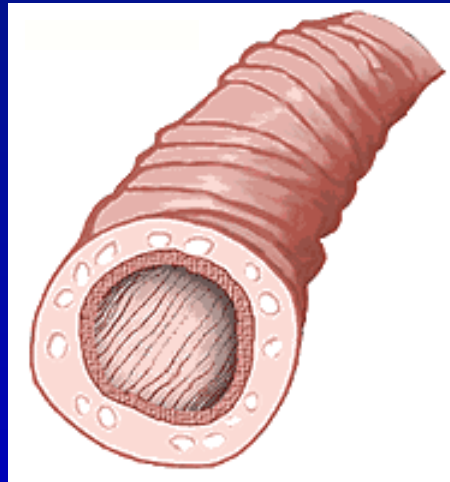


Obstructive Airway Disease



Dr. Khalid Al-Mobaireek
King Khalid University Hospital

Obstructive airway Disease:

- Physiological:
 - Reversible = Asthma
 - Irreversible: Bronchiectasis
 - Anatomical:
 - Upper
 - Lower
- Obstructive > happens in the air way
- Restriction > any thing prevent lung from expansion

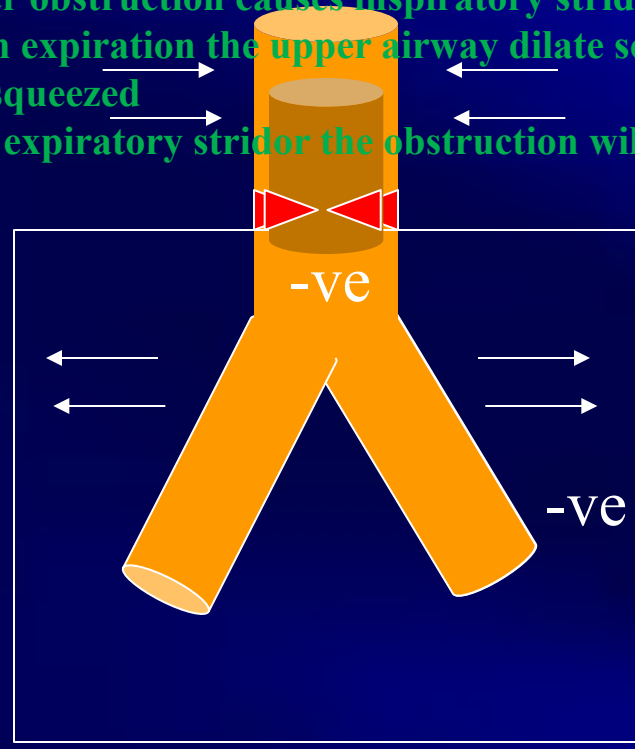
The upper airway is outside the thoracic cage, we create negative pressure by inspiration because we are lowering the diaphragm and increasing the space with the -ve pressure the air way inside thoracic cage will be dilated. If there is an obstruction it will not be very clear during this phase

But upper airway will be squeezed so if the obstruction is here it will be obvious

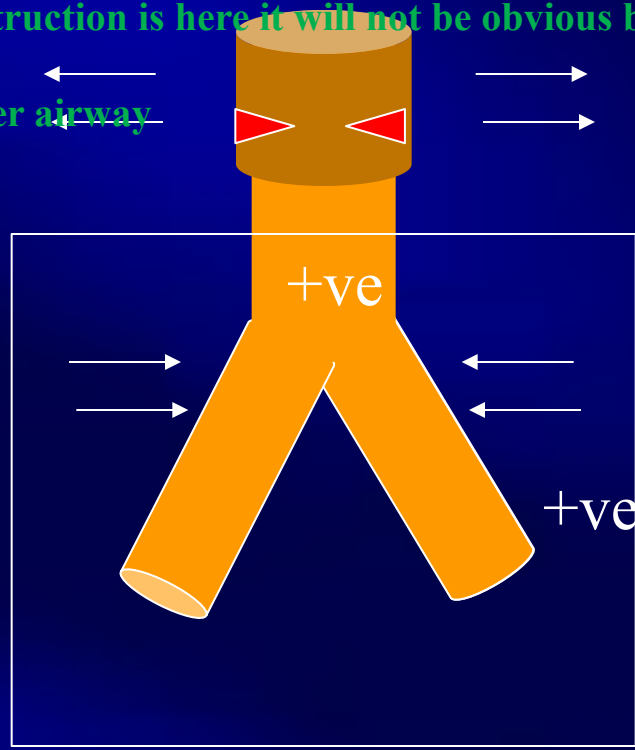
So upper obstruction causes inspiratory stridor

While in expiration the upper airway dilate so if the obstruction is here it will not be obvious but lower airway will be squeezed

So with expiratory stridor the obstruction will be in lower airway

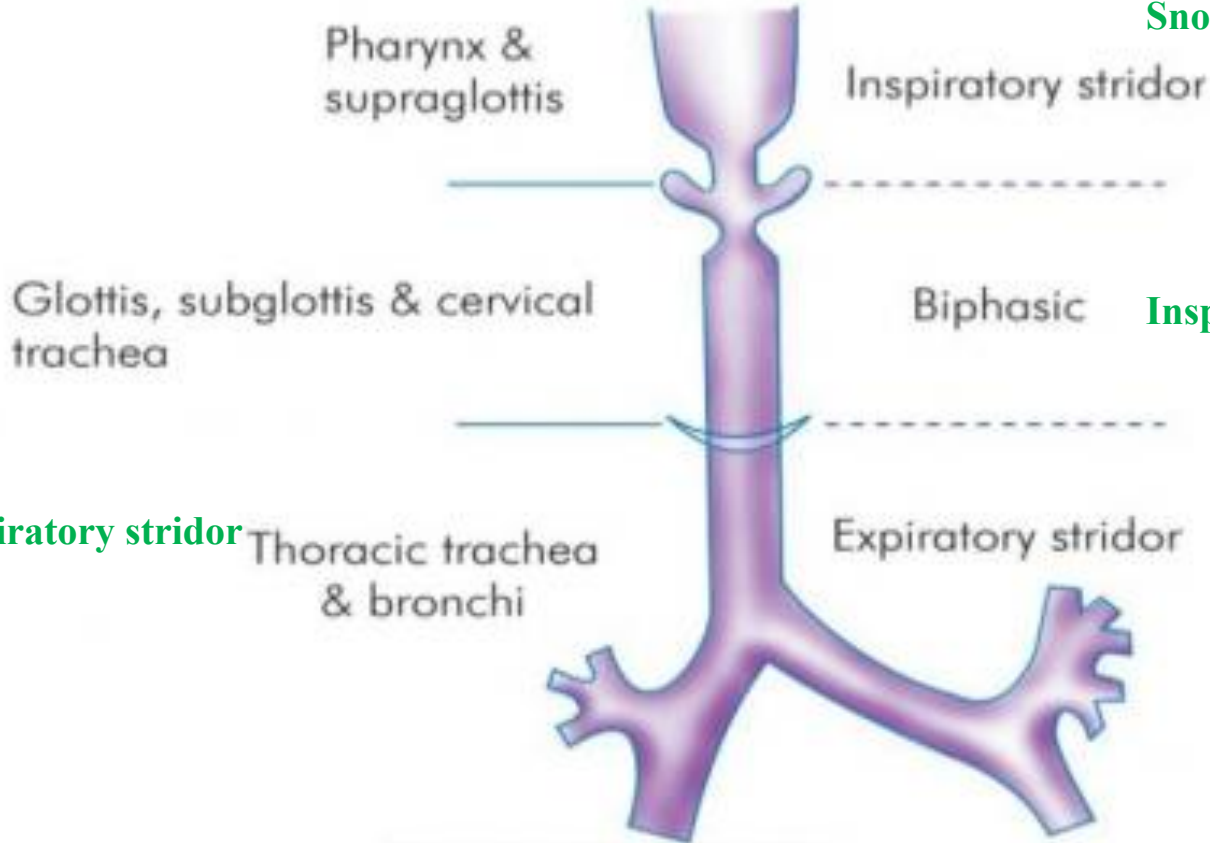


Inspiration



Expiration

Types Of Stridor And Probable Site of Obstruction



Snoring During inspiration

Inspiratory stridor

Expiratory stridor

Both phases
So the
problem in
these areas

Bronchiectasis

– Localized:

