

# DISEASES OF THE RESPIRATORY SYSTEM LECTURES 3 AND 4

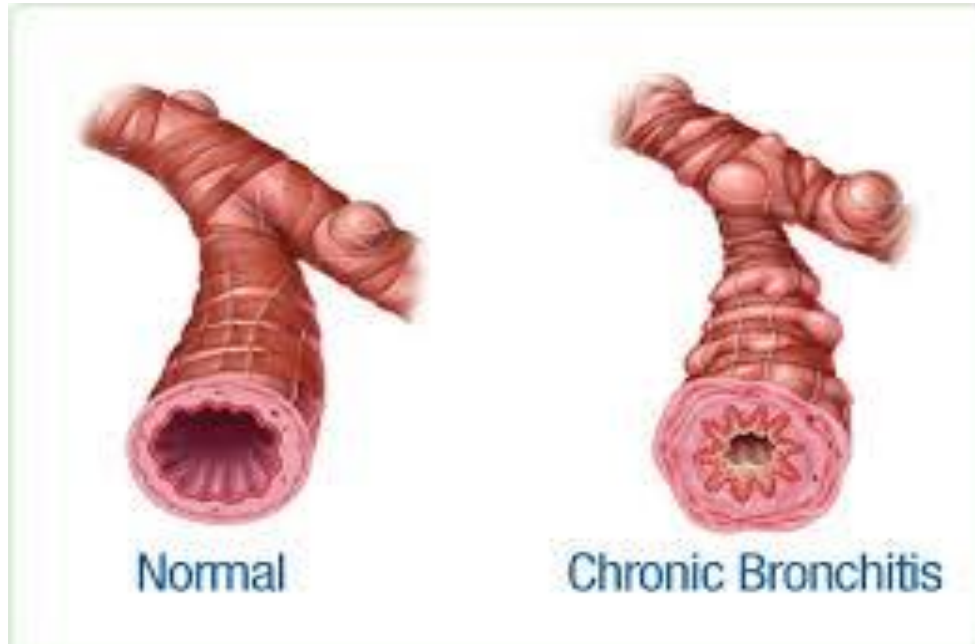
DR HEYAM AWAD

FRCPATH

# CHRONIC BRONCHITIS

PERSISTENT **PRODUCTIVE COUGH** FOR AT LEAST  
**THREE CONSECUTIVE MONTHS** FOR AT LEAST  
**TWO CONSECUTIVE YEARS.**

# CHRONIC BRONCHITIS



# CAUSES

- **SMOKING RELATED.**
- AIR POLLUTION.. SO<sub>2</sub>, NO.

# PATHOGENESIS

- HYPERSECRETION OF MUCUS.
- DUE TO HYPERTROPHY OF MUCUS SECRETING GLANDS IN TRACHEA AND MAIN BRONCHI.
- INCREASE IN MUCIN SECRETING GOBLET CELLS IN THE EPITHELIUM OF SMALL BRONCHIA.

# PATHOGENESIS

- SMOKING ALSO CAUSES INFLAMMATION AND INFILTRATION BY LYMPHOCYTES, MACROPHAGES AND NEUTROPHILS.

# CAUSE OF AIRFLOW OBSTRUCTION

## CHRONIC BRONCHIOLITIS

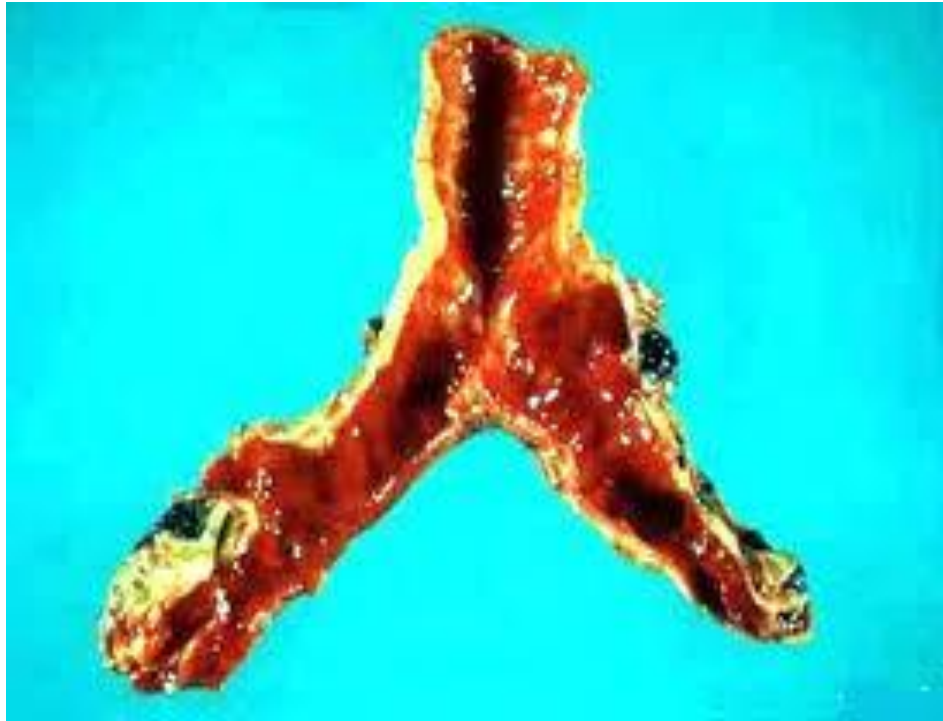
- SMALL AIRWAY DISEASE CAUSED BY GOBLET CELL METAPLASIA, MUCUS PLUGGING, INFLAMMATION AND FIBROSIS.

