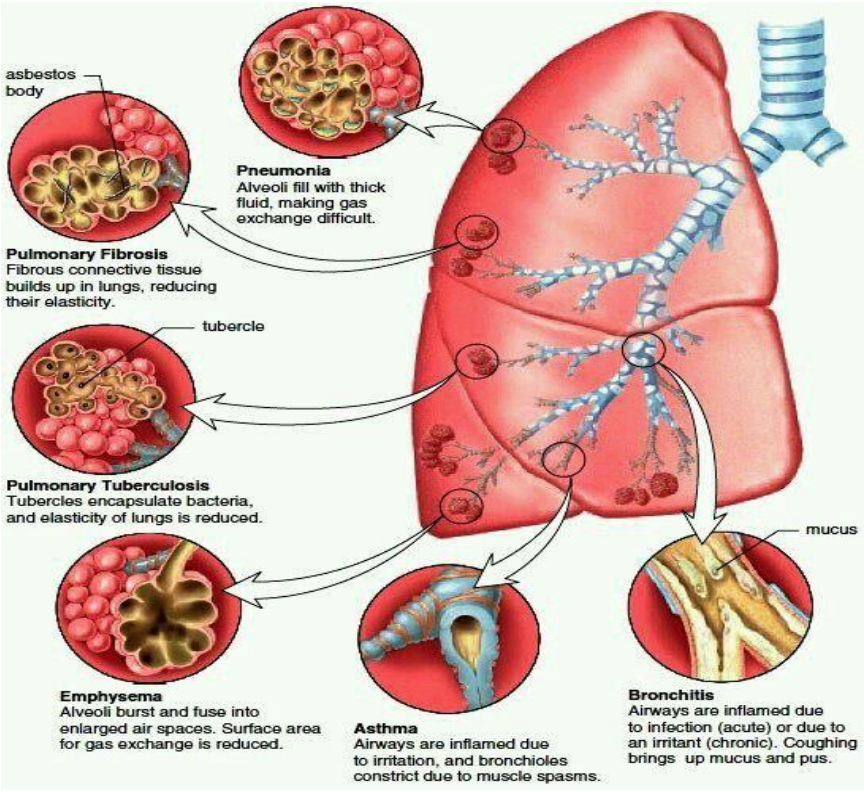


OVERVIEW : BY DR.RIKABI

- **Dr.rikabi's note** :any time you have patient with respiratory disease we have to listen to his symptoms and do some investigation and then think about one of the category :

obstructive airway disease → **restrictive lung disease** → **infection** → **tumors of the lungs** → **congenital diseases**

1- **chronic obstructive airway disease** like : bronchial asthma - chronic bronchitis - emphysema - bronchiectasis
2- **restrictive lung disease:** will affect the **interstitium** of lung and cause **fibrosis** and reduce lung volume.
3- **infection by viral or bactira:** TB, viral pneumonia, bacterial pneumonia
4-**tumors of the lungs. :**Most of them are malignant tumors. And mainly caused by smoking
5-**congenital diseases**



OBSTRUCTIVE VS RESTRICTIVE LUNG DISEASE (EXTRA)

Obstructive	restrictive
characterized by limitation of airflow due to partial or complete obstruction	characterized by reduced expansion of lung parenchyma accompanied by decreased total lung capacity.
Eg are emphysema, chronic bronchitis, bronchiectasis, and asthma	Eg are ILD like Fibrosing alveolitis, idiopathic pulmonary fibrosis, interstitial pneumonia, Pneumoconiosis,Sarcoidosis; and chest wall neuromuscular diseases
total lung capacity normal	decreased
forced vital capacity (FVC) normal	reduced
decreased expiratory flow rate, measuerd as forced expiratory volume at 1 second (FEV_{1})	Normal or reduced
FEV1/FVC ratio < 0.80	normal

EXTRA EXPLANATION :

- **FEV1**: Forced Expiratory Volume in one second: It is the amount of air expired in one second.

What do you think would happen with this number in asthma?

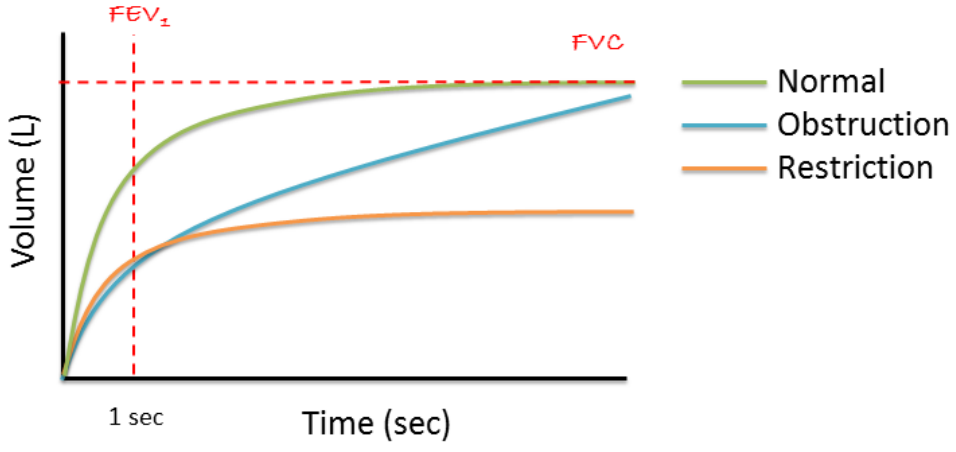
-Well, since it is an obstructive disease, there must be problems with air coming out of the lung. This will make the patient able to expire less air. Thus, **FEV1** will be decreased .

*المصابين بالأزمه عادةً لا يعانون من مشاكل بال Inspiration..لماذا ؟ لأن بعمليه inspiration يزيد lung volume بحيث يدخل الهواء فيكون الوضع سهل، لكن عند expiration بيقل ال lung volume بحيث يخرج الهواء ، فيضيق فوق ما هو ضيق بسبب الازمه فيكون فيه صعوبه بإخراج الهواء .

- **FVC:** Forced Vital Capacity: this is the total lung volume – residual volume. In patients with obstructive lung disease, there is no problem with the amount of air in the lungs. In fact, **there may be an increase in the amount of air in the lung. WHY ?** because when the patient can't exhale the air, it will stay inside the lung causing hyper inflation. **FVC will be normal or slightly increased** .

- **PEF:** Peak Expiratory Flow: This is the **ratio between FEV1/FVC**. Since FEV1 is **decreased**, and FVC is **normal** or **increased**, the ratio between them would be **lower** than normal .

*Conclusion: In COPD and asthma patients, FEV1 is less, FVC is normal or increased, and PEF is lower than normal.



*Dr.rikabi's note : -
-in asmatic people FEV1 is reduced while FAC is normal .
-the ratio between them is altered.
-this is the pattern in COPD .

