# Clinical Round in Medicine

## QMA Notes

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# Chapter 1

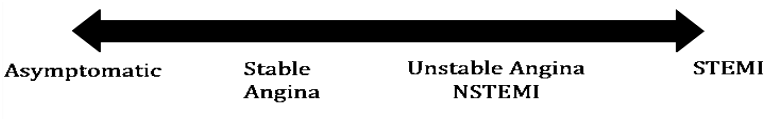
# Cardiovascular System

# Ischemic Heart disease

## Coronary Artery disease (CAD)

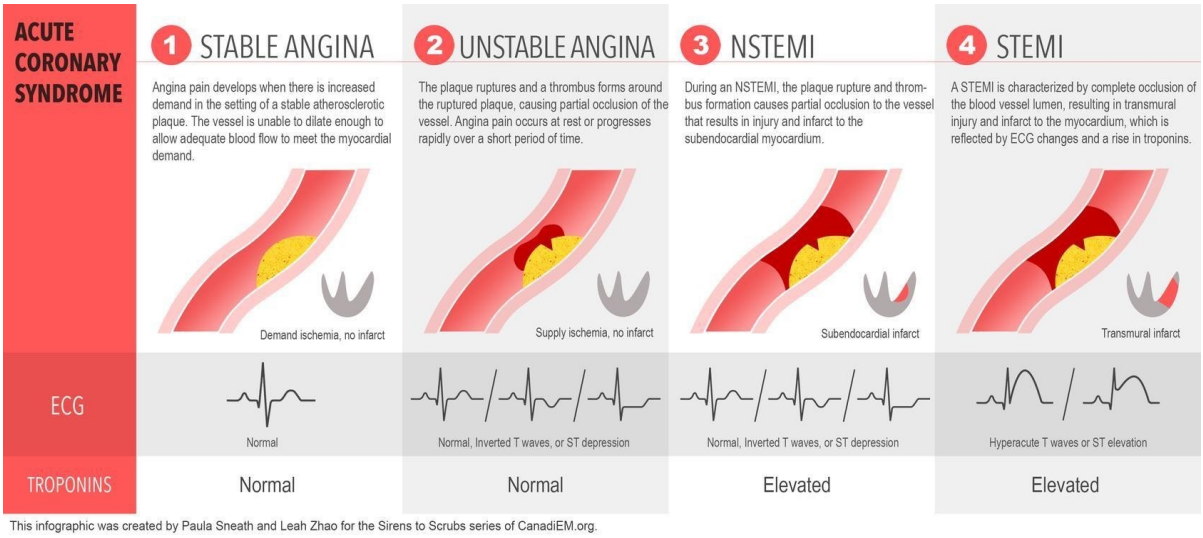
### Pathophysiology

- **Narrowing of coronary artery usually caused by atherosclerosis.**
- **Asymptomatic until ~75 % artery lumen occluded.**
- **If symptomatic :Chest pain (angina), may also cause dyspnea, other symptoms.**




## IHD (Pathophysiology)

- **Coronary ischemia is due to an imbalance between blood supply and oxygen demand, leading to inadequate perfusion.**




## Stable angina (Clinical features)

| Characteristics of Angina pain | |
|---|---|
| Site | Retrosternal. |
| Onset | Progressive, increase in intensity over1-2 minutes. |
| Character | Constricting, heavy. |
| Radiation | Sometimes arm, neck, epigastrium. |
| Associated features | Dyspnea. |
| Timing | Intermittent, with episode lasting 2-10minutes. |
| Exacerbating/relieving factors | Triggered by emotion, exertion, cold, large m eal.<br>Relieved by rest, nitrates. |
| Severity | Mild to moderate. |

## ACS (Clinical features)

| Characteristics of MI pain | |
|---|---|
| Site | Retrosternal |
| Onset | Rapid over a few minutes |
| Character | Constricting, heavy |
| Radiation | Often to arm(s ,(neck, jaw, sometimes epigastrium |
| Associated features | Sweating, nausea, vomiting, breathlessness. |
| Timing | Acute presentation ;prolonged duration > 10 – 30minutes |
| Exacerbating/relieving factors | Stress 'and exercise rare triggers , usually spontaneous.<br>Not relieved by rest or nitrate |
| Severity | Usually severe |

## Major Risk Factors

- Diabetes.
- Chronic kidney disease.
- Hypertension.
- Hyperlipidemia (LDL).
- Age) M< 45 F > 55 ).
- Family History of premature CAD (1° relative, M < 55, F < 65).
- Smoking (quitting smoking → significantly ↓ risk).
- Obesity, sedentary lifestyle.

**CAD Equivalents**

1. Diabetes
2. CKD

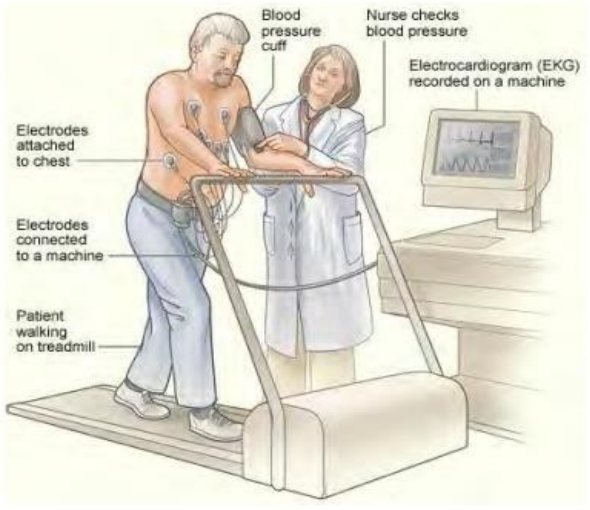
## Stable Angina

- EKG at rest :normal.
- Cardiac enzyme :normal.
- Diagnosis :symptoms or stress testing.
- Stress testing key when diagnosis uncertain.

| Classification of angina | |
|---|---|
| Classic | • Typical location (eg, substernal), quality & duration 2-10m<br>• Provoked by exercise or emotional stress<br>• Relieved by rest or nitroglycerin |
| Atypical | • 2 of the 3 characteristics of classic angina e.g: epigx |
| Nonanginal | • <2 of the 3 characteristics of classic angina |

## Stress Testing

- **Patient must be asymptomatic.**
- **Goal :provoke ischemia and detect ischemia.**
- **Provocation: (Exercise VS .Drugs) exercise always preferred.**
- **Detection:**
  1. **EKG changes (ST-depressions).**
  2. **Nuclear imaging (usually Technetium)**
  3. **Echocardiography.**

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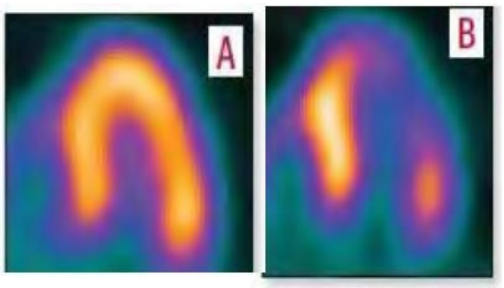
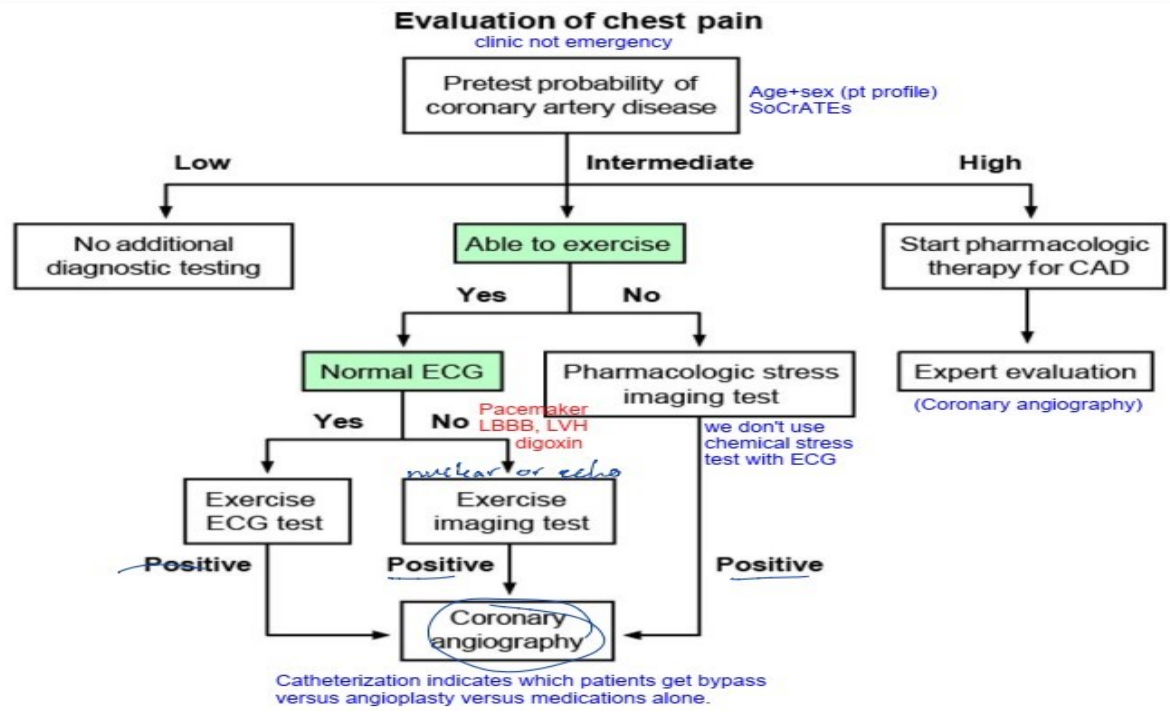
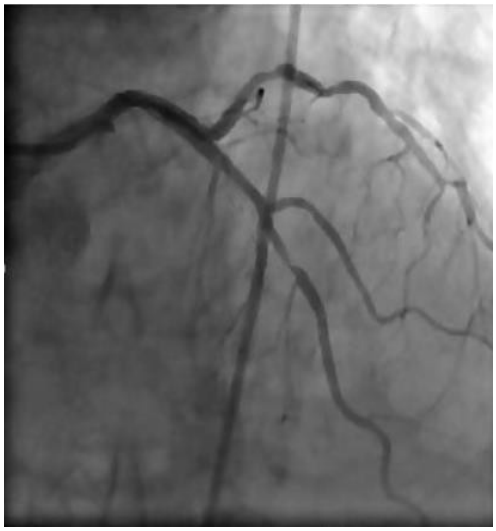


## Angiography

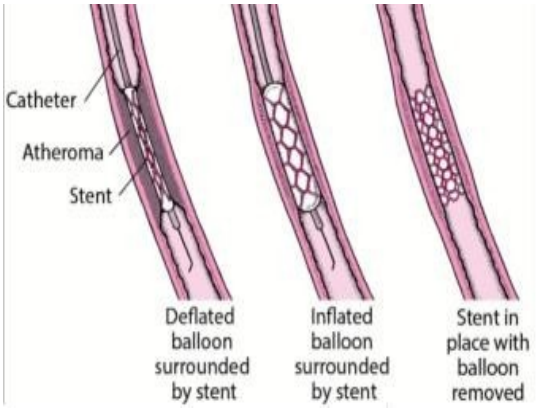


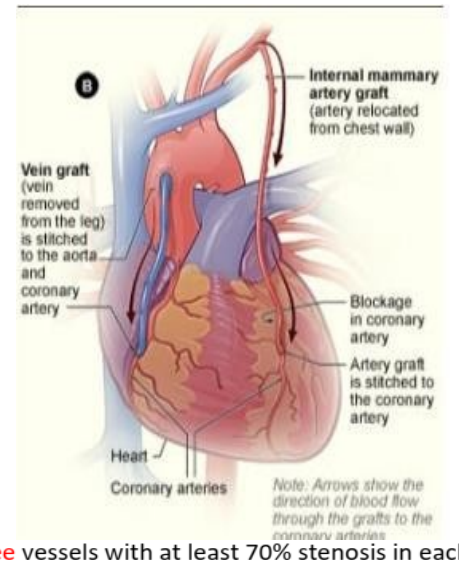
## Treatment of stable angina (NBAs)

- Nitroglycerin :Decrease **preload** by dilation of veins .Short acting :used in acute setting (**sublingual**, IV).
  Long acting :for persistent angina) Oral, Transdermal patch).
- **Beta blockers ( 1st line therapy )** Decrease **myocardial contractility and HR.** (Non - Dihydropiridine CCBs).
- **Aspirin** :Antiplatelet therapy.
- Statin + smoking cessation.

Statins inhibit **HMG-CoA reductase**, a rate-limiting enzyme in the intracellular biosynthesis of cholesterol.

## Revascularization



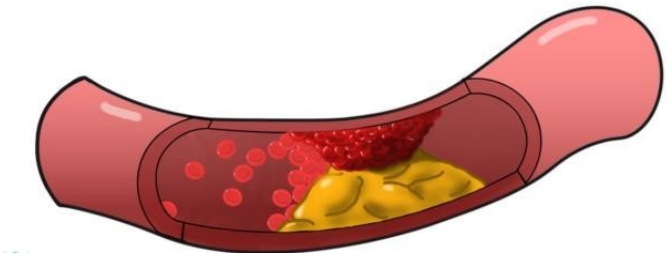




Three vessels with at least 70% stenosis in each vessel.
Two-vessel disease in a patient with diabetes.
Left main coronary artery occlusion.
Persistent symptoms despite maximal medical therapy.

## Stent Complications

**Thrombosis**:
Acute closure of stent same as STEMI :life threatening event

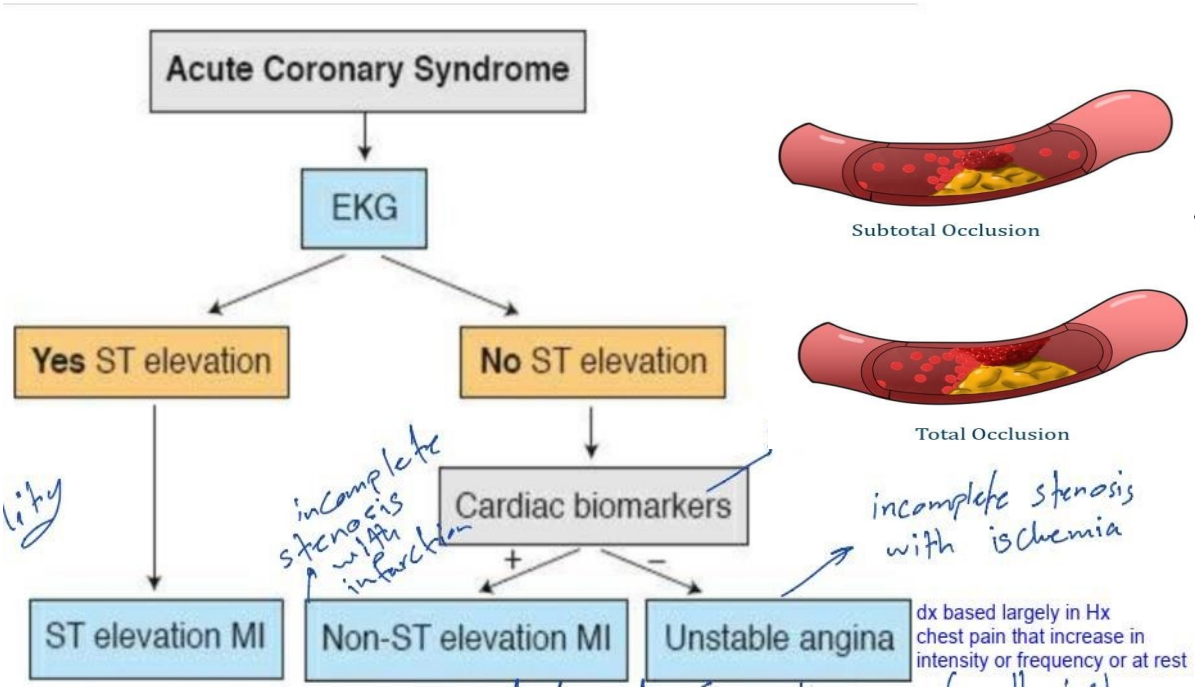
**Stent Thrombosis Prevention**
- "Dual antiplatelet therapy"
- Typically one year of:
- Aspirin
- Clopidogrel



## Stable Angina :Typical Case

- 65-year-old man with **chest pain while walking.**
- Relieved with **rest**.
- Presents to ED:
- EKG **normal**
- Biomarkers **normal**
- Stress test
- Walks on **treadmill** → chest pain, **EKG changes**
- Cardiac **catheterization** performed
- %90LAD artery blockage
- Stent placed → angina resolved

## Acute Coronary Syndrome



## Cardiac enzymes

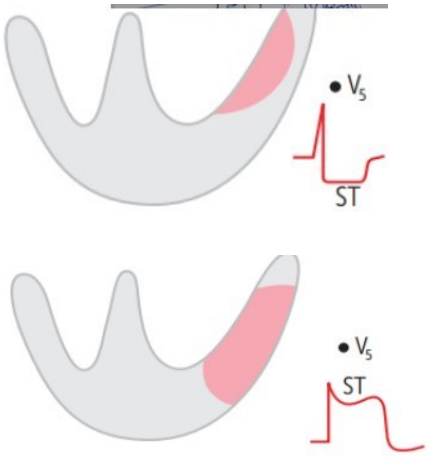
| Test | Time to becoming abnormal | Duration of abnormality |
|---|---|---|
| Myoglobin | 1-4 hours | 1-2 days |
| CK-MB | 4-6 hours | 1-2 days |
| Troponin | 4-6 hours | 10-14 days |

**Most common marker used :Troponin I or T CK-MB also used :** Very good for re-infarction.

- Normal enzyme: in stable angina/UA.
- Abnormal cardiac enzyme in MI.

## ECG

- **Normal in stable angina.**
- **UA/NSTEMI:**
  1. **ST depressions.**
  2. **T-wave inversions.**
- **STEMI:**
  1. **ST Segment elevation in certain lead.**
  2. **May show MI complications( Such as: Sinus tachycardia /bradycardia and heart block).**



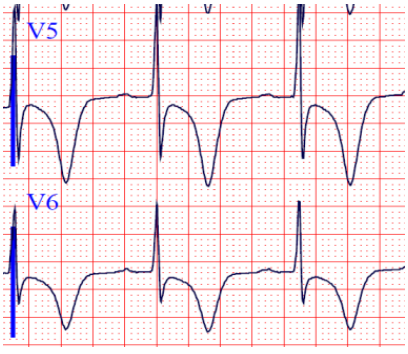

## Unstable angina/NSTEMI

Chest pain (**at rest** or angina that is increasing in **frequency or duration**).
Other symptoms :Dyspnea, nausea/vomiting.

**Diagnosis** :Clinical features, plus ECG and Biomarkers (CK-MB)

**ECG Changes**

- ST depressions
- T-wave inversions



## UA/NSTEMI Treatment

- **Thrombotic and ischemic syndrome (like STEMI)**
- **No“ ticking clock (”unlike STEMI)**
  1. **Subtotal occlusion.**
  2. **Some blood flow to distal myocardium.**
  3. **No emergency angioplasty.**
  4. **No benefit to thrombolysis.**

This is a **thrombotic** problem:

**1-** **Aspirin**-&

**2-** **Clopidogrel** to inhibit platelet aggregation (best initial)

This is also an **ischemic** problem:

**3-** **Beta-blockers** to reduce O2 demand (Non-dependent on time)

**4-** **Nitrates** to reduce O2 demand

**5-** ACEI :to reduce re-modeling of heart. (within 24 hours of MI)

**6-** Statin :to reduce recurrence.

**7-** **Heparin**

**8-** **PCI** :within 48-24 h.

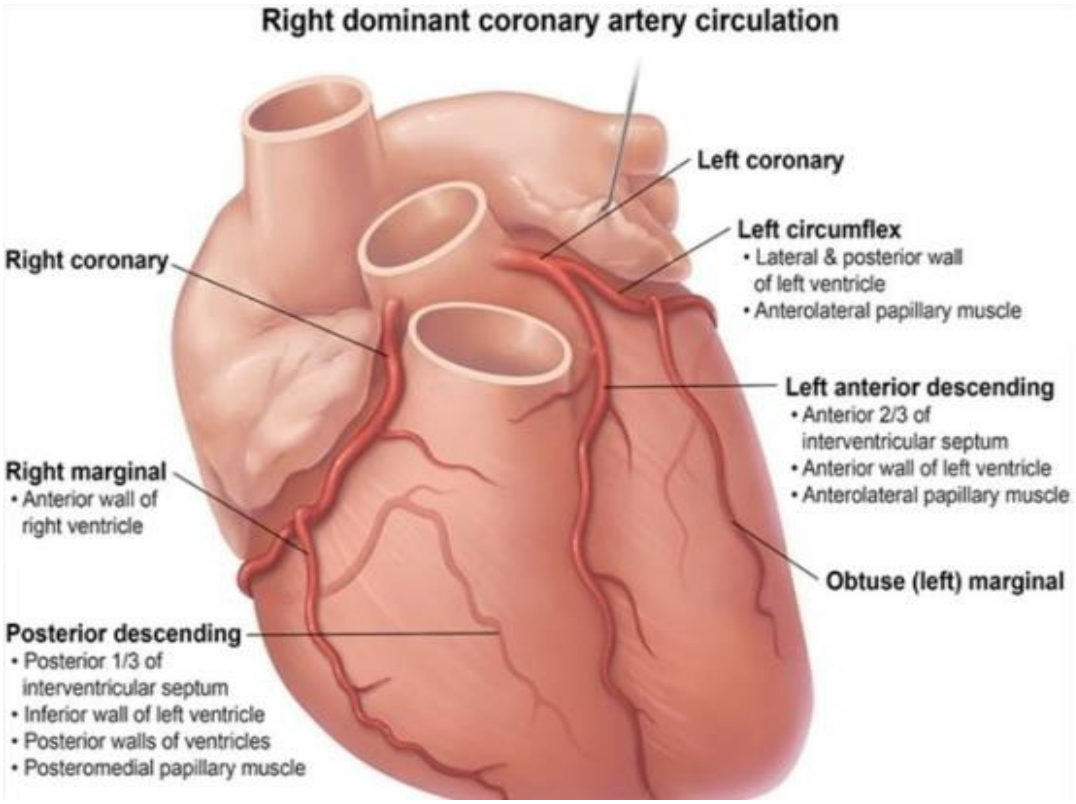
- **Long-term therapy**
  - **Goal ↓ :mortality and recurrent infarction.**
    1. **Aspirin.**
    2. **Statin.**
  - **Beta-blocker for prevention of recurrent disease.**
    - **Used for prevention in NSTEMI only.**

## Typical UA/NSTEMI Course

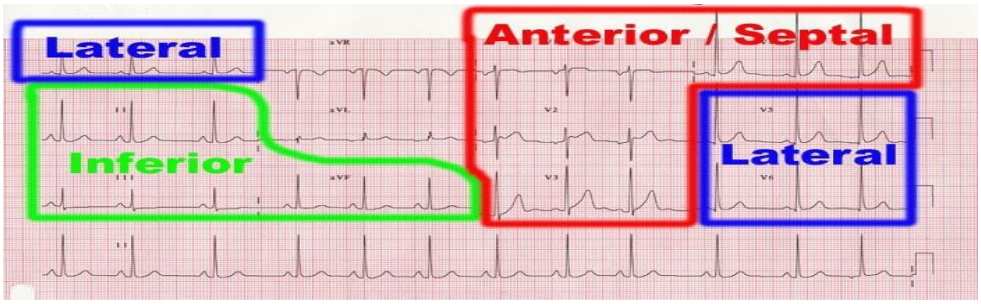
- Presents to ER with chest pain
- Biomarkers **elevated**
- **Absence** of ST-segment elevations
- Medical Therapy:
- Aspirin & clopidogrel
- Metoprolol
- Heparin drip
- Admitted to cardiac floor
- Hospital day → 2 angiography
- %90blockage of LAD → Stent

## STEMI

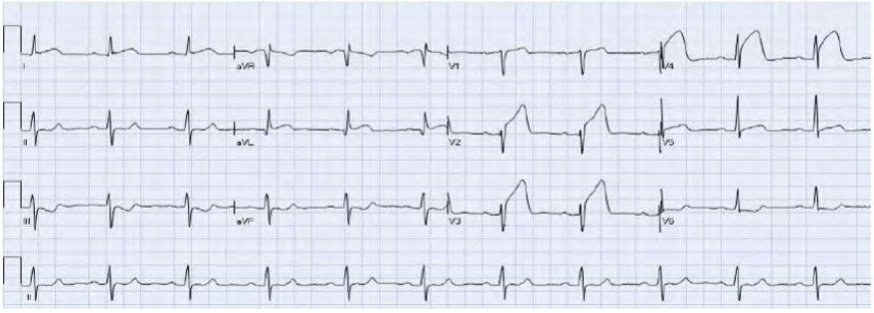
- Chest pain + ECG + Cardiac enzymes.
- Radiation of pain) jaw, shoulder ,(diaphoresis, dyspnea.
- > **10 - 30** minutes.
- **Not relieved** by rest or nitrate.