# MORPHOLGY

- COEXISTENT EMPHYSEMA ALSO CAUSES AIRWAY OBSTRUCTION.
- CHRONIC BRONCHITIS THAT IS ACCOMPANIED BY SIGNIFICANT AIRFLOW OBSTRUCTION IS ALMOST ALWAYS ASSOCIATED WITH EMPHYSEMA.



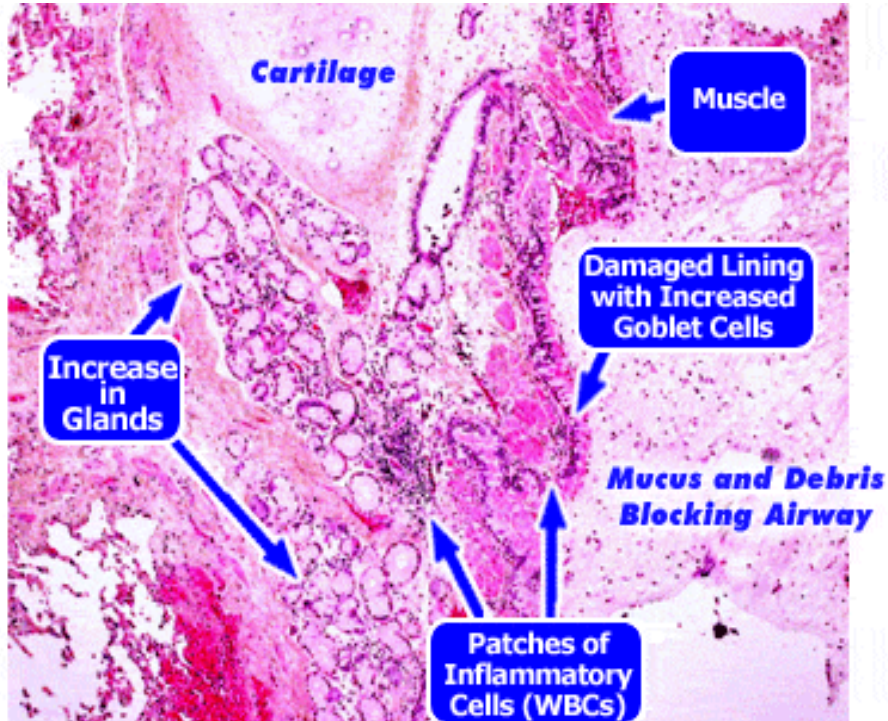
# MORPHOLOGY

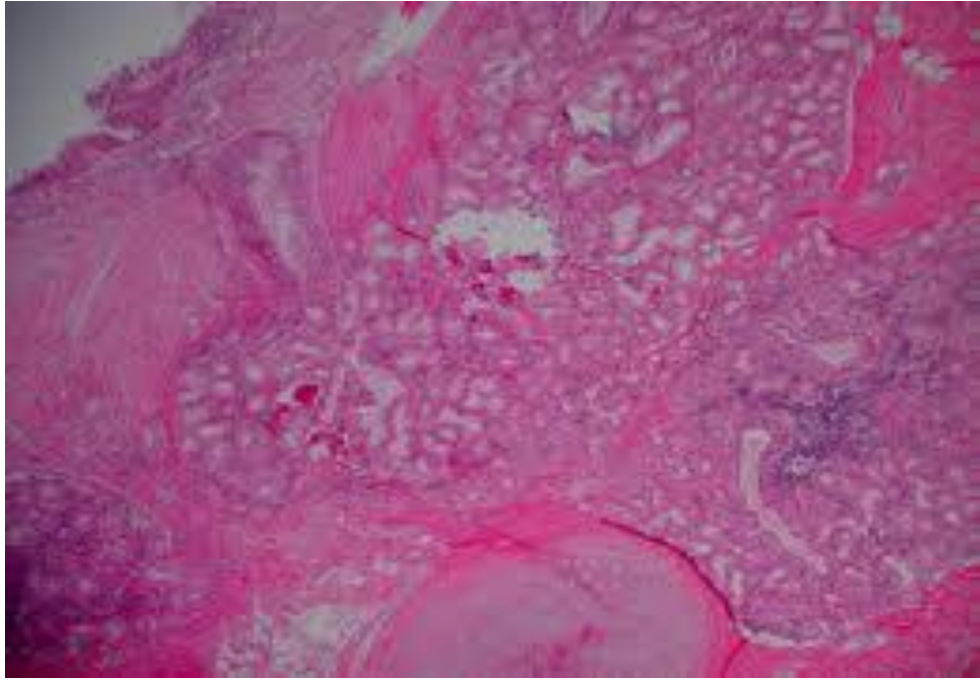


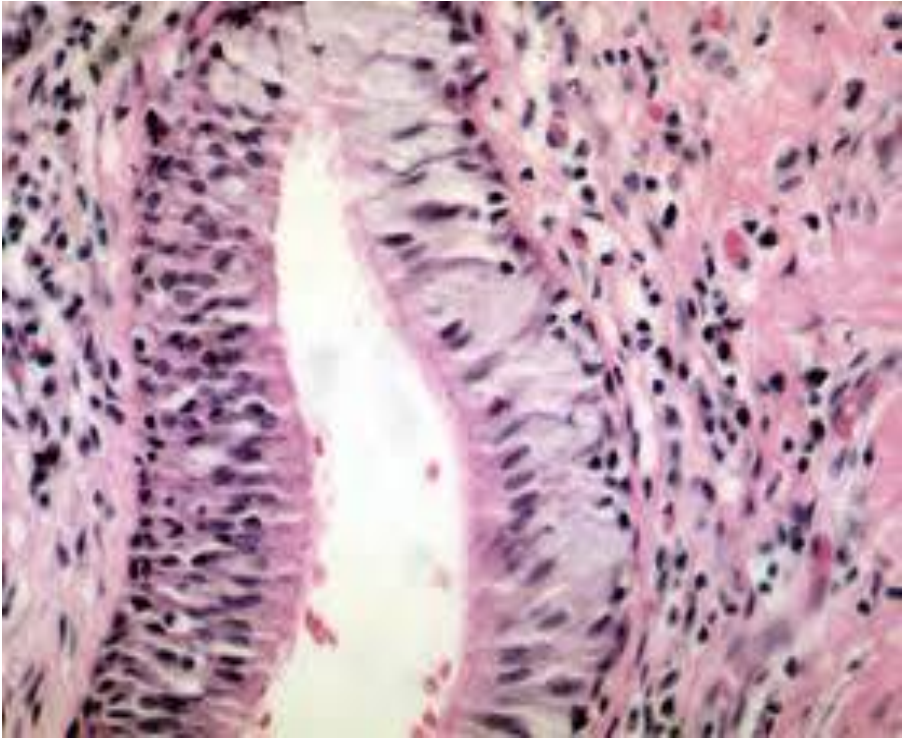
# MORPHOLOGY

- MUCOSA OF THE LARGE AIRWAYS IS HYPEREMIC AND SWOLLEN AND COVERED BY MUCINOUS SECRETIONS.
- ENLARGEMENT OF THE MUCUS SECRETING GLANDS.

- THICKNESS OF THE MUCOSAL GLAND LAYER TO THE BRONCHIAL WALL = REID INDEX.
- NORMAL REID INDEX IS 0.4.







# CLINICAL FEATURES

- PRODUCTIVE COUGH.
- HYPERCAPNIA.
- HYPOXEMIA.
- CYANOSIS.
  
- BLUE BLOATERS





# COMPLICATIONS

- PULMONARY HYPERTENTION.
- CARDIAC FAILURE.
