Chronic bronchitis

- Persistent cough with expectoration on most days for at least 3 months in a year for 2 or more consecutive years.
- More common in middle aged males
- 20% of males 5% of females have ch bronchitis.
- Minority develop serious COPD

Aetiopathogenesis

- **Factors:**
- Cigarette smoking
- Atmospheric pollution
- **Contributory factors:**
- Occupation
- Infection
- Familial & genetic factors

Smoking: More prone (4-10 times)

- Impairs ciliary movement
- Inhibit function of alveolar macrophages
- Causes hypertrophy and hyperplasia of mucus secreting glands
- Causes obstruction of small airways
- Stimulates vagus – bronchoconstriction

Atmospheric pollution: More incidence in industrilised area

- Sulphur dioxide, NO_2, toxic fumes, particulate dust

Occupation: Workers engaged in

- Cotton mills(Byssinosis), plastic factories

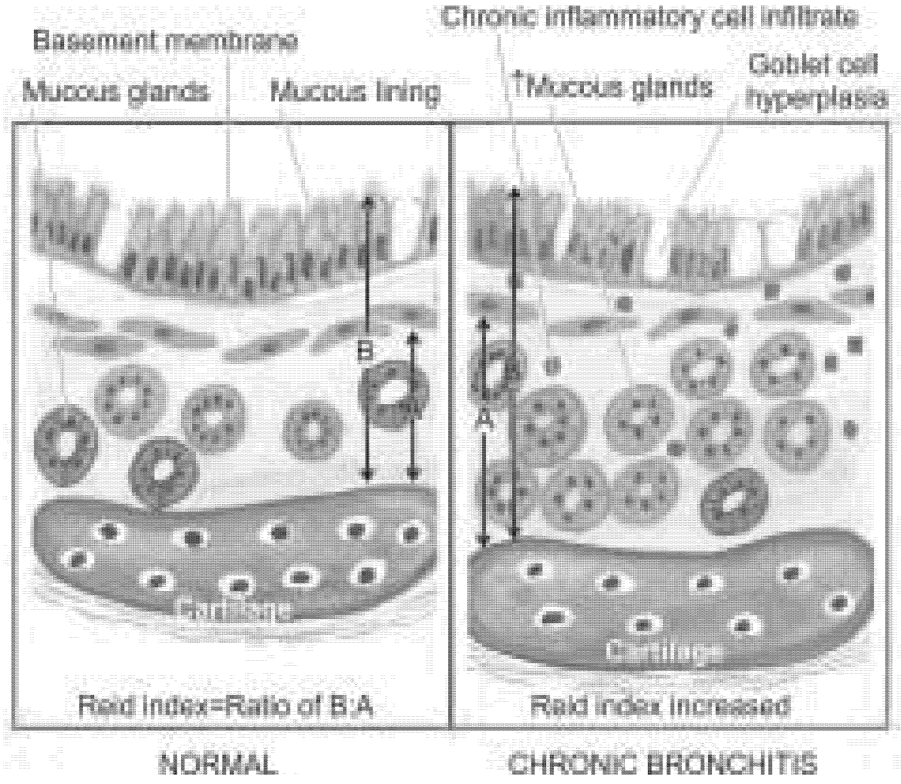
Infection: Usually secondary to bronchitis

- Bacterial
- Viral
- Mycoplasma

Familial & genetic: Family residing in air polluted home more prone

Morphologic changes

- **G/A**: Bronchial wall thickened, hyperemic & oedematous. Lumina of bronchi/bronchioles show mucus plugs & purulent exudate.
- **M/E:**
- Reid index-Ratio between thickness of submucus glands to thickness of total bronchial wall.
- Bronchial epithelium show squamous metaplasia. Small airways show goblet cell hyperplasia, intraluminal and peribronchial fibrosis.