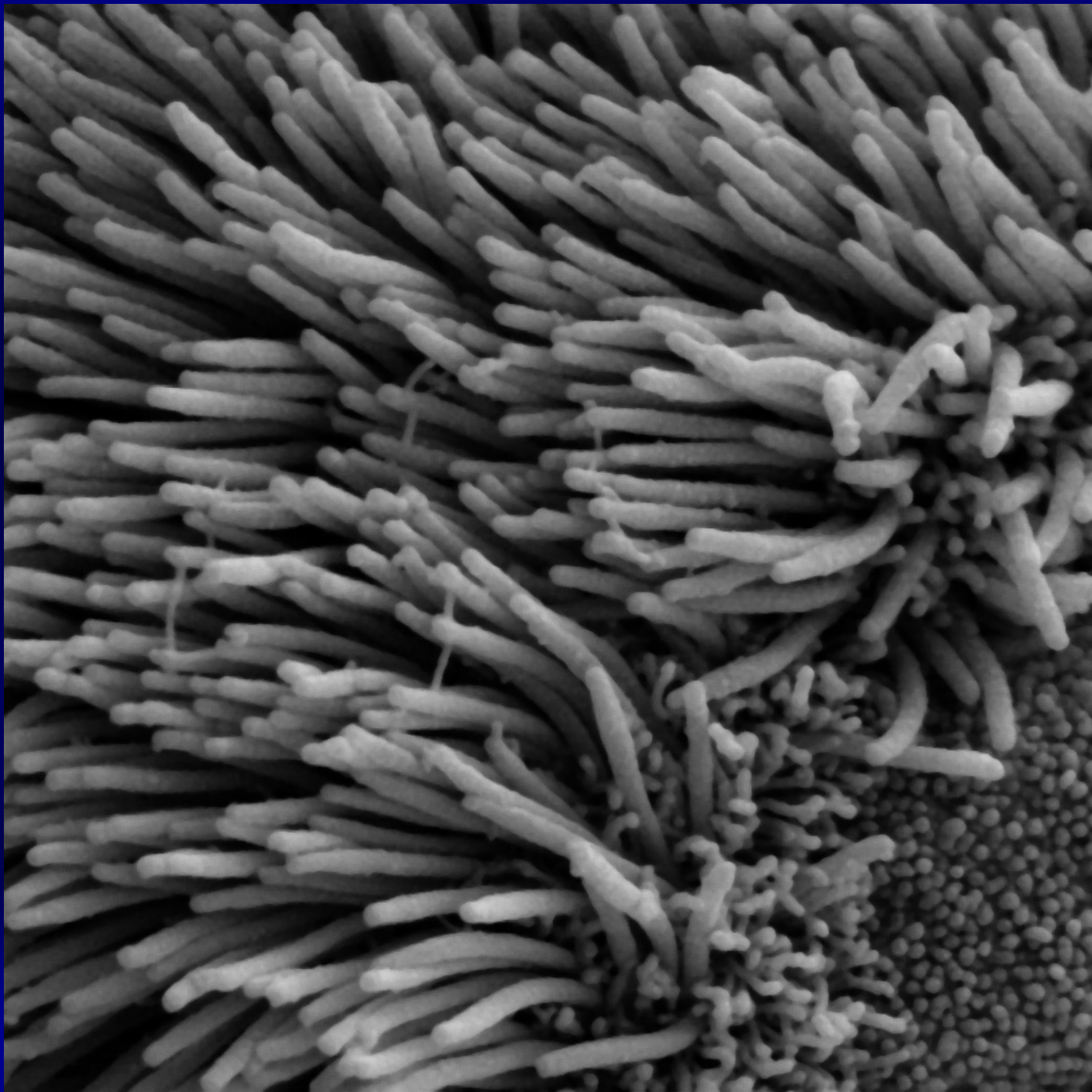
- Anatomical **if only one lobe think about anatomical**
 - Airway: Internal, External,
 - Parynchymal **congenital or infection or cyst**

– Diffuse:

- Aspiration **babies who can't coordinate there swallowing like children with neurological problems**
- Mucociliary clearance: PCD, CF
- Immune deficiency
- Congenital **they born with no cartilage but very rare**
- Post-infectious: Pertusis, TB, adenovirus..
- **Like: cystic fibrosis (thick mucus because there is mutation so there are no chloride channel > no pumping of chloride so no Na then no water so the secretion will be dehydrated and heavy so the cilia will not be able to move the mucus) even if the child cough it will not help, ciliary dyskinesia (here the secretion is normal but the cilia are not moving in one direction so there will be ineffective clearance) coughing can help in airway clearing (patient will have chronic cough and the cilia is present also in the sinus so they present with recurrent sinusitis, and in the middle ear so they will have recurrent otitis media, males infertility because sperm motility depends on the cilia, females it may affect pregnancy because the movement of ovum in fallopian tube**
- **Kartagener's syndrome it is ciliary dyskinesia with situs inversus**

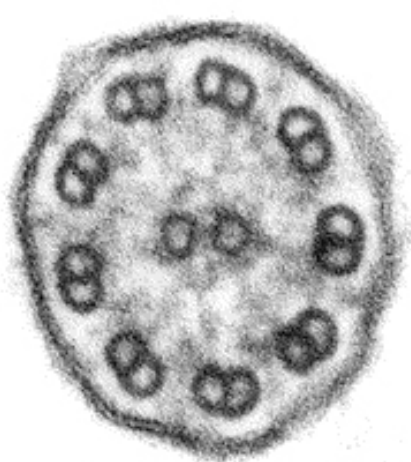
It is permanent dilatation of airway
The cartilage maintains the size of the air way so if it destroyed by infection then you will have dilatation

So if it happens there will be ineffective clearance there will be stagnation of secretions and mucus plugging, inflammation, and bacteria will colonize and causes infections.





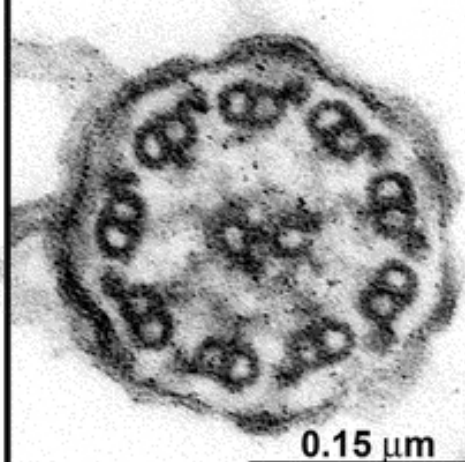
Normal



**Both
Arms
Absent**



**Absent
ODA**



**Absent
IDA**

Nose cilia cross-section

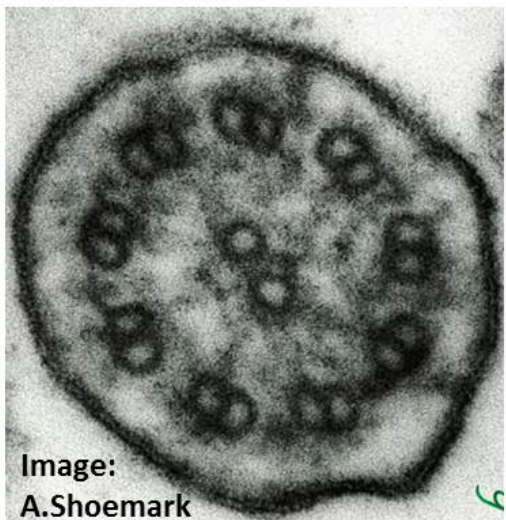
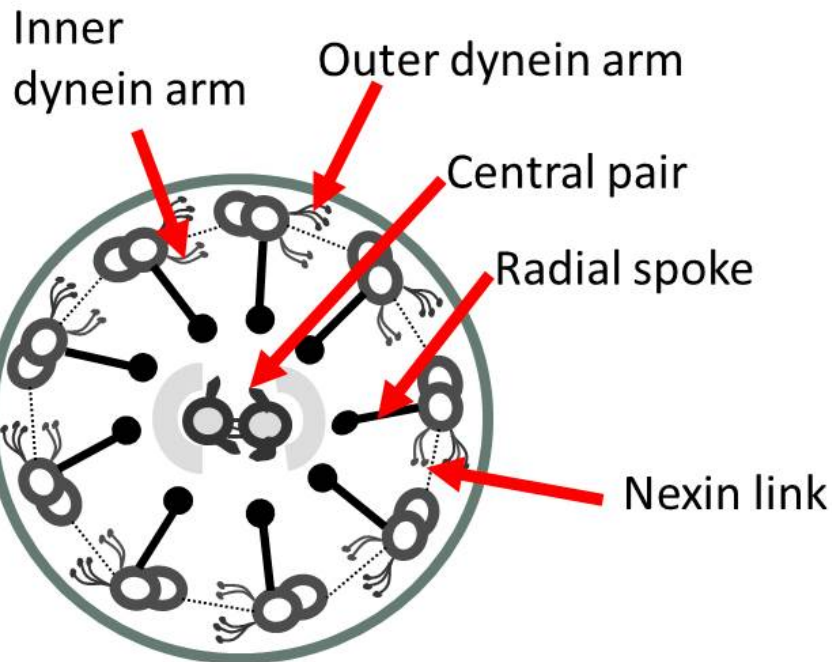


