

# **CARDINAL SIGNS AND SYMPTOMS IN CLINICAL MEDICINE**

## **Lec 3**

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# Cough

**Explosive expiration that provides a protective mechanism for clearing the bronchotracheal tree from foreign bodies & secreted material which are sometimes excessive or bothersome.**

**It is also one of the most common symptoms for which medical attention is sought.**

# Causes

- 1) Airway irritants, which enter bronchotracheal tree by inhalation (smoke, dust, foreign bodies in the upper airway), Secretion (Post nasal drip), Gastric content (gastro-esophageal reflux).
- 2) Inflammation, constriction, infiltration and compression of airway.  
Inflammation → Airway infection [Viral, Bacterial], bronchitis, bronchiectasis, pertussis, bronchial asthma (S.T without dyspnea & wheeze).

- Neoplastic infiltration of the airway wall (bronchogenic CA & carcinoid Tumor), granulomatous infiltration with Sarcoidosis & Tuberculosis (TB).
  - Compression of airway from extrinsic masses (left lung malignant tumor, aortic aneurysm).
- 3) Parenchymal lung disease: interstitial lung disease (pneumonia & lung abscess).
- 4) Congestive Heart Failure → interstitial & peribronchial edema.

## 5) Use of ACE (Angiotensin Converting Enzyme)

### inhibitors:

- Non-productive cough usually within one week but may be delayed up to 6 months.
- 5-20% of patient (accumulation of bradykinin or substance P which are degraded by ACE).

# Approach to the Patient

## 1. History

- Is the cough acute or chronic?
- Is it productive of sputum or associated with blood?
- Any associated symptoms suggestive of respiratory infection?
- Is it seasonal or associated with wheeze?
- Is there any postnasal drip or evidence of reflux?
- Any associated disease or risk factors for disease? (cigarette smoking, HIV, environmental exposure)
- Is the patient taking ACE inhibitors?

## 2. Physical Examination

- Inspiratory stridor → upper airway disease.
- Rhonchi → lower airway disease. Mainly expiratory with wheeze.
- Inspiratory crackles → interstitial lung disease, pneumonia & pulmonary edema.