In Restrictive lung disease the FEV1 and FAC both are reduce
the ration between them is normal

Bronchial Asthma:

What is **Bronchial Asthma**? is an episodic, reversible bronchoconstriction caused by increased responsiveness of the tracheobronchial tree to various stimuli.

-It's a chronic inflammatory disorder of the airways that causes recurrent episodes of **wheezing**[1], **breathlessness (Dyspnea)**[2]**, chest tightness**, and **cough**, particularly at night and/or early in the morning.

-What is The hallmarks of the disease? **intermittent** and **reversible** airway **obstruction**, chronic bronchial **inflammation** with **eosinophils**, **bronchial smooth muscle, cell hypertrophy**[3] **and hyper-reactivity**[4], **and increased mucus secretion.**

*EXTRA NOTES :

-1***why does the asthmatic patient wheez ?** Wheezing occurs when the small airways of the lungs become **narrow** or constricted. This makes it difficult to breathe, and can cause a **whistling sound when breathing out**. **WHY**? Because of a combination of :swelling, mucus and muscles tightening can cause narrowing of the airways.

2*DR.Rikabi's note :when the patient comes to the doctor and says that he has **Dyspnea all the time** then the doctor should know that this is **not asthma.** Why ? Because the asthma actually is **episodic** it occurs in attacks that lasts for one or two hours then stopes.

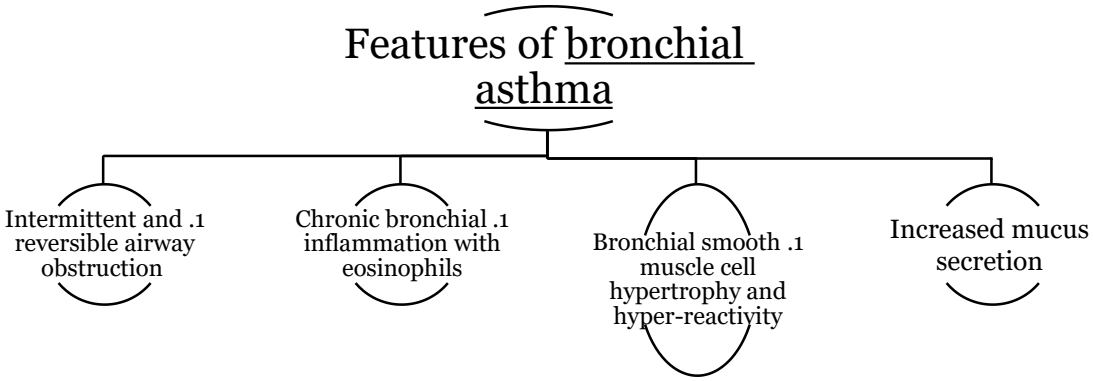
3*- why there is a bronchial smooth muscle hypertrophy ? Because with repeated contraction the muscle size will increase

-4* what do we mean by hyper-reactivity? Some of the stimuli that trigger attacks in patients would have little or no effect in persons with normal airways**.**

***DR.Rikabi's note** : Asthma is attacks , periodic, episodic that lasts for half hour or hour or two hours it will finish ether spontaneously or by taking bronchodilators. In most cases asthma is reversible only in rare cases it can be irreversible.

***DR.Rikabi's note:** why does asthma cases increases in the month of March in Saudi Arabia?
-In march the sand storms in KSA will increase.

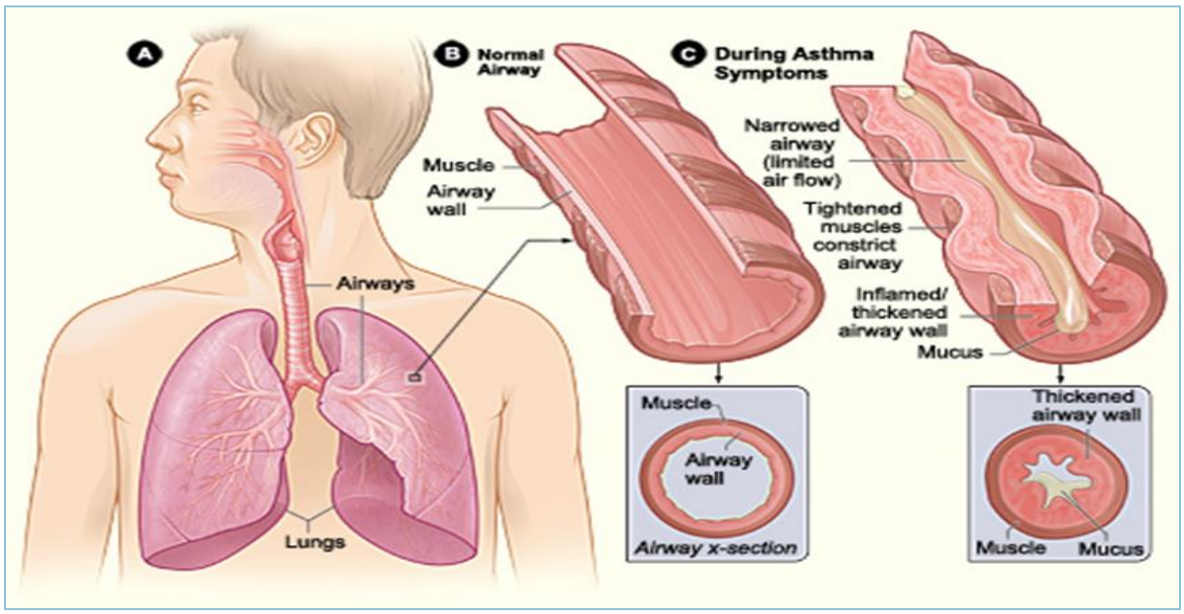
*So as a summary the features of asthma are :



•Asthma primarily targets: the **bronchi** and **terminal bronchioles**

•Most **common** chronic respiratory disease in **children**.(More common in children than adults Especially in intrinsic asthma

- In asthma, There is an increased **irritability** of the bronchial tree with paroxysmal narrowing of the airways, which may **reverse** either spontaneously or after treatment with **bronchodilators**.