- RECURRENT INFECTIONS.
- RESPIRATORY FAILURE.



# ASTHMA

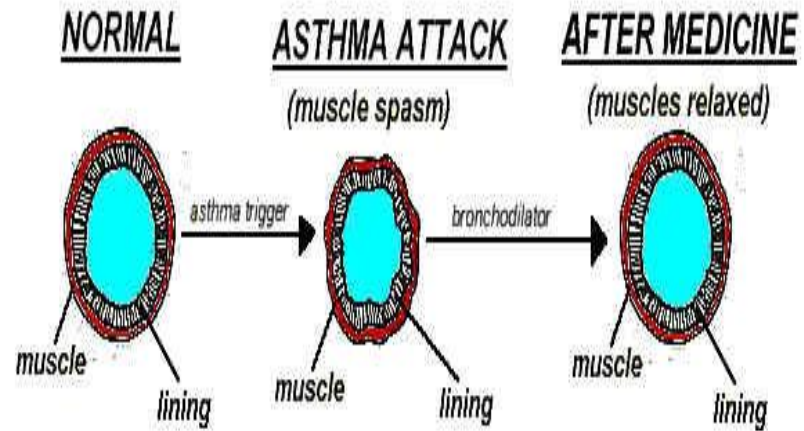
- CHRONIC INFLAMMATORY DISORDER WHICH CAUSES RECURRENT EPISODES OF WHEEZING, BREATHLESSNESS, COUGH, AND CHEST TIGHTNESS.

# ASTHMA

## **INTERMITTENT, REVERSIBLE :**

- AIRWAY OBSTRUCTION.
- CHRONIC BRONCHIAL INFLAMMATION WITH **EOSINOPHILS**.
- BRONCHIAL SMOOTH MUSCLE HYPERTROPHY AND HYPERREACTIVITY.
- INCREASED MUCUS SECRETION.

# REVERSIBLE CHANGES



# EPIDEMIOLOGY

- SIGNIFICANT INCREASE IN ASTHMA IN THE WESTERN WORLD IN THE LAST FOUR DECADES.
- HYGEINE HYPOTHESIS: DECREASED INFECTIONS CHANGES THE IMMUNE BALANCE AND PROMOTE ALLERGIC IMMUNE RESPONSES.

# TYPES OF ASTHMA

- **ATOPIC**: EVIDENCE OF ALLERGIC SENSITIZATION.
- **NONATOPIC**.