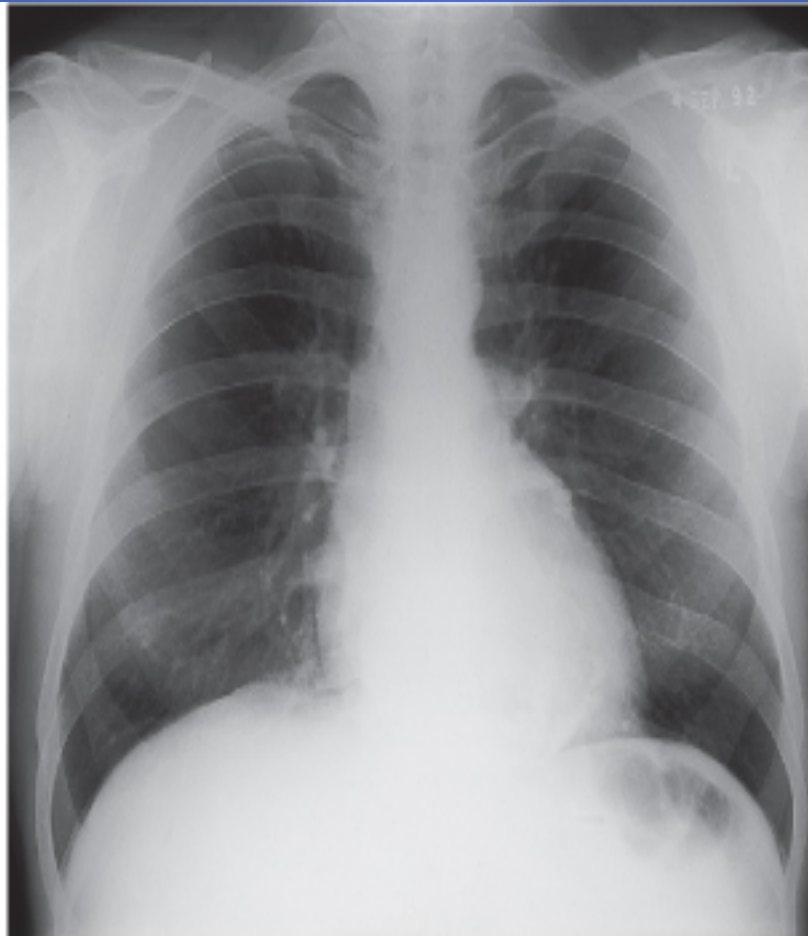
## 3. Investigation

### A. Chest X-Ray (CXR):

- Intra-thoracic mass lesion: (tumor)
- Honey combing: bronchiectasis
- Non-homogenous opacity: pneumonia
- Hilar adenopathy: Tumor, Tuberculosis (TB), Sarcoidosis
- Homogenous opacity: pleural effusion