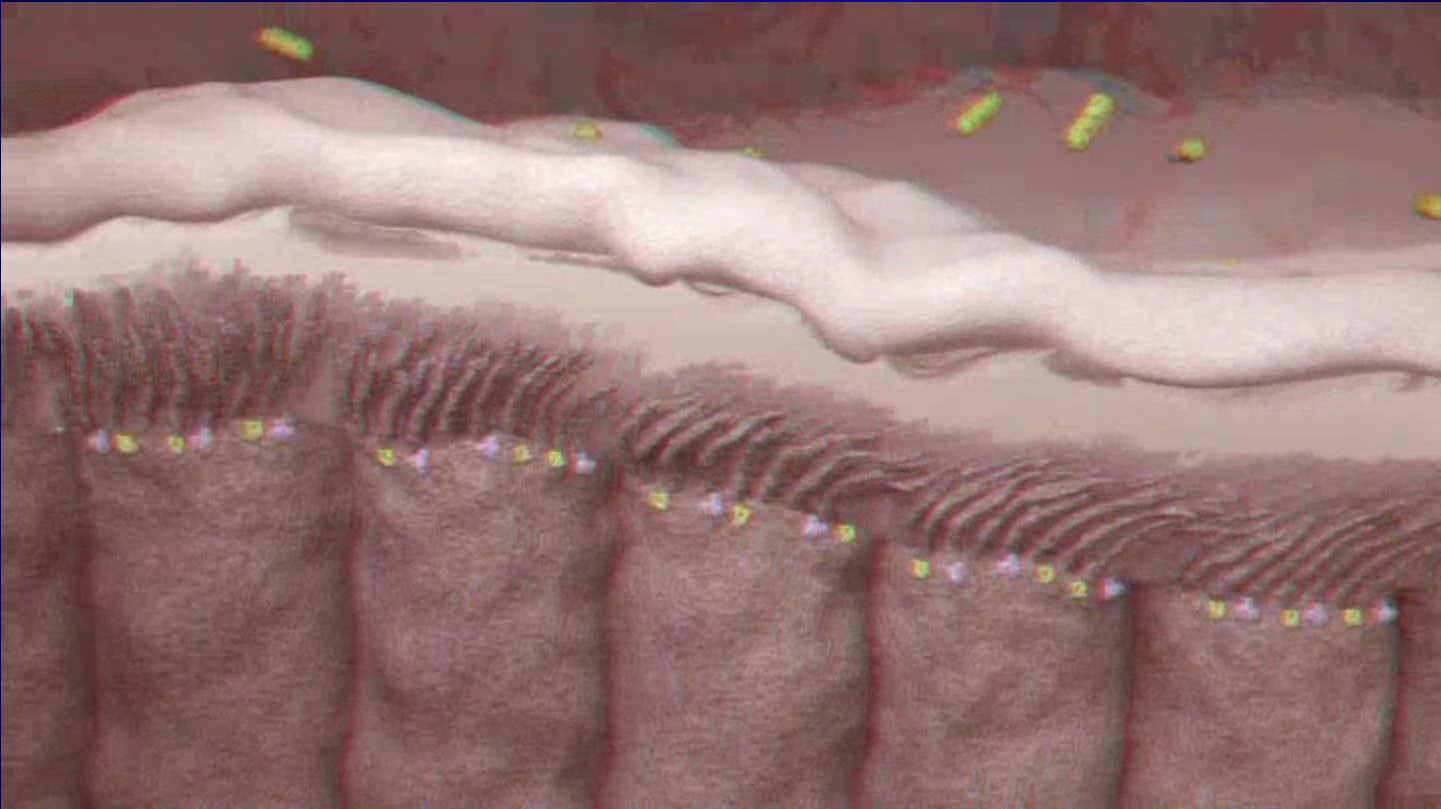
Image:
A. Shoemark



PCD

The normal cilia have 2 central tubules and 9 peripheral pairs of tube so 20 tubes

The central tubules is connected to the peripheral by spikes



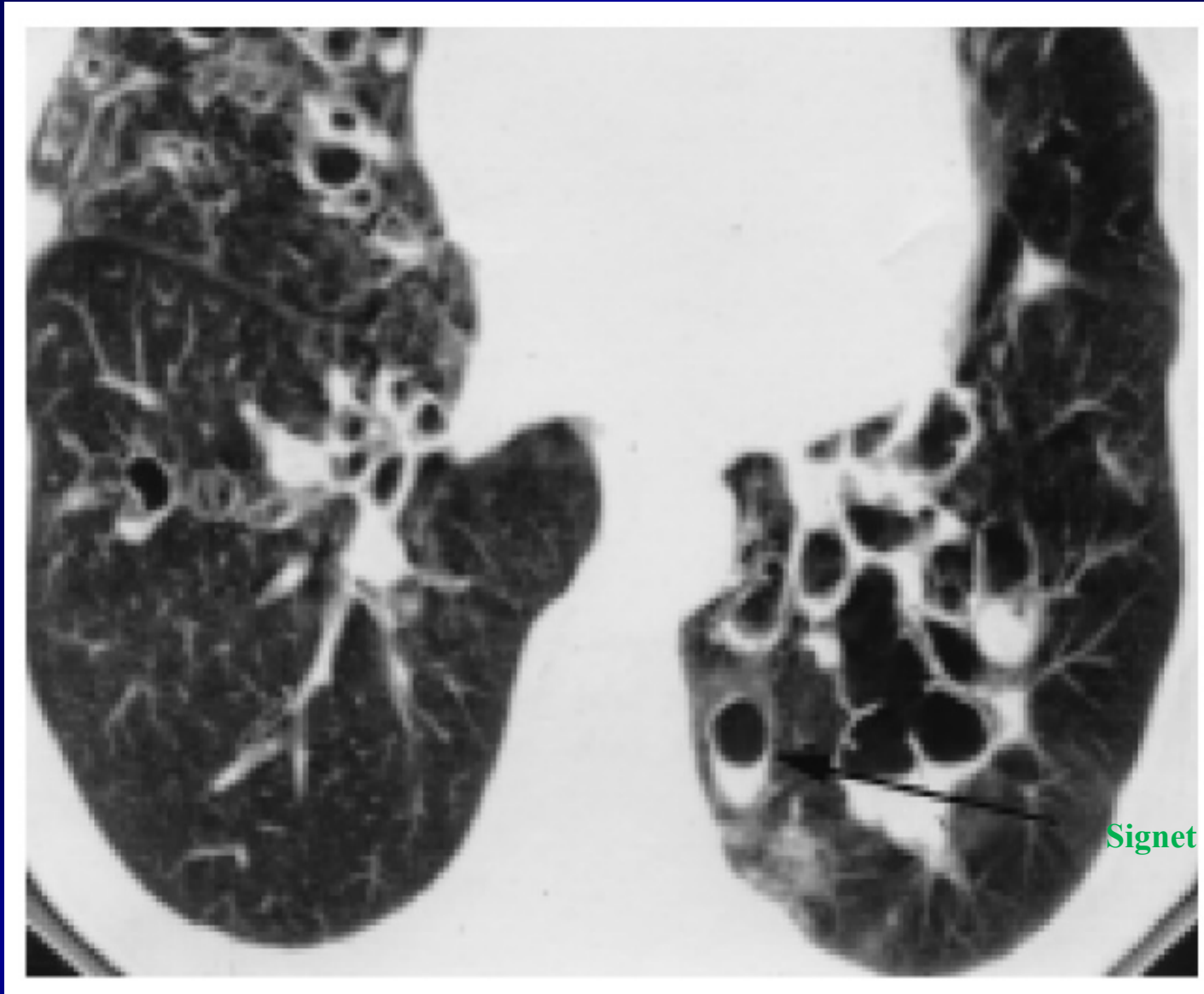
Diagnosis:

CT scan

Here you can see dilated airway

Normally the airway is smaller than blood vessels

If its larger this is bronchiectasis



Signet ring sign



Definition of Asthma

- A chronic **inflammatory** disorder of the airways
- Many cells and cellular elements play a role
- Chronic inflammation is associated with airway hyperresponsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing
- Widespread, variable, and often reversible airflow limitation