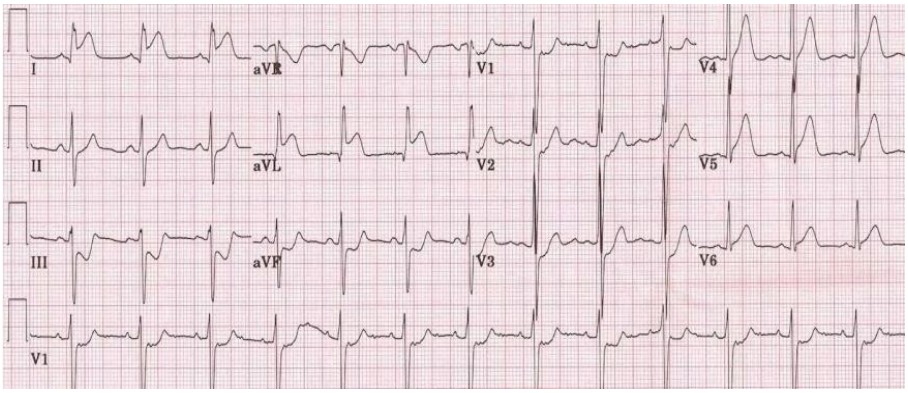

## ECG Leads



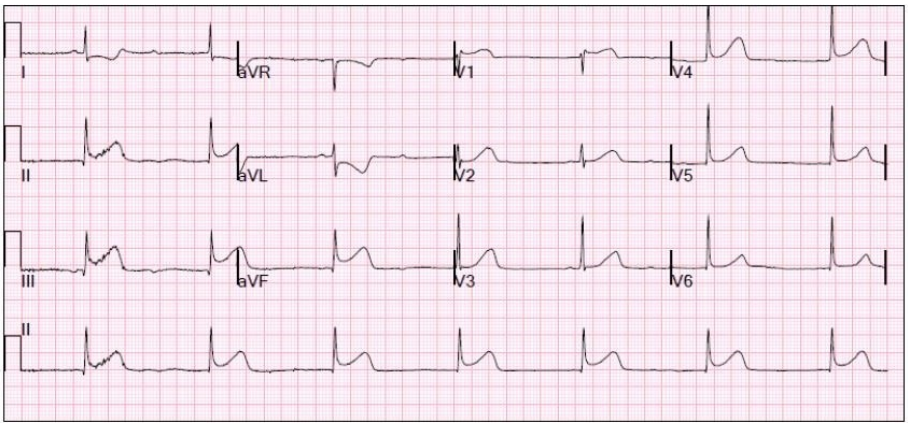

## ST Elevations -Anterior



## ST Elevations -Lateral

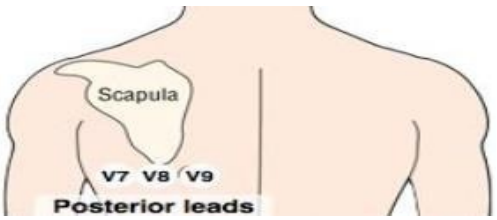


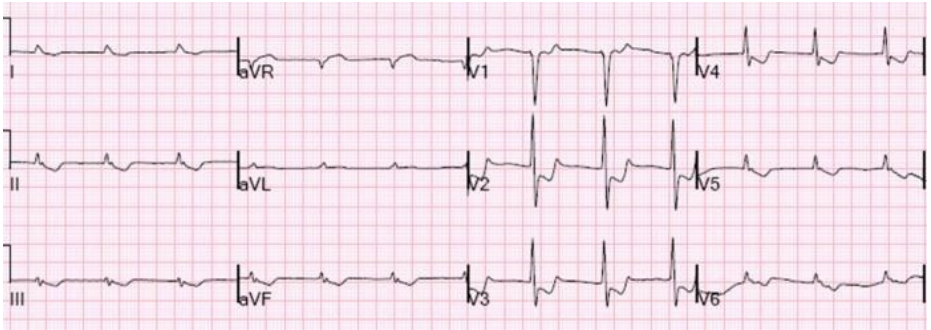

## ST Elevations -Inferior

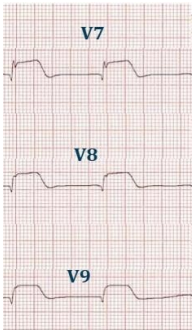



## Posterior MI

Anterior ST **depressions** with standard leads ST-elevation in **posterior leads (V7-V9)**



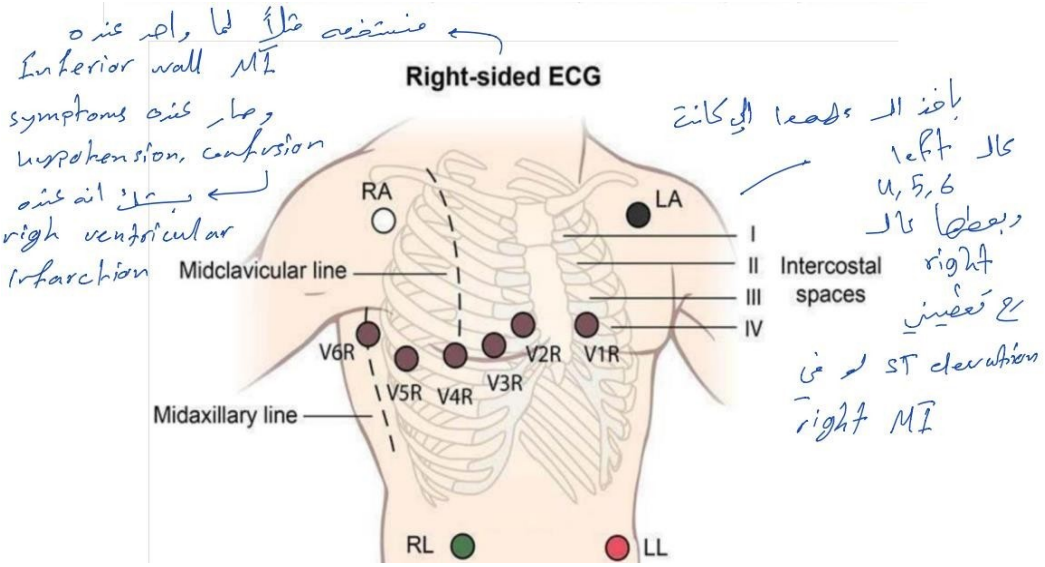



## Special complication (Inferior MI)

### Right ventricular infarction

- Occurs in inferior STEMI(II, III, aVF)
- Loss of right ventricular contractility
- **Elevated jugular venous pressure**
- Decreased preload to left ventricle →**hypotension**
- **Diagnosis :right-sided chest leads**
- Avoid nitroglycerine
- Treatment :IV fluids ↑( preload)




### Sinus bradycardia and heart block

- **Inferior wall ischemia**
- **RCA :SA node ,%60 AV node%90 cautions: BB (AV block and sinus bradycardia.)**

## Treatment of STEMI

- **“Time is muscle”**
- **Coronary artery occluded by thrombus**
- **Longer occlusion → more muscle dies**
  1. **More likely the patient may die**
  2. **More heart failure symptoms**
  3. **More future hospitalization for heart disease**
- **Medical emergency**

• Main objective is to open the artery“ Revascularization”

• Option :1 Emergency angioplasty) PCI :(Mechanical opening of artery
Stent placemnt
Should be done **90 > min**


• Option :2 Thrombolysis
Alteplase, TPA
Should be done **30 > min**
“ Door to balloon ”or“ door to needle

This is a **thrombotic** problem

1-**Aspirin** & 2- **Clopidogrel** to inhibit platelet aggregation

This is also an **ischemic** problem:

3- **Beta-blockers** to reduce O2 demand

4- **Nitrates** to reduce O2 demand

5- ACEI :to reduce re-modeling of heart

6-Statin :to reduce recurrence.

## Typical STEMI Course

- Arrival in ER with chest pain 5:42 pm•
- EKG done 5:50 pm
- STEMI identified
- Cardiac cath lab activated for emergent angioplasty
- Meds given in ER
- Aspirin
- Metoprolol
- Nitro drip
- Heparin bolus
- Transport to cath lab 6:15 pm
- Artery opened with balloon 6:42 pm
- DTB time 60 minutes (ideal < 90min)
- Arrival in ER with chest pain 5:42 pm
- EKG done 5:54 pm
- STEMI identified
- Meds given in ER
- Aspirin
- Metoprolol
- Nitro drip
- Heparin bolus
- tPA given based on weight 6:07 pm
- IV push
- Door to needle time 25 min) ideal(30 >

## MI Complications

**First 4 days:**

- Arrhythmia :Ventricular tachycardia → ventricular fibrillation → cardiac arrest

**5-10 days** :**Transthoracic Echocardiogram**

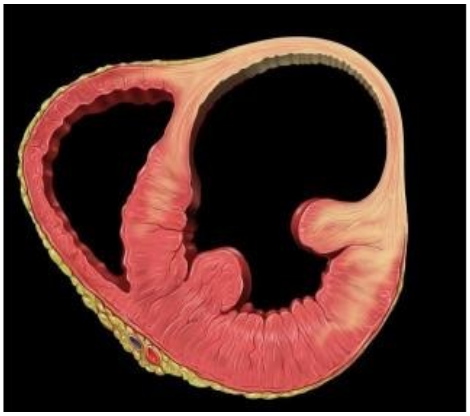
- Free wall rupture :Cardiac Tamponade
- Papillary muscle rupture :mitral regurgitation) holosystolic murmur ,(Heart failure, respiratory distress
- VSD (septal rupture) :Hypotension, right heart failure (↑ JVP, edema)

**Weeks later:**

- Dressler's syndrome
- Aneurysm
- LV Thrombus/CVA

**Ventricular Aneurysm**

- More common **anterior infarction**
- Risk of thrombus → stroke, peripheral embolism
- Causes persistent ST elevations



## Dressler's Syndrome

Weeks to months after MI

- Form of pericarditis
  - Chest pain
  - Friction rub
- Immune-mediated (details not known)
- Diagnosis: clinical
- Treatment: NSAIDs or steroids

## Fibrinous Pericarditis

- Occurs ***days*** after MI
  - Sometimes called "post-MI" pericarditis
  - Not autoimmune
  - Extension of myocardial inflammation
- Dressler's occurs ***weeks*** after MI
  - Sometimes called "post cardiac injury" pericarditis
- Rarely life-threatening
- Diagnosis: clinical

**Variant ( Prinzmetal) Angina**

**Episodic** vasoconstriction of coronary vessels Episodes usually **at rest**
Midnight to **early morning**
Sometimes symptoms **improve with exertion**
Associated with **smoking**
Diagnosis :usually based on history.

**Treatment**

- Quit smoking
- **Calcium channel blockers, nitrates**
  - Dihydropyridine CCB :amlodipine
  - **Vasodilators**
  - Dilate coronary arteries, oppose spasm
  - Avoid propranolol (Nonselective beta blocker)
  - Can cause unopposed alpha stimulation
  - Symptoms may worsen

# Heart Failure

## HF (Etiology)

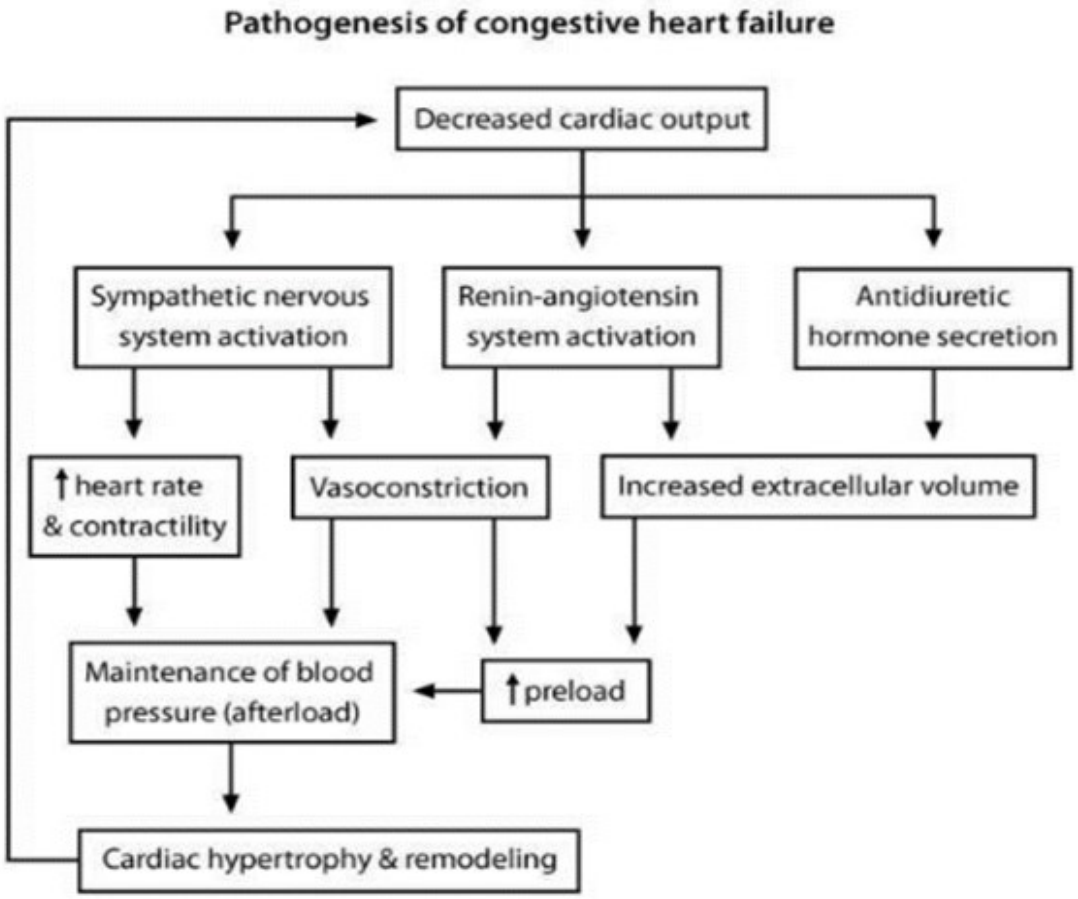
**What is the definition of heart failure?**
Abnormality of cardiac structure and/or function resulting in clinical symptoms (e.g., dyspnea, fatigue) and signs (e.g., edema, pulmonary crackles), hospitalizations, poor quality of life, and shortened survival.

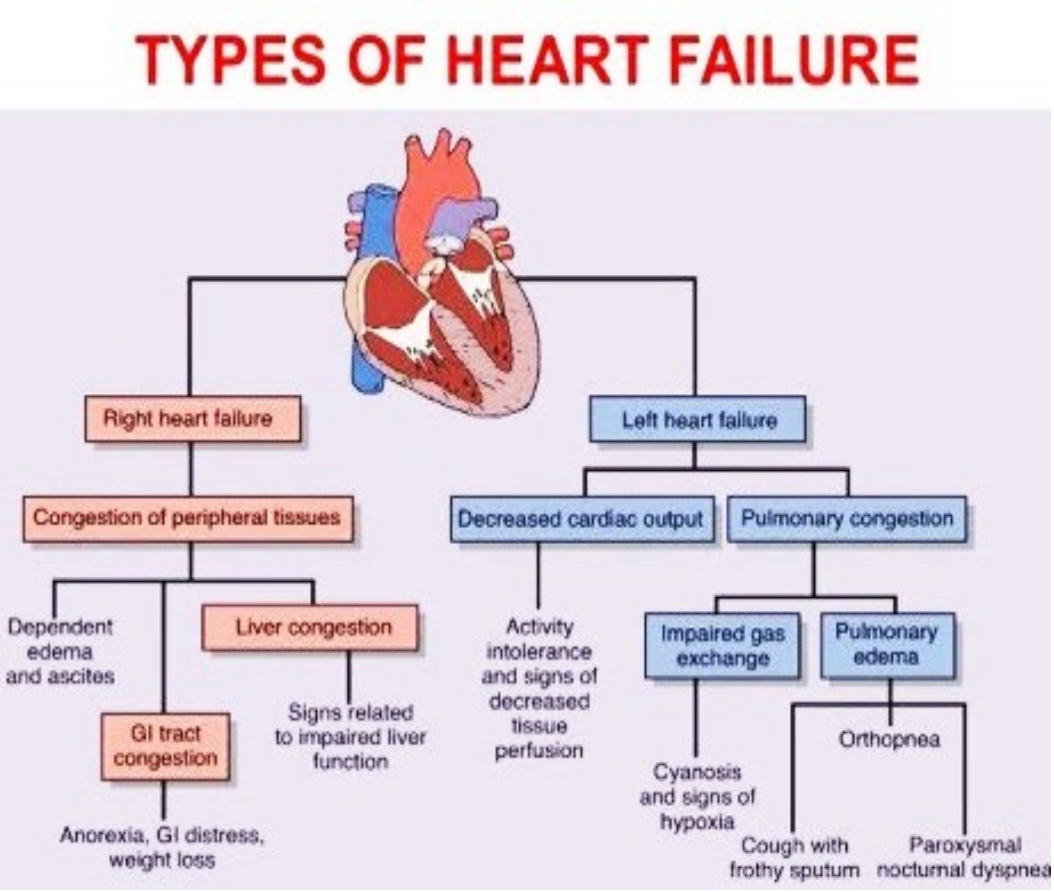
**UNDERLYING CARDIAC DISEASE**

**(1) States that depress systolic ventricular function with reduced ejection fraction** (**HFrEF**; e.g., coronary artery disease [CAD], dilated cardiomyopathies, valvular disease, congenital heart disease).

**(2) States of HF with preserved ejection fraction** (**HFpEF**; e.g., restrictive cardiomyopathies, hypertrophic cardiomyopathy, fibrosis, endomyocardial disorders), also termed diastolic failure.

## Pathophysiology






## HF (Acute Participating Factors)

(1) **Excessive Na+ intake.**

(2) **Noncompliance with HF medications.**

(3) Acute **MI** (may be silent).

(4) Exacerbation of hypertension.

(5) Acute **arrhythmias**.

(6) **Infection and/or fever.**

(7) Pulmonary embolism

(8) Anemia.

(9) Thyrotoxicosis.

(10) Pregnancy.

(11) Acute myocarditis or infective endocarditis.

(12) **Certain drugs (e.g., no steroidal anti-inflammatory agents).**

## Heart Failure (Clinical Presentation)

• **SYMPTOMS:**
Due to **inadequate perfusion of peripheral tissues** (**fatigue**) and **elevated intracardiac filling pressures** (**dyspnea, orthopnea, paroxysmal nocturnal dyspnea, peripheral edema**).

• **PHYSICAL EXAMINATION:**
- **Jugular venous distention**.
- **S3 = Ventricular gallop** (in HFrEF/volume overload).
- **Pulmonary congestion** (crackles, dullness over pleural effusion), peripheral edema, hepatomegaly, and ascites.
- **Sinus tachycardia** is common.
- In patients with **HFpEF, S4 = Atrial gallop** is often present.