Asthma animation:
https://www.youtube.com/watch?v=7ED09pUYvPE

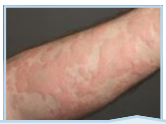
BRONCHIAL ASTHMA TYPES:

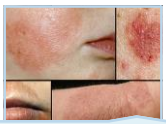
Extrinsic asthma :

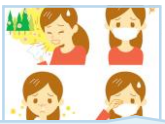
- **Other names**: allergic asthma / immune mediated asthma /atopic asthma /reaginic asthma.
- Bronchospasm is induced by **inhaled antigens**, usually in **children** with a personal or family history of allergic disease.
- Symptoms are brought about by IgE mediated type I hypersensitivity reaction to inhaled allergens And exposure to **extrinsic** allergens e.g. food, pollen, dust, animal dander..etc

- Serum levels of **IgE** and **eosinophils** usually **are elevated**.
- Atopic (allergic) asthma is the **most common type** and **begins** in **childhood**
- Other **allergic manifestation** may be present:


allergic rhinitis

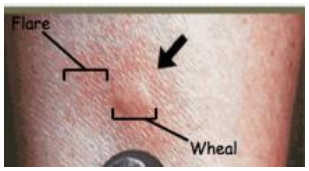

urticaria


eczema


hay fever

*الفحص يكون بطريقتين skin test OR serologic test "blood test "RAST

- Skin test with antigen is **positive** and results in an immediate wheal and flare reaction.



- Atopic asthma also can be diagnosed based on **A radioallergosorbent test (RAST)** That identify the presence of IgE in the blood

Subtypes include:

- 1- Atopic (allergic) asthma.
- 2- Occupational asthma.
- 3- Allergic bronchopulmonary aspergillosis (ABPA).

*DR.rikabi's note :There are a lot of allergic conditions (Eczema , allergic rhinitis , allergic conjunctivitis , bronchial asthma ... etc)

-some patient could have asthma for all their life ,or. it might get improved and the symptoms disappear due to tolerance in immune system.

*مثال شخص عنده حساسية تجاه الحيوانات الأليفة ف بمجرد ما يبتعد عنهم يتحسن او تجاه اكل معين بس ما ياكله يتحسن وتخف عليه

INTRINSIC/ NON-ATOPIC/ IDIOSYNCRATIC BA (NON-IMMUNE MEDIATED ASTHMA)

- **Intrinsic asthma** is a disease of adults in which the bronchial hyper- reactivity is precipitated by a variety of factors **unrelated to immune mechanisms.**
  - It has an **unknown** basis. Symptoms are precipitated by **non allergic factors** such as inhaled irritants/pollutants (e.g. sulfur dioxide, ozone) or infection (viruses). It is thought that virus-induced inflammation of the respiratory mucosa lowers the threshold of the sub-epithelial vagal receptors to irritants.
  - Positive family history is uncommon. (No family history of bronchial asthma)
  - Serum IgE : normal. (Not an immunological reaction)
  - No other associated allergies. (No urticaria, eczema, allergic rhinitis.. Etc.)
  - Skin test : **negative**.
  - Subtypes:
    - **Drug-induced asthma** (aspirin or nonsteroidal drug sensitivity)
    - **Occupational asthma** (fumes, dusts, gases)

*why do people who take aspirin develop asthma?

aspirin inhibits the cyclooxygenase pathway of arachidonic acid metabolism without affecting the lipoxygenase route, thereby shifting the balance of production toward leukotrienes that cause bronchial spasm.

*Further information :

There is another type which is : Exercised-**induced asthma** is a narrowing of the airways in the lungs that is triggered by strenuous **exercise**. It causes shortness of breath, wheezing, coughing and other symptoms during or after **exercise**. ... Among people with **asthma**, **exercise** is likely just one of several factors that can induce breathing difficulties.

❖ Major basic mechanism in both extrinsic and intrinsic asthma is inflammation. This only to have an IDEA about it.

Inflammatory cells:
- Mast cells
- Eosinophils
- Th2 cells
- Basophils
- Platelets

Structural cells:
- Epithelial cells (produce chemicals to stimulate eosinophils)
- Smooth muscle cells (hypertrophy)
- Endothelial cells (produce histamine and chemical mediators)
- Fibroblast
- Nerves (could lead to bronchoconstriction)

Mediators:
- Histamine
- Leukotrienes
- Prostanoids
- PAF
- Kinins
- Adenosine
- Endothelins
- Nitric oxide
- Cytokines
- Chemokines
- Growth factors

Effects: (end result)
- Bronchospasm
- Plasma exudation
- Mucus secretion
- AHR
- Structural changes

EXTRINSIC VS INTRINSIC

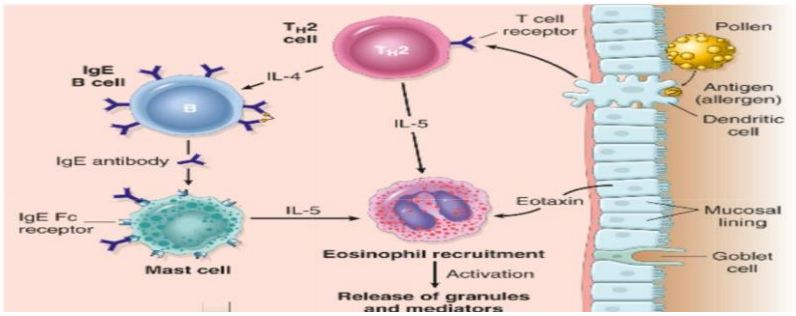
	Extrinsic Asthma	Intrinsic Asthma
	extrinsic**(atopic, allergic)** Asthma **70%**	Intrinsic (non-atopic) Asthma **30%**
	Initiated by **type 1 hypersensivity reaction** induced by exposure to **extrinsic** antigen/allergens e.g. food, pollen, dust, etc.	Initiated by **diverse**, non-immune mechanisms e.g. infections, drugs like aspirin, pollutants, inhaled chemical irritants, cold, stress and exercise.
	Subtypes include: 1)1- Atopic (allergic) asthma. 2)2- Occupational asthma. 3)3- Allergic bronchopulmonary aspergillosis (ABPA).	**No personal or family history of allergic reaction.**
	Develop early in life.	**Develop later** in life.

Pathogenesis of Bronchial Asthma

- **What is the etiology of asthma?** The asthma has a genetic predisposition to type I hypersensitivity (atopy), which causes bronchial hyper-responsiveness to a variety of stimuli that leads to acute and chronic airway inflammation.
- The inflammation involves many cell types and numerous inflammatory mediators, but the role of **type 2 helper T (TH2)** cells may be critical to the pathogenesis of asthma.
- The pathogenesis mechanisms have been best studied in atopic asthma the mechanism of non-atopic asthma is still unknown

Principal cells in asthma

- mast cells
- eosinophils
- epithelial cells
- macrophages
- activated T lymphocytes (TH2 subset)
  - play an important role in the regulation of airway inflammation through the **release of numerous cytokines**
- neutrophils



1. Antigen enters the body
2. Recognized by the dendritic cell or any other APC
3. Activate TH2 cell and the release of cytokines such as **IL-5** and **IL-4**
   - IL-4 will play a role in cross-linking of immunoglobulins in B lymphocytes, promote production of **IgE**
   - IL-5 stimulates production and activation (recruitment) of eosinophils
4. igE attach to mast cell and stimulate the release cytokines such as histamine and IL-5
5. Eosinophils will release the granules and mediators which are are toxic to epithelial cells
6. injured bronchial mucosal lining cells will secrete **eotaxin** which helps in the eosinophils recruitment.