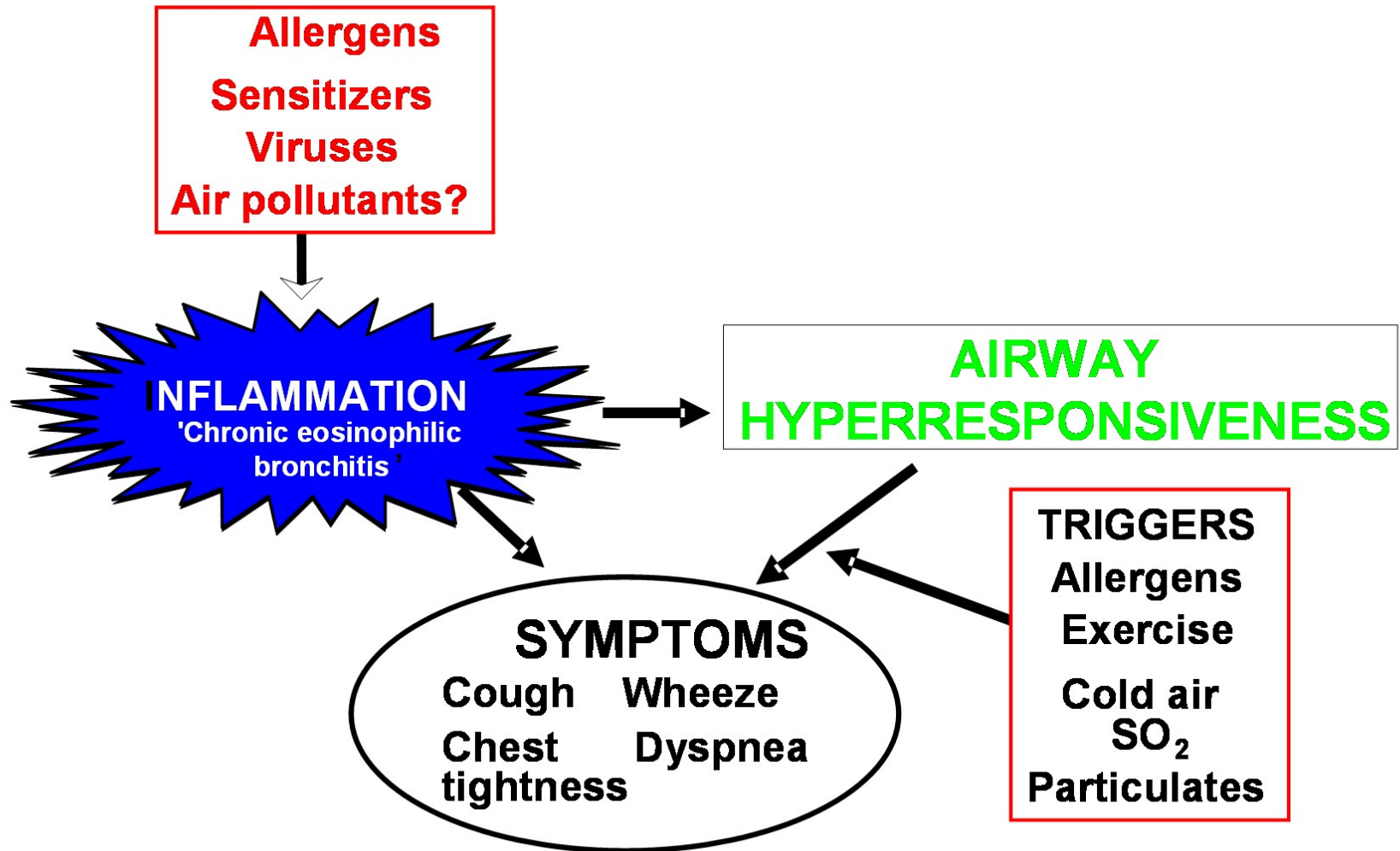


Bronchospasm
Edema, Mucus

Hyperresponsiveness

INFLAMMATION

Asthma Inflammation: Cells and Mediators



Asthma Inflammation: Cells and Mediators

Inflammatory cells

Mast cells
Eosinophils
Th2 cells
Basophils
Neutrophils
Platelets

Structural cells

Epithelial cells
Sm muscle cells
Endothelial cells
Fibroblast
Nerves



Mediators

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



Effects

Bronchospasm
Plasma exudation
Mucus secretion
AHR
Structural changes

Hypertrophied muscle happens after
recurrent bronchospasm
Not reversible



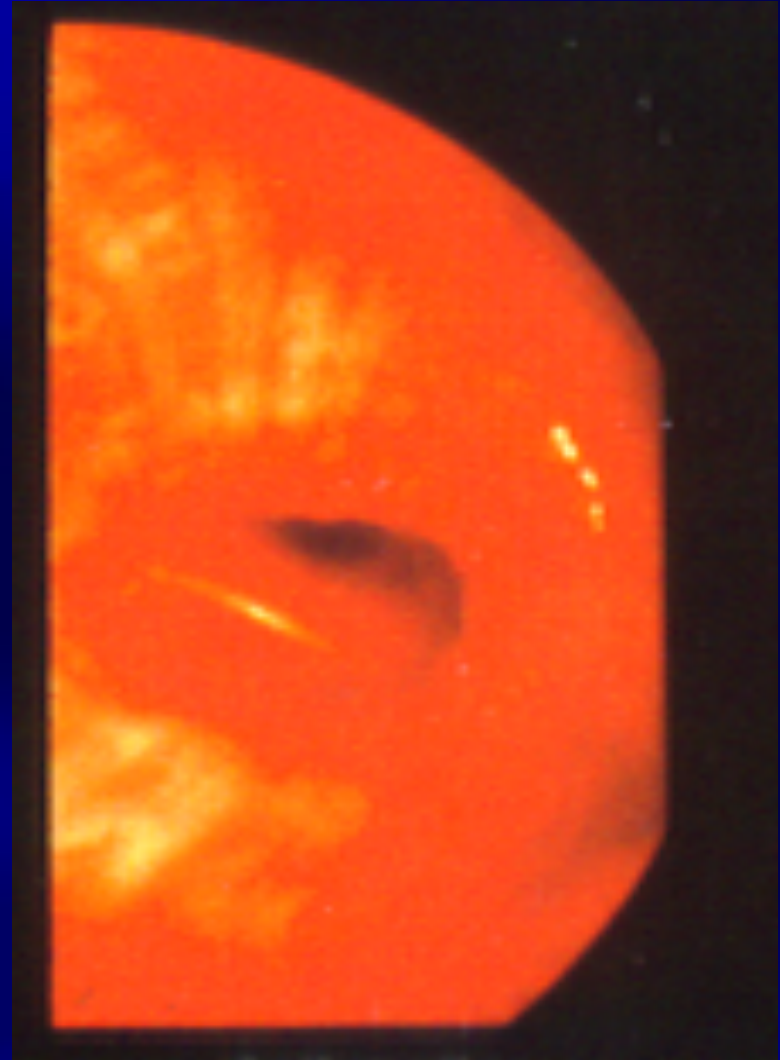


No body will do bronchoscopy to asthma patient because the airway is hyper responsive and it will cause spasm and it will cause obstruction

NORMAL

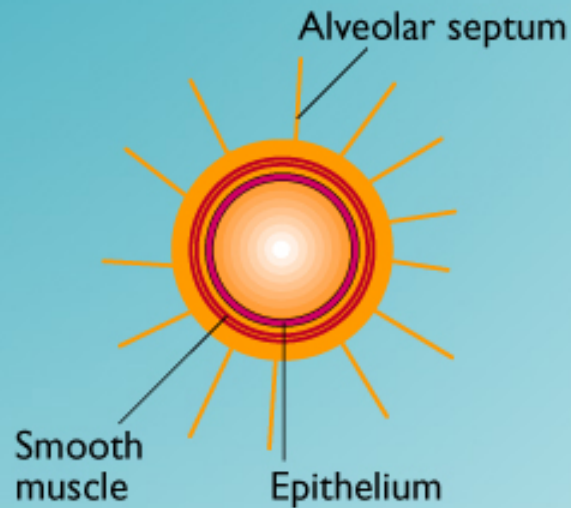


ASTHMA

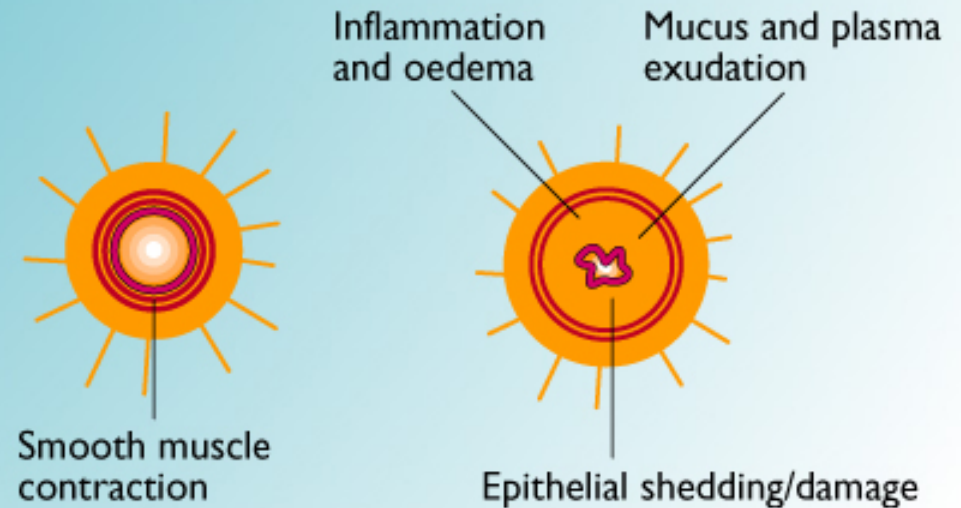


Asthma mainly is in the lower airways so expiratory stridor
So during inspiration they will have dilated airway and during expiration more obstructed airway