## New York Heart Association (NYHA) Classification

- **NYHA class I:** Symptoms only occur **with vigorous activities**, such as playing a sport. Patients **are nearly asymptomatic.**
- **NYHA class II:** Symptoms occur with **prolonged or moderate exertion**, such as **climbing a flight of stairs or carrying heavy packages**.
**Slight limitation of activities.**
- **NYHA class III:** Symptoms occur with **usual activities of daily living**, such as **walking across the room or getting dressed. Markedly limiting.**
- **NYHA class IV:** Symptoms occur **at rest**
**Incapacitating.**

## HF (Work-up)

- **CXR:** may reveal cardiomegaly, pulmonary vascular redistribution, interstitial edema, pleural effusions.
- Left ventricular systolic and diastolic dysfunction are most readily evaluated by **echocardiography with Doppler**, and **EF calculated or estimated**.
- Measurement of **B-type natriuretic peptide (BNP) or N-terminal pro-BNP** differentiates cardiac from pulmonary causes of dyspnea.

## Treatment

**Drugs that decrease mortality in HFrEF:**

1. **ACEI, ARBS.**
2. **Beta-Blocker.**
3. **Aldosterone antagonist.**
4. **Hydralazine/ Nitrate.**
5. **Ivabradiine if PR> 70, LEF < 35%, Or in maximum dose of beta-blocker, or if contraindicated beta-blocker.**

- **Sacubitril/valsartan:**

- Used **instead of** an ACE inhibitor.
- Sacubitril is **added only** to an ARB.
- This **neprilysin inhibitor** (which is responsible for the degradation of atrial and brain natriuretic peptide) does provide a mortality benefit for systolic dysfunction.

- **Pts with HFpEF are treated with:**

1. **Salt restriction and diuretics**, and attention to **underlying causes** (e.g., treatment of hypertension).
2. **Beta blockers.**
3. **ACE inhibitors** may be of benefit in blunting neurohormonal activation, but have not been shown to lower mortality in this population.

**TABLE 126-1 Therapy for Chronic Heart Failure**

1. General measures
   a. Restrict salt intake
   b. Avoid NSAIDs
   c. Immunize against influenza and pneumococcal pneumonia
2. Diuretics
   a. Use in volume-overloaded pts
   b. Weigh daily to adjust dose
   c. For diuretic resistance, administer IV or use two diuretics in combination (e.g., furosemide plus metolazone)
3. ACE inhibitor or angiotensin receptor blocker
   a. For all pts with LV systolic heart failure or asymptomatic LV dysfunction
   b. Contraindications: Serum $K^+$ >5.5, advanced renal disease (e.g., creatinine >3 mg/dL), bilateral renal artery stenosis, pregnancy
4. Beta blocker
   a. For pts with symptomatic or asymptomatic heart failure and LVEF <40%, combined with ACE inhibitor and diuretics
   b. Contraindications: Bronchospasm, symptomatic bradycardia or advanced heart block, unstable heart failure
5. Aldosterone antagonist
   a. Consider for class II–IV heart failure and LVEF <35%
   b. Avoid if $K^+$ >5.0 or creatinine >2.5 mg/dL
6. Digitalis
   a. For persistently symptomatic pts with systolic heart failure (especially if atrial fibrillation present) added to ACE inhibitor, diuretics, beta blocker
7. Other measures
   a. Consider combination of hydralazine and oral nitrate if not tolerant of ACE inhibitor/ARB, and as additive therapy in African-Americans
   b. Consider ivabradine for LVEF ≤35%, if in sinus rhythm, rate >70, already on maximum tolerated beta blocker, or if contraindication to beta blocker
   c. Consider ventricular resynchronization (biventricular pacemaker) for pts with class III–IV heart failure, LVEF <35%, and prolonged QRS (especially LBBB with QRS ≥150 msec)
   d. Consider implantable cardioverter-defibrillator in pts with class II–III heart failure and ejection fraction <35%
   e. Assess and treat sleep apnea

***Abbreviation:*** LBBB, left bundle branch block.

## Acute Pulmonary Edema

- **Definition:**
  - Pulmonary edema is the worst, or most severe, form of CHF.
  - Pulmonary edema is the rapid onset of fluid accumulating in the lungs.

## Presentation

- Pulmonary edema presents with the **acute onset of shortness of breath associated with:**
  - **Orthopnea.**
  - **Rales.**
  - **JVD.**
  - **S3 gallop.**
  - **Edema.**

- There may also be **ascites and enlargement of the liver and spleen** if there has been sufficient time for the chronic passive congestion of the right side of the heart to prevent filling of the heart.

## Diagnostic Tests

**A. Brain natriuretic Peptide:** A normal BNP level excludes pulmonary edema.

**B. Chest X-ray.**

**C. Oximetry/Arterial Blood Gases:** Hypoxia, Respiratory alkalosis.

**D. EKG:**

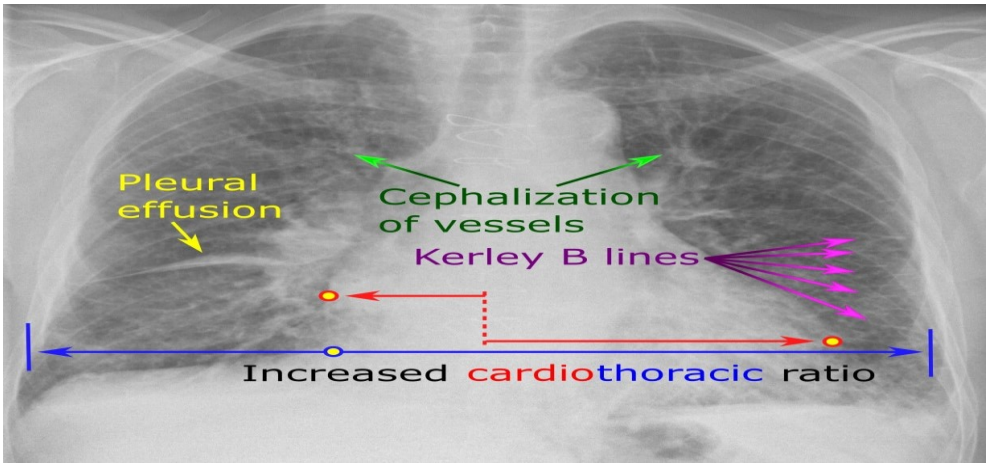
- This is the most important test to do acutely, because the EKG can lead to a change in immediate therapy.

- If atrial fibrillation, atrial flutter, or ventricular tachycardia is the cause of pulmonary edema, the first thing to do is to perform rapid, synchronized cardioversion in order to restore atrial systole and to return the atrial contribution to cardiac output.

**E. Echocardiography:**

- This should be done in all patients to determine if there is systolic or diastolic dysfunction.
- This makes no difference acutely if there is pulmonary edema because the initial therapy does not differ.

## ADHF

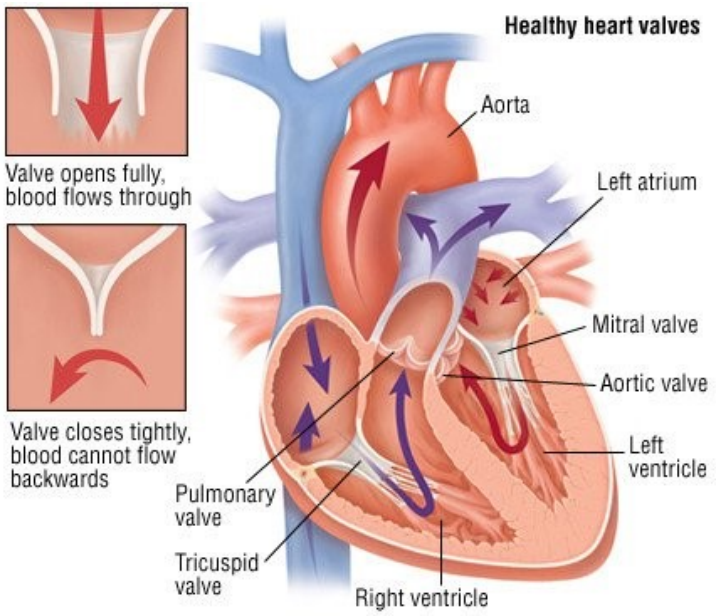



## Treatment

| | |
|---|---|
| **Treatment** | **Normal or elevated blood pressure with adequate end-organ perfusion**<br>• Supplemental oxygen<br>• Intravenous loop diuretic (eg, furosemide)<br>• Consider intravenous vasodilator (eg, nitroglycerin)<br>**Hypotension or signs of shock**<br>• Supplemental oxygen<br>• Intravenous loop diuretic (eg, furosemide) as appropriate<br>• Intravenous vasopressor (eg, norepinephrine) |

# Murmurs

## Anatomy & Physiology




## Heart sounds

- S1 and S2 are **systolic sounds**.
- S3 and S4 are **diastolic sounds.**

**A. First Heart Sound (S1):**

- Closure of mitral, then tricuspid valve. - **Loudest at mitral area.**

**B. Second Heart Sound (S2):**

- Closure of the aortic, then the pulmonic valve.
- Two components: A2 aortic valve closure, and P2, pulmonic valve closure.
- **Loudest at left upper sternal border.**

**C. Third Heart Sound (S3):**

- Occurs during the **rapid filling of a very compliant ventricle.**
- **Normal** in children and pregnancy.
- Is often associated with **a volume-overloaded ventricle** (mitral regurgitation, aortic regurgitation, CHF).
- **Called Ventricular gallop.**

**D. Fourth Heart Sound (S4):**

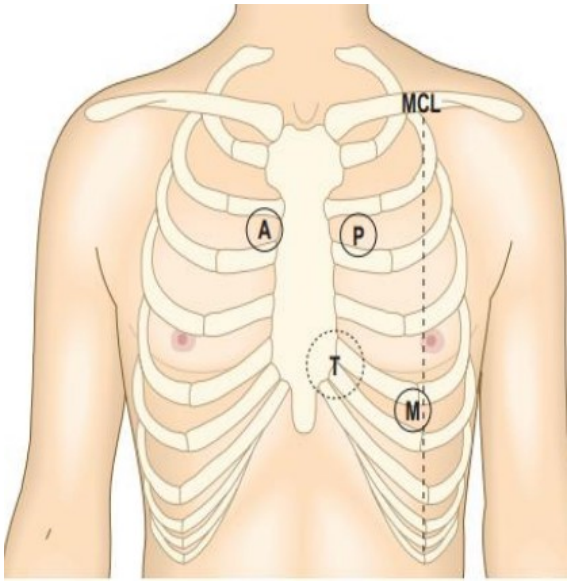
- **Atrial contraction** against **a stiff ventricle** (diastolic dysfunction).
- Presents in any condition that causes **reduced ventricular compliance** (hypertensive heart disease, aortic stenosis, hypertrophic cardiomyopathy).

## Murmurs

- **Heart murmurs produced by:**

  A – Turbulent flow across **an abnormal valve, septal defect or outflow obstruction.**

  B – **Increased stroke volume** or **velocity of flow through a normal valve (innocent murmur),** as in pregnant women, athletes with resting bradycardia or patients with fever.




**Presentation:**

- All valvular heart disease can be **congenital in nature.**
- **Rheumatic fever** can lead to any form of valve disease, but mitral stenosis is most common.
- **Aging** can automatically be associated with **aortic stenosis.**
- **Regurgitant disease** is most commonly caused by **hypertension and ischemic heart disease**.
- **All forms of valvular heart disease** are associated with **shortness of breath** and many of the signs and symptoms of CHF.
- Lesions **on the right side of the heart** (tricuspid and pulmonic valve) increase in **intensity or loudness with inhalation**. Inhalation will **increase venous return to the right side of the heart.**
- **Left-sided lesions** (mitral and aortic valve) **increase with exhalation**. Exhalation will "squeeze" **blood out of the lungs and into the left side of the heart.**

### Diagnostic Tests

- The best initial test is the **echocardiogram.** Transesophageal echo is generally both more sensitive and more specific than transthoracic echo.
- Catheterization is **the most accurate test.**
- Chest x-ray will also show **hypertrophy and enlargement of various cardiac chambers**, but the precise anatomic correlation with the chest x-ray is poor.

**Treatment**

- All forms of valvular heart disease are associated with **fluid overload** in the lungs, all of them will benefit from **diuretics.**
- **Mitral stenosis** is dilated with **a balloon**.
- **Aortic stenosis** needs **surgical removal.**
- Regurgitant lesions seem to respond best to **vasodilator therapy with ACEIs/ARBs, nifedipine, or hydralazine.**
- **Surgical replacement** of regurgitant lesions must be done before **the heart dilates too much**.

**Mitral Stenosis**

- Caused by **Rheumatic Fever.**
- The main indication for treatment is **the presence of symptoms.**
- **Presentation:**

Besides the usual **shortness of breath** and CHF

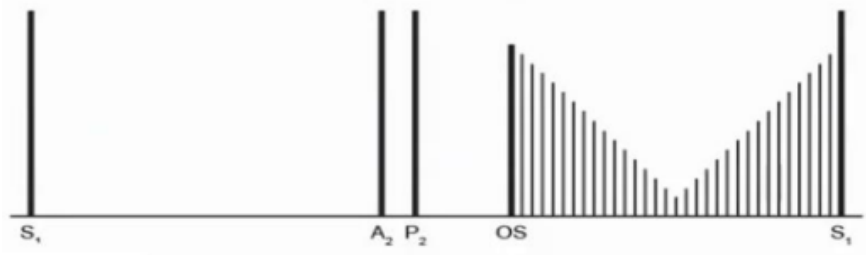
**Unique features of presentation in MS**:

- **Dysphagia** from **left atrium** pressing on the **esophagus.**
- **Hoarseness** (LA pressing on laryngeal nerve).
- **Atrial fibrillation** and stroke from enormous LA. **Up to 70% of** patients with mitral stenosis develop **atrial fibrillation.**
- **Hemoptysis**

**Mitral Stenosis…2**

**Physical Findings:**

- A **mid-diastolic rumbling murmur** with presystolic accentuation will be heard after the **opening snap**.
- **Squatting and leg raising** increase the intensity from increased venous return to the heart.




## Aortic Stenosis

- **Etiology:**
  - **Senile calcific aortic stenosis**
  - **Bicuspid aortic valve**
  - **Rheumatic heart disease.**
- **Presentation:**
  - **Angina**: **most common presentation**.
  - **Syncope**.
  - **CHF.**

**Murmur:**

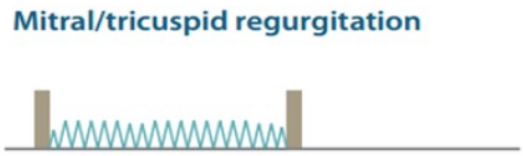
- **A systolic**, crescendo-decrescendo murmur peaking in a diamond shape in mid-systole.
- **The murmur of AS** is heard best at the second right intercostal space, and **radiates to the carotid artery.**
- **Valsalva and standing** improve or decrease the intensity of the murmur from **decreased venous return to the heart**.
- **Handgrip** softens the murmur because of **decreased ejection of blood.**

## Mitral Regurgitation

• **Etiology:**

- Mitral regurgitation (MR) is an **abnormal backward flow of blood through a mitral valve that does not fit together.**
- **Causes :**

**1- Hypertension**.
**2- Endocarditis.**
**3- Myocardial infarction with papillary muscle rupture.**
**4- Any reason that cause the heart dilates will lead to MR.**




▪ **Presentation:**

- The murmur of MR is classically **a holosystolic murmur heard best over the apex with radiation to the axilla.**
- **Handgrip will worsen the murmur of MR** by pushing more blood backwards through the valve.
- **Squatting and leg raising** will also worsen MR by increasing venous return to the heart.
- **All left-sided murmurs** except mitral valve prolapse (MVP) and hypertrophic obstructive cardiomyopathy will increase with **expiration.**

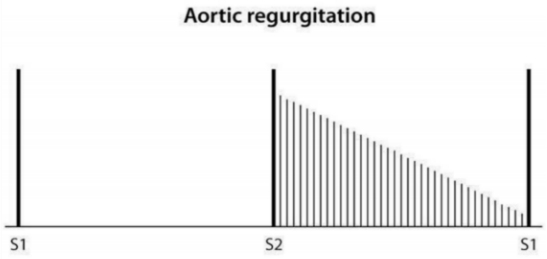
## Aortic Regurgitation

**Etiology:**

- **Aortic regurgitation (AR) is caused by**
  - **Myocardial infarction.**
  - Hypertension.
  - Endocarditis.
  - **Marfan syndrome .**
  - Inflammatory disorders such as **ankylosing spondylitis** or Reiter syndrome.
  - Syphilis.
  - Congenital bicuspid aortic valve is a common type of congenital heart disease in adults.

▪ **Presentation:**

CHF , AR **has a large array of relatively unique physical findings such as:**




**1- Wide pulse pressure.**

**2- Quincke pulse (pulsations in the nail bed).**

**3- Head bobbing (de Musset sign).**

▪ **Murmur:**

- AR leads to an **early decrescendo diastolic murmur**, best heard along the left sternal border at the 3rd and 4th intercostal space.
- **Valsalva and standing** make it better.
- **Handgrip**, which increases afterload by compressing the arteries of the arms, **makes it worse.**

## Mitral Valve Prolapse

▪ **Etiology:**

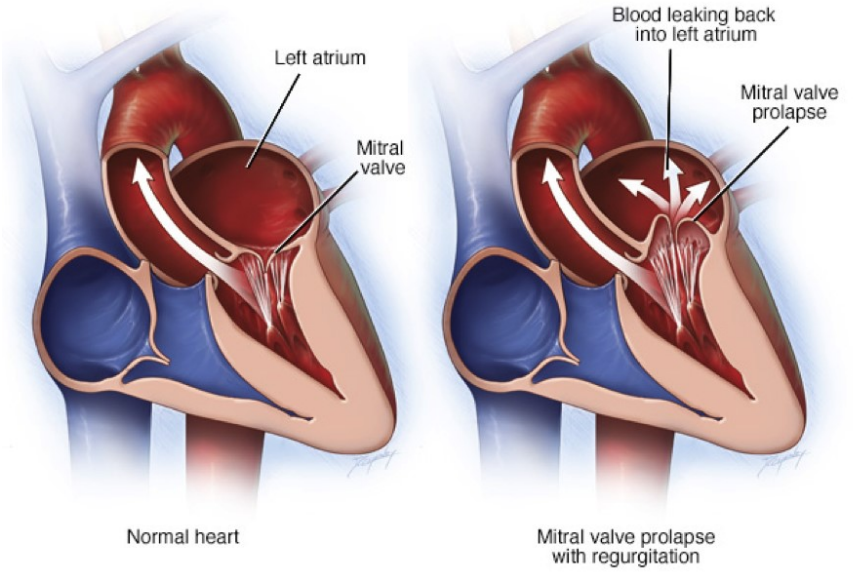
- MVP occurs due to **myxomatous degeneration** of the mitral valve leaflets and chordae **(Marfan and Ehlers-Danlos syndrome)**.

▪ **Presentation:**

- The symptoms of CHF are usually **absent.**
- The most common presentation is:
  - **Atypical chest pain.**
  - **Palpitations.**
  - **Panic attack.**

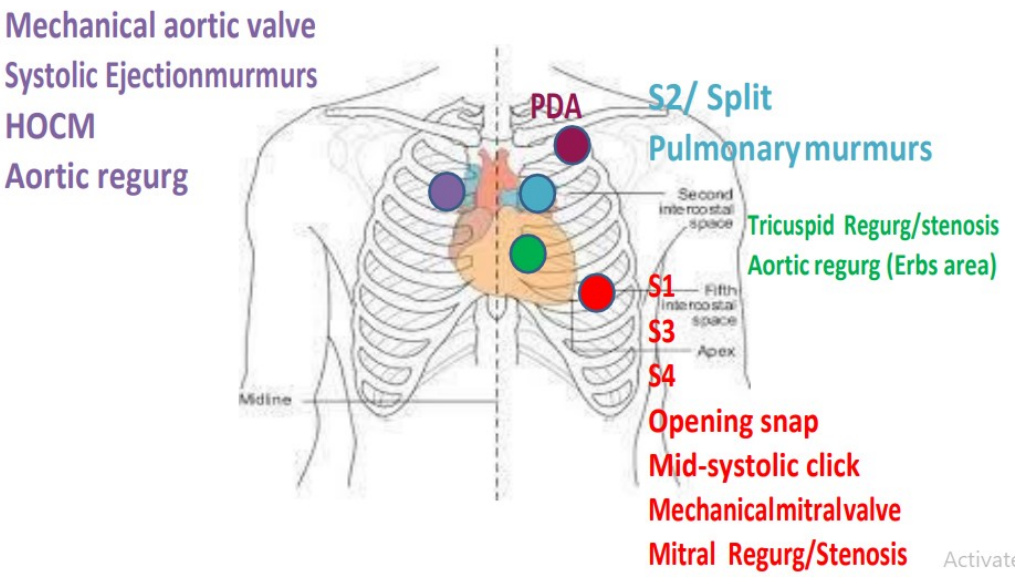
▪ **Murmur:**

- MVP presents with a **midsystolic click** (due to sudden tensing of chordae tendineae) that,
- **When severe,** is associated with a murmur just after the click from **mitral regurgitation (mid to-late systolic murmur).**



## Murmurs and the Effects of Maneuvers

| Murmurs and the Effects of Maneuvers | Squatting/leg raising | Standing/Valsalva |
|---|---|---|
| Mitral and aortic stenosis | Increases both | Decreases both |
| Mitral and aortic regurgitation | Increases both | Decreases both |
| Mitral valve prolapse | Decrease | Increase |
| HOCM | Decrease | Increase |




## Case Scenario

**50 YO male, smoker, has HTN, & hyperlipidemia came to you with chest pain, effort dizziness or lightheadedness, easy fatigability, & progressive inability to exercise. After Chest examination you found mid-systolic ejection murmur & you felt in left systolic thrill in left mediastinum. What is the most likely diagnosis of this patient ?**

# Hypertension

1. **Essential hypertension (HTN)** (i.e., there is no identifiable cause) applies to more than 95% of cases of HTN.
2. **Secondary HTN has many identifiable causes:**
   a. **Renal/ renovascular disease—renal artery stenosis (most common cause of secondary HTN),** chronic renal failure, polycystic kidneys.
   b. **Endocrine causes—**hyperaldosteronism, thyroid or parathyroid disease, Cushing syndrome, pheochromocytoma, acromegaly.
   c. **Medications—oral contraceptives, decongestants, estrogen, appetite suppressants, chronic steroids, tricyclic antidepressants (TCAs), nonsteroidal anti-inflammatory drugs (NSAIDs).**
   d. **Coarctation of the aorta.**
   e. **Cocaine, other stimulants.**
   f. **Obstructive sleep apnea (OSA).**

## Risk Factors

1. **Age**.
2. **Gender**—more common in men.
3. **Race**—it is twice as common.
4. **Obesity, sedentary lifestyle, dyslipidemia**.
5. **Family history.**
6. **Increased sodium intake.**
7. **Alcohol.**

## HTN (Definition)

1. **Hypertensive urgency—severe HTN (typically systolic BP >180 and/or diastolic BP >120) in an asymptomatic patient.**
2. **Hypertensive emergency—severe HTN with end-organ damage (e.g., neurologic changes, myocardial ischemia, aortic dissection).**
3. **Target Organ Damage: Heart—(LVH, MI, CHF), Brain— (stroke, TIA), Chronic kidney disease, (Peripheral arterial disease, AAA, aortic dissection), Retinopathy.**

**ACC/AHA 2017 HYPERTENSION GUIDELINES (13TH NOV 2017)**
**New Classification for Hypertension**

| CATEGORY | SYSTOLIC BP (MM HG) | | DIASTOLIC BP (MM HG) | COMPARISON WITH JNC 7 |
|---|---|---|---|---|
| NORMAL | <120 | AND | <80 | -- |
| ELEVATED BP | 120-129 | AND | <80 | *Was classified as Pre-hypertension under JNC7* |
| STAGE 1 | 130-139 | OR | 80-89 | |
| STAGE 2 | ≥ 140 | OR | ≥ 90 | *SBP of 140-159 OR DBP of 90-99 mm Hg was classified as Stage 1 under JNC7* |
| HYPERTENSIVE CRISIS | ≥ 180 | OR | ≥ 120 | -- |

*Compiled by PlexusMD*

## HTN (History)

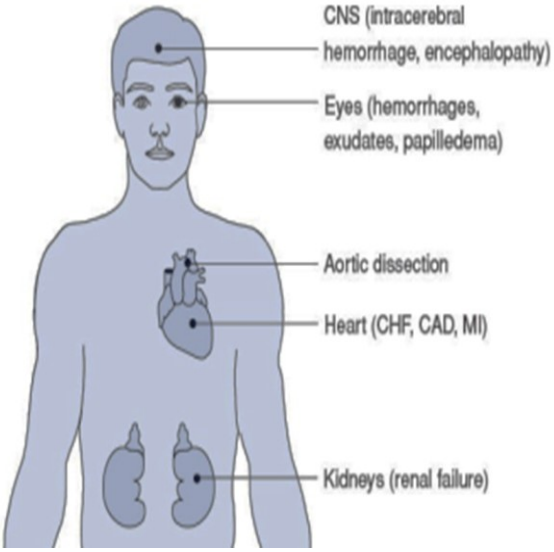
- **History:** Most pts are **asymptomatic**. Severe hypertension may lead to headache, dizziness, or blurred vision.
- **Clues to specific forms of secondary hypertension:** Use of **medications** (e.g., birth control pills, glucocorticoids, decongestants, erythropoietin, NSAIDs, cyclosporine); **paroxysms of headache**, **sweating, or tachycardia** (pheochromocytoma); history of **renal disease or abdominal trauma** (renal hypertension); **daytime somnolence and snoring** (sleep apnea).