# AETIOLOGY

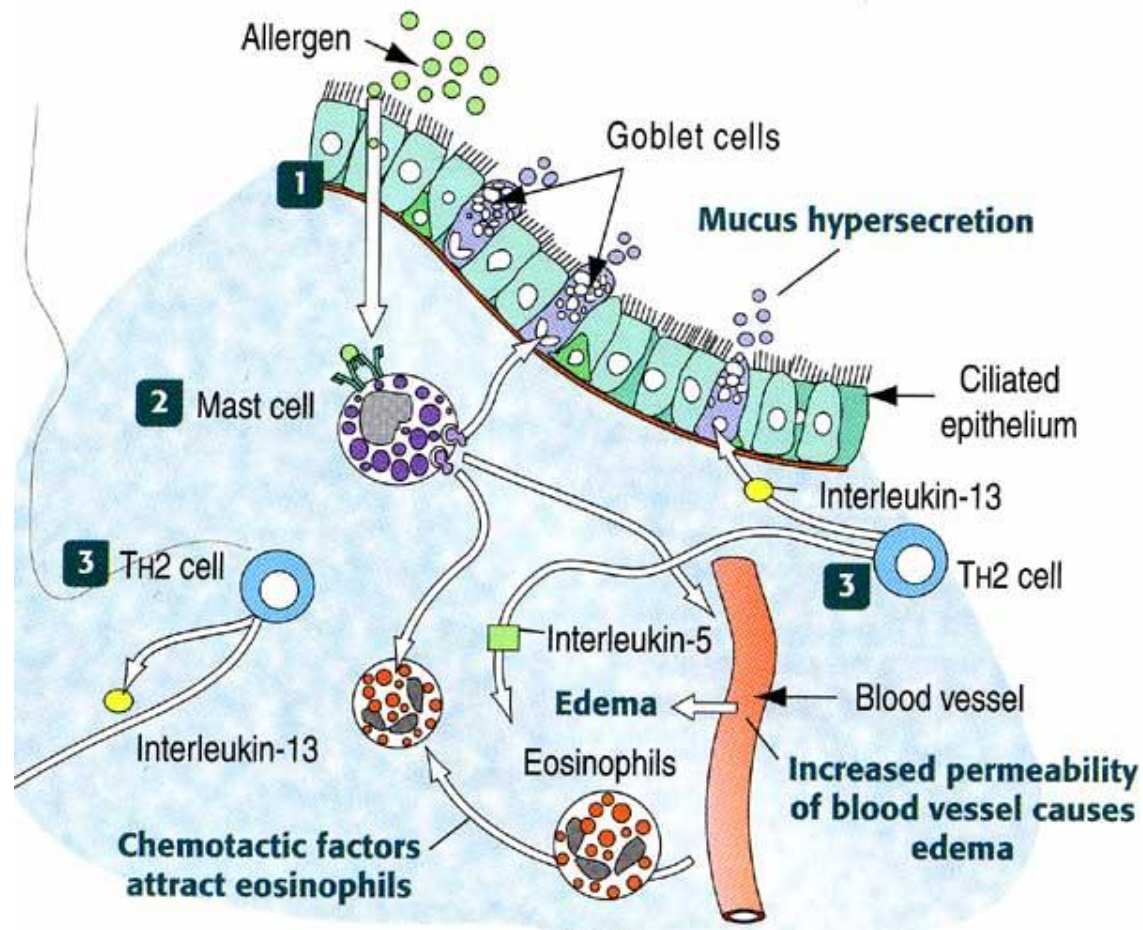
- GENETIC PREDISPOSITION TO TYPE 1 HYPERSENSITIVITY.
- BRONCHIAL HYPERRESPONSIVENESS TO VARIETY OF STIMULI.
- ACUTE AND CHRONIC INFLAMMATION.



# INFLAMMATION

- TYPE 2 T HELPER CRITICAL IN THE PATHOGENESIS.
- T2H PRODUCES CYTOKINES :
- IL4...IGE PRODUCTION.
- IL5...EOSINOPHIL ACTIVATION.
- IL3...MUCUS PRODUCTION.

- IGE ...COATES MAST CELLS WHICH, ON EXPOSURE TO ALLERGENS, RELEASES GRANULE CONTENT.



# INFLAMMATION

- DEGRANULATION OF MAST CELLS PRODUCES TWO WAVES OF REACTION:
- **EARLY**, IMMEDIATE PHASE.
- **LATE** PHASE.

# EARLY REACTION

- BRONCHO-CONSTRICTION..STIMULATION OF SUEPITHELIAL VAGAL RECEPTORS.
- INCREASED MUCUS.
- VASODILATION.

# LATE PHASE REACTION

- INFLAMMATION WITH ACTIVATION OF EOSINOPHILS. NEUTROPHILS AND T CELLS.

- REPEATED BOUTS OF INFLAMMATION LEAD TO STRUCTURAL CHANGES IN THE BRONCHIAL WALL.
- THIS IS CALLED AIRWAY REMODELING.

# AIRWAY REMODELING

- HYPERTROPHY OF BRONCHIAL SMOOTH MUSCLE .
- HYPERTROPHY OF MUCUS GLANDS.
- INCREASED VASCULARITY.
- DEPOSITION OF SUBEPITHELIAL COLLAGEN.