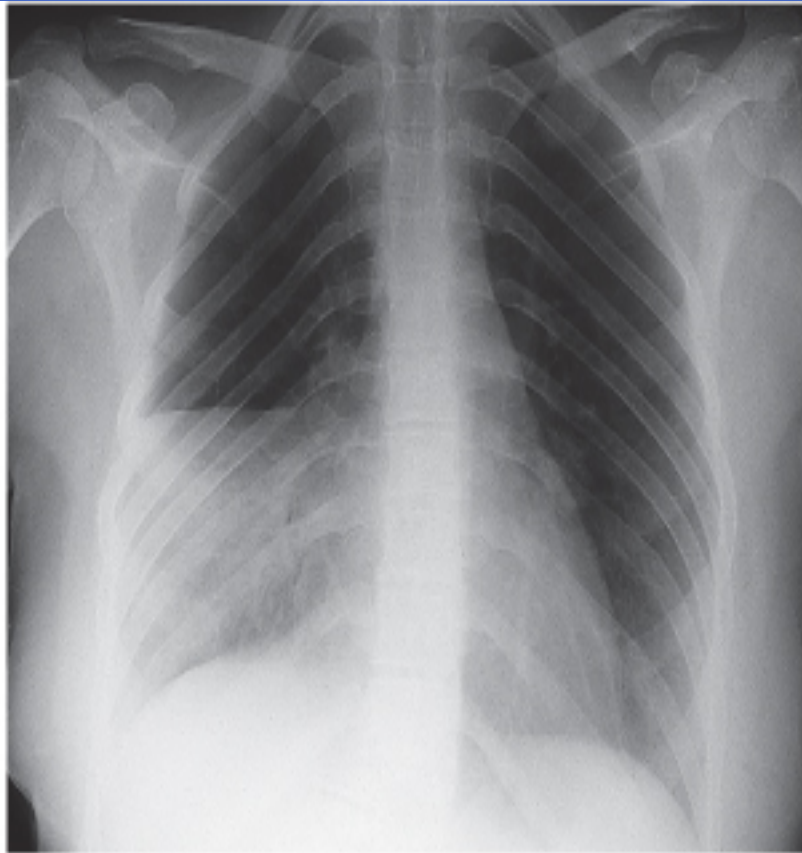


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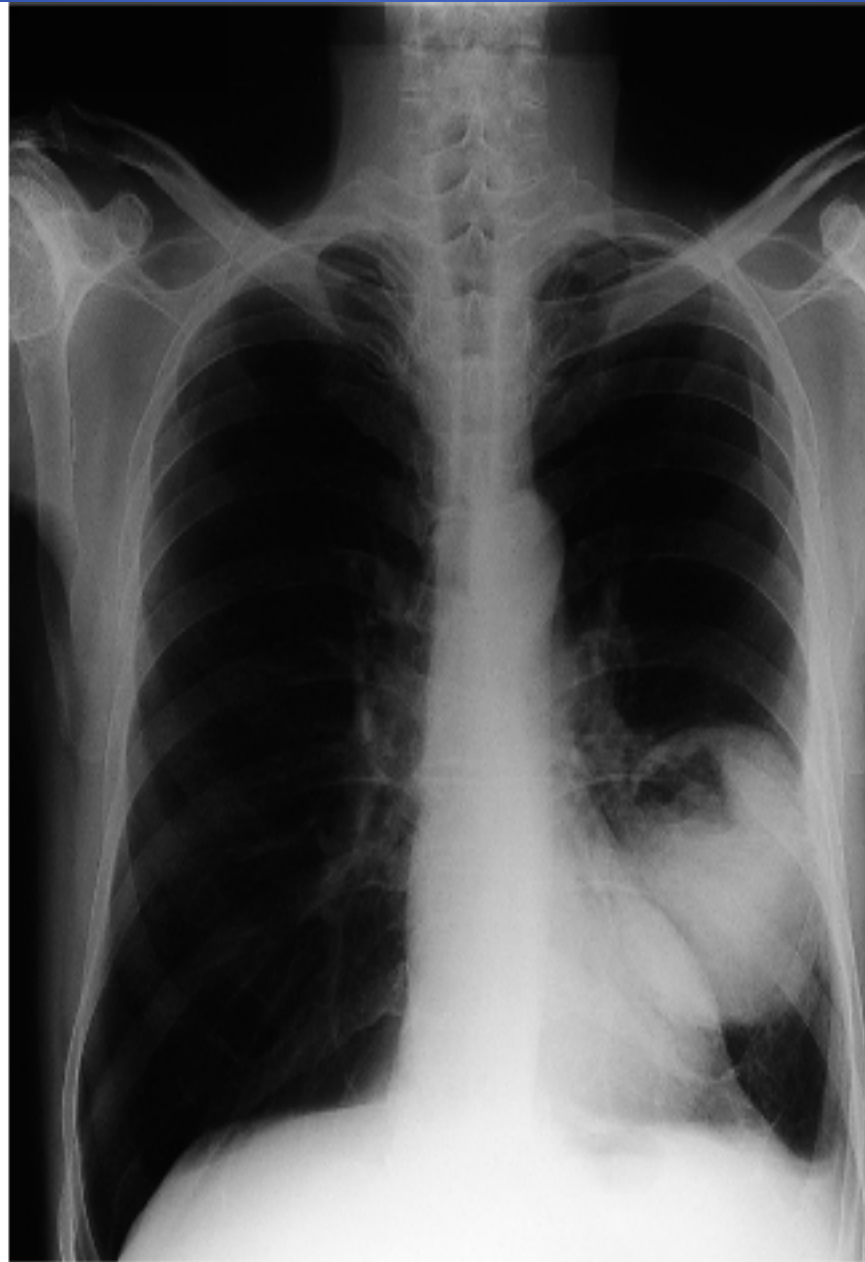


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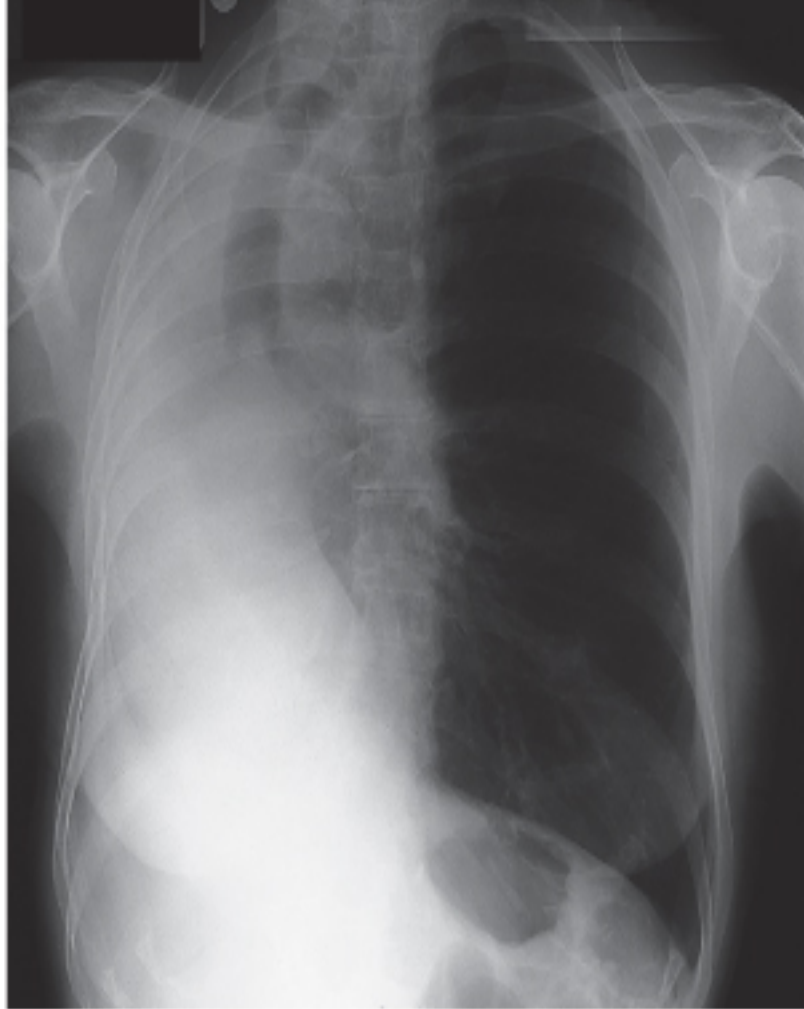
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**CTPA  
Saddle  
embolism  
in the  
bifurcation  
of PA**