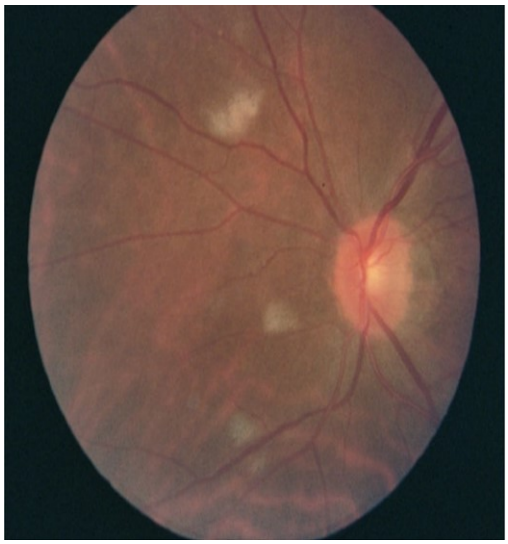
## HTN (Physical examination)

1. **Measure bp with appropriate-sized cuff** (large cuff for large arm).
2. **Measure bp in both arms as well as a leg** (to evaluate for aortic Coarctation).
3. **Signs of hypertension** include retinal arteriolar changes (narrowing/nicking); left ventricular lift, loud A2, S4.
4. **Clues to secondary forms of hypertension** include cushingoid appearance, thyromegaly, abdominal bruit (renal artery stenosis), delayed femoral pulses (coarctation of aorta).

## HTN (Complications)

1. **The major complications of HTN are cardiac complications.**
2. **HTN affects the following organs:**



## HTN (Diagnosis)

**BP measurement:**

a. Unless the patient **has severe HTN or evidence of end-organ damage**, never diagnose HTN on the basis of one BP reading.
b. Establish **the diagnosis on the basis of at least two readings up to 4 weeks apart**.

**Order the following laboratory tests to evaluate target organ damage and assess overall cardiovascular risk**

**a. Urinalysis**
**b. Chemistry panel:** serum K, BUN, Cr
**c. Fasting glucose** (if patient is diabetic, check for microalbuminuria)
**d. Lipid panel**
**e. ECG**

**If the history and physical examination (H&P) or laboratory tests suggest a secondary cause of HTN, order appropriate tests.**

## Treatment (Life-style modification)

**Treat confirmed mild and moderate hypertension with non-pharmacologic modifications in lifestyle:**

- **Weight loss for the obese (most effective).**
- Dietary sodium restriction.
- Aerobic exercise.
- Reduced alcohol intake.
- DASH "Dietary Approaches to Stop Hypertension" diet includes increased fruits/vegetables, low-fat dairy).

## Treatment approach

- Goal is to **control hypertension with minimal side effects**.
- A **combination of medications** with complementary actions is often required.
- **First-line agents include diuretics, ACE inhibitors, angiotensin receptor antagonists, calcium channel antagonists**, and sometimes beta blockers.
- **On-treatment blood pressure goal is <130/80.**

## Treatment (First- line agents)

**TABLE 119-2 Oral Drugs Commonly Used in Treatment of Hypertension (*Continued*)**

| DRUG CLASS | EXAMPLES | USUAL TOTAL DAILY DOSE (DOSING FREQUENCY/DAY) | POTENTIAL ADVERSE EFFECTS |
|---|---|---|---|
| ACE inhibitors | Captopril | 25–200 mg (2) | Cough, hyperkalemia, azotemia, angioedema |
| | Lisinopril | 10–40 mg (1) | |
| | Ramipril | 2.5–20 mg (1–2) | |
| Angiotensin II receptor blockers | Losartan | 25–100 mg (1–2) | Hyperkalemia, azotemia |
| | Valsartan | 80–320 mg (1) | |
| | Candesartan | 2–32 mg (1–2) | |
| Calcium channel antagonists | | | |
| Dihydropyridines | Nifedipine long-acting | 30–60 mg (1) | Edema, constipation |
| Nondihydropyridines | Verapamil long-acting | 120–360 mg (1–2) | Edema, constipation, bradycardia, heart block |
| | Diltiazem long-acting | 180–420 mg (1) | |

## Treatment (Compelling indications)

**TABLE 119-3 Guidelines for Selecting Initial Drug Treatment of Hypertension**

| CLASS OF DRUG | COMPELLING INDICATIONS | POSSIBLE INDICATIONS | COMPELLING CONTRAINDICATIONS | POSSIBLE CONTRAINDICATIONS |
|---|---|---|---|---|
| Diuretics | Heart failure<br>Elderly pts<br>Systolic hypertension | | Gout | |
| Beta blockers | Angina | Heart failure | Uncontrolled asthma and COPD | Athletes and physically active pts |
| | After MI | Pregnancy | Heart block[a] | |
| | Tachyarrhythmias | | | Peripheral vascular disease |
| ACE inhibitors | Heart failure | Chronic renal parenchymal disease | Pregnancy | |
| | LV dysfunction | | Hyperkalemia | |
| | Following an MI | | Bilateral renal artery stenosis | |
| | Diabetic nephropathy | | | |
| Angiotensin receptor blockers | ACE inhibitor cough | Chronic renal parenchymal disease | Pregnancy | |
| | Heart failure | | Bilateral renal artery stenosis | |
| | Diabetic nephropathy | | Hyperkalemia | |
| Calcium channel blockers | Angina<br>Elderly pts<br>Systolic hypertension | Peripheral vascular disease | Heart block[b] | Heart failure with reduced ejection fraction[c] |

[a]Second- or third-degree atrioventricular block.
[b]Second- or third-degree atrioventricular block with verapamil or diltiazem.
[c]Verapamil or diltiazem.
***Abbreviations:*** ARB, angiotensin receptor blocker; COPD, chronic obstructive pulmonary disease.

## Treatment of HTN in pregnancy

| Antihypertensive medications in pregnancy | | |
|---|---|---|
| **First-line (safe)** | **Second-line** | **Contraindicated** |
| • **Methyldopa**<br>• Beta blockers (**labetalol**)<br>• **Hydralazine**<br>• Calcium channel blockers (nifedipine) | • Thiazide diuretics<br>• Clonidine | • ACE inhibitors<br>• Angiotensin receptor blockers<br>• Aldosterone blockers<br>• Direct renin inhibitors<br>• Furosemide |

## Hypertensive emergency

- **Treatment:**
- **IV therapy is indicated**; **nitroprusside and labetalol** are the best agents. For those with evidence of **myocardial ischemia**, use **nitroglycerin**.
- **Enalapril at is an IV ACE inhibitor** that is now being used as well.
- Other less commonly used agents include **esmolol and nicardipine**.
  1. The most important point in management is **not to lower the pressure too far** (not <95-100 mm Hg diastolic) so as not to compromise myocardial or cerebral perfusion.
  2. **The initial goal is to reduce BP by no more than 25% within the first 1−2 hours.**
  3. Because nitroprusside needs monitoring with an arterial line, this is not usually the first choice

# Chapter 2
# Respairtoy System

# Asthma

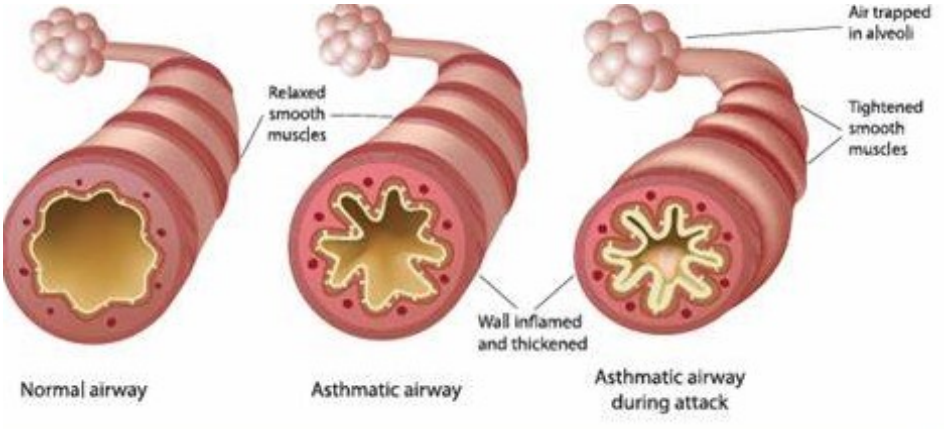
## Asthma (Definition & Pathophysiology)

### What is the definition of Asthma?

**Asthma** is a reversible obstructive lung disease, which is the main difference between this disorder and chronic obstructive pulmonary disease (COPD).

**Asthma is a disease characterized by:**

1. **Inflammatory hyperreactivity** of the respiratory tree to various stimuli, resulting in reversible airway obstruction.
2. **A combination of mucosal inflammation**, **bronchial musculature constriction**, and **excessive secretion of viscous mucus-causing mucous plugs** will produce bronchial obstruction.




There are **2 types of asthma**. Many patients have features of both types:

**A. Intrinsic or idiosyncratic asthma:**

- A bronchial reaction occurs secondary to **nonimmunologic stimuli**, such as infection, irritating inhalant, cold air, exercise, and emotional upset.

**B. Extrinsic (allergic, atopic) asthma:**

- Specific **immunoglobulins (IgE class [type 1])** are produced, and **total serum IgE concentration is elevated**.
- There is a **positive family history of allergic disease**.
- Extrinsic asthma is precipitated by **allergens**.
- Other symptoms include **allergic rhinitis, urticaria, and eczema**.

## Asthma (Clinical presentation)

1. **Characterized by intermittent symptoms** that include **SOB, wheezing, chest tightness, and cough**. Symptoms have **variable severity and may not be present simultaneously**. Usually occur **within 30 minutes of exposure to triggers.**
2. **Symptoms are typically worse at night**.
3. **Wheezing** (commonly during expiration, but can occur during inspiration) is the **most common finding on physical examination**.

"**All That Wheezes Is Not Asthma**"

The most common cause of wheezing is asthma. However, any condition that mimics large airway bronchospasm can cause wheezing.

1. **CHF—due** to edema of airways and congestion of bronchial mucosa
2. **COPD**—inflamed airways may be narrowed, or bronchospasm may be present.
3. **Cardiomyopathies, pericardial diseases** can lead to edema around the bronchi.

- **In a mild attack**, slight tachypnea (increased respiratory rate), prolonged expirations, and mild, diffuse wheezing are seen.
- Asthma **can present exclusively as a cough**.
- In a **severe attack**, use of accessory muscles of respiration, diminished breath sounds, loud wheezing, hyper-resonance (increased vocal fremitus), unable to speak in full sentences, and intercostal retraction are noted.

- **Poor prognostic factors** include fatigue, diaphoresis, inaudible breath sounds, decreased wheezing, cyanosis, and bradycardia.

## Asthma (Causes of acute exacerbations)

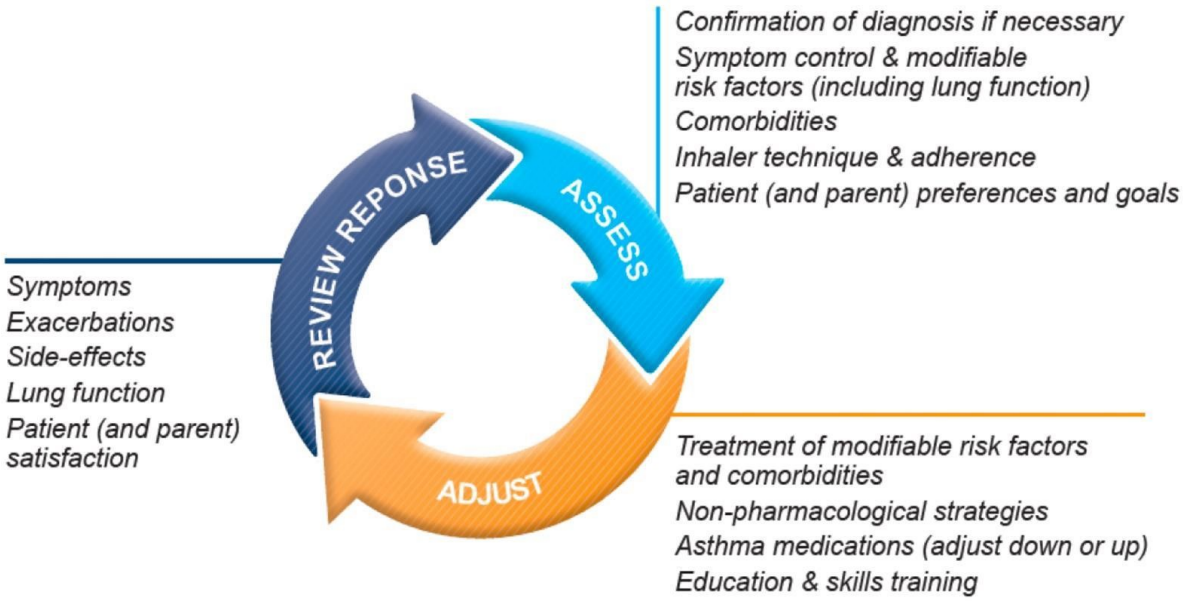
- **Infection and cold air.**
- **Allergens** such as pollen, dust mites, cockroaches, and cat dander.
- **Emotional stress or exercise**.
- **Catamenial**
- **Aspirin, NSAIDs, beta blockers, histamine, any nebulized medication, tobacco smoke.**
- Gastroesophageal reflux disease (**GERD**).

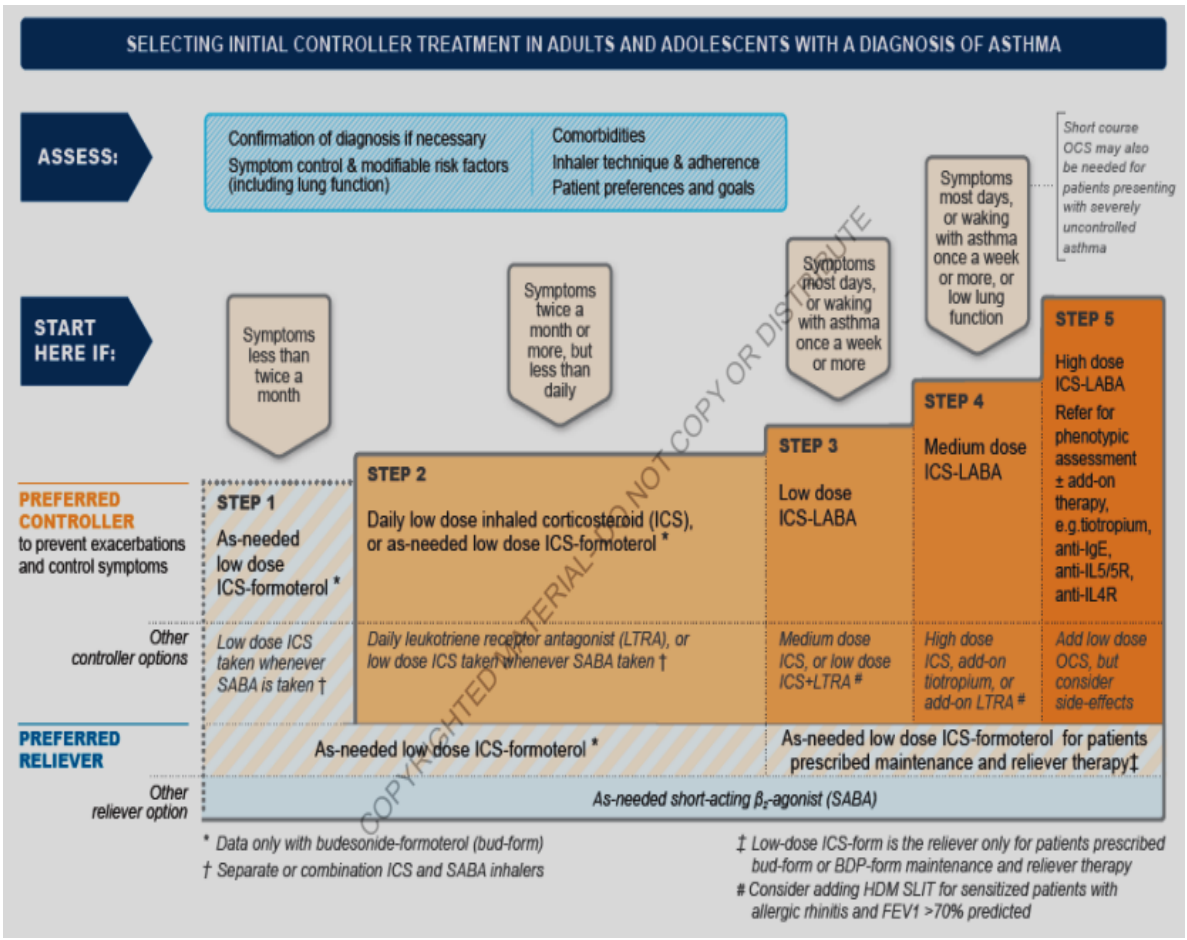
## Asthma (Diagnosis)

• **Pulmonary function tests (PFTs) in asthma show:**
- **PFTs show an obstructive pattern that typically reverses with bronchodilation.**
- Decreased FEV1 and decreased FVC with a decreased ratio of FEV1/FVC.
- **Increase in FEV1 of more than 12% and 200 mL with the use of albuterol**.
- **Decrease in FEV1 of more than 20%** with the use of **methacholine or histamine**.

• **Additional testing options include:**
- **CBC may show an increased eosinophil count**.
- **Skin testing** is used to identify specific allergens that provoke bronchoconstriction.
- **Increased IgE levels** suggest an **allergic etiology**. IgE levels may also help guide therapy such as the use of the anti-IgE medication omalizumab. Increased IgE levels are also associated with allergic bronchopulmonary aspergillosis.