Healthy Airway

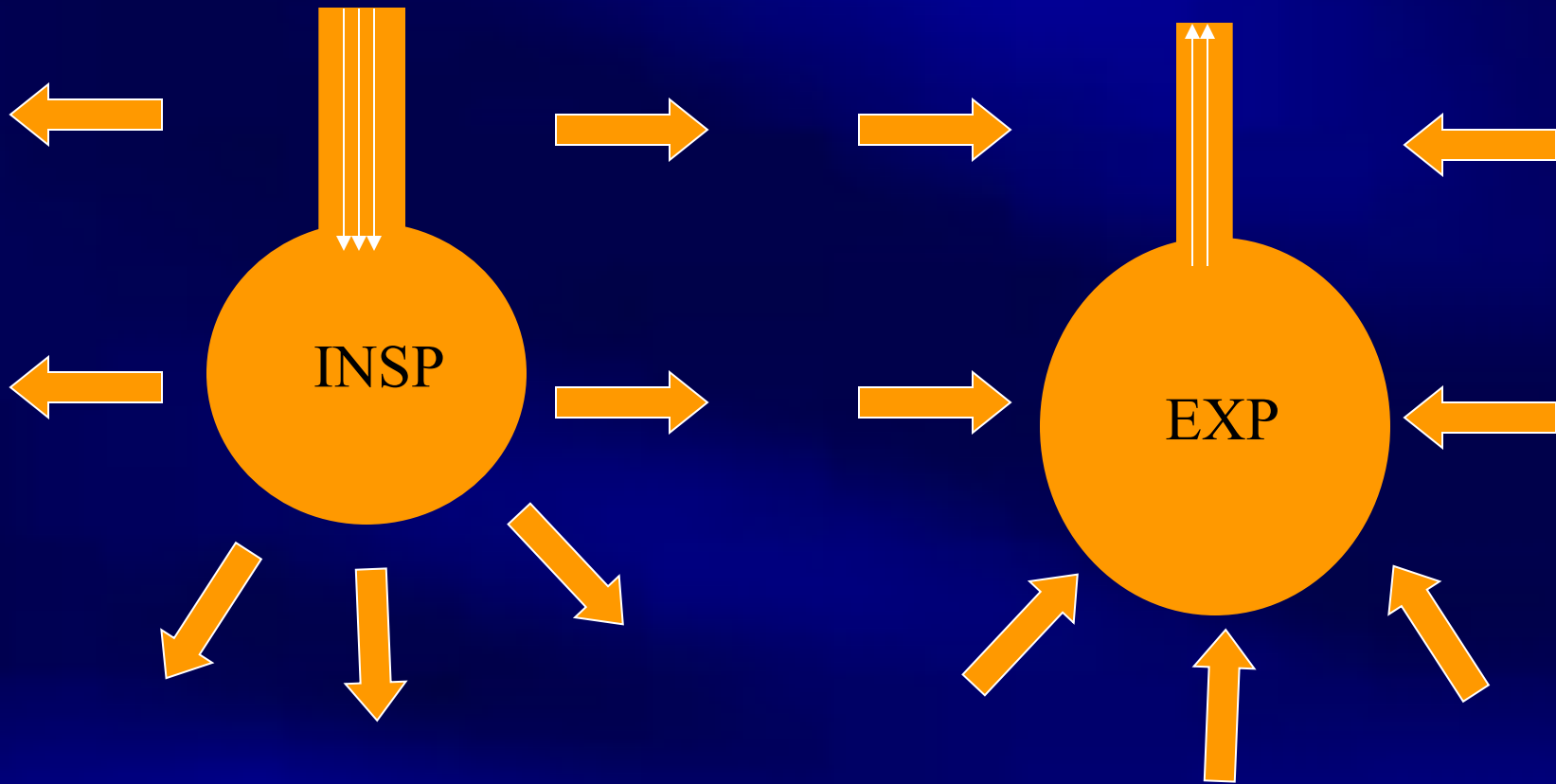


Asthmatic Airway

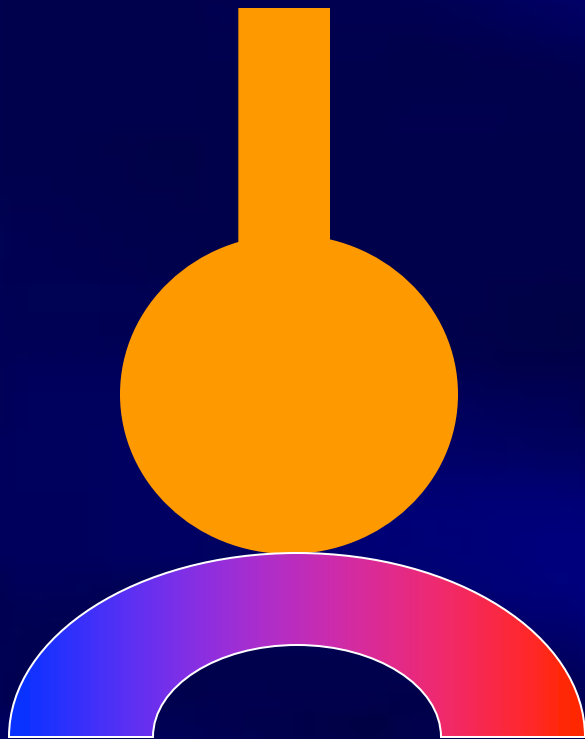


During inspiration air will enter the lung normally but during expiration the air will not be able to go out because of the lower airway obstruction so there will be air trapping and this will cause air leak because the lung is full > pneumothorax, Pneumomediastinum , subcutaneous emphysema
Also the hyperinflation will also cam compromise the circulation by decreasing cardiac output

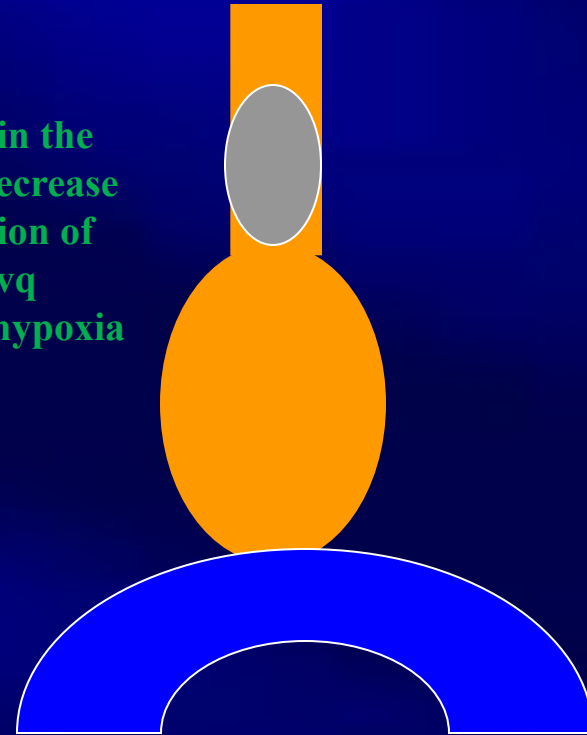
AIR TRAPPING



Ventilation Perfusion (V/Q) Mismatch



Obstruction in the alveoli will decrease the oxygenation of the blood so vq mismatch > hypoxia





Burden of Asthma

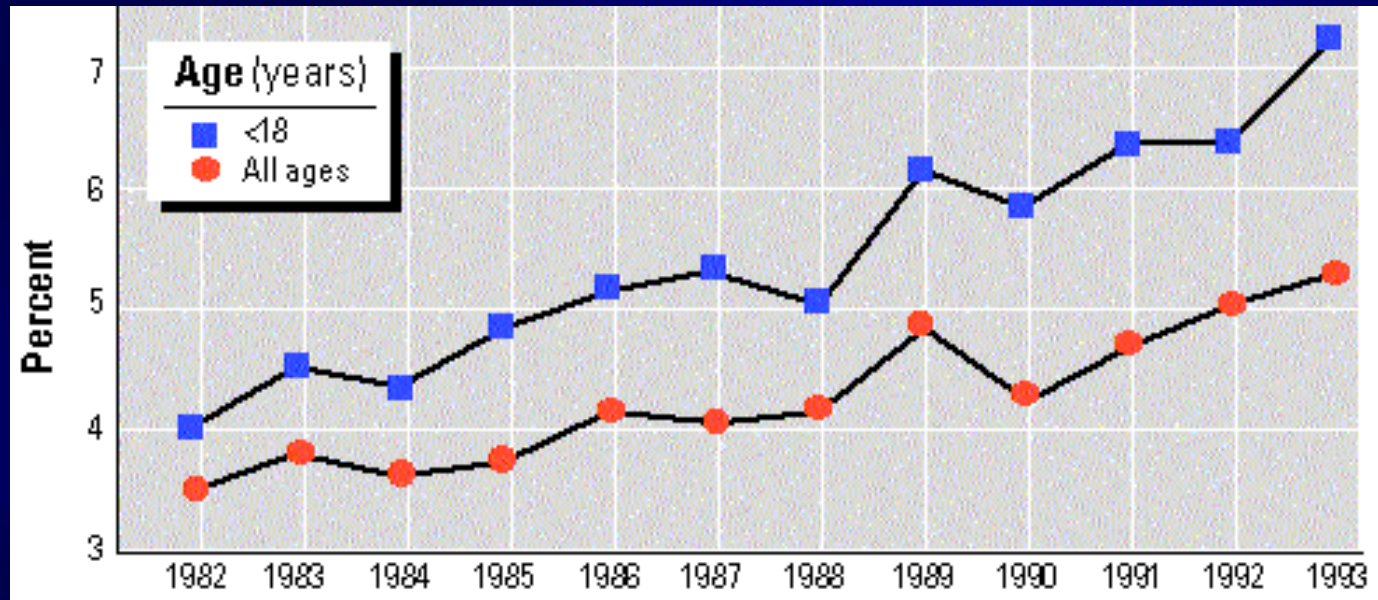
- Asthma is one of the most common chronic diseases worldwide with an estimated 300 million affected individuals
- Prevalence increasing in many countries **now is plateauing**, especially in children
- A major cause of school/work absence