Pathogenesis of Bronchial Asthma using type 1 IgE-mediated Atopic Asthma as a model

- First there is **initial sensitization** or priming:
first time exposure to an inhaled allergen which stimulates induction of Th2-type T cells (CD4 TH2) to produce cytokines(interleukin IL- 4, IL-5 and IL-13)

  - IL-4: plays a role in cross-linking of immunoglobulins in B lymphocytes, promote production of **IgE** and mast cells.
  - IL-5: stimulates production and activation (recruitment) of **eosinophils**.
  - IL-13: stimulates **mucus production** and also promotes IgE production by B cells. Has the same function of IL-4 + mucus production

- Then there is **subsequent re-exposure** to the allergen will leads to an IgE mediated reaction.

- This IgE-mediated reaction to inhaled allergens elicits:
  1) an acute response (within minutes)
  2) a late phase reaction (after 4-8 hours)

In the first exposure to the allergen it will start the sensitization of igE with no symptoms but in the second exposure the igE is already exists so it will lead to IgE mediated reaction which has to phases the first one start within minutes and called the acute phase and after 4-8 hours the late phase will start

Acute-phase response

- Begin 30 to 60 minutes after inhalation of antigen/aeroallrgens (e.g. allergens, drugs, cold, exercise)

- The exposure results in the stimulation and degranulation of **mast cells** ,**eosinophils** , dna**basophils** with the release of inflammatory mediatorsfrom these cells and also from activated macrophages.

- The released mediators induce :
  - bronchoconstriction/spasm .
  - increased vascular permeability ,
  - inflammation and injury of the bronchial walls and bronchialepithelium
  - excess mucous secretion.

-The early reaction is dominated by bronchoconstriction, increased mucus production and variable vasodilation (hyper-reactivity reaction)

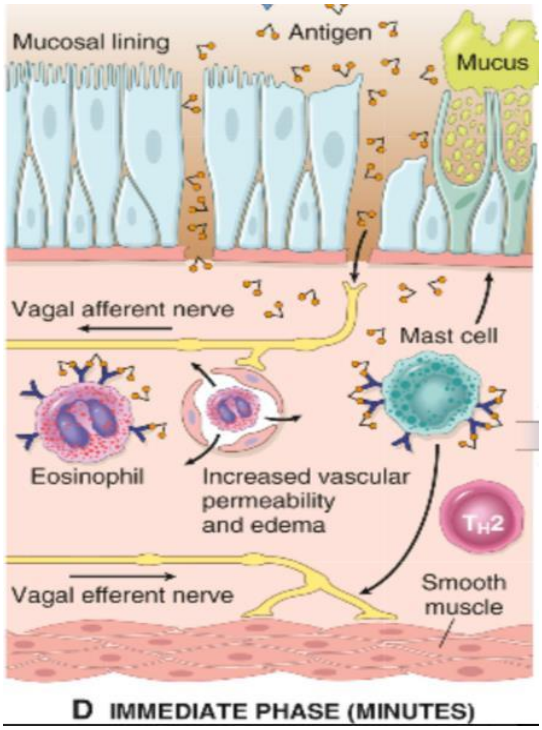
-Bronchoconstriction is triggered by direct stimulation of sub-epithelial vagal receptors

Late phase reaction/ late asthmatic response:

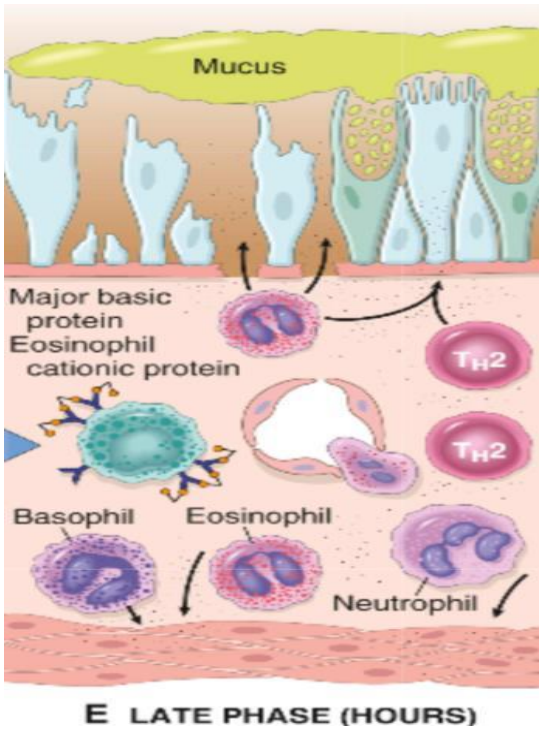
- Occurs 6-24 hours following allergen exposure .

The arrival of leukocytes at the site of mast cell degranulation (Due to some of the cytokines that realesed in the acute phase) leads to release of moremediators to activate more mast cells.

- Discharge of eosinophil granules releases major basic protein, eosinophilic cationic protein and eosinophil peroxidase into the **bronchial lumen**. These substances are **toxic to epithelial cells.**
- Eotaxin is secreted **by injured bronchial mucosal lining cells** and helps in the eosinophils recruitment.
- Moreover, chemotactic factors like leukotriene B4, eosinophil chemotactic factor and PAF recruit more **eosinophils**, **neutrophils** and **platelets** to the **bronchial wall.**
- The vicious circle continues and prolongs and amplifies the asthmatic attack.
- All these factors amplify and sustain injury without additional antigen.



1- Acute-phase response



2- Late phase reaction

Still confused ? Read this. The story from the beginning ;

1)The asthma occurs due to the **abnormal responsiveness** to stimulus that stimulate and irritate the bronchial epithelium to create the attack.

2)What causes this abnormal responsiveness in patients? is an antigen (certain smells, some medications, sand ,dust , ...)

3)the antigen is going to enter in to the bronchial tree and present to the epithelium cells by antigen presenting cells such as dendritic cells (mostly found in the lymphoid tissues) and macrophages .

4)They are going to stimulate and activate the **T lymphocytes** making new population of activated T lymphocytes called (CD4-TH2 lymphocytes) these lymphocytes are going to **secrete certain cytokines** called **interleukins**

5) **IL-4** stimulate the B lymphocytes to **produce IgE** which is responsible for type one hypersensitivity reaction (anaphylactic).

6) **IgE has receptors** on the external surface of the **mast cells** the FC part of IgE is going to sit on those

7)once they sit on it the mast cells are going to **release histamine and serotonin** that present in granules in the cytoplasm of these cells. (degranulation)

8) Both histamine and serotonin acts on bronchial tree causing **edema, bronchospasm , hypersensitivity reaction and vasodilation.**

Acute phase

9) IL-5 stimulates the **eosinophils** , it is very important in the pathogenesis because it is going to be secreted in the sputum of asthmatic patient.