# Asthma (Medical Treatment)



## Acute asthma exacerbation

- **Definition:** It is the acute and sub-acute worsening in symptoms and lung function from the patient's usual status for an asthmatic patients.
- Exacerbations are **commonly triggered by viral URIs**, but other triggers also can be involved.
- Symptoms often include increased **dyspnea, wheezing, and chest tightness**.
- Physical examination can **reveal tachypnea, tachycardia, and lung hyperinflation**.
- Pulmonary function testing reveals a **reduction in FEV1 and PEF**.
- **Hypoxemia can result; Pco2 is usually reduced due to hyperventilation. Normal or rising Pco2 can signal impending respiratory failure.**

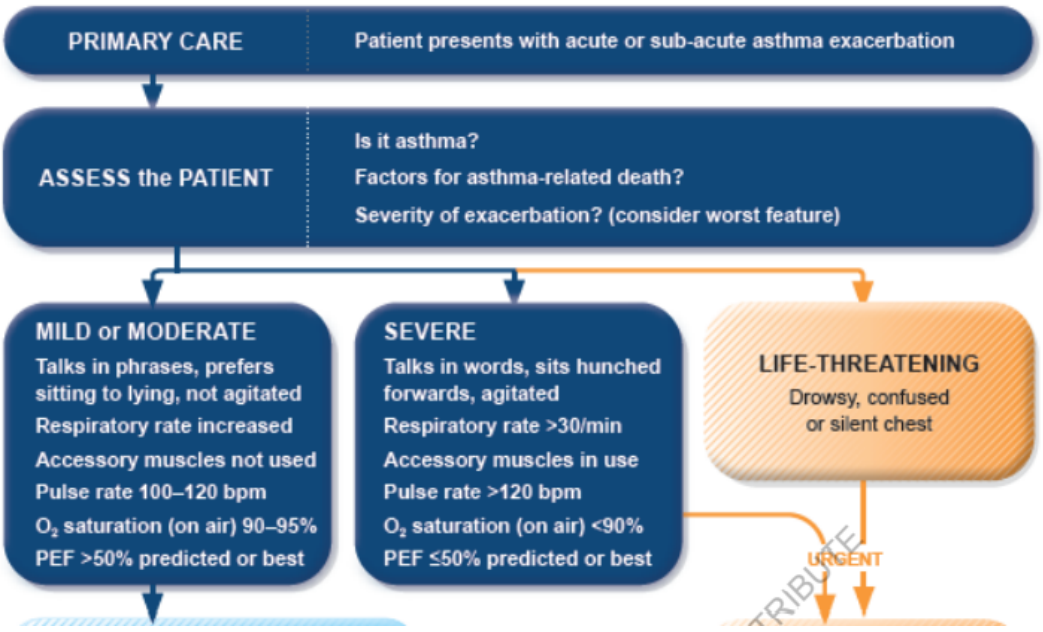
**Box 73-1 Risk Factors for Death from Asthma**

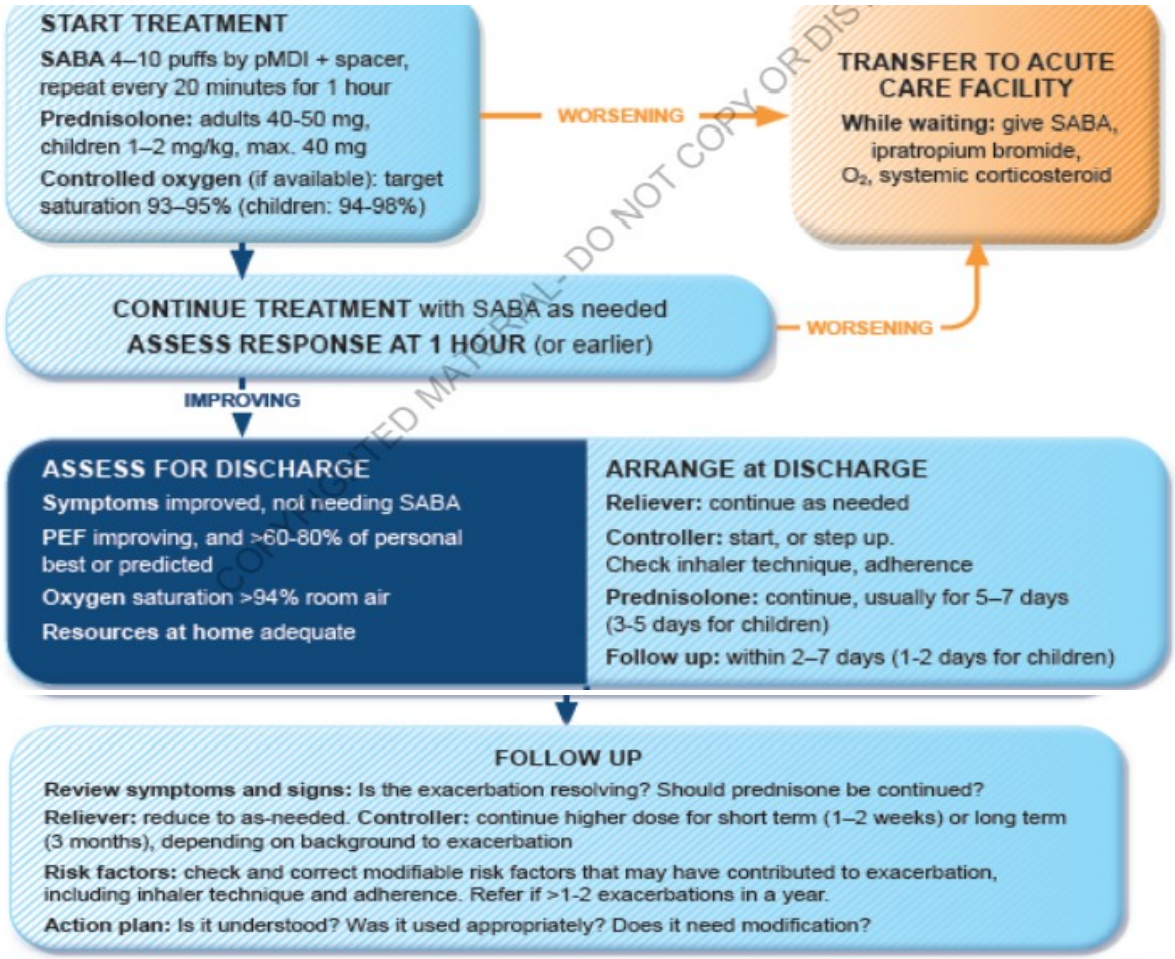
**Asthma History**
Previous severe exacerbation (intubation or ICU admission for asthma)
Two or more hospitalizations for asthma in the past year
Three or more ED visits for asthma in the past year
Hospitalization or an ED visit for asthma in the past month
Use of more than two MDI short-acting beta$_2$-agonist canisters per month
Current use of or recent withdrawal from systemic corticosteroids
Difficulty perceiving asthma symptoms or severity of exacerbations

**Social History**
Low socioeconomic status or inner-city residence
Serious psychosocial problems
Illicit drug use, especially inhaled cocaine and heroin

**Comorbidities**
Cardiovascular disease
Other chronic lung disease
Chronic psychiatric disease

*ED,* emergency department; *ICU,* intensive care unit; *MDI,* metered-dose inhaler.






$O_2$: oxygen; PEF: peak expiratory flow; SABA: short-acting beta$_2$-agonist (doses are for salbutamol)

## Case scenario

You are paged to come and evaluate a 33-year-old female with **SOB and tachycardia**. She normally takes **inhaled fluticasone and albuterol** for asthma, however she is **not currently responding to the albuterol**. On examination, she **appears anxious and is moderately short of breath**. There are **loud bilateral wheezes** on examination, **with a prolonged inspiratory and expiratory phase and use of accessory muscles of respiration**. Temperature = 99.4°F, pulse = 116, **BP = 116/56, RR = 36**. Oxygen saturation **is 91%** on room air. Examination shows **use of accessory muscles on inspiration**. An **arterial blood gas is obtained and shows a pH of 7.4,** PO2 of 60 mm Hg, and **PCO2 of 40 mm Hg**. What is the appropriate next step to be taken in the management of this patient?

A. Increase supplemental oxygen flow rate
B. IV corticosteroids
C. Azithromycin
D. Intubation
E. Nebulized albuterol

# Chronic Obstructive Pulmonary Disease

## Introduction

**Emphysema and bronchitis must be identified as separate entities**, **but most patients with COPD have characteristics of both conditions.**

Chronic bronchitis is defined as a chronic productive cough for ≥ 3 months (not necessarily consecutive) in 2 successive years, with cigarette smoking as the leading cause emphysema abnormal permanent dilation of air spaces distal to the terminal bronchioles with destruction of air space walls.

Both of these processes are defined by **nonreversible** obstruction of the airways. This is the pathognomonic differentiating finding **on PFTs** when compared with asthma (Reversibility).

## Etiology

- Cigarette smoking is a cause of COPD (80–90% of COPD patients are cigarettesmokers )
- Alpha-1 antitrypsin deficiency as the most likely cause in young and anonsmoker

## Pathophysiology

After long-term exposure to cigarette smoke, inflammatory cells arerecruited in the lung. These inflammatory cells in turn secrete proteinases, which may lead to air space destruction and permanentenlargement. Eventually, decreased elastic recoil (mainly in emphysema)

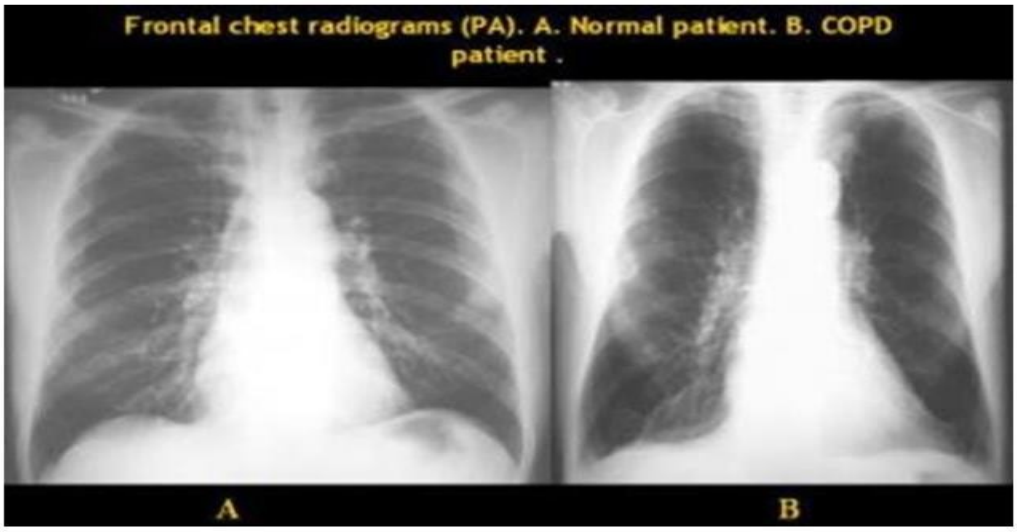
## Presentation

**Shortness of breath worsened by exertion.**
**Productive cough** due to excessive mucus production in chronic bronchitis **increased anterior-posterior diameter of chest (barrel-chest)**
**Muscle wasting and cachexia**

## Diagnosis

The best initial test is chest x-ray:

- ❖ Increased anterior-posterior (AP) diameter.
- ❖ Air trapping and flattened diaphragms.



- The most accurate diagnostic test is PFT:
  Decreased FEV1, decreased FVC , decrease FEV1/FVC ratio (< 70%) **Increased TLC**

  Atter a bronchodilator is given, you would expect the FEV1/FVC to remainthe same

- Arterial blood gas (ABG): **CO2 retention**
- CBC: **Polycythemia**
- EKG: **MAT**
- Echocardiography: **Right ventricular Hypertrophy**

## Improves Mortality and Delays Progression of Disease:

- **The only interventions which have been shown to decrease mortality in patients with COPDare home oxygen and smoking cessation:**
  1. Smoking cessation: Smoking cessation is associated with a mortality benefit and reduced progressionof disease in patients with chronic obstructive pulmonary disease.
  2. O2 use: Long-term supplemental oxygen therapy (LTOT) has demonstrated prolonged survival andimproved quality of life in patients with COPD with significant chronic hypoxemia.

- **The criteria for initiating LTOT in such patients include:**
  **Resting arterial oxygen tension (PaO2) ≤55 mm Hg or pulse oxygen saturation (SaO2) ≤88% on room airPaO2 <60 mm Hg or SaO2 <90% in patients with cor pulmonale, evidence of right heart failure, or hematocrit >55%.**
  **In addition to continued abstinence from smoking, initiation of LTOT will have the greatest benefit tothis patient's survival.**

**Although the "hypoxic drive elimination" concept is not correct, you would still avoid reflexively placinga patient with COPD on a very high-flow 100% nonrebreather mask. Use only as much oxygen as is necessary to raise the pO2 above 90% saturation**

**All patients with COPD must have the pneumococcal vaccine (Pneumovax) every 5 years and theinfluenza vaccine yearly**

- Definitely Improves Symptoms (But Does Not Decrease Disease Progressionor Mortality):
  - Anticholinergic agents (ipratropium and tiotropium) are the first-linedrugs in COPD. Ipratropium is the only one used in acute exacerbation.
  - β2-adrenergic agonists (albuterol) are used after anticholinergic agents.
  - The inhaled route is the preferred administration.
  - Beta agonists are not first-line agents in the management of COPDbecause many of the patients have underlying heart disease and the tachycardia commonly associated withthese agents may precipitate heart failure.
  - Chronic inhaled corticosteroids are reserved for severe cases of COPD.

| Acute exacerbation of chronic obstructive pulmonary disease | |
|---|---|
| **Cardinal symptoms** | • Increased dyspnea<br>• Increased cough (more frequent or severe)<br>• Sputum production (change in color or volume) |
| **Diagnostic testing** | • Chest x-ray: Hyperinflation<br>• ABG: Hypoxia, $CO_2$ retention (chronic &/or acute) |
| **Management** | • Oxygen (target $SpO_2$ of 88%-92%)<br>• Inhaled bronchodilators<br>• Systemic glucocorticoids<br>• Antibiotics if ≥2 cardinal symptoms<br>• Oseltamivir if evidence of influenza<br>• NPPV if ventilatory failure<br>• Tracheal intubation if NPPV failed or contraindicated |

**ABG** = arterial blood gas; **NPPV** = noninvasive positive-pressure ventilation; **$SpO_2$** = peripheral oxygen saturation.

## ETIOLOGY OF ACUTE EXACERBATION

Although viruses cause 20% to 50% of episodes, coverage should beprovided against

Streptococcus pneumonia, H. influenza, and Moraxella catarrhal is:

Macrolides: azithromycin, clarithromycin.

Quinolones: levofloxacin, moxifloxacin

# Restrictive Lung Disease

## Interstitial Lung Disease

- Interstitial lung disease is a group of heterogeneous diseases characterized by **chronic inflammation** and **fibrosis** of the **interstitium and lung parenchyma**.
- The interstitium of the lung **(supporting structure)** is the area in and around the small blood vessels and alveoli where **the exchange** of oxygen and carbon dioxide takes place.

Inflammation and scarring of the interstitium (and eventually extension into the alveoli) will **disrupt normal gas exchange**.

## Causes of apical ILD (Breasts clap)

- Berylliosis
- Radiation
- Extrinsic Allergic Alveolitis
- Allergic Bronchopulmonary Aspergillosis
- Sarcoidosis
- Tuberculosis
- Coal worker's Pneumoconiosis
- Langerhans cell histiocytosis
- Ankylosing Spondylitis
- Psoriasis

## Causes of basal ILD

- **Drugs** (amiodarone, bleomycin, busulfan, methotrexate, vincristine, nitrofurantoin).
- Rheumatoid Arthritis.
- Connective Tissue Disease.
- Idiopathic Pulmonary Fibrosis.
- Asbestosis.

## Idiopathic pulmonary fibrosis (IPF)

Inflammatory lung disease of unknown origin that causes lung fibrosis and restrictive lung disease .It characteristically involves only the lung and has no extra pulmonary manifestations except clubbing.
Typically seen in decade 5 of life, it affects men and women equally.

## Pneumoconiosis

Occupational lung diseases in which inhalation of certain fibers initiates an inflammatory process and eventually leads to fibrosis of the lung .Alveolar macrophages engulf offending agents, causing inflammation and fibrosis of the lung parenchyma in pneumoconiosis.

| Exposure | Disease |
|---|---|
| **Coal** | Coal worker's pneumoconiosis<br>preference of upper lobes |
| **Sandblasting, rock mining, tunneling** | Silicosis: affect upper lobes, eggshell calcifications o |
| **Shipyard workers, pipe fitting, insulators** | Asbestosis |
| **Cotton** | Byssinosis |
| **Electronic manufacture** | Berylliosis |

The most common cancer associated with asbestosis is **bronchogenic carcinoma** (adenocarcinoma or squamous cell carcinoma)

Pleural or peritoneal **mesotheliomas** are also associated with asbestos exposure but are not a common as bronchogenic cancer.

It is thought that **silica** may disrupt phagolysosomes and impair macrophages, increasing susceptibility to **TB**.

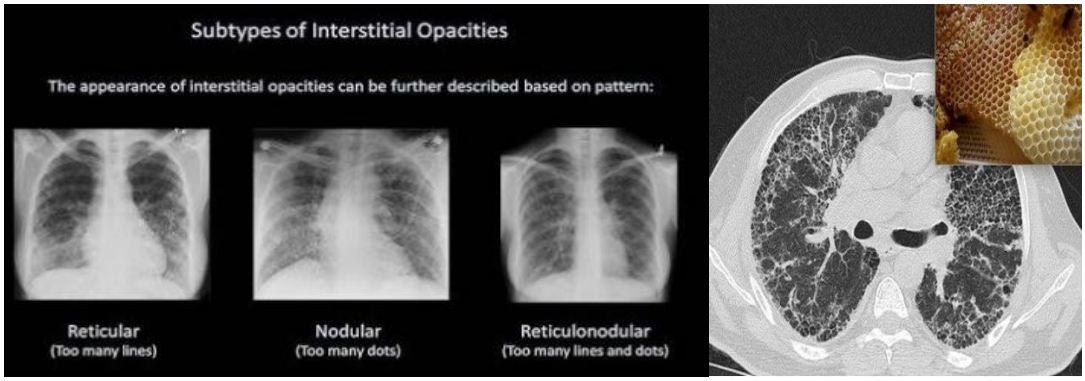
## Clinical picture

All forms of pulmonary fibrosis present with:

- Dyspnea, worsening on exertion, dry cough.
- Fine rales or" crackles "on examination.
- Loud P2 heart sound.
- Clubbing of the fingers.

## Diagnostic tests

- PFTs :Restrictive lung disease with decrease of everything proportionately .The FEV1, FVC, TLC, and residual volume will all be decreased, but since everything is decreased, the FEV1/FVC ratio will be normal with decreased DLCO.
- The best initial test is always a **CXR** .High resolution **CT scan** is more accurate than CXR
- Echocardiography will often show pulmonary hypertension and possibly right ventricular hypertrophy.
- Biopsy shows granulomas in berylliosis.
- Those without an identifiable environmental, infectious, or autoimmune etiology likely have idiopathic pulmonary fibrosis (IPF).




## Treatment

- Most types of interstitial lung diseases are **untreatable**.
- If the biopsy shows white cell or inflammatory infiltrate, **prednisone** should be used . **Berylliosis** is the most likely to respond to treatment with steroids .This is due to the presence of **granulomas**, which are a sign of inflammation.
- In patients who do respond to steroids, switch to **azathioprine** for long-term treatment to get the patient off steroids .If there is no response to steroids or azathioprine, try **cyclophosphamide**.

- Agents to Decrease the Rate of Progression of Idiopathic Pulmonary Fibrosis(IPF):
  - **Pirfenidone** and **nintedanib** slow the rate of fibrosis.
  - Pirfenidone is an antifibrotic agent that inhibits collagen synthesis.
  - Nintedanib is a tyrosine kinase inhibitor that blocks fibrogenic growth factors and
  - inhibits fibroblasts.

# Pulmonary Embolism & Deep Vein Thrombosis

## Thromboembolic Disease

**1- Deep vein thrombosis (DVT)**

Is a type of **venous thrombosis** involving the **formation of a blood clot in a deep vein**, most commonly in the **legs or pelvis**. may occur in the **arms**.

**Symptoms**:
Pain, Swelling, Redness and Enlarged Veins in the affected area, but some DVTs have no symptoms.

**2- Pulmonary Embolism (PE)**

The **most common life-threatening** concern with DVT is the potential for a clot to embolize & travel as an embolus through the **right side of the heart, and become lodged in a pulmonary artery.**

## Etiology

DVT is Predisposed by **Virchow triad (SHE)** :
1- **S**tasis (post-op, long drive/flight).
2- **H**ypercoagulability (defect in coagulation cascade proteins, such as factor V Leiden).
3- **E**ndothelial damage (exposed collagen triggers clotting cascade).

## Presentation

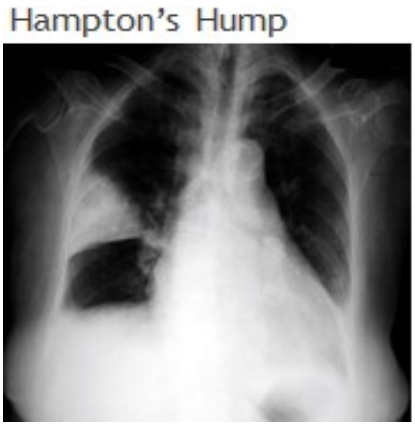
- **Acute-onset dyspnea, Pleuritic chest** pain are the most common symptoms. May be associated with **Tachypnea, tachycardia, and cough**.
- **Hemoptysis.**
- **Unilateral leg pain** from DVT.
- **Low-grade fever**
- **Extremely severe emboli** will produce **hypotension**.

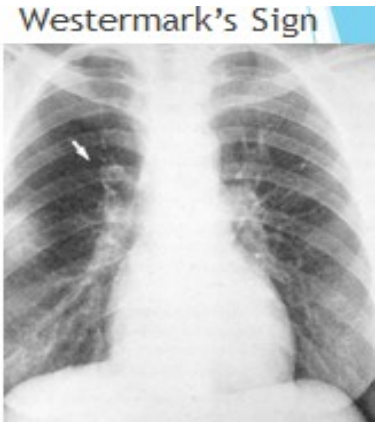
**On Physical Examination:**
Always **increased respiratory rate** with **tachycardia**; **increased Pulmonic sound (P2).**

## Diagnostic Tests

**A. Chest x-ray:**

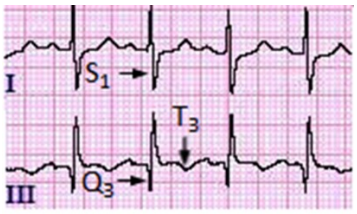
- **Usually normal in PE.**
- The most common abnormality is **atelectasis.**
- Wedge-shaped infarction, **Hampton hump** (peripheral wedge of lung opacity due to pulmonary infarction), **Westermark sign** (peripheral hyperlucency due to oligemia that occur distal to the pulmonary embolus).



**B. ECG:**

- **Most common** finding on the ECG is **sinus tachycardia.**
- **Most Specific** findings are **S1, Q3, T3 due to increase tension on RV wall** .