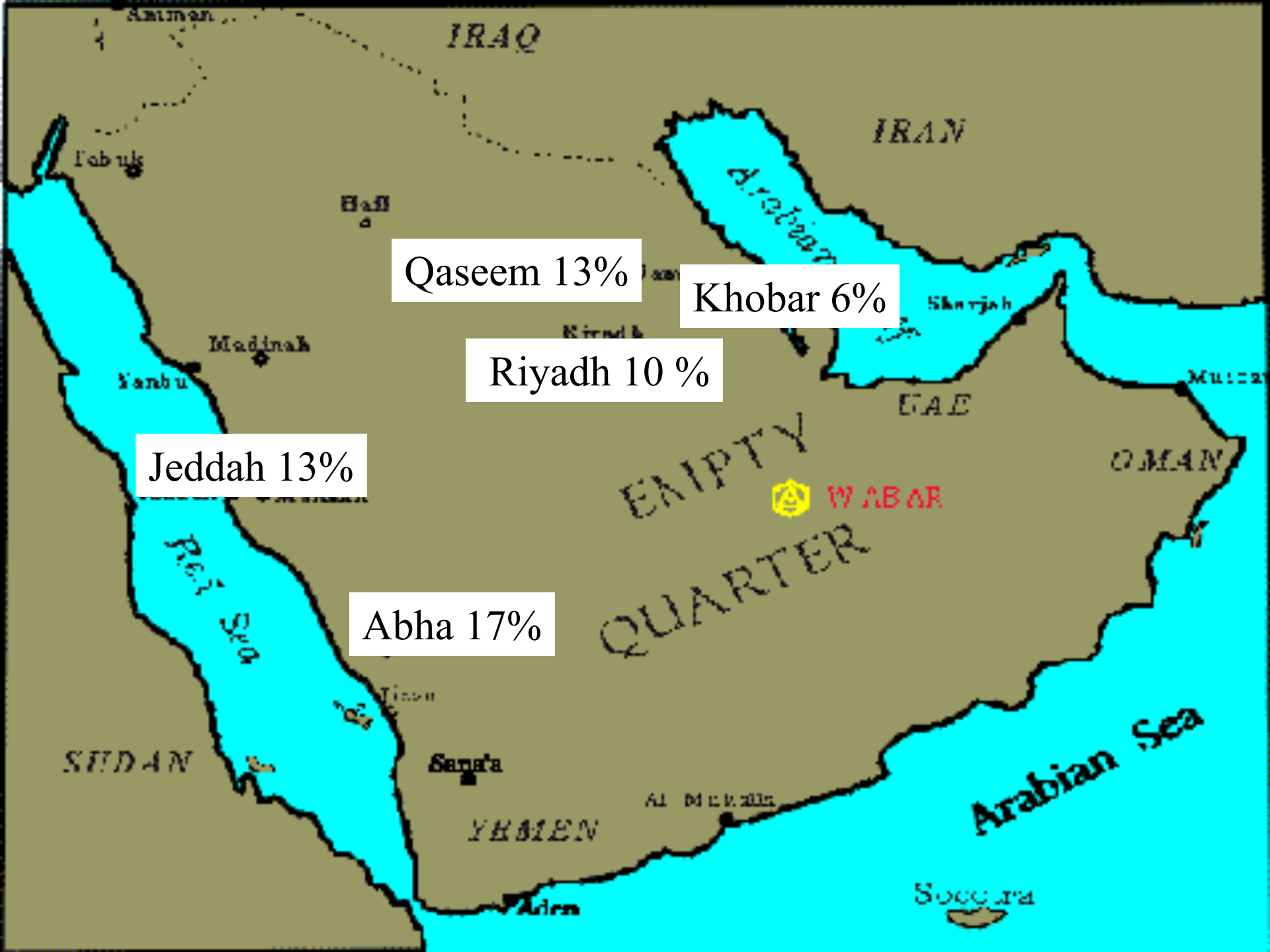
Asthma Prevalence



10 - 15%

Asthma Prevalence





Qaseem 13%

Khobar 6%

Riyadh 10 %

Jeddah 13%

Abha 17%

WABAR



Factors that Influence Asthma Development and Expression

Host Factors You need both to develop asthma

- Genetic
 - Atopy
 - Airway hyperresponsiveness
- Gender
- Obesity

Environmental Factors

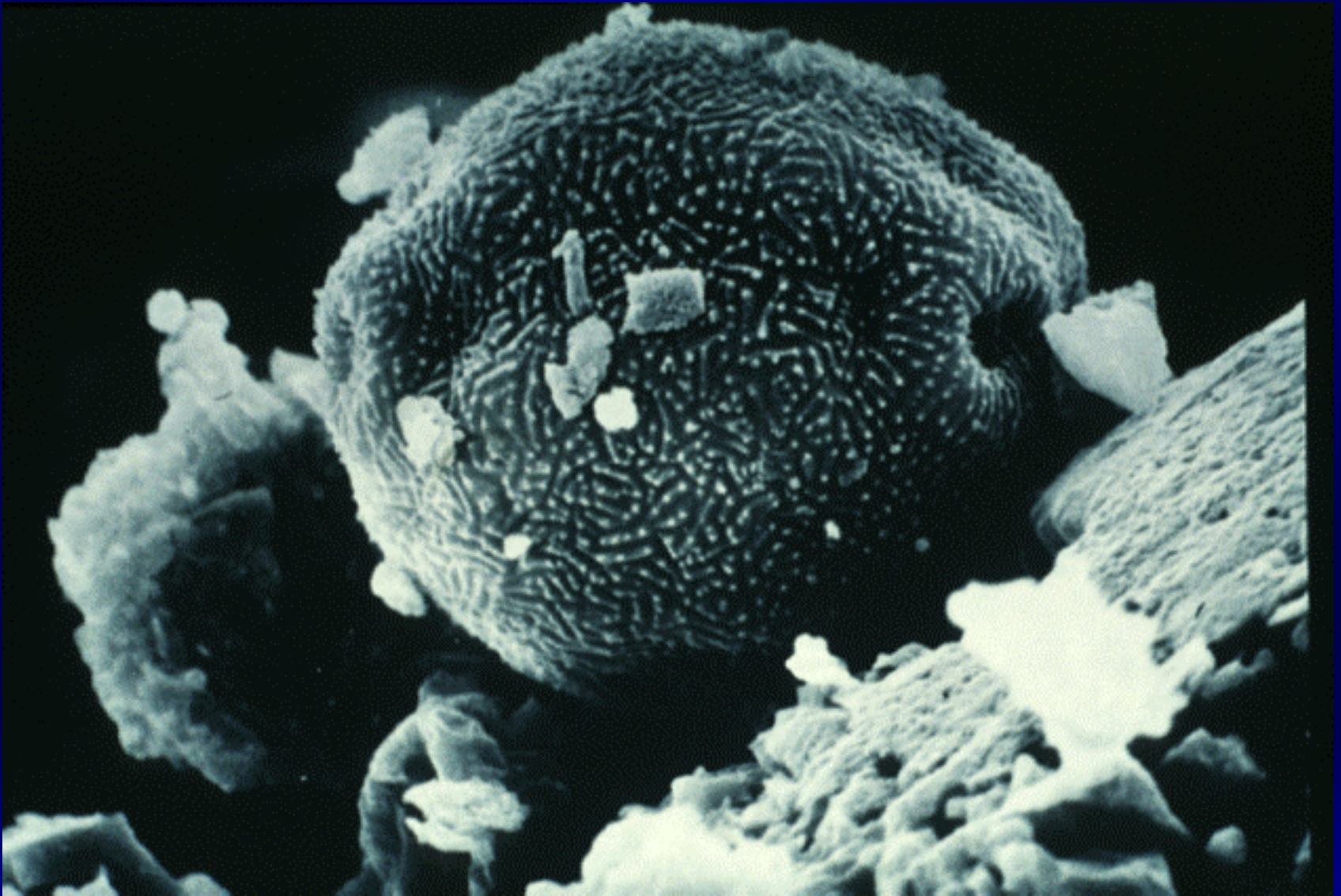
- Indoor allergens
- Outdoor allergens
- Occupational sensitizers
- Tobacco smoke
- Air Pollution
- Respiratory Infections
- Diet

Environmental Allergens and Childhood Asthma

- Dust mites *عثة غبار المنزل*
They produce fecal material
- Furry pets The effect will take months to be cleared after you remove the pets
- Molds
- Cockroaches
- **Cigarette Smoking**



POLLENS



Management of Chronic Asthma

History

Daytime symptoms how many time in a day

Nocturnal symptoms how many time in month

Reliever use how many time in a weak Exacerbation

Activities

These questions tells you Is it controlled or not

- Symptoms (cough, wheeze, SOB)
- Onset, duration, frequency and severity
- Activity and nocturnal exacerbation
- Previous therapy
- Triggers
- Other atopies
- Family history
- Environmental history, SMOKING
- Systemic review



Usually the child is normal between the attack
For example if a child presented with cough and Diarrhoea or failure to thrive think about another disease





Physical Examination

- Growth parameter
- ENT Not helpful
- Features of atopy
- Chest findings
- PEF
- Asthma doesn't cause clubbing because clubbing come with suppuratif lung disease

Investigations

- Pulmonary Function *the only thing can help*
- Chest X ray in some.
- Allergy testing in some

Doesn't help you





Skin Testing

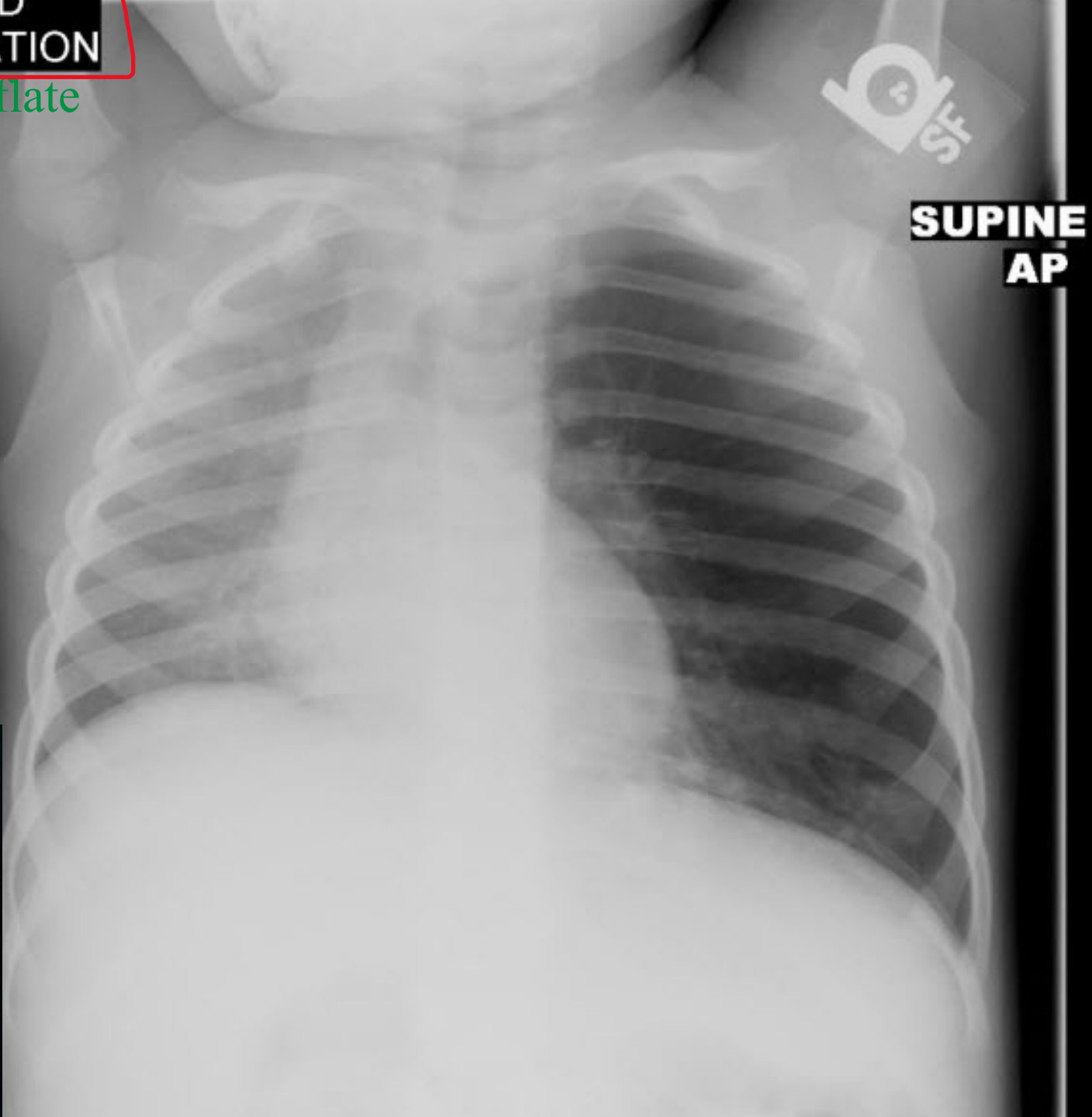
Differential Diagnosis

- Recurrent aspiration
- Bronchiolitis
- Cardiac failure
- Bronchopulmonary Dysplasia
- Inhaled foreign body
- Bronchiectasis
- Gastroesophageal Reflux
- Primary Ciliary Dyskinesia
- Cystic Fibrosis
- Vocal cord dysfunction/
Hyperventilation
- Structural anomalies:
Tracheomalacia/
Bronchomalacia..etc.