10) After stimulation the eosinophils are going to accumulate in the bronchial tree and secrets granules that contain ;
-major basic protein.
-eosinophilia cationic protein.

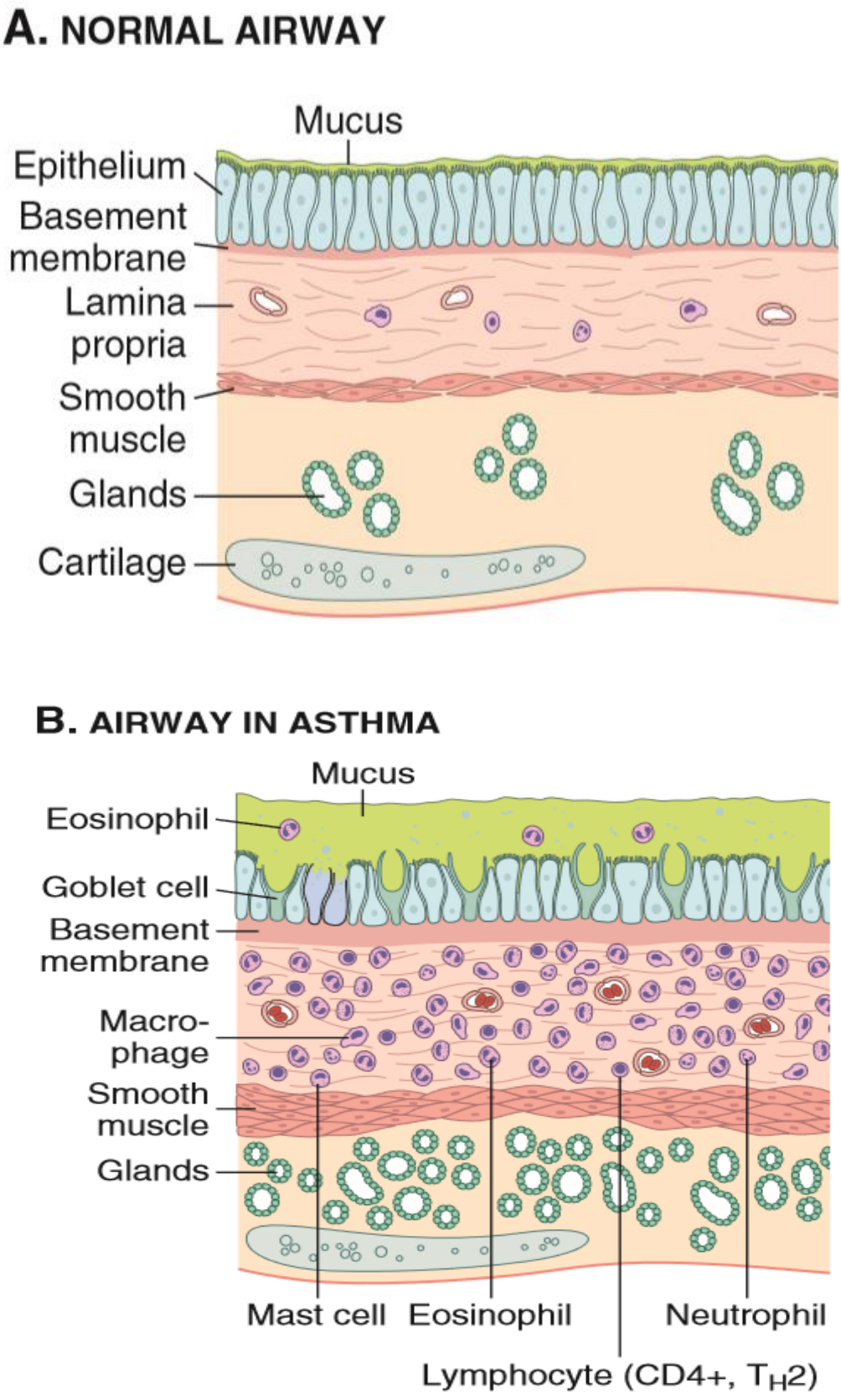
11) These proteins are going to cause damage of bronchial epithelium (ciliated) -> which is going to increase the number of goblet cells (goblet cells metaplasia) -> which will cause -> excessive mucus production which will block the bronchial tree .

12) Will it stop at this stage? NO.. chemotactic factors like leukotriene B4, eosinophil chemotactic factor and PAF recruit more **eosinophils**, **neutrophils** and **platelets** to the **bronchial wall and even T-cells.**

13) E-otaxin is secreted **by injured bronchial mucosal lining cells** and helps in the eosinophils recruitment

late phase

*تأملوا الفرق بين الشخص الطبيعي والمصاب بالأزمه :



MORPHOLOGY OF ASTHMA:

THE PATHOLOGIC FINDINGS ARE SIMILAR IN BOTH TYPES OF BA

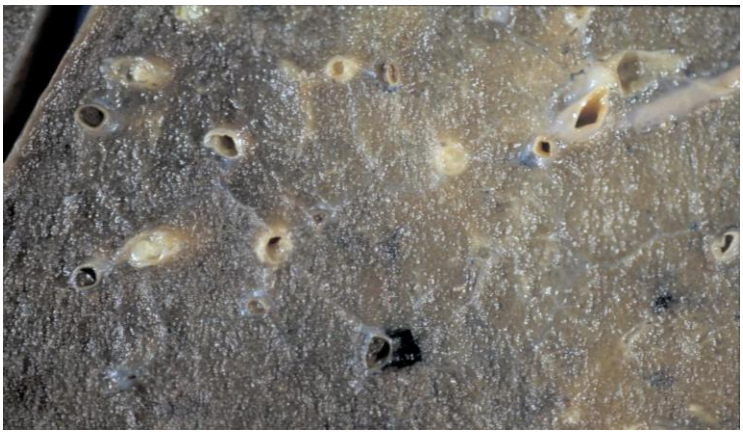
Grossly:

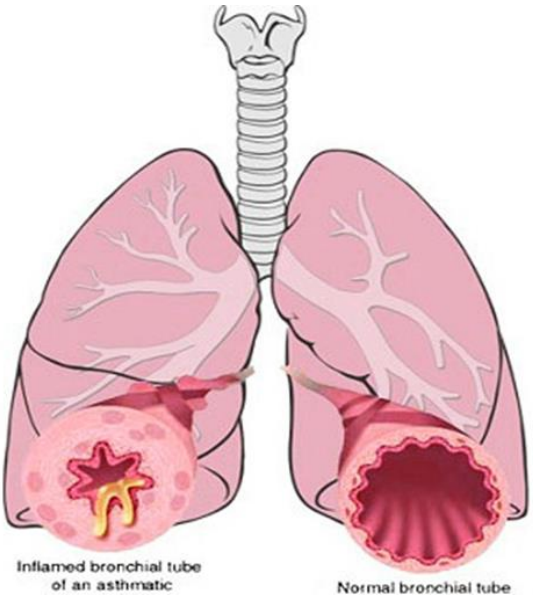
- Lung over distended (over inflation), occlusion of bronchi and bronchioles by thick mucous.