**C. ABG:**

**Hypoxia** and **Respiratory Alkalosis** (high pH and low pCO2).

**Wells Score for Deep Vein Thrombosis**

*Wells score 2≤, DVP is Unlikely*
*Wells score > 2, DVP is Likely.*

| | |
|---|---|
| Active Cancer | +1 |
| Paralysis, paresis, recent cast on lower extremity | +1 |
| Recent immobilization that lasted more than 3 days, or, surgery within the past month | +1 |
| Localized tenderness of deep venous system | +1 |
| Swelling of entire leg | +1 |
| Calf swelling of more than 3 cm (compare to non-swollen leg) | +1 |
| Pitting edema (unilateral) | +1 |
| Collateral superficial veins | +1 |
| Prior DVT | +1 |
| Alternative likely diagnosis | -2 |

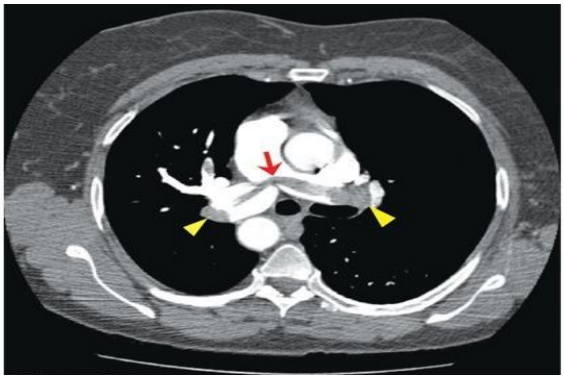
| Features | Score (points) |
|---|---|
| Clinical signs and symptoms of DVT | 3.0 |
| No alternative diagnosis | 3.0 |
| Heart rate >100 beats/min | 1.5 |
| Immobilization ≥3 days or surgery in the previous 4 weeks | 1.5 |
| Previous DVT or PE | 1.5 |
| Hemoptysis | 1.0 |
| Malignancy with active treatment in the past 6 months or under palliative care | 1.0 |
| Pretest clinical probability | |
| PE unlikely | ≤4.0 |
| PE likely | >4.0 |

PE = Pulmonary embolism, DVT = Deep vein thrombosis

**D- CT Angiogram :**

It is used to **confirm the presence of a PE** after the x-ray, EKG, and ABG are done.

- It allows **direct visualization of the pulmonary embolus**.
- It is **Specific and Sensitive**



**E- D-dimer:**

This test is **very Sensitive** , but **the Specificity** is **poor** since any cause of clot or increased bleeding **can elevate the d-dimer level.**

**F. Lower extremity (LE) Doppler study**:

**70% of PEs** originate in the legs, it will miss **30% of cases.**

**G. Ventilation/Perfusion (V/Q) scan**.

**H. Angiography:**

**The most accurate test** with nearly 100% specificity and a false negative rate **under 1% .**

## Treatment

- Give **Oxygen** and **start heparin immediately** before the diagnosis is confirmed.

Once the diagnosis is confirmed:

1- **Heparin**: LMWH (Enoxaparin) or unfractionated for 5-7 days.

2- **Warfarin**: should be started with heparin and continued for 6 months for both **pulmonary emboli and DVT. (INR 2-3)**

   - **Warfarin** is contraindicated in **pregnant patients**. **LMWH for 6 months is the best alternative.**

3- **Novel oral anticoagulants (NOAC)** can be used instead of **Warfarin :** Rivaro**Xa**ban, api**Xa**ban, edo**Xa**ban (Direct factor **Xa** inhibitors), and **dabigatran** (Direct thrombin inhibitors).

**Thrombolytics (tPA, streptokinase) or Surgical thrombectomy:**

Are not used routinely in pulmonary embolism and should be reserved for patients that become **hemodynamically unstable (hypotension [systolic BP < 90] and tachycardia).**

**Inferior Vena Cava (IVC)** filter Used when:

- **Contraindication to the use of anticoagulants** (melena, CNS bleeding).
- **Recurrent emboli** while on a NOAC or fully therapeutic warfarin (INR of 2-3).

## Case Scenario

**A 28-year-old female presented to the ER complaing of low grade fever , Acute onset SOB and chest pain that is increase with inspiration of 2 hour duration . She is also complaining of pain & Swelling in her right calf for 2 days.**

**- What is the most likely diagnosis?**

**- If Her BP was 80/60 what is the best next step?**

# Chapter 3

# Renal System

# Acute Kidney Injury

## AKI (Etiology)

• **Definition**:
• A **rapid decline in renal function**, reflected by GFR.

• **KIDIGO:**
1. **Abrupt (< 48hr) increase in Cr by > 0.3 mg/dl.**
2. **Increase in creatinine 50% from baseline in last week.**
3. **Reduction in urine out-put to < 0.5 ml/kg/hr for > 6hr.**

| RIFLE | Creatinine | GFR | Urine Output |
|---|---|---|---|
| Risk | 1.5 fold ↑ | ↓ 25% | < 0.5 mL/kg/hr for 6 hours |
| Injury | 2.0 fold ↑ | ↓ 50% | < 0.5 mL/kg/hr for 12 hours |
| Failure | 3.0 fold ↑ | ↓ 75% | < 0.3 mL/kg/hr for 24 hours or Anuria |
| Loss | - Complete loss of kidney function for > 4 weeks | | |
| ESRD | - Complete loss of kidney function for > 3 months (requiring dialysis) | | |

| | Pre-renal | Renal | Post-renal |
|---|---|---|---|
| Etiology | - Hypovolemia.<br>- Hypoperfusion.<br>- Hepatorenal.<br>- Cardiorenal. | - Glomerular disease.<br>- ATN.<br>- AIN.<br>- Vascular (PAN, TTP). | - Obstruction (BPH, nephrolithiasis, neoplasm, neurogenic bladder). |
| BUN/ Cr | > 20 | Varies | Varies |
| FE $_{Na}$ | < 1% | > 2% | > 2% |
| U $_{Osm}$ | > 500 | < 350 | < 350 |
| U $_{Na}$ | < 40 | > 40 | > 40 |

## AKI (Clinical presentation)

- **Usually asymptomatic.**
- Can cause **edema, hypertension, urine out-put**.
- **Uremia:** Anorexia, vomiting, pericarditis, encephalopathy.

## AKI (Diagnosis)

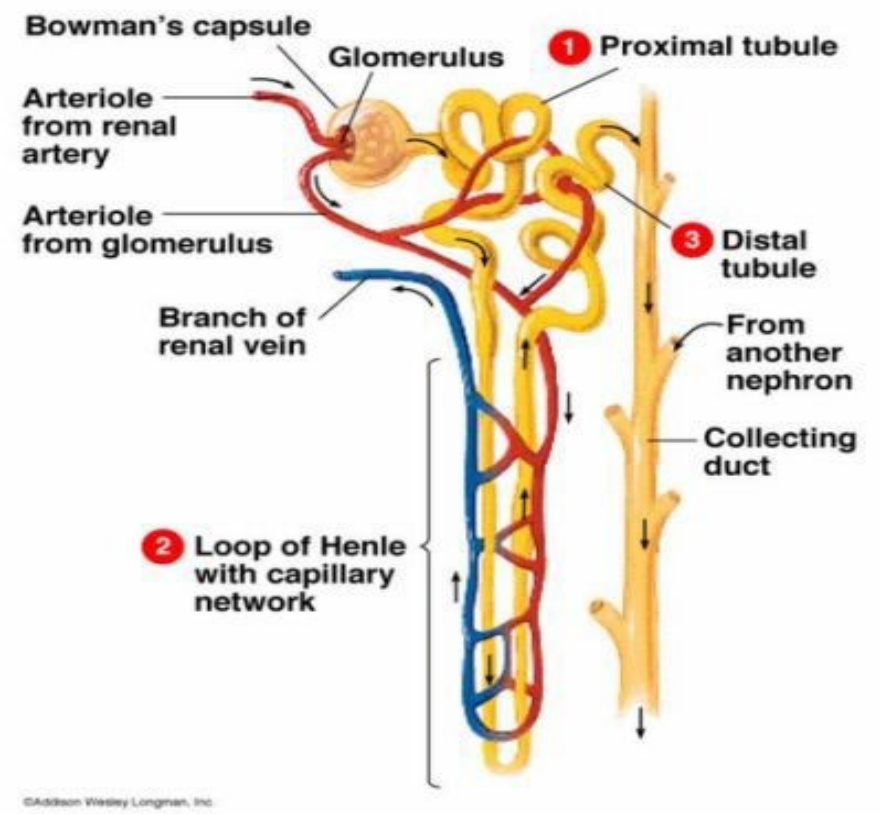
• **Renal function test** (as seen before).
• **Urinalysis**.
• **Urine sodium excretion.**
• **Urine volume monitoring.**

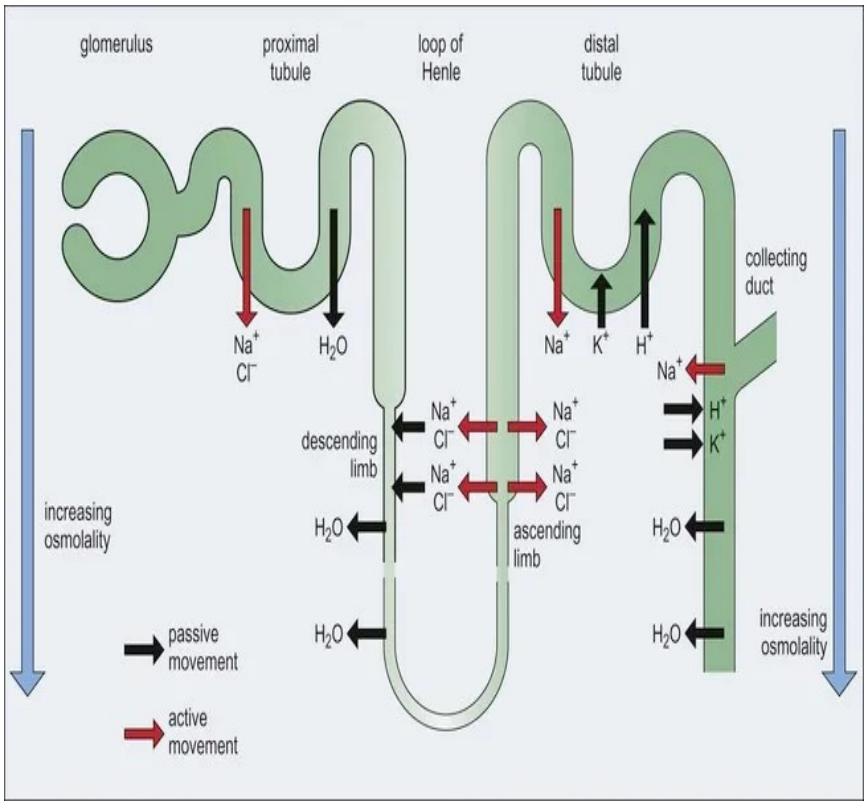
## Management

• **Correct volume status, underlying disorder.**
• Monitor for below complications, which (if severe) are dialysis indications.
• **Post-renal: Urinary catheterization.**

# Chronic Kidney Disease

## Anatomy



## CKD (Etiology)

• **Definition**:
• **Structural/Functional kidney abnormalities X3 months, with or without decrease in GFR<60.**
• May results from **glomerular, tubular, inflammatory, or vascular insult.**
• More common **in African-American** than in Caucasian patients.

• **Causes:**
**A. Diabetes is the most common cause (30% of cases).**
**B. HTN** is responsible for **25% of cases.**
**C. Chronic GN** accounts for **15% of cases.**
D. Interstitial nephritis, polycystic kidney disease, obstructive uropathy.
E. Any of the causes of AKI may lead to CKD if prolonged and/or if treatment is delayed.

## Pathophysiology

**A. Plasma Cr varies inversely with GFR.**

**B. Cr clearance is the most common clinical measure of GFR** (although remember this is an indirect estimation and can lead to miscalculations).

**C. An increase in plasma Cr indicates disease progression**, whereas a **decrease suggests recovery of renal function** (assuming muscle mass has not changed).

**Most laboratories now also report an estimated GFR (eGFR) each time the creatinine is ordered.**

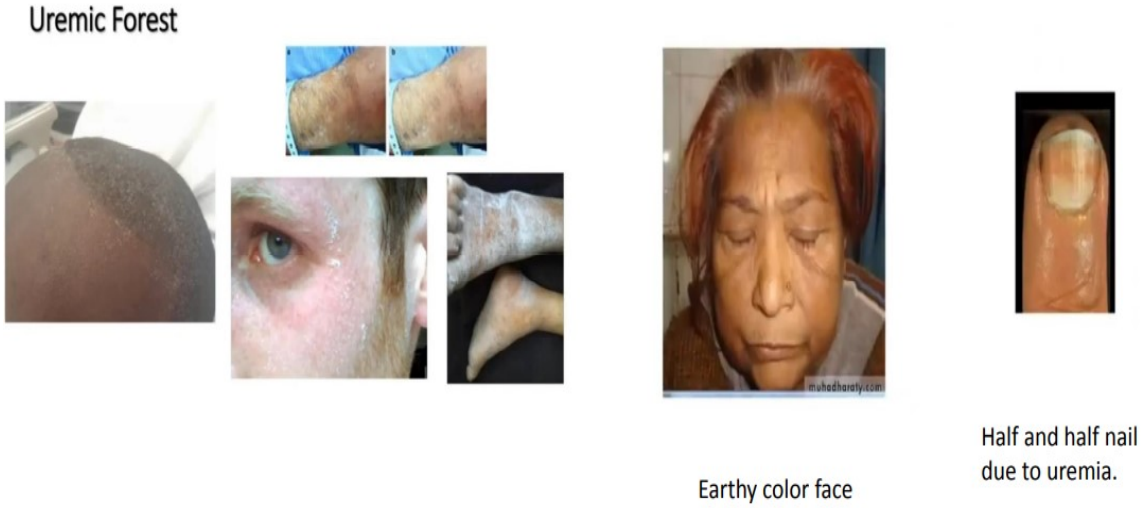
**Estimation of GFR**

| | |
|---|---|
| **1. Cockcroft-Gault Equation (mL/min)** | $CCr = \frac{(140 - age) \times LBW\ [kg]}{Cr\ [mg/dL] \times 72}$ <br> *For women Multiply by 0.85* |
| **2. MDRD study equation (mL/min/1.73 m²)** | $GFR = 186.3 \times (SCr)^{-1.154} \times (Age)^{-0.203}$ <br> *Multiply by 0.742 for women* <br> *Multiply by 1.21 for African ancestry* |
| **3. CKD-EPI equation** | $GFR = 141 \times \min(SCr/\kappa)^{\alpha} \times \max(SCr/\kappa)^{-1.209} \times 0.993^{age}$ <br> *Multiply by 1.018 for women* <br> *Multiply by 1.159 for African ancestry* <br> **κ** is 0.7 for females and 0.9 for males, **α** is −0.329 for females and −0.411 for males, **min** indicates the minimum of SCr/κ or 1, & **max** indicates the maximum of SCr/κ or 1 |

## CKD (Clinical presentation)

• **Clinical features:**
**1. Urinary** (Polyuria to Anuria, frothy .. Etc), Uremia (increased BUN in blood (azotemia) results in **nausea, anorexia, pericarditis, platelet dysfunctions, encephalopathy with asterixis, and deposition of urea crystals in the skin**).
**2. Salt and water retention** (**Hypertension**).
**3. Hyperkalemia with metabolic acidosis.**
**4. Anemia** (due to decreased EPO production).
**5. Hypocalcemia** (Due to decrease 1a hydroxylation of vitamin d, and due to hyperphosphatemia), **Renal osteodystrophy, osteomalacia, osteoporosis.**
**6. Infections.**

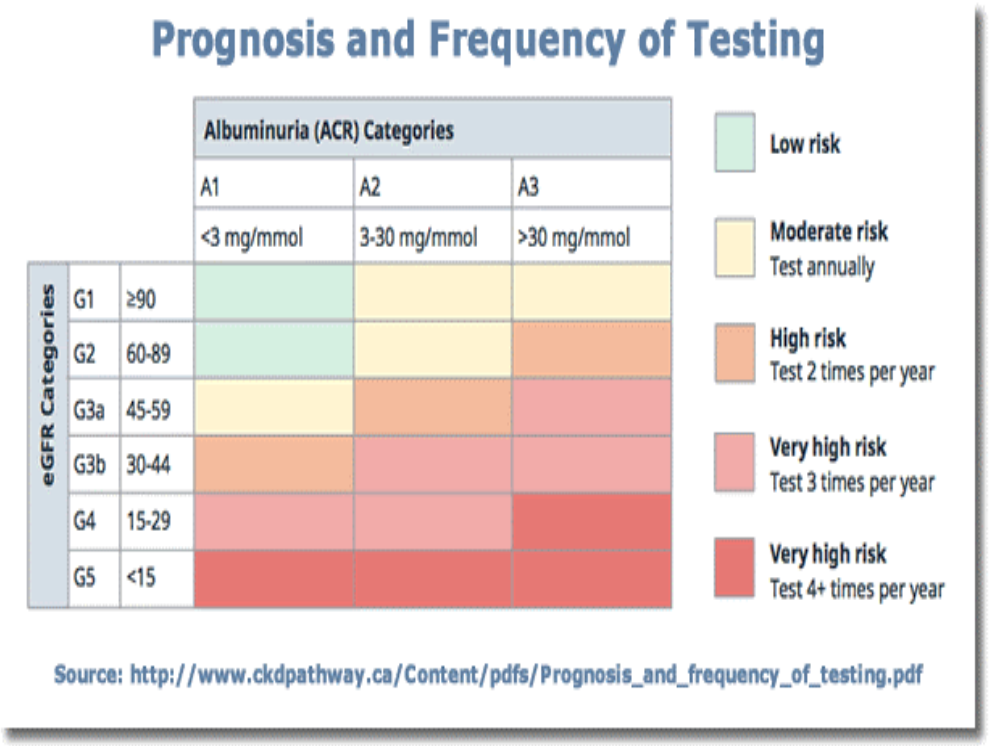
## Uremia




## Life-Threatening Complications in CKD:

1. **Hyperkalemia**—obtain an ECG (be aware that potassium levels can be high without ECG changes).
2. **Pulmonary edema** secondary to volume overload—look for recent weight gain.
3. **Infection** (e.g., pneumonia, UTI, sepsis).

## CKD (stages)




- **The higher the albuminuria the more severe and more cardiovascular complications.**
- **At 3b the complications will start (Anemia).**
- **Stage 5 is either 5D or 5-without D. (D= Dialysis).**

## Diagnosis

1. **Urinalysis**:
   a. Level of proteinuria is increasingly being reported alongside GFR as it is associated with more rapid progression of CKD.

**2. Measure Cr clearance to estimate GFR.**

**3. CBC (anemia, thrombocytopenia).**

**4. Serum electrolytes (e.g., K+, Ca2+, PO43−, serum protein).**

**5. Renal ultrasound—evaluate size of kidneys/rule out obstruction.**
   a. **Small kidneys are suggestive of chronic renal insufficiency** with little chance of recovery.
   b. Presence of normal-sized or large kidneys does not exclude CKD.
   c. **Renal biopsy**—in select cases to determine specific etiology.

## CKD (Treatment)

**1. Diet:**
   a. **Low protein—to 0.7 to 0.8 g/kg body weight per day**: data suggests this may slow progression of CKD.
   b. **Use a low-salt diet if HTN, CHF, or oliguria are present.**
   c. **Restrict potassium, phosphate, and magnesium intake.**

**2. ACE inhibitors—dilate efferent arteriole of glomerulus**
   a. If used early on, they reduce the risk of progression to ESRD because they slow the progression of proteinuria.
   b. Use with great caution because they can cause hyperkalemia.

**3. BP control**
   a. Strict control decreases the rate of disease progression.
   b. ACE inhibitors are the preferred agents. Multiple drugs, including diuretics, may be required.
   c. The target is < 140/90, then if proteinuria the target will be < 130/80.

**4. Glycemic control** (if the patient is diabetic) prevents worsening of proteinuria.

**5. Smoking cessation** has been associated with slower rates of progression.

**6. Correction of electrolyte abnormalities**
   a. Correct hyperphosphatemia with calcium citrate (a phosphate binder).
   b. Patients with chronic renal disease are generally treated with long-term oral calcium and vitamin D in an effort to prevent secondary hyperparathyroidism and uremic osteodystrophy.
   c. Acidosis—treat the underlying cause (renal failure). Patients may require oral bicarbonate replacement.

**7. Anemia**—treat with **erythropoietin**, which kidneys normally secrete (**10-11 g/dl**).

**8. Pulmonary edema—**arrange for dialysis if the condition is unresponsive to diuresis.

**9. Pruritus**—try capsaicin cream or cholestyramine and UV light.

**10. Dialysis** (See indications in the Dialysis section.)

**11. Transplantation is the only cure**.

## Renal replacement therapy (Dialysis & Transplant)

- Dialysis is the **artificial mechanism by which fluid and toxic solutes are removed from the circulation when the kidneys cannot do so sufficiently**.
- In all forms of dialysis, the blood interfaces with an **artificial solution resembling human plasma (called the dialysate)**, and diffusion of fluid and solutes occurs across **a semipermeable membrane**.
- The two major methods of dialyzing a patient are **hemodialysis and peritoneal dialysis**.

### Absolute Indications for Dialysis:

1. **Acidosis** - significant, intractable metabolic acidosis.
2. **Electrolytes** - severe, persistent hyperkalemia.
3. **Intoxications** - methanol, ethylene glycol, lithium, aspirin.
4. **Overload** - hypervolemia not managed by other means.
5. **Uremia** (**severe**) - based on clinical presentation, not laboratory values (e.g., uremic pericarditis or encephalopathy are absolute indications for dialysis).