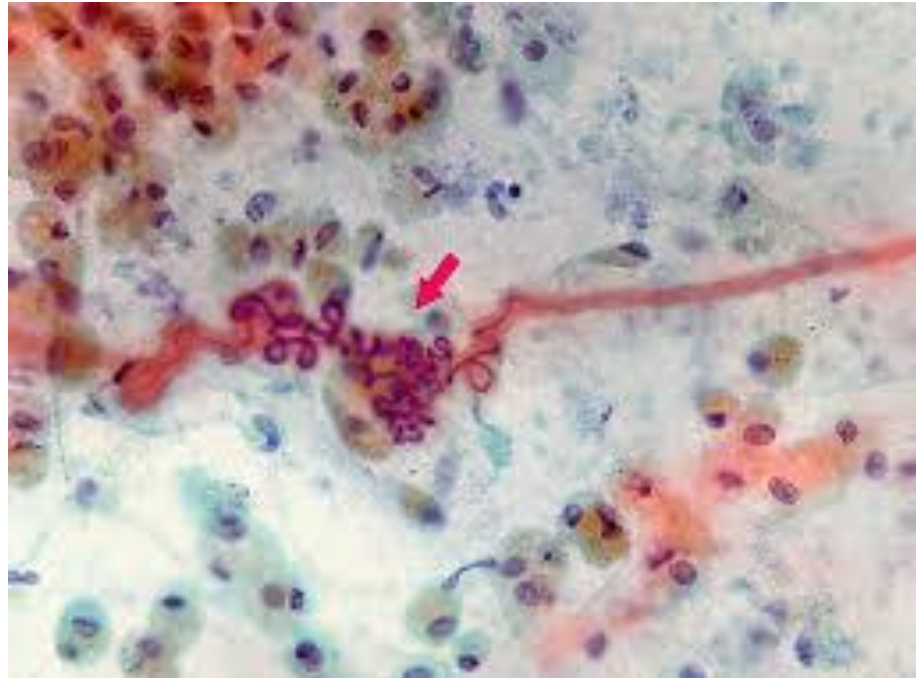
# MORPHOLOGY

- LUNGS OVERDISTENDED DUE TO OVERINFLATION.
- MUCUS PLUGS.

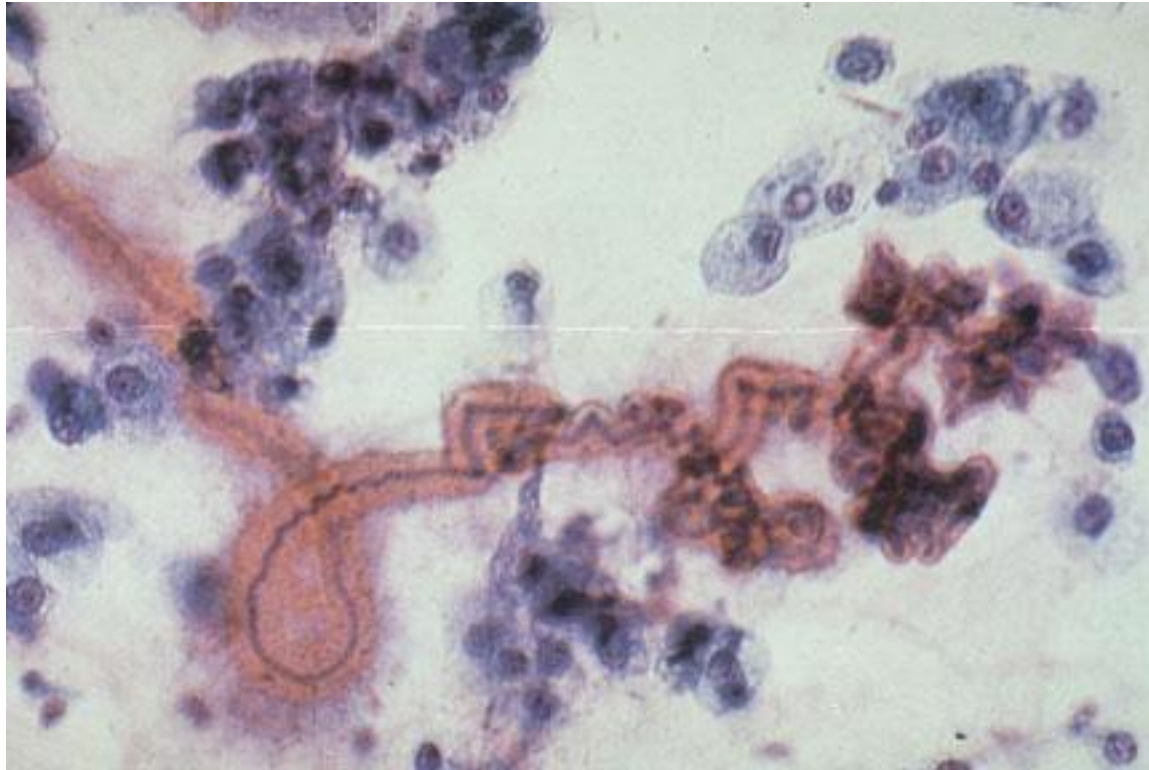
# HISTOLOGY

- MUCUS PLUGS:
- WHORLS OF SHED EPITHELIUM = CURSCHMAN SPIRALS.
- EOSINOPHILS.
- CHARCOT LADEN CRYSTALS = CRYSTALLOIDS MADE UP OF EOSINOPHIL PROTEINS.