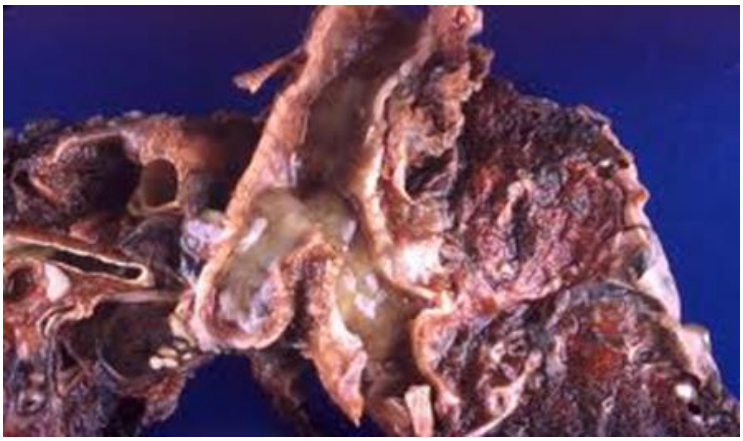
(Expiration needs effort –becomes an active process-, patients have a wheezing sound. As a result, there will be residual air in the lung and that leads to over inflation)

- The bronchi have thickened walls with narrowed lumina and generally are filled with plugs of mucus in acute attack.

- Mucus plugs





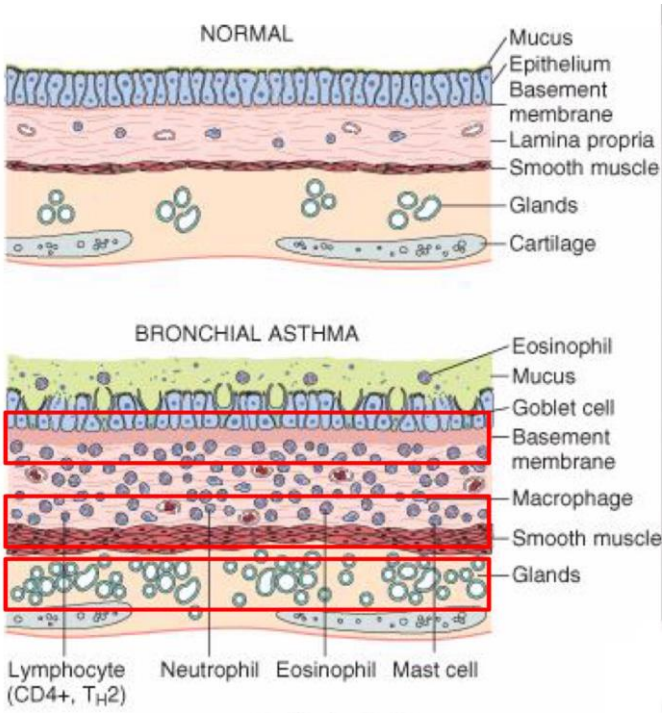


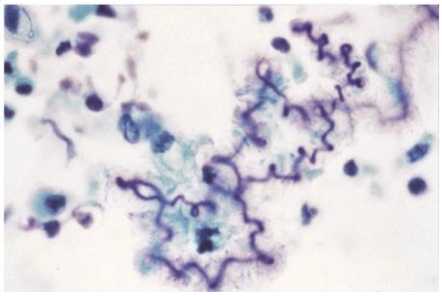
Narrowed lumen because of:

- Mucus secretion
- Thickened wall by inflammatory cells and edema
- Hypertrophied constricted muscle

MORPHOLOGY OF ASTHMA

- **Histologic finding:**
- 1. Thick Basement Membrane.
- 2. Edema and inflammatory infiltrate
- in bronchial wall.
- 3. Submucosal glands increased.
- 4. Hypertrophy of the bronchial wall muscle.
- 5. mucous contain **Curschmann spirals**,
- **Charcot-Leyden crystals and eosinophil**

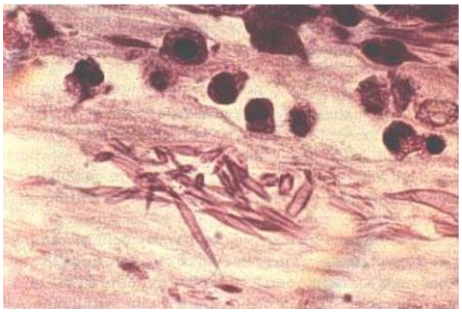


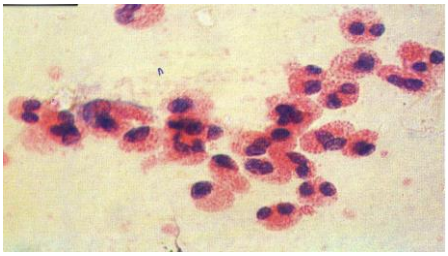


Curschmann spirals
Coiled, basophilic plugs of mucus formed in the lower airways and found in sputum and tracheal washings

* توجد بالمريض اللي فيه ازمه فقط، لما تشوفها راح تعرف ان المريض مصاب بالازمه

Charcot-Leyden crystals
Eosinophilic needle-shaped crystalline structures.





Eosinophils from a case of Bronchial Asthma

CLINICAL COURSE OF BRONCHIAL ASTHMA

1- The clinical manifestations vary from occasional **wheezing**[1] **to paroxysms of dyspnea**[2] and **respiratory distress.**

2- **Nocturnal cough** (سعال ليلي)

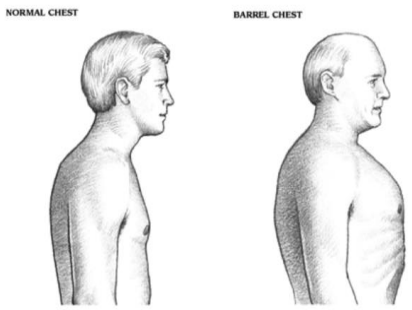
3- Increased **anteroposterior diameter**[3], due to air trapping and increase in residual volume

4-**Status asthmaticus** – **severe cyanosis** and **persistent dyspnea**, may be fatal

5- In a classic asthmatic attack there **is dyspnea, cough, difficult expiration, progressive hyperinflat**ion of lung and **mucous** plug in bronchi. This may resolve spontaneously or with treatment.