## Hemodialysis

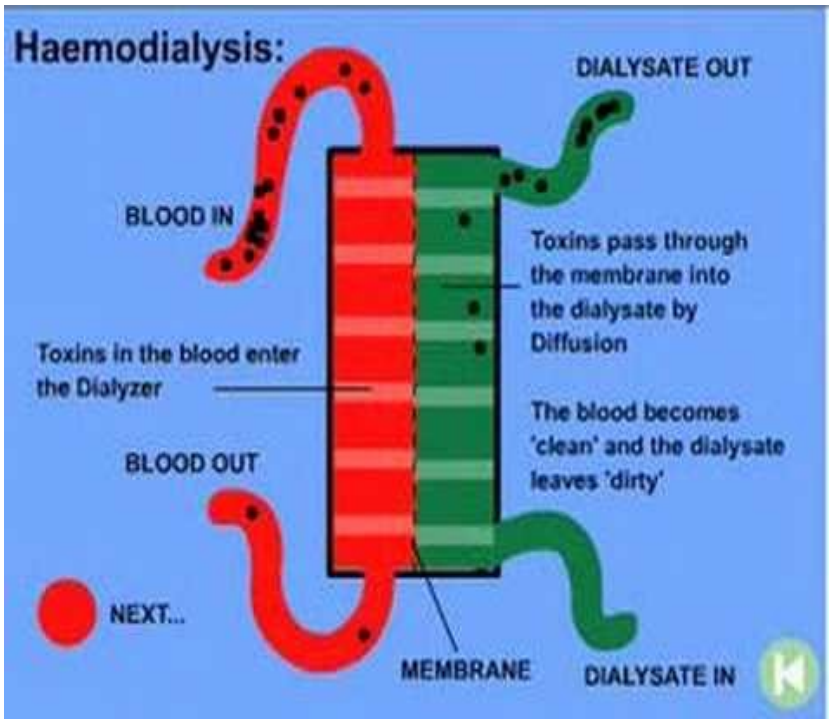
**1. Process:**

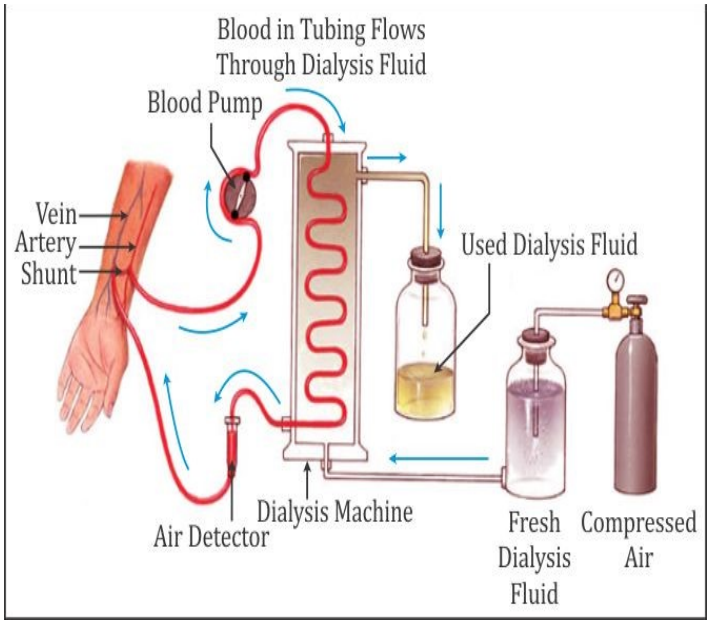
a. The patient's **blood is pumped by an artificial pump outside** of the body **through the dialyzer**, which typically consists **of fine capillary networks of semipermeable membranes**. The **dialysate flows on the outside of these networks, and fluid and solutes diffuse across the membrane**.

b. The **patient's blood must be heparinized to prevent clotting** in the dialyzer.

**2. Frequency:** Most hemodialysis patients **require 3 to 5 hours of dialysis 3 days per week.**

**3. Access:** e.g. Arteriovenous fistula (Check for **thrills and audible bruit**).






**5. Advantages of hemodialysis.**

a. It is **more efficient** than peritoneal dialysis. High flow rates and efficient dialyzers **shorten the period** of time required for dialysis.

b. It can **be initiated more quickly** than peritoneal dialysis, using temporary vascular access in the **emergent setting**.

**6. Disadvantages of hemodialysis.**

a. It is **less similar** to the physiology of **natural kidney function** than is peritoneal dialysis, predisposing the patient to the following:

1. **Hypotension** due to rapid removal of intravascular volume leading to rapid fluid shifts from the extravascular space into cells.
2. **Hypo-osmolality** due to solute removal.

b. **Requires vascular access**.

## Peritoneal Dialysis

**1. Process**

a. The **peritoneum serves as the dialysis membrane**. Dialysate **fluid is infused into the peritoneal cavity**, then fluids and solutes from the peritoneal capillaries diffuse into the dialysate fluid, which is drained from the abdomen.

b. **A hyperosmolar (high-glucose) solution is used**, and water is removed from the blood via **osmosis**.

**2. Frequency:** dialysate **fluid is drained and replaced every hour in acute peritoneal dialysis**, but **only once every 4 to 8 hours in CAPD**.

**3. Access:** dialysate is infused into the peritoneal fluid via an implanted catheter.

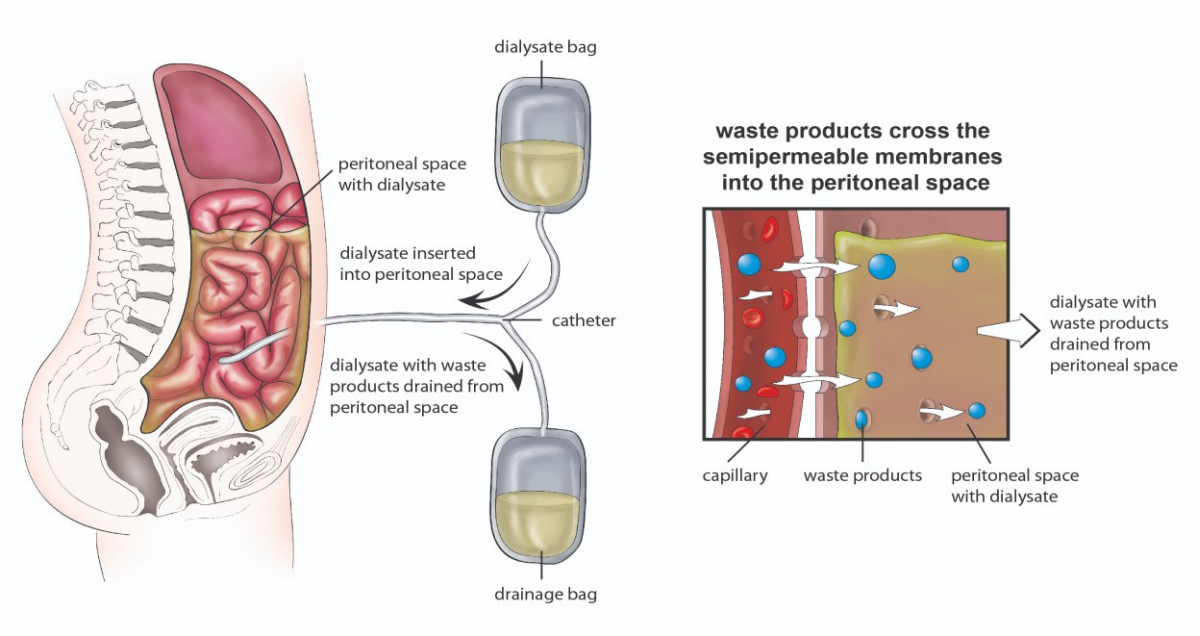
**4. Advantages:**

a. The patient can learn to **perform dialysis on his or her own**.

b. It **mimics the physiology of normal kidney function** more closely than hemodialysis in that it is more continuous.

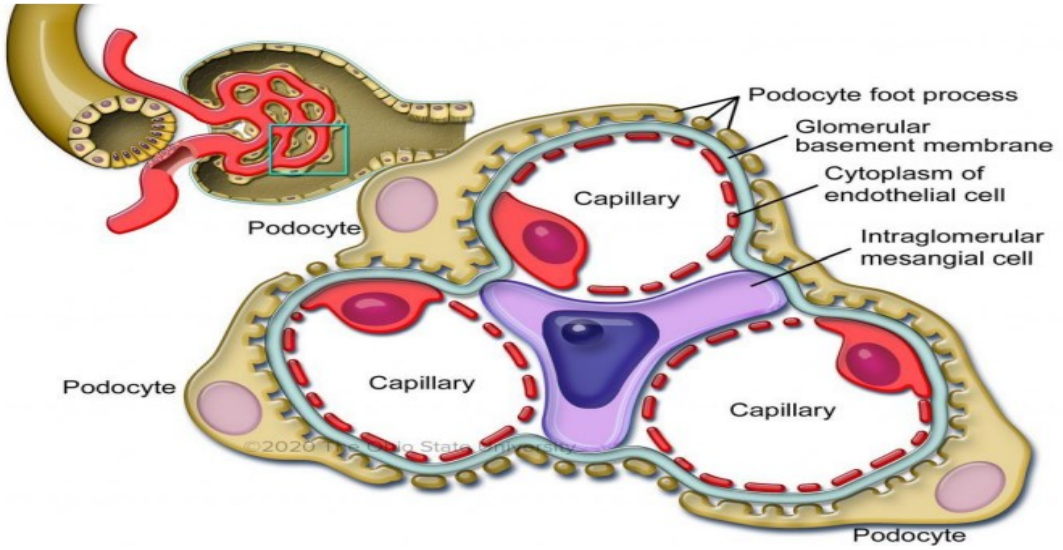
**5. Disadvantages:**

a. **High glucose load may lead to hyperglycemia and hypertriglyceridemia**.
b. **Peritonitis** is a significant potential complication.
c. The **patients must be highly motivated to self-administer it**.
d. **Cosmetic** —there is increased abdominal girth due to dialysate fluid.



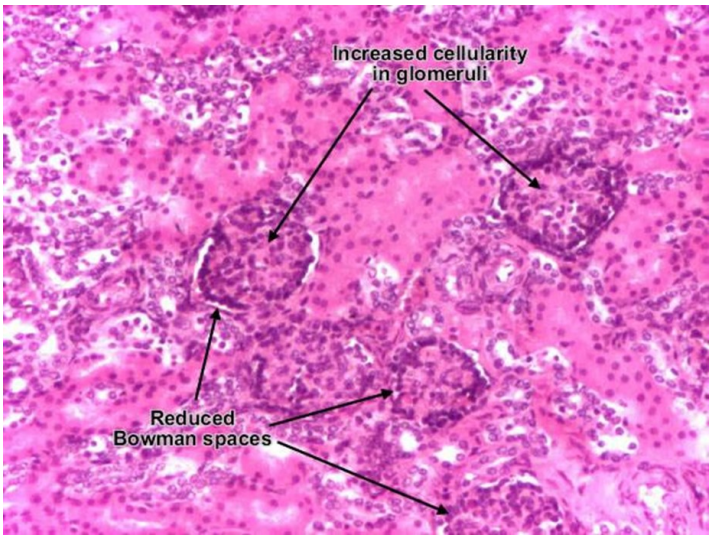
# Nephrotic & Nephritic Syndrome

## Anatomy




## Nephritic (Pathophysiology)

• Glomerular disease characterized **by glomerular inflammation and bleeding.**
• It is usually due to **the involvement of GBM**. (Glomerulonephritis)
• Biopsy reveals **hyper-cellular inflamed glomeruli** ©.

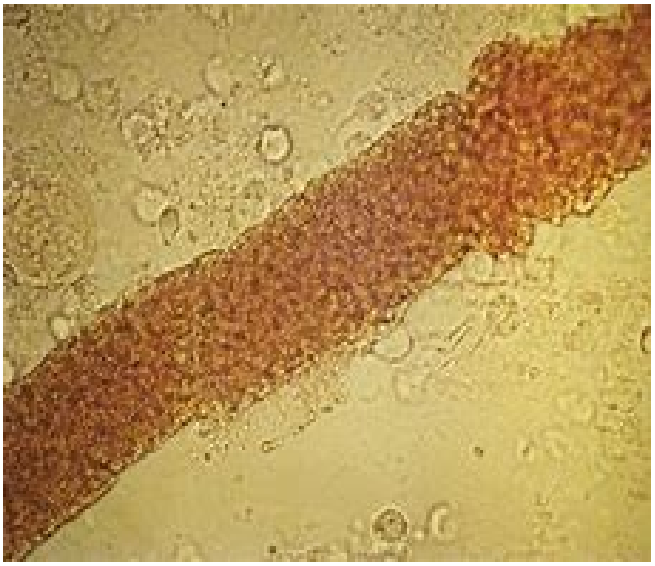



## Nephritic (Etiology)

- **Post streptococcal GN**.
- Crescentic GN.
- Lupus Nephritis.
- **IgA Nephropathy.**
- **Hereditary nephritis**.

## Nephritic (Clinical presentation)

- Characterized by:
  1. Limited proteinuria <3.5 gm/d.
  2. Oliguria and azotemia = elevated BUN/SCr.
  3. Salt retention with periorbital edema and hypertension.
  4. RBCS cast and dysmorphic RBC'S in urine.



## Post-Strep GN

- Occurs ~ **2 weeks after group A strep infection of skin or respiratory tract**.
- **More common in children.**
- Diagnosis:
  1. **ASO increases.**
  2. **Decrease complement.**
  3. LM: G enlarged/ hypercellular.
  4. EM: **Subepithelail hump**.
  5. Management**: Self-limited.**

## IgA Nephropathy (Berger Disease)

1. **Asymptomatic recurrent hematuria/mild proteinuria is common**. This is the **most common cause of glomerular hematuria**. Gross hematuria **1 to 3 days after an upper respiratory infection** (or other viral infection or exercise) is common (as opposed to weeks after a URTI in PSGN).

2. **Renal function is usually normal**.

3. Mesangial deposition of IgA and C3 are seen on electron microscopy.

4. The **prognosis in most patients is good** with preservation of renal function (renal insufficiency may develop in 25%).

5. Some **advocate steroids for unstable disease**, but no therapy has been proven to be effective.

## Hereditary Nephritis (Alport syndrome)

1. **X-linked or autosomal dominant inheritance** with variable penetrance.
2. Features include **hematuria, pyuria, proteinuria**, **high-frequency hearing loss without deafness.**
3. No effective treatment.
4. **Progressive renal failure.**

## Nephrotic syndrome

- Nephrotic syndrome: **Defined by protein excretion > 3.5 g/day**, due to a variety of glomerular process that allow for leakage of protein through GBM and into urine.

- **Etiology:**
  1. **Primary causes:** (Minimal changer disease, Focal segmental glomerulosclerosis, Membranous nephropathy, Membrenoproliferative).
  2. **Secondary causes:** (Amyloidosis, Diabetic nephropathy).

- **Management:**
  - Treat underlying disease
  - Proteinuria: ACEi/ ARB
  - Edema: Loop diuretics.
  - Hyperlipidemia: Statin.

## Nephrotic syndrome (Primary)

- **Minimal change disease**: Most common in **children—Hodgkin disease and non-Hodgkin lymphoma** have been associated with MCD.
- **Focal Segmental Glomerulosclerosis**: This accounts for 25% of cases of nephrotic syndrome in adults and is more common in **blacks**. It may be primary but it **may be associated with (HIV, Sickle cell, Heroin).**
- **Membranous Glomerulonephritis**: Primary disease is idiopathic (this is the most common etiology). The secondary form is due **to infection (hepatitis C virus, hepatitis B virus, syphilis, malaria), drugs (gold, captopril, penicillamine), neoplasm, or lupus**.

## Nephrotic syndrome (Secondary)

**Diabetic nephropathy:**

- **Most common cause of nephropathy in adults**.
- Leading cause of **ESRD** in US.
- **30% with type 1 DM**, **20% with type 2 DM** develop diabetic nephropathy.
- Non enzymatic glycosylation affects the efferent more than afferent lead to high GFR.
- **Initially starts as microalbuminuria** due to hyprefiltration followed by heavy proteinuria due to sclerosis of mesangium and decline in renal function.
- Diagnosis is made **on clinical grounds** (unless there is no retinopathy).
- Treatment: **ACEI/ARBS to slow down the progression**.

## TABLE 7-5 Nephritic Versus Nephrotic Syndrome

| | Nephritic Syndrome | Nephrotic Syndrome |
|---|---|---|
| **Pathogenesis** | Inflammation of glomeruli due to any of the causes of glomerulonephritis | Abnormal glomerular permeability due to a number of conditions |
| **Causes** | Poststreptococcal glomerulonephritis is the most common cause, but may be due to any of the causes of glomerulonephritis | Many conditions. Membranous glomerulonephritis is the most common cause in adults. Other causes include diabetes, SLE, drugs, infection, glomerulonephritis (focal segmental and others)<br>Minimal change disease is the most common cause in children |
| **Laboratory Findings** | Hematuria<br>AKI—azotemia, oliguria<br>Proteinuria, if present, is mild and not in nephrotic range | Urine protein excretion rate >3.5 g/24 hr<br>Hypoalbuminemia<br>Hyperlipidemia, fatty casts in urine |
| **Clinical Findings** | HTN<br>Edema | Edema<br>Hypercoagulable state<br>Increased risk of infection |

# Hypo/Hyperkalemia

## Causes

a. Acute or chronic kidney disease.
b. Medications.
c. Disorders that impair renin-angiotensin axis.
d. ↑ Potassium movement out of cells (eg, uncontrolled hyperglycemia, metabolic acidosis).
e. ↑ Tissue catabolism (eg, trauma, tissue lysis syndrome).
f. Pseudohyperkalemia (eg, hemolyzed blood sample).

**MEDICATION-INDUCED HYPERKALEMIA**

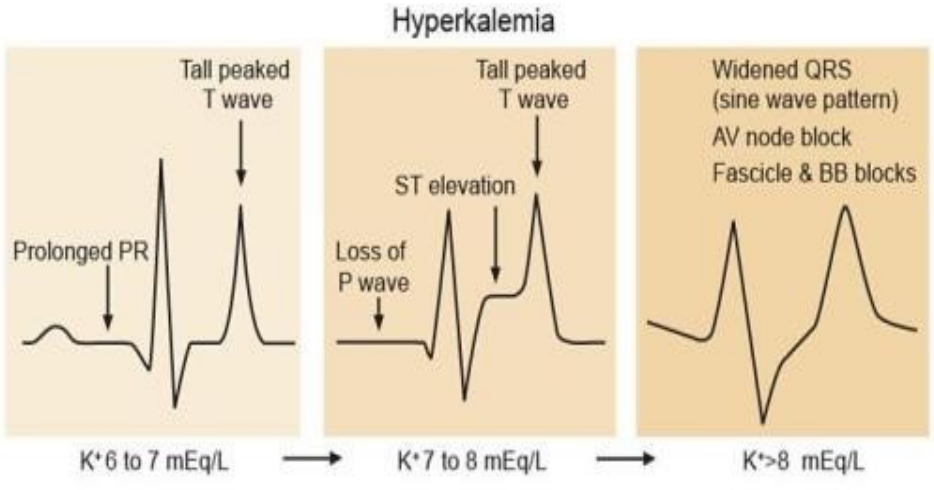
| Medications that can cause hyperkalemia | |
|---|---|
| **Medication** | **Mechanism** |
| Nonselective beta-adrenergic blockers | Inhibit beta-2–mediated intracellular potassium uptake |
| ACE inhibitors | Inhibit angiotensin II formation, leading to decreased aldosterone secretion |
| ARBs | Inhibit $AT_1$ receptor, leading to decreased aldosterone secretion |
| $K^+$-sparing diuretics | Inhibit ENaC or aldosterone receptor |
| Cardiac glycosides (eg, digoxin) | Inhibit the $Na^+/K^+$-ATPase pump |
| NSAIDs | Inhibit local prostaglandin synthesis, leading to decreased renin & aldosterone secretion |

## Clinical features

a. Ascending muscle weakness with flaccid paralysis

b. ECG changes (in order of presentation):
   i. Peaked T waves
   ii. Short QT interval
   iii. QRS widening
   iv. Sine wave with ventricular fibrillation

Patients with chronic hyperkalemia may be asymptomatic until potassium gradually rises ≥7 mEq/L
a. Acute hyperkalemia may become symptomatic at lower levels




## Management

a. Initial evaluation with ECG to evaluate for conduction abnormalities

b. Acute therapy (eg, calcium gluconate [or calcium chloride], insulin with glucose) is given in following cases:
   i. ECG changes
   ii. Potassium ≥7 mEq/L without ECG changes
   iii. Rapidly rising potassium due to tissue breakdown

c. Dialysis

i. It should be reserved for patients with renal failure and those with severe life threatening hyperkalemia unresponsive to initial therapy

d. If patient does not require acute therapy, the next step is to exclude acute treatable secondary causes (eg, uncontrolled hyperglycemia [>300 mg/dL], tumor lysis syndrome)

i. Patients should have a review of recent/current medications

| Clinical features of hyperkalemia | |
|---|---|
| **Sequence of ECG changes** | • Tall **peaked T waves** with shortened QT interval<br>• **PR prolongation & QRS widening**<br>• Disappearance of P wave<br>• Conduction blocks, ectopy, or **sine wave** pattern |
| **Cardiac membrane stabilization** | • **Calcium** infusion |
| **Rapidly acting treatment options** | • Insulin with glucose<br>• Beta-2 adrenergic agonists<br>• Sodium bicarbonate |
| **Removal of potassium from the body (slow-acting)** | • Diuretics<br>• Cation exchange resins<br>• Hemodialysis |