# CURSCHMAN SPIRALS



# CURSCMAN SPIRALS

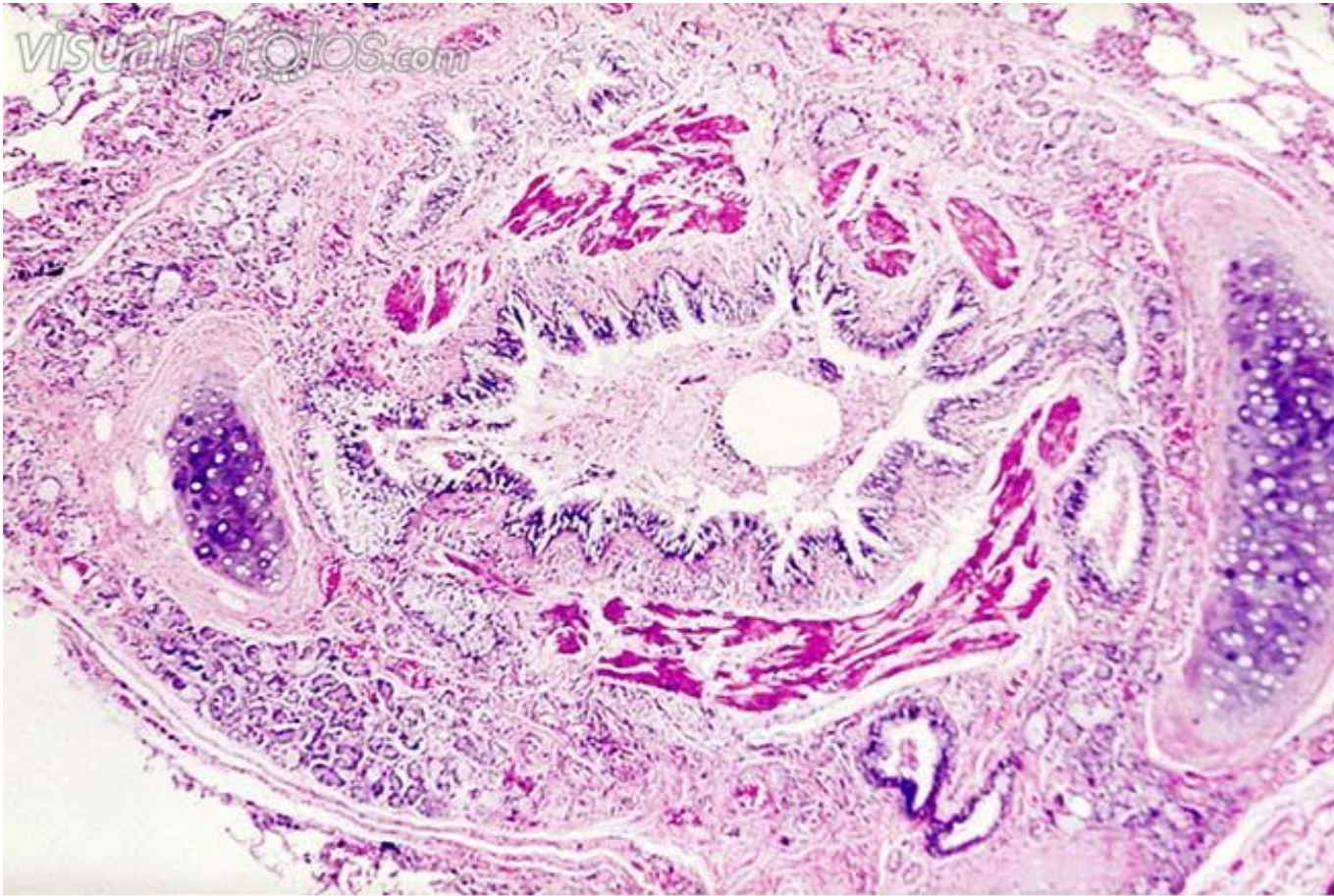


# CHARCOT LAREN CRYSTALS

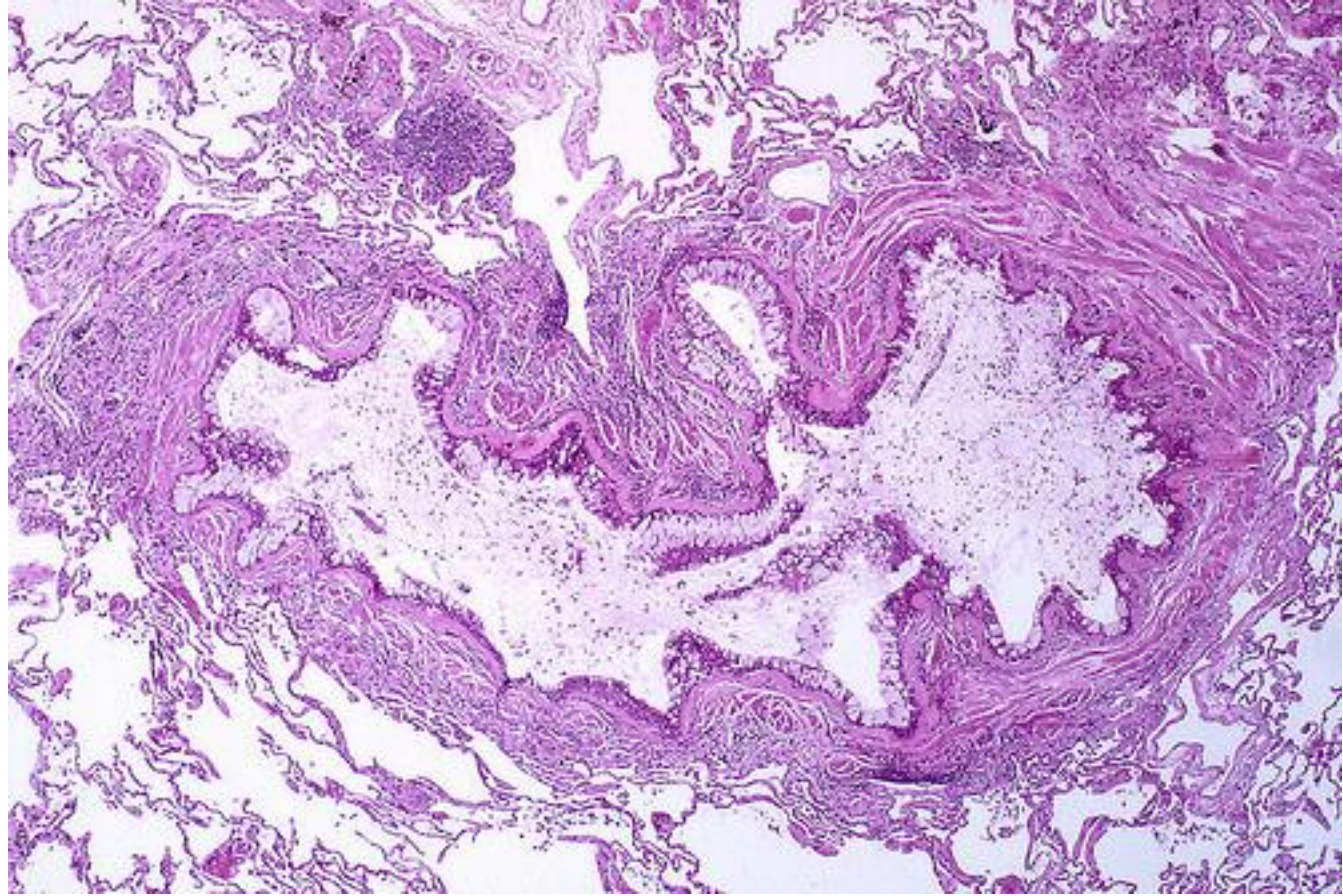


# AIRWAY REMODELING

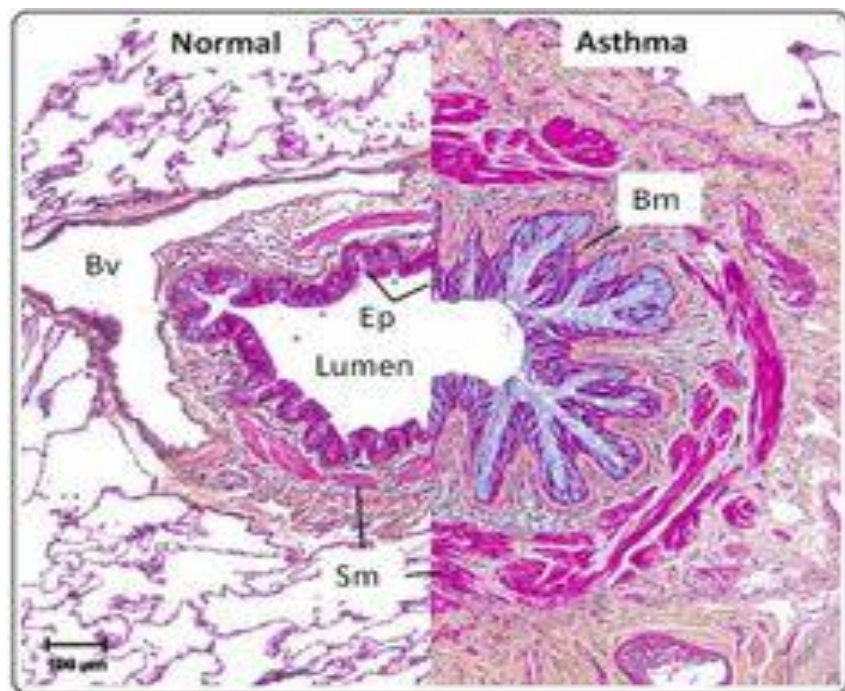
- THICKENED AIRWAYS.
- SUB-BASEMENT MEMBRANE FIBROSIS.
- INCREASED VASCULARITY.
- INCREASED SIZE OF SUBMUCOSAL GLANDS
- GOBLET CELL METAPLASIA.
- HYPERTROPHY AND/OR HYPERPLASIA OF BRONCHIAL MUSCLE



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# TYPES OF ASTHMA

## ATOPIC :

- THE MOST COMMON TYPE.
- BEGINS IN CHILDHOOD.
- TYPE 1 HYPERSENSITIVITY REACTION.
- POSITIVE FAMILY HISTORY.
- TRIGGERED BY ENVIRONMENTAL ANTIGENS OR INFECTIONS.

# TYPES OF ASTHMA

## NON ATOPIC ASTHMA

- NO EVIDENCE OF ALLERGEN SENSITIZATION
- SKIN TEST NEGATIVE.
- POSITIVE FAMILY HISTORY IS LESS COMMON.
- INFECTIONS COMMON.
- VIRAL INFLAMMATION LOWERS THRESHOLD OF THE SUBEPITHELIAL VAGAL RECEPTORS TO IRRITANTS.
- HUMORAL AND CELLULAR MEDIATORS SIMILAR TO ATOPIC ASTHMA.

# TYPES OF ASTHMA

## DRUG INDUCED ASTHMA

- ASPIRIN
- MECHANISM UNKNOWN
- ASPIRIN INHIBITS COX WITHOUT AFFECTING LIPOOXYGENASE PATHWAY SHIFTING THE BALANCE TO BRONCHOSPASM.

# TYPES OF ASTHMA

## OCCUPATIONAL ASTHMA

- PLASTIC FUMES.
- ORGANIC AND CHEMICAL DUST E.G WOOD, COTTON.
- GASES: TOLUENE

# CLINICAL FEATURES

- SEVERE DYDPNEA AND WHEEZING.
- Labor to inspire and can not expire. This results in hyperinflation.
- Attacks last for 1 to several hours.