Basement membrane
Mucous glands
Mucous lining
Chronic inflammatory cell infiltrate
↑Mucous glands
Goblet cell hyperplasia
B
A
Cartilage
Cartilage
Reid index=Ratio of B:A
Reid index increased
NORMAL
CHRONIC BRONCHITIS

Bronchial asthma

- Episodic disease, 4% of world population suffer from it. More common in children

- Increased responsiveness of tracheobronchial tree to variety of stimuli leading to widespread narrowing of air passages.
- Clinically paroxysms of dyspnoea, cough and weezing
- Status asthmaticus: Severe and unremitting form of disease

Aetiopathogenesis

Two aetiologic types:

- Extrinsic (Allergic)
- Intrinsic (Idiosyncratic)
- Mixed
- **Extrinsic asthma:**
- Most common
- Begins in childhood
- Family history of allergic disease
- Inhalation of allergens ie house dust, pollen, animal danders, moulds
- Increased levels of IgE in serum

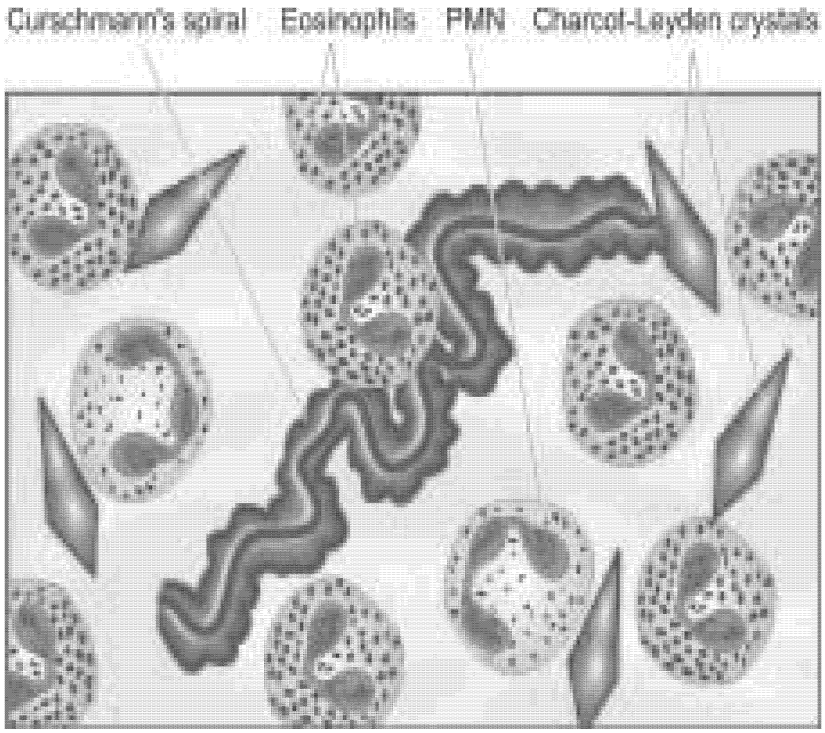
- IgE mediated Type I hypersentivity reaction causing mast cell degranulation with release of
- Histamine, leukotriene, prostaglandins, PAF, chemotaxis eosinophils and neutrophils
- Bronchoconstriction, oedema, mucus hypersecretion

Intrinsic asthma:

- In adult life, no family history, no h/o allergy, normal IgE
- Symptoms after URTI by viruses
- Nasal polypi and ch bronchitis commonly present

Morphological changes

- **G/A:** Lungs overdistended, occlusion of bronchi and bronchioles by vicid mucus plugs
- **M/E:**
- Mucus plugs contain Curschmann’s spiral
- Sputum contain Charcoal leyden crystal
- Bronchial wall show thickened BM, submucosa show oedema, infiltration by eosinophils, lymphocytes, plasma cells,
- Hypertrophy of submucosal glands and smooth muscles


Curschmann's spiral
Eosinophils
PMN
Charcot-Leyden crystals