*1 لماذا يخرج صوت ال wheezing؟ لأنه مثل ما قلنا الممرات الهوائية تضيق في حالة الأزمه فبالتالي عندما يخرج الهواء من مكان ضيق بيسبب صوت صفير اثناء خروجه

2* Shortness of breath



*3 : increased anteroposterior diameter :

STATUS ASTHMATICUS

- **What is status asthmaticus?** It is the **most severe** form of asthma. It refers to severe bronchoconstriction that does not respond to the drugs that usually abort the acute attack. very severe type of asthma .

  - This situation is **potentially serious** and requires hospitalization. Patients in status asthmaticus have hypoxemia and often hypercapnia (high co2) .

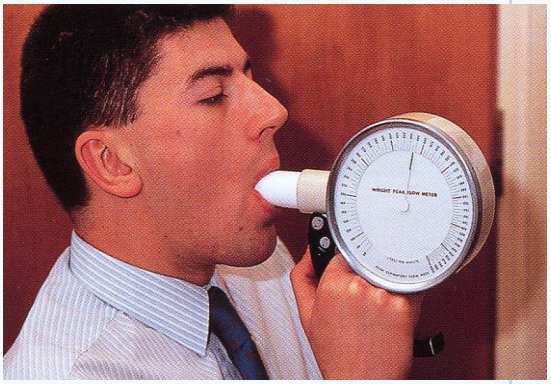
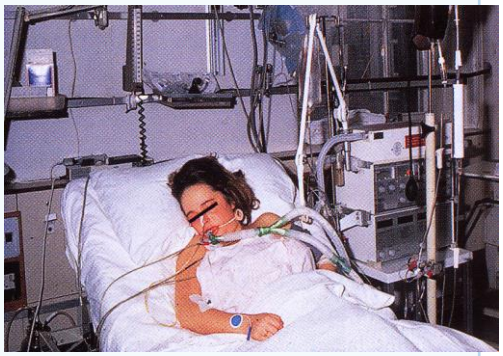
  - In particularly severe episodes the ventilatory functions may be so impaired so as to cause severe cyanosis and even death.

  - They require **oxygen** and other pharmacologic interventions.

  - It may persists for days and even weeks.

***Dr. alrikabi's note**: as a doctors the patient should be given :
-steroid , Bronchodilator , and keep monitoring.

- The range of presentation in asthma. الازمه تتدرج من حيث الخطوره، فممكن تكون بسيطة – متوسطه – خطيره جدا لدرجة ممكن تؤدي الى الوفاه

This patient was found incidentally to have a degree of reversible airways obstruction during a routine medical examination.	medical emergency with acute severe breathlessness, diagnosed as a case of **status asthmaticus** which required immediate intensive care including intermittent **positive-pressure ventilation**.
	

*Don't take much time with the diseases mentioned here because we will study them in other lectures

COMPLICATIONS OF ASTHMA

- **Airway remodeling**: some persons with long standing asthma develop permanent structural changes in the airway with hypertrophy of muscle and progressive loss of lung function that increase airflow obstruction and airway responsiveness.

Extra :
-Airway remodeling refers to the structural changes that occur in both large and small airways relevant to miscellaneous diseases including asthma. In asthma, airway structural changes include subepithelial **fibrosis**, increased smooth muscle mass, gland enlargement, neovascularization and epithelial alterations.

- remodeling is associated with poor clinical outcomes among asthmatic patients

- **Superimposed infection** i.e. pneumonia
- About pneumonia (https://www.youtube.com/watch?v=IAQp2Zuqevc)

- **Chronic bronchitis** (i.e.Asthmatic bronchitis: chronic bronchitis with superimposed asthma)
- About chronic bronchitis (https://www.youtube.com/watch?v=Y29bTzKK_P8)

- **Emphysema, pneumothorax and pneumomediastinum**
- About emphysema (https://www.youtube.com/watch?v=TEuSV_7gWA8)
- About pneumothorax (https://www.youtube.com/watch?v=DgU1HE_6ueI)

- **Bronchiectasis**
- About Bronchiectasis (https://www.youtube.com/watch?v=uOVZXzl6Qro)

- **Respiratory failure** requiring intubation in severe exacerbations i.e. status asthmaticus

- In some cases **cor pulmonale and heart failure** develop.
- About cor pulmonale (https://www.youtube.com/watch?v=Dx4QgdN_hI4)

PROGNOSIS OF ASTHMA

Approximately half the **children diagnosed** with asthma in childhood outgrow their disease by late adolescence or early adulthood and **require no further treatment**

Patients with **poorly** controlled **asthma develop long-term changes** over time (i.e. with airway remodeling). This can lead to **chronic** symptoms and a significant irreversible component to their disease.

Remission—approximately 50% of cases of childhood asthma resolve spontaneously but may recur later in life; remission in adult-onset asthma is less likely.

Many patients who develop asthma at an **older** age **also tend to have chronic symptoms.**

Mortality—death occurs in approximately 0.2% of asthmatics. Mortality is usually (but not always) preceded by an **acute attack** and about 50% are more than 65 years old.

PREVENTION OF ASTHMA

Control of factors contributing to asthma severity. **Exposure to irritants or allergens** has been shown to increase asthma symptoms and cause exacerbations.

Clinicians should evaluate patients with persistent asthma for allergen exposures and sensitivity to seasonal allergens**. Skin testing** results should be used to assess sensitivity to common indoor allergens.