# CLINICAL FEATURES



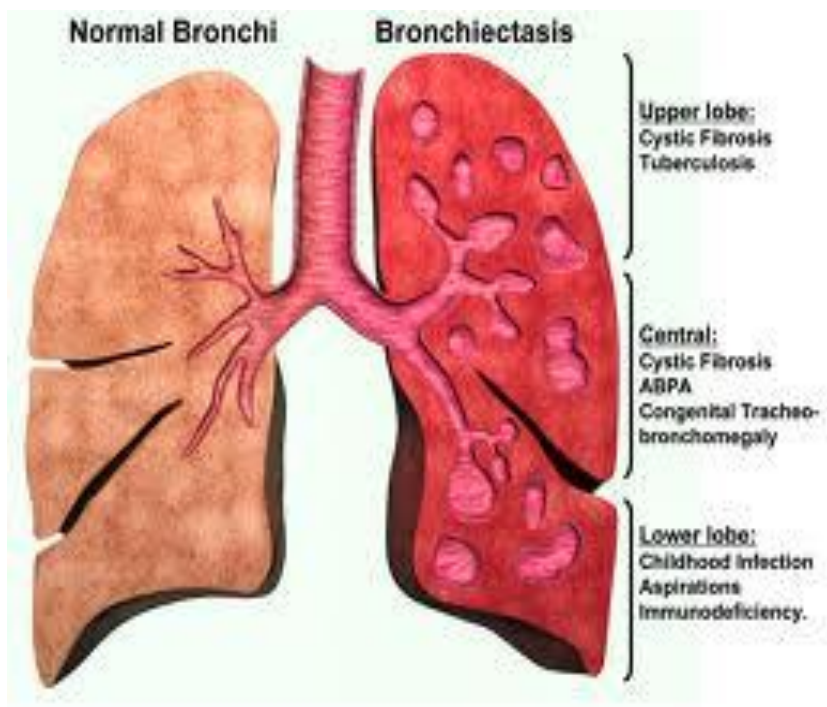
## Common Asthma Symptoms:

- Coughing (at night or with activity)
- Wheezing (whistling sound in the lungs)
- Shortness of breath
- Chest feels "tight" or Hurts"

# BRONCHIECTASIS

- **PERMANENT** DILATION OF BRONCHI AND BRONCHIOLES .
- CAUSED BY **DESTRUCTION** OF MUSCLE AND ELASTIC TISSUE.
- RESULTING FROM **CHRONIC NECROTISING INFECTIONS.**







# BRONCHIECTASIS

## SECONDARY DISEASE CAUSED BY:

- NECROTISING OR SUPPURATIVE PNEUMONIA: STAPHYLOCOCCUS, KLEBSIELLA OR TB.
- BRONCHIAL OBSTRUCTION: TUMOURS, FOREIGN BODY, MUCUS.
- CONGENITAL AND HERIDETARY CONDITIONS.

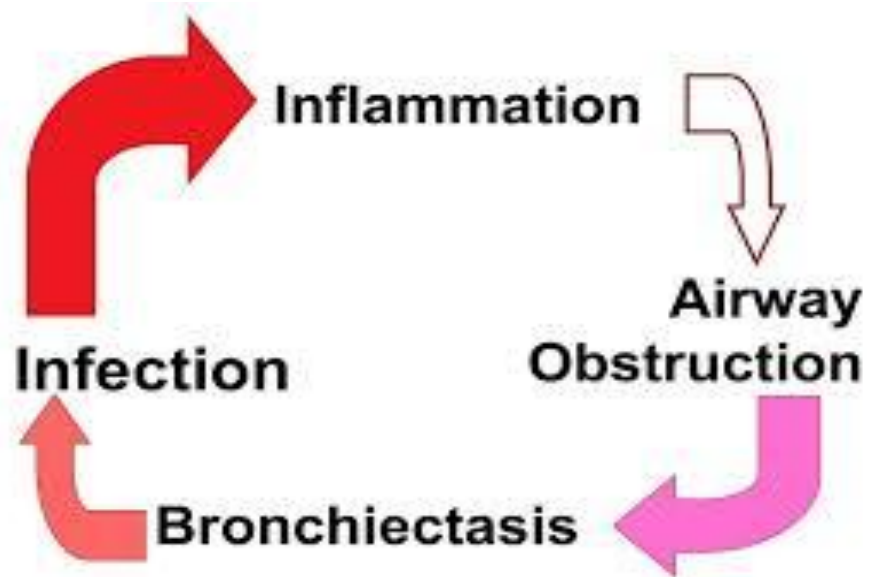
# CONGENITAL CAUSES OF BRONCHIECTASIS

- **CYSTIC FIBROSIS**....ABNORMAL VISCOUS MUCUS, PREDISPOSES TO INFECTIONS AND CAUSES BRONCHIECTASIS.
- **IMMUNODEFICIENCY** : INCREASED INFECTIONS.
- **KARTAGNER SYNDROME**: STRUCTURAL ABNORMALITY OF THE CILIA... IMPAIRED CLEARANCE OF AIRWAYS..INFECTIONS

# PATHOGENESIS

- OBSTRUCTION AND INFECTIONS.
- EITHER CAN COME FIRST.

- INFLAMMATION CAUSES DAMAGE TO THE BRONCHIAL WALLS, AND ACCUMULATION OF EXUDATE LEADING TO DILATION.



# MORPHOLOGY

- USUALLY AFFECTS LOWER LOBES BILATERALLY.
- IF THE CAUSE IS OBSTRUCTION BY TUMOUR OR FOREIGN BODY IT CAN BE LOCALISED.



# MORPHOLOGY

- DILATED AIRWAYS.
- INFLAMMATORY EXUDATE.
- PERIBRONCHIAL FIBROSIS.

# CLINICAL FEATURES

- SEVERE PERSISTENT COUGH.
- HEMOPTYSIS.
- CLUBBING OF FINGERS.

## COMPLICATIONS

- METASTATIC ABSCESSSES
- REACTIVE AMYLOIDOSIS