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**B. PFT: Obstructive Pulmonary Defect, e.g., asthma or chronic bronchitis.**

**Restrictive Pulmonary Defect, e.g., Pulmonary Fibrosis.**

## **C. Sputum Examination**

- **Purulent sputum → chronic bronchitis, bronchiectasis pneumonia, lung abscess.**
- **Blood with sputum → TB, bronchogenic CA.**
- **Gram & acid fast stain & culture → particular infection.**
- **Sputum cytology → pulmonary malignancy.**



**D. Fiberoptic bronchoscopy → Visualization of endobronchial tumor & collecting cytological & histological specimens.**

**E. CT (Computed Tomography) → interstitial lung disease, bronchiectasis.**

# Treatment

1. Specific therapy of underlying pathology
2. Avoiding airway irritants.
3. Symptomatic therapy considered if the cause of cough is not known or specific TX is not possible & if it causes marked discomfort.
4. Irritant non-productive cough → suppressed by anti-tussive agent which increases threshold of cough center.e.g., codein → 15 mg, dextromethorphan 15 mg 3-4 times daily.

- Cough productive of significant amount of sputum should not be suppressed → why?
- Inhaled glucocorticoids: betamethasone, triamcinolone in patients with airway inflammation (asthma).
- *Answer: Retention of sputum may interfere with distribution of ventilation, alveolar aeration and ability to resist infection.*
- Inhaled anti-cholinergic agent: ipratropium bromide 2-4 puffs tid → inhibits the efferent limb of cough reflex.

# Hemoptysis

Coughing up blood irrespective of the amount. Alarming symptom that bring the patient to the doctor.

- It should be differentiated from haematemesis (bloody vomiting) and epistaxis (nose bleeding).
- This symptom must always be assumed to have a serious cause until appropriate investigation have excluded bronchial CA, thromboembolic disease and TB.

# causes

**Note: the star sign (\*) means common**

- 1. Bronchial diseases → \*carcinoma, \*bronchiectasis, bronchial adenoma, foreign body, \*acute bronchitis.**
- 2. Parenchymal diseases → \*TB, lung abscess, \*pneumonia, trauma, actinomycosis and aspergillosis.**
- 3. Pulmonary vascular diseases → \*pulmonary infarction, polyartitis nodosa, good pastures syndrome, idiopathic pulmonary hemosiderosis.**
- 4. Cardiovascular diseases → \*acute LVF, \*mitral stenosis & aortic aneurysm.**
- 5. Blood disorders → leukemia, hemophilia, anti coagulants.**

# Diagnosis

- History of repeated small hemoptysis is highly suggestive of bronchial carcinoma in a smoker.
- Chronic form & weight loss → TB.
- Chronic hemoptysis in the young female → bronchial adenoma.
- Chronic productive cough + hemoptysis which is sometimes massive associated with crepitation on lung → bronchiectasis.