**FORCED
EXPIRATION**

Left lung didn't deflate
do to obstruction

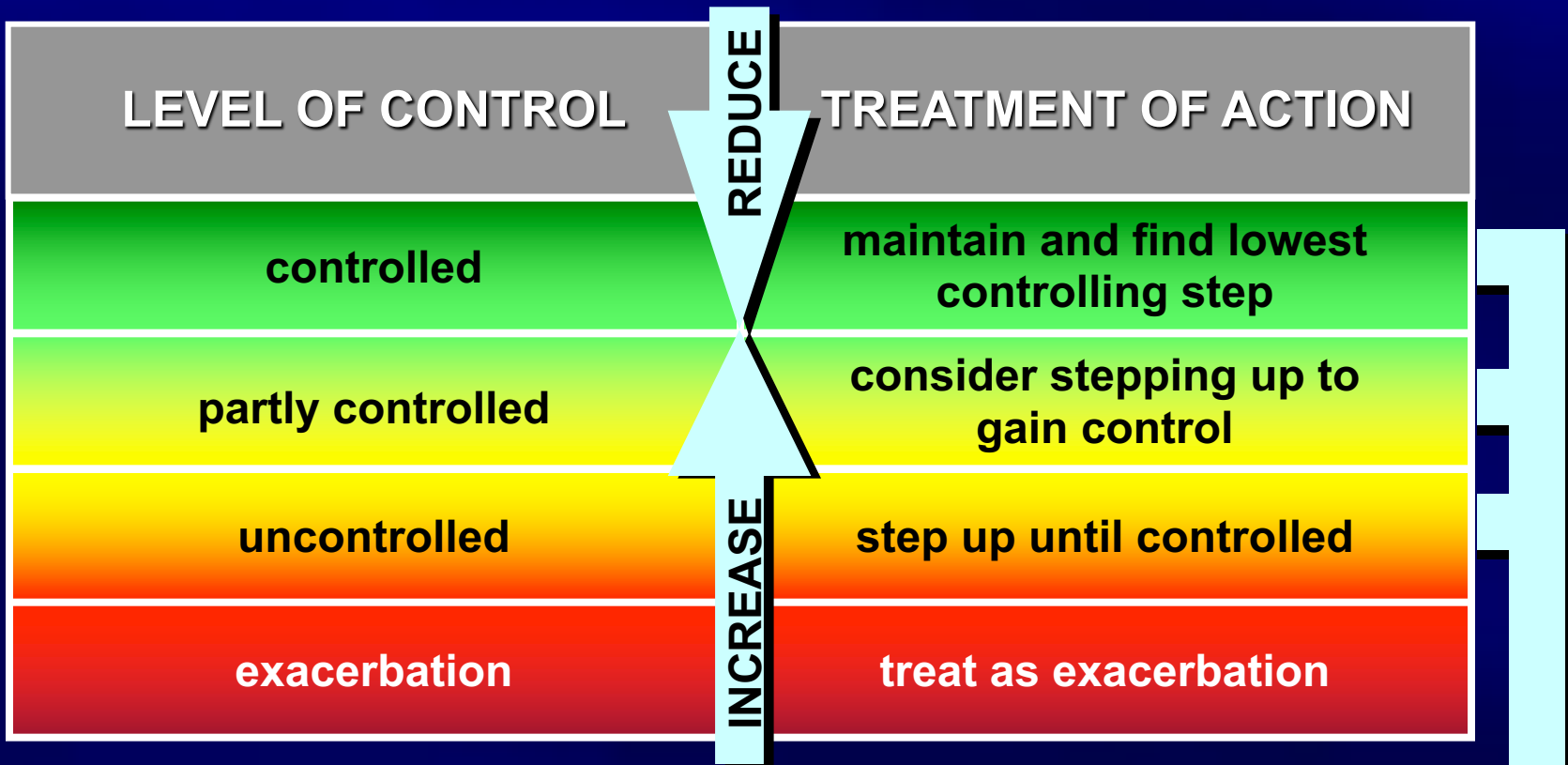
**SUPINE
AP**





Levels of Asthma Control

<i>Characteristic</i>	Controlled (All of the following)	Partly controlled (Any present in any week)	Uncontrolled	
Daytime symptoms	None (2 or less / week)	More than twice / week	3 or more features of partly controlled asthma present in any week	
Limitations of activities	None	Any		
Nocturnal symptoms / awakening	None	Any		
Need for rescue / "reliever" treatment	None (2 or less / week)	More than twice / week		
Lung function (PEF or FEV₁)	Normal	< 80% predicted or personal best (if known) on any day		
Exacerbation	None	One or more / year		1 in any week



You will not be asked about the details

REDUCE		TREATMENT STEPS					INCREASE	
		STEP 1	STEP 2	STEP 3	STEP 4	STEP 5		
		asthma education						
		environmental control						
		as needed rapid-acting β_2 -agonist	as needed rapid-acting β_2 -agonist					
CONTROLLER OPTIONS		SELECT ONE	SELECT ONE	ADD ONE OR MORE	ADD ONE OR BOTH			
		low-dose ICS*	low-dose ICS <i>plus</i> long-acting β_2 -agonist	medium- <i>or</i> high-dose ICS <i>plus</i> long-acting β_2 -agonist	oral glucocorticosteroid (lowest dose)			
		leukotriene modifier**	medium- <i>or</i> high-dose ICS	leukotriene modifier	anti-IgE treatment			
			low-dose ICS <i>plus</i> leukotriene modifier	sustained-release theophylline				
			low-dose ICS <i>plus</i> sustained-release theophylline					

*inhaled glucocorticosteroids

** receptor antagonist or synthesis inhibitors



Treatment objectives

- Achieve and maintain control of symptoms
- Maintain normal activity levels, including exercise
- Maintain pulmonary function as close to normal levels as possible
- Prevent asthma exacerbations
- Avoid adverse effects from asthma medications
- Prevent asthma mortality

Treatment strategy

1. Develop Patient/Doctor Partnership
2. Identify and Reduce Exposure to Risk Factors
3. Assess, Treat and Monitor Asthma
4. Manage Asthma Exacerbations
5. Special Consideration

Pharmacological therapy

■ Relievers

- Inhaled fast-acting β_2 -agonists
- Inhaled anticholinergics

■ Controllers

- Inhaled corticosteroids
- Inhaled long-acting β_2 -agonists **use it with ICS never alone**
- Inhaled cromones
- Oral anti-leukotrienes
- Oral theophyllines
- Oral corticosteroids

Why don't patients comply with treatment?

Intentional

- Feel better
- Fear of side effects
- Don't notice any benefit
- Fear of addiction
- Fear of being seen as an invalid
- Too complex regimen
- Can't afford medication

Unintentional

- Forget treatment
- Misunderstand regimen / lack information
- Unable to use their inhaler
- Run out of medication

Cromolyn Sodium

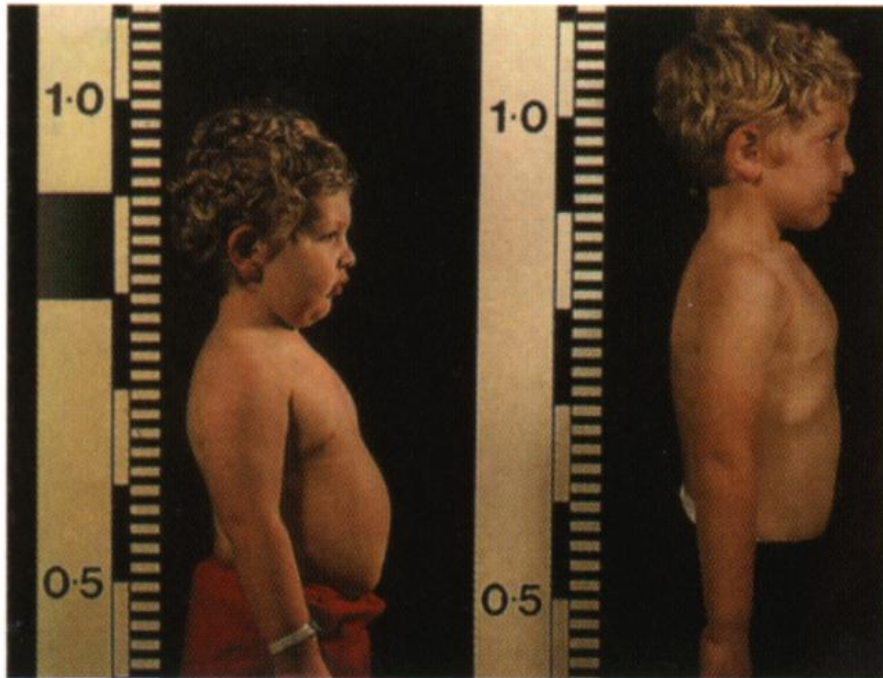
- Non-steroidal anti-inflammatory
- Weak action on Early and late phases
- Slow onset of action
- If no response in 6 weeks change to ICS
- Side effects: Irritation