## Hyperkalemia

- **Causes:**

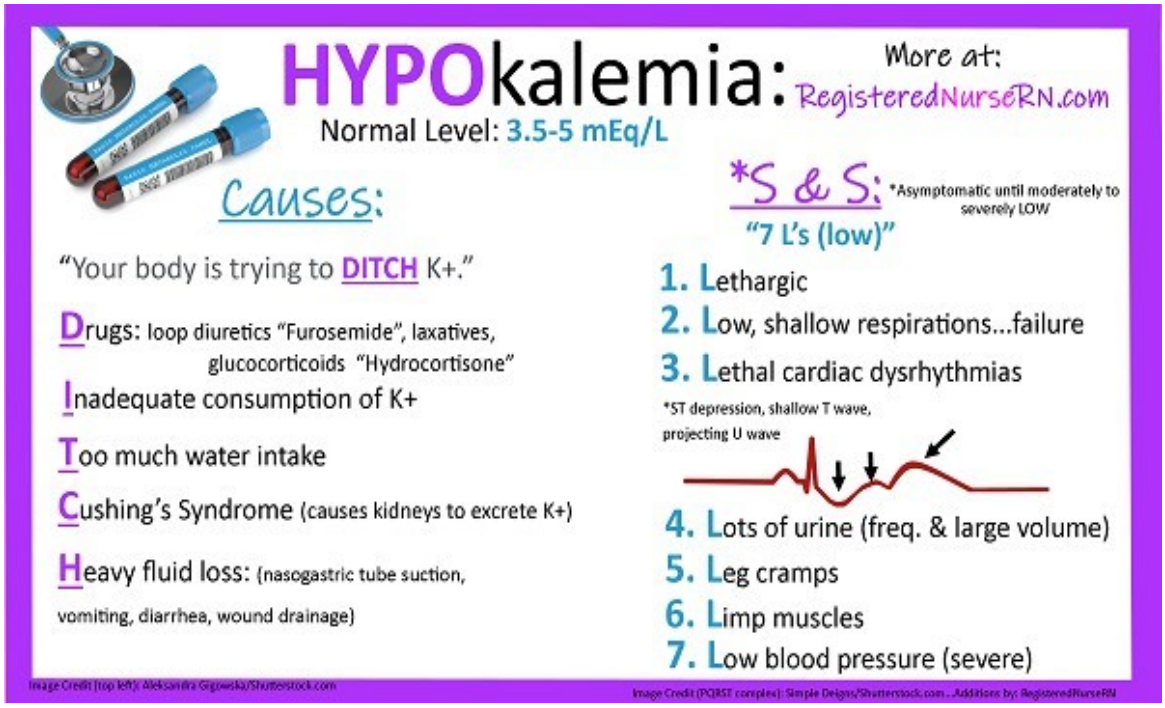
**a. ↑ potassium entry into cells**

1- This may be due to beta-2 adrenergic agents stimulating sodium- potassium ATPase pump

2- beta-2 adrenergic stimulated release of insulin

**b. Renal potassium wasting**

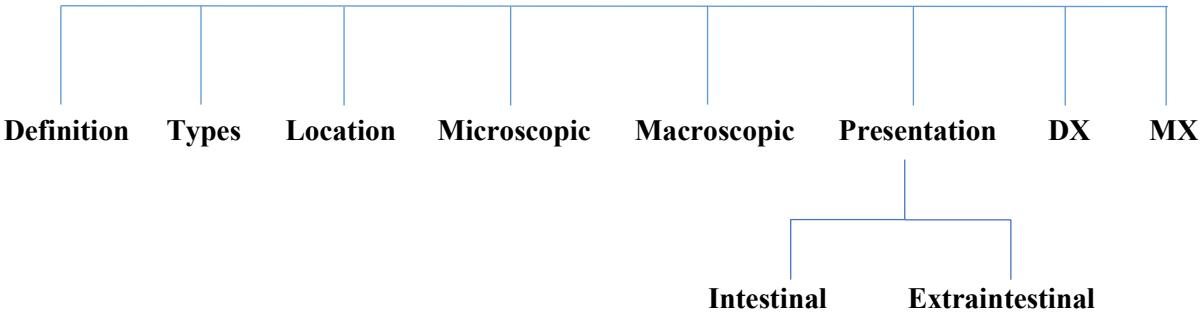
**c. Gastrointestinal fluid loss**



# Chapter 4

# Gastrointestinal System

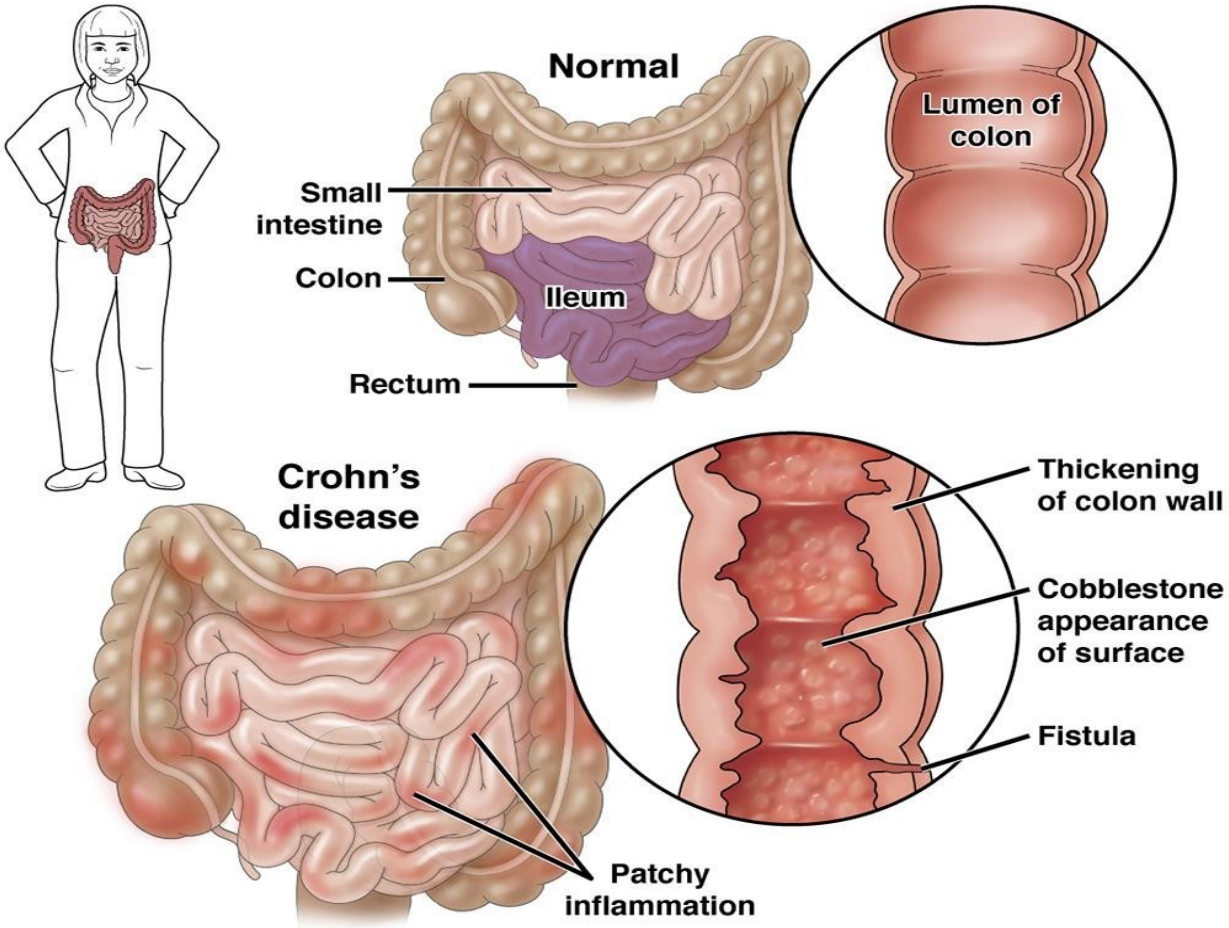
# Inflammatory Bowel Disease




## Introduction

**Crohn's disease and ulcerative colitis are often collectively referred to asinflammatory bowel disease (IBD).**

**The cause of IBD is unknown, although genetic predisposition and immune hyperreactivity to an unknown antigen are thought to play an important role.**



## Crohn’s disease

**Crohn's disease is a chronic inflammatory condition of the gut that caninvolve any part of the GI tract from the mouth to the anus**

**It is most classically a disease of the small bowel, with the terminalileum is one of the most common locations**

**Non caseating granulomas (accumulation of epithelioid macrophages without central necrosis) and an inflammatory infiltrate that involves alllayers of the intestinal wall (transmural) are characteristic of Crohn's disease.**

**The affected bowel appears hyperemic and edematous on macroscopicexamination. Mucosa of the involved area contains linear ulcers.**

**Normal-looking mucosal areas intervene between the areas involved •**

**in pathologic process, leading to the classic "cobblestoneappearance”.**

## Crohn’s disease-presentation

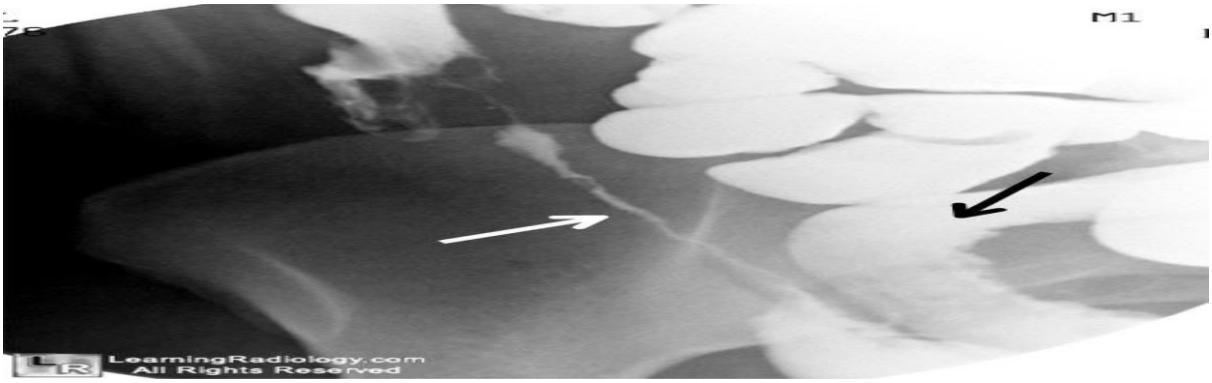
**Typically, the disease presents insidiously over the course of years,marked by bouts of abdominal pain, diarrhea (bloody if colitis), constitutional symptoms**

**The most common presentation of Crohn's disease is abdominal pain,which represents transmural inflammation.**

**Transmural inflammation explains the most common complications ofCrohn's disease: strictures, abscesses, and fistulas.**

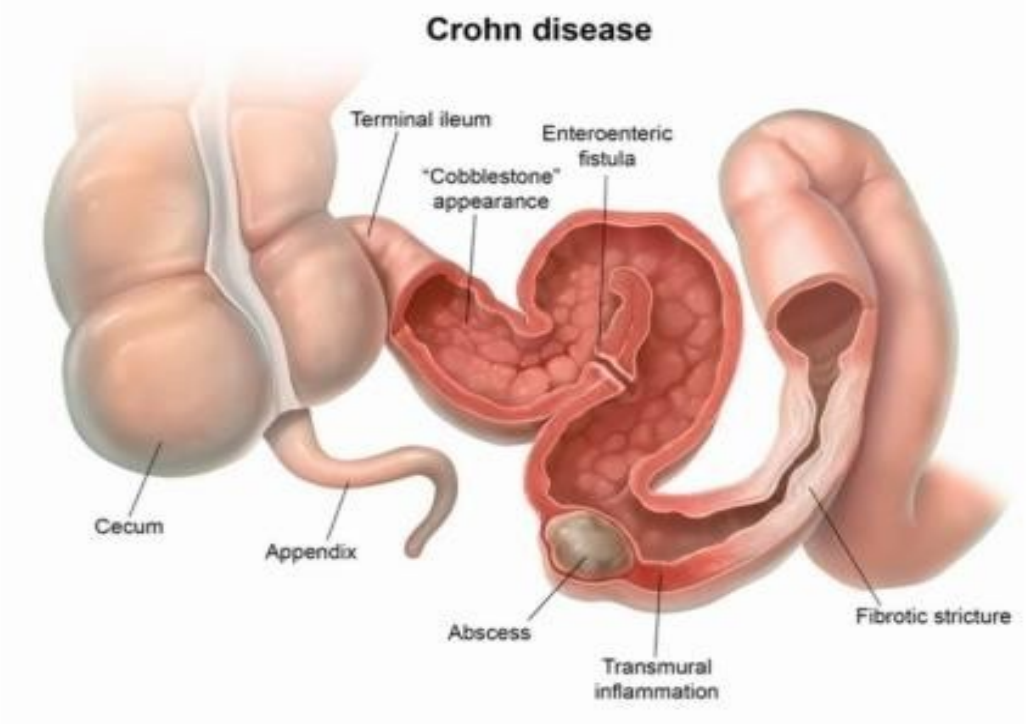
**The disease may progress to intestinal obstruction resulting from fibrotic narrowing of the intestinal lumen (as a result of bowel wall edema, fibrosis, and hypertrophy of the muscularis mucosae), requiring surgical bowel resection and placement of an ostomy (contrast barium studies may show the"string sign").**

**Fistulas occur when ulcers penetrate the entire thickness of the intestinalwall, leading to a sinus tract that communicates between multiple organs(enterovesicular, enterovaginal, enteroenteric)**



- Normally, the mucosa of the terminal ileum plays an important role in "recycling" bile acids that are necessary for the absorption of fat. Bile acids are produced in the liver, excreted with bile, and then reachthe terminal ileum.

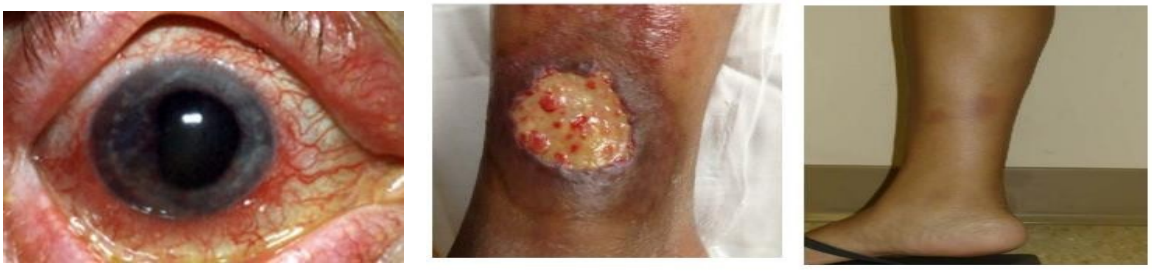
- When the mucosa of terminal ileum is inflamed (as in Crohn's disease), bile acids are not reabsorbed, becoming lost with feces. As aresult, a lesser amount of bile acid is present in bile, and the ratio of cholesterol/bile acids increases → Cholesterol precipitates in bile of the gallbladder and forms gallstones.

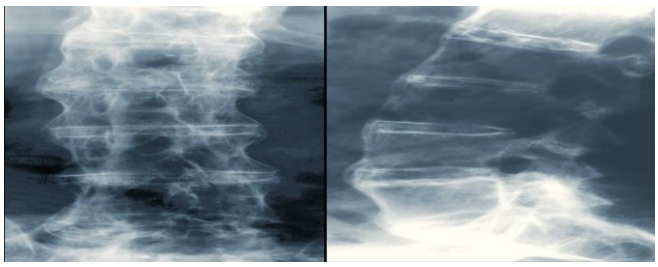



**There are also a number of extraintestinal manifestations that are eitherimmune-mediated or occur due to deficient absorption of nutrients.**

**The most important complications of Crohn's disease are as follows:**

- Skin: pyoderma gangrenosum (more common with ulcerative colitis), erythemanodosum
- Joints: arthritis, ankylosing spondylitis.
- Eyes: iritis, uveitis, episcleritis





**Malabsorption: oxalate kidney stones, anemia, hypoproteinemia, B12and folate deficiencies , AKED VITAMINES**

**Liver: primary sclerosing cholangitis (more common with ulcerativecolitis),increased risk of cholangiocarcinoma**

| | Crohn disease |
|---|---|
| Involvement | • Anywhere mouth to anus (mostly ileum & colon)<br>• Perianal disease with rectal sparing<br>• Skip lesions |
| Microscopy | • Noncaseating granulomas |
| Gross findings | • Transmural inflammation<br>• Linear mucosal ulcerations<br>• Cobblestoning, creeping fat |
| Clinical manifestations | • Abdominal pain (often RLQ)<br>• Watery diarrhea (bloody if colitis) |
| Intestinal complications | • Fistulas, abscesses<br>• Strictures (bowel obstruction) |

## Ulcerative colitis

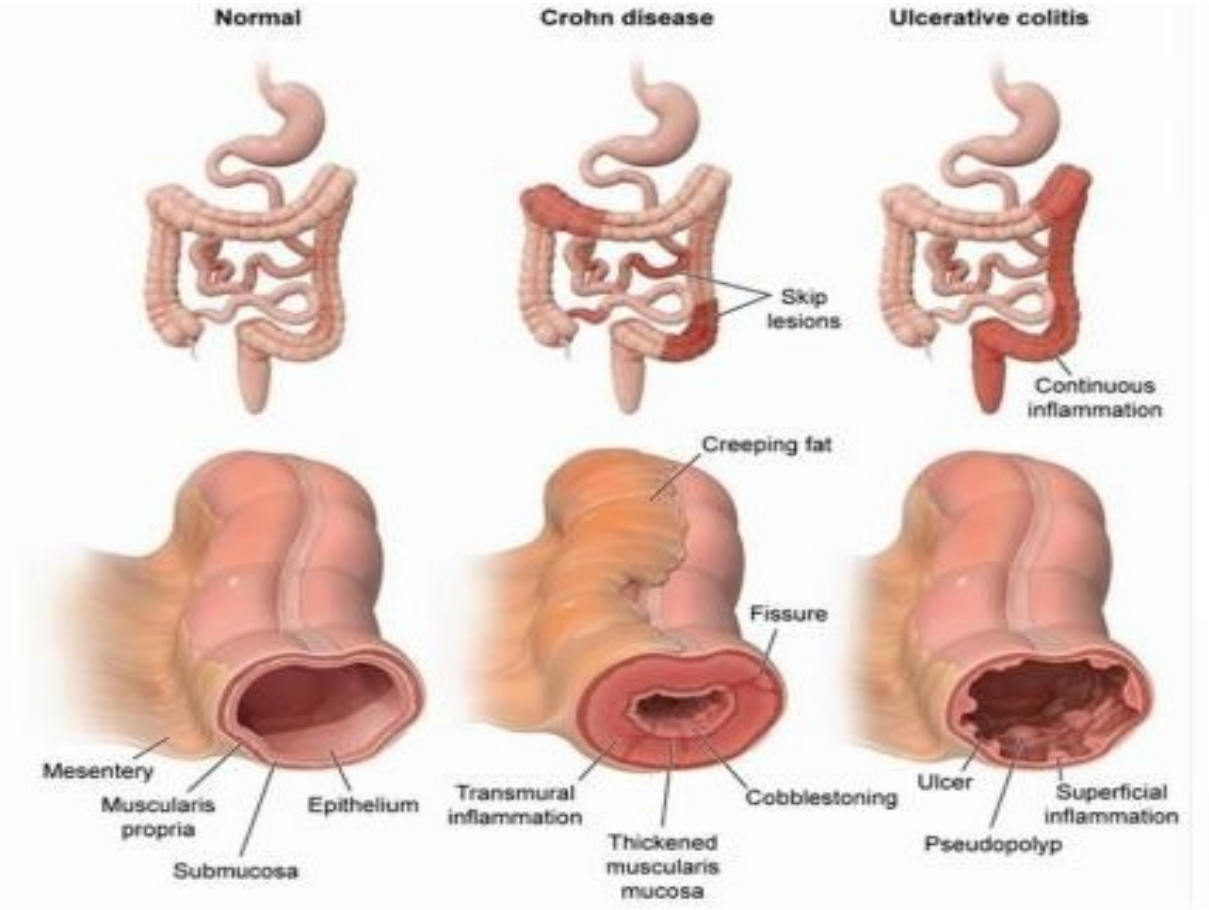
**Ulcerative colitis has the following unique characteristics:**

- **The rectum is always involved**; involvement of other areas of the intestine isvariable
- Inflammation **is limited to the mucosa and submucosa** only, so strictures andfistulas are not common.
- **Mucosal damage is continuous.** There are no areas of normal mucosabetween the affected segments.
- **Bloody diarrhea,** with or without abdominal pain, is the **hallmark of ulcerativecolitis** (In Crohn's disease, there may also be bloody diarrhea, but abdominal pain is virtually always present).

**Ulcerative colitis has a number of complications:**

**The most dangerous is toxic megacolon (severe dilatation of thebowel), which can lead to perforation.**

**Ulcerative colitis significantly increases the risk of adenocarcinoma of the colon**




**When should screening occur?**

❖ After 8 to 10 years of colonic involvement, with colonoscopy every 1to 2 years.

**Diagnostic Tests**

❖ Endoscopy is the most accurate test

## Treatment

**Acute exacerbations of disease are treated with steroids in both CD and UC.**

**Chronic maintenance of remission is with 5-ASA derivatives such as mesalamine:Asacol (mesalamine) is used for UC and Pentasa (mesalamine) for CD.**

**Rowasa (mesalamine) is for UC largely limited to the rectum.**

**Steroids used are prednisone or budesonide. Budesonide is a steroid specific forIBD. First pass effect is good for IBD treatment**

| | Crohn disease | Ulcerative colitis |
|---|---|---|
| **Involvement** | • Anywhere mouth to anus (mostly ileum & colon)<br>• Perianal disease with rectal sparing<br>• Skip lesions | • Rectum (always) & colon<br>• Continuous lesions |
| **Microscopy** | • Noncaseating granulomas | • No granulomas |
| **Gross findings** | • Transmural inflammation<br>• Linear mucosal ulcerations<br>• Cobblestoning, creeping fat | • Mucosal & submucosal inflammation<br>• Pseudopolyps |
| **Clinical manifestations** | • Abdominal pain (often RLQ)<br>• Watery diarrhea (bloody if colitis) | • Abdominal pain (varying locations)<br>• Bloody diarrhea |
| **Intestinal complications** | • Fistulas, abscesses<br>• Strictures (bowel obstruction) | • Toxic megacolon |

# Irritable Bowel Syndrome

## Irritable bowel syndrome (IBS)

Is a pain syndrome that can have diarrhea, constipation, or both .IBS is further **sub-classified** as:

1. Diarrhea-predominant.
2. Constipation-predominant.
3. Mixed.

- Most common in **middle-aged women.**
- The cause of IBS is **unknown**.
- Affects up to **15 ~ 20%** adults in the industrialized world.

## ROME criteria

**Recurrent abdominal pain 1 ≤day/week for past 3 months associated with 2 ≤ of the following:**

- Related to **defecation (**improves or worsen).
- Change in **stool frequency**.
- Change in form **(consistency)** of stool.

## Diagnosis

- IBS was previously considered a diagnosis of exclusion.
- However, patients with IBS symptoms based on the ROME criteria, no alarm features, and no family history of inflammatory bowel disease or colorectal cancer **do not require extensive workup.**
- Colonoscopy performed on IBS patients typically shows **normal colonic mucosa.**