- In hospitalised patient pulmonary embolism is the most common cause.
- Major risk factors: immobilization, malignant disease, cardiac failure, pregnancy.
- Physical examination → may reveal clue to the underlying diagnosis,  
E.g., → finger clubbing in bronchogenic CA & bronchiectasis  
other signs of malignancy: cachexia, hepatomegaly, lymphadenopathy.  
fever, chest pain, sign of consolidation and pleurisy (pneumonia).  
pulmonary infarction: Leg swelling and tenderness (DVT).

# Management

In massive hemoptysis patient should be nursed on the side of suspended source of bleeding; Hemodynamically resuscitated, then bronchoscoped ideally under general anesthesia using rigid bronchoscope to attempt bronchial suction & to maintain ventilation during anesthesia.

- Angiography & arterial embolization can be life saving in acute situations.
- In majority hemoptysis is not life threatening & searching for a cause is logical by doing investigation:



1. CXR: localized lesion → pulmonary infarction, tumor, pneumonia, TB.
2. Full blood count & coagulation screen.
3. Bronchoscopy for Dx of central CA & for tissue Dx.
4. Ventilation perfusion lung scan: pulmonary thrombo-embolic disease; pulmonary angiography sometimes needed
5. CT (Computed Tomography): investigation of peripheral chest radiographic lesion which is not accessible to bronchoscopy.

# Cyanosis

Bluish discoloration of skin & mucous membrane resulting from an increased amount of reduced Hb in small blood vessel of skin and mucous membrane..

- Most marked in lips, nail bed, ears, malar eminences.
- Cyanosis becomes apparent when the reduced Hb conc.  $> 50$  g/L.
- Cyanosis can be masked in anemic patient.

Central cyanosis → results from arterial blood desaturation or abnormal Hb derivatives. Both skin & mucous membrane are affected.

**a) Decreased arterial O<sub>2</sub> saturation**

**1- impaired pulmonary function: alveolar hypoventilation and pulmonary embolism (ventilation-perfusion mismatch & impaired O<sub>2</sub> diffusion).**

**2- Anatomic Shunt:**

**(TOF/TOG) Cyanotic Congenital Heart Disease (CHD), pulmonary A-V fistula, and multiple small intra-pulmonary shunts.**

**3- Hb with low affinity for O<sub>2</sub> (Hb Kansas).**

## b) Hb abnormalities:

- Methemoglobinaemia → hereditary or acquired.
- Sulfhemoglobinaemia → acquired.

Peripheral Cyanosis → results from slowing of blood flow & abnormally great extraction of O<sub>2</sub> from normally saturated arterial blood.

- Reduced cardiac output.
- Cold exposure.
- Redistribution of blood from extremities.
- Arterial obstruction: Raynaud's, Arterial embolus, arterial constriction.
- Venous obstruction.  
often the mucous membrane is spared.

- **Clinical differential between central & peripheral cyanosis may be difficult & in conditions like cardiogenic shock with pulmonary edema there may be a mixture of both.**

# Approach

1. **History: duration** → cyanosis since birth usually due to cyanotic CHD  
exposure to drugs or chemicals → abnormal Hb.
2. **Clinical differentiation between central & peripheral cyanosis**
  - Physical or radiographic examination of cardiac & respiratory system.
  - Massage or gentle rewarming of cyanotic extremities will improve peripheral blood flow in peripheral but not central cyanosis.
3. **Presence or absence of finger clubbing**
  - Clubbing without cyanosis → infective endocarditis, ulcerative colitis or familial.
  - Slight cyanosis of lips, checks without clubbing → mitral stenosis.

- Cyanosis & clubbing → cyanotic CHD, pulmonary A-V shunt.
- Peripheral cyanosis & acutely developing central cyanosis is not associated with finger clubbing.

4. Measuring arterial blood  $O_2$  tension or  $O_2$  saturation, spectroscopic examination of blood for abnormal type of Hb.