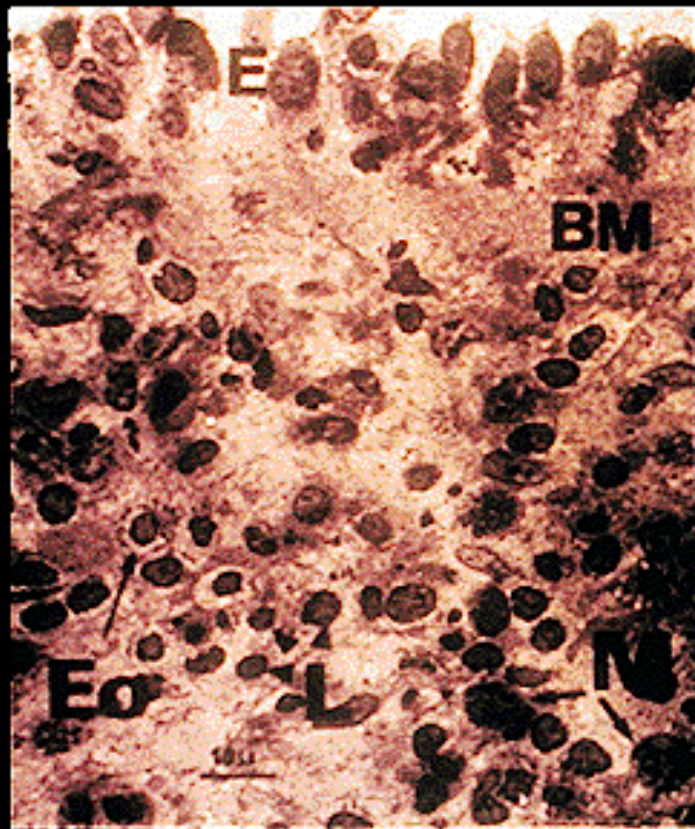


Amni Visnaga, also known as khellin, from which the cromone for DSCG was derived

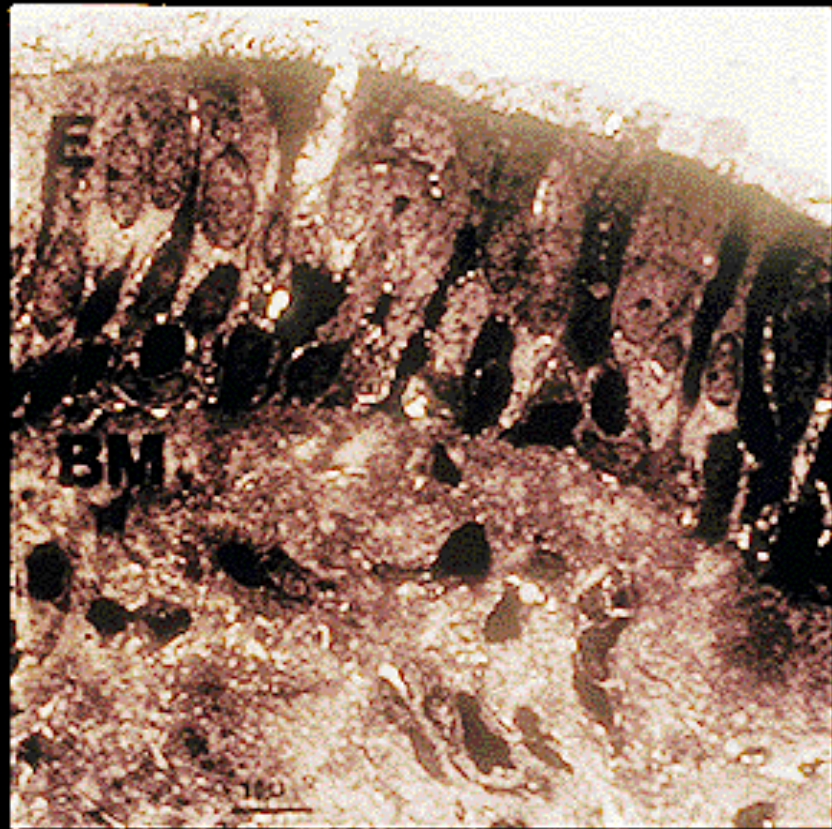
Inhaled Corticosteroids

- Effective in most cases
- Safe especially at low doses
- The anti-inflammatory of choice in asthma





Asthmatic



Steroid-treated asthmatic

Inhaled Steroids

Side Effects

- Growth: No significant effect at low to moderate doses.
- Bones: not important
- HPA axis: No serious clinical effect (high doses)
- Alteration of glucose and lipid metabolism: Clinical significant is unclear (high doses)
- Cataract: No increase risk
- Skin: Purpura, easily bruising, dermal thinning
- Local side effects

Nebulizers



MDI and spacer



Dry powder inhalers



MANAGEMENT OF ACUTE ASTHMA

Assessment: History

- Symptoms
- Previous attacks
- Prior therapy
- Triggers

Physical examination:

Signs of airway obstruction:

- **Fragmented speech**
- **Unable to tolerate recumbent position**
- **Expiration > 4 seconds**
- **Tachycardia, tachypnea and hypotension**
- **Use of accessory muscles**
- **Pulsus paradoxus > 10 mmhg**
- **Silent hyperinflated chest** *in very sever cases*
- **Air leak**

- **Cyanosis**
- **Wheezing is a good sign it tells you there is airflow**

Physical examination:

Signs of tissue hypoxia:

- Cyanosis
- Cardiac arrhythmia and hypotension
- Restlessness, confusion, drowsiness and obtundation

Physical examination:

Signs of Respiratory muscles fatigue:

- Increase respiratory rate
- Respiratory alterans (alteration between thoracic and abdominal muscles during inspiration)
- Abdominal paradox (inward movement of the abdomen during inspiration)

Investigations:

- Peak expiratory flow rate
- Pulse oxymetry
- ABG



- CXR



ONLY IN FEW CASES

The First Hour

Oxygen

- Hypoxemia is common
- It worsens airway hyperreactivity
- Monitor saturation

Inhaled β_2 agonist

Every 20 minutes in the first hour

Assess after each nebulizer

You can give it with spacer to
avoid infections specifically
now to avoid Covid-19



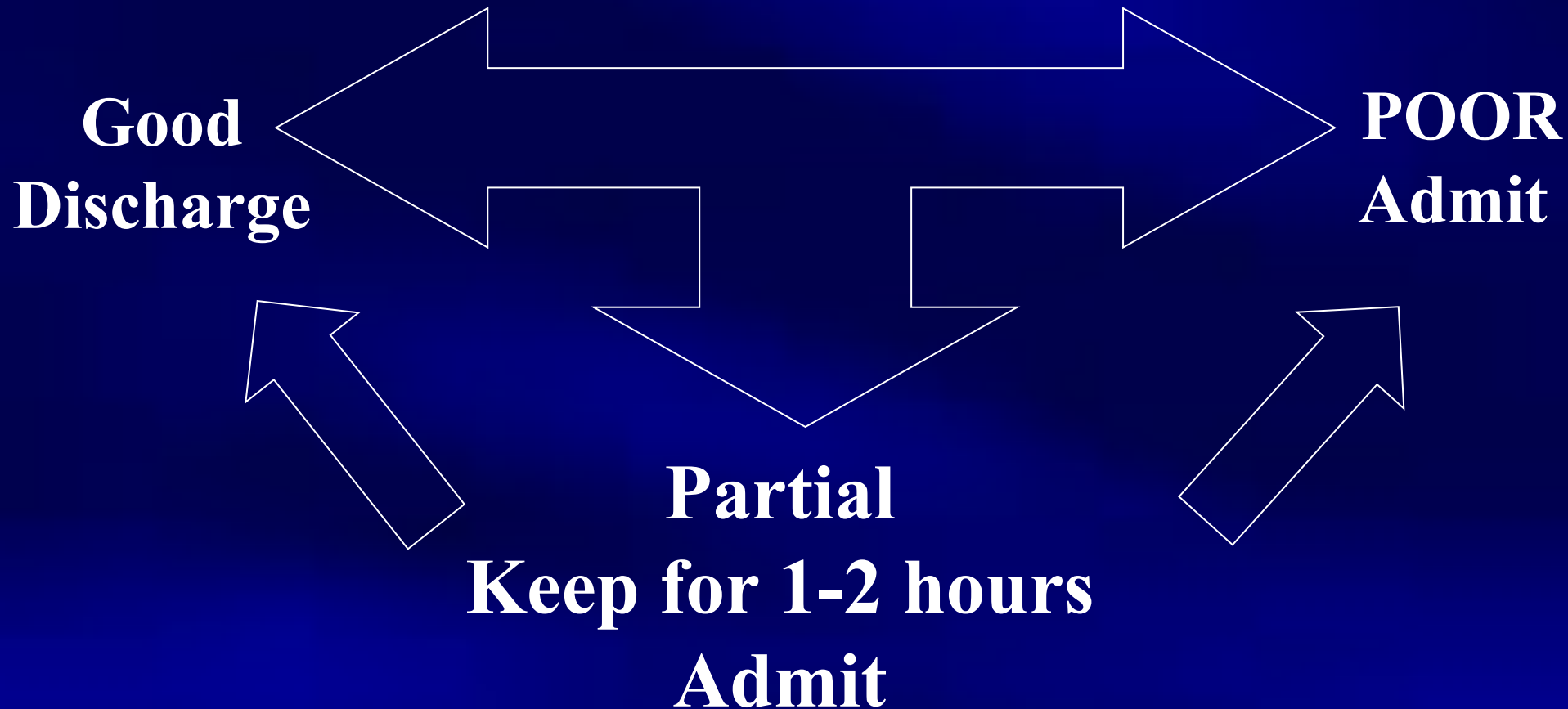
Steroids

- If not responding to the β agonist
- If severe in the beginning
- If on PO prednisone or high dose inhaled steroids.
- Previous severe attacks

Ipratropium Bromide

- **Anti-cholinergic**
- **For severe cases**
- **Along with β 2 agonist**

Response to the first hour



Discharge

- Follow up
- Give inhaled β_2 agonist
- Steroids
- When to come back?