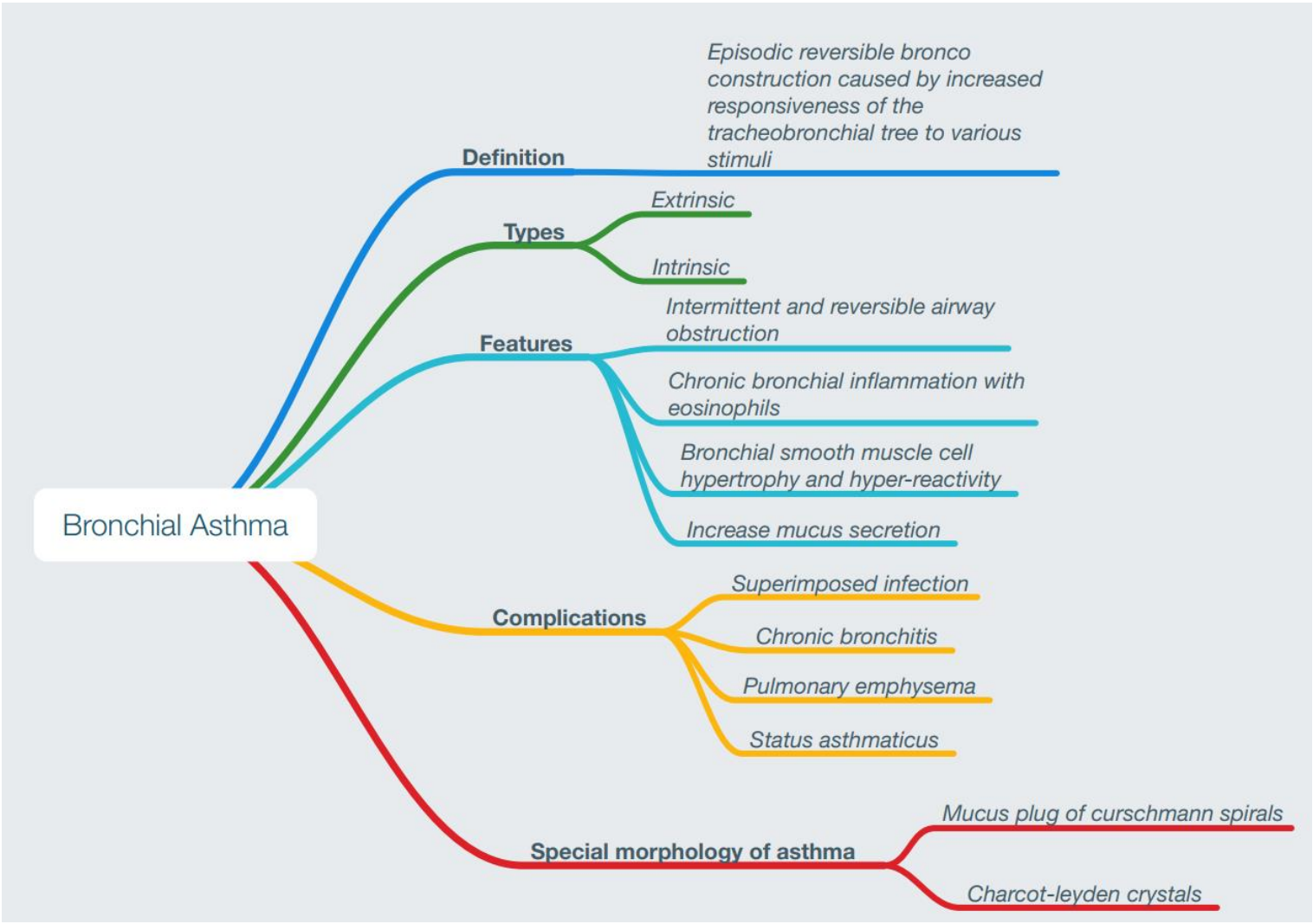
All patients with asthma should be **advised to avoid exposure to allergens** to which they are sensitive.

SUMMARY:

Asthma: Episodic attacks of bronchoconstriction

Types	• **Extrinsic asthma**: Type 1 Hypersensitivity reaction, IgE, childhood, family Hx of allergy. • **Intrinsic asthma:** associated e intake of aspirin, exercise, cold induced. No Hx of allergy
Morphology	• Hypertrophy of bronchial smooth muscle & hyperplasia of goblet cells e eosinophils, thickened basement membrane • Mucous plug , Curschmann spirals & Charcot-Leyden crystals.
Complication	• Superimposed infection • Chronic bronchitis • Pulmonary emphysema • Status asthmaticus

SUMMARY :


Bronchial Asthma
Definition
Episodic reversible bronco construction caused by increased responsiveness of the tracheobronchial tree to various stimuli
Types
Extrinsic
Intrinsic
Features
Intermittent and reversible airway obstruction
Chronic bronchial inflammation with eosinophils
Bronchial smooth muscle cell hypertrophy and hyper-reactivity
Increase mucus secretion
Complications
Superimposed infection
Chronic bronchitis
Pulmonary emphysema
Status asthmaticus
Special morphology of asthma
Mucus plug of curschmann spirals
Charcot-leyden crystals

TEST YOURSELF :

1- which of the following activate eosinophils in bronchial asthma ?
A- IL-4
B- IL-5
C- IL-28
D- Th2

2- which of the following is the most common type of asthma ?
A- type I hypersensitivity.
B- type II hypersensitivity.
C- type III hypersensitivity.
D- type IV hypersensitivity.

3- A patient of 19 years old come to ER with his friend , after a bad dusty storm and he complains from
shortness of breathing and cough and feeling tired at night so he could not sleep well. Which of the
following do you think is correct ?
A- bonchial asthma
B- lung TB
C- pulmonary eosinophilia
D- acute pneumonia

4- Asthma is more common in:
A. Adult
B. Same
C. Children

5- In Extrinsic Asthma the serum levels of IgE is:
A. Low
B. High
C. Normal

- **6. Which type of Asthma is related to allergic reactions?**
- A. Extrinsic Asthma
- B. Intrinsic Asthma
- C. Idiosyncratic

- **7. Which one of the following types of asthma is induced when exposure to allergen:**
- A. Chronic asthma
- B. Extrinsic asthma
- C. Intrinsic asthma

- **8.Which one of the following statements is true about allergic bronchial asthma:**
- A. IG E-mediated type I hypersensitivity.
- B. Increased amount of basophils
- C. It is common in adults.
- D. None of them

- **9. A 23-years old male came to the clinic complaining from tachypnea, tightness of the chest and dry**
- cough particularly at night.
- On auscultation, the doctor was able to hear the wheezing sounds over his chest while expiration.

- **What is the most likely diagnosis:**
- A. Bronchial asthma
- B. Sinusitis
- C. Bronchitis
- D. Tuberculosis

- ANWERS:
- 1- B
- 2-A
- 3-A
- 4-C
- 5-B
- 6-A
- 7-B
- 8-A
- 9-A

Males:

Leader: محمد باحذاق

فهد عبدالله الفايز
سيف المشاري
محمد الأصقه
خالد المطيري
داود اسماعيل
خالد محمد العقيلي
احمد وليد الراشد
فهد النحابي
عبدالله السرحاني
رشيد سليمان البلاع
معاذ ابراهيم الحمود
فايز غياث الدرسوني
عبد الكريم إبراهيم العنيق
خالد محمد العقيلي
عبدالله سليمان احمد بالعبيد
أنس عبدالله السيف
سعد محمد الفوزان
عبدالعزيز بن خالد السرحاني

Females:

Leader: بثينة آل ماجد

فاطمة بالشرف
روان الحربي
رهف الشمري
رناد الفرم
هديل عورتاني
منيره المسعد
الجوهرة الشنيفي
رزان الزهراني
روان مشعل
نوف العتيبي
غرام جليدان
بلقيس الراجحي
نورة القاضي
آلاء الصويغ
ريم القحطاني
شيرين حمادي
منى العمري
مجد البراك
رناد الغويري
دورة الحامدي
فاطمة الديحان

PATHOLOGY
TEAM 437

Kindly contact us if you have any questions/comments and suggestions:

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* TWITTER : @pathology437

GOOD LUCK! ☺

*references;

-Robbins Basic Pathology
- doctor's slides

Pathology
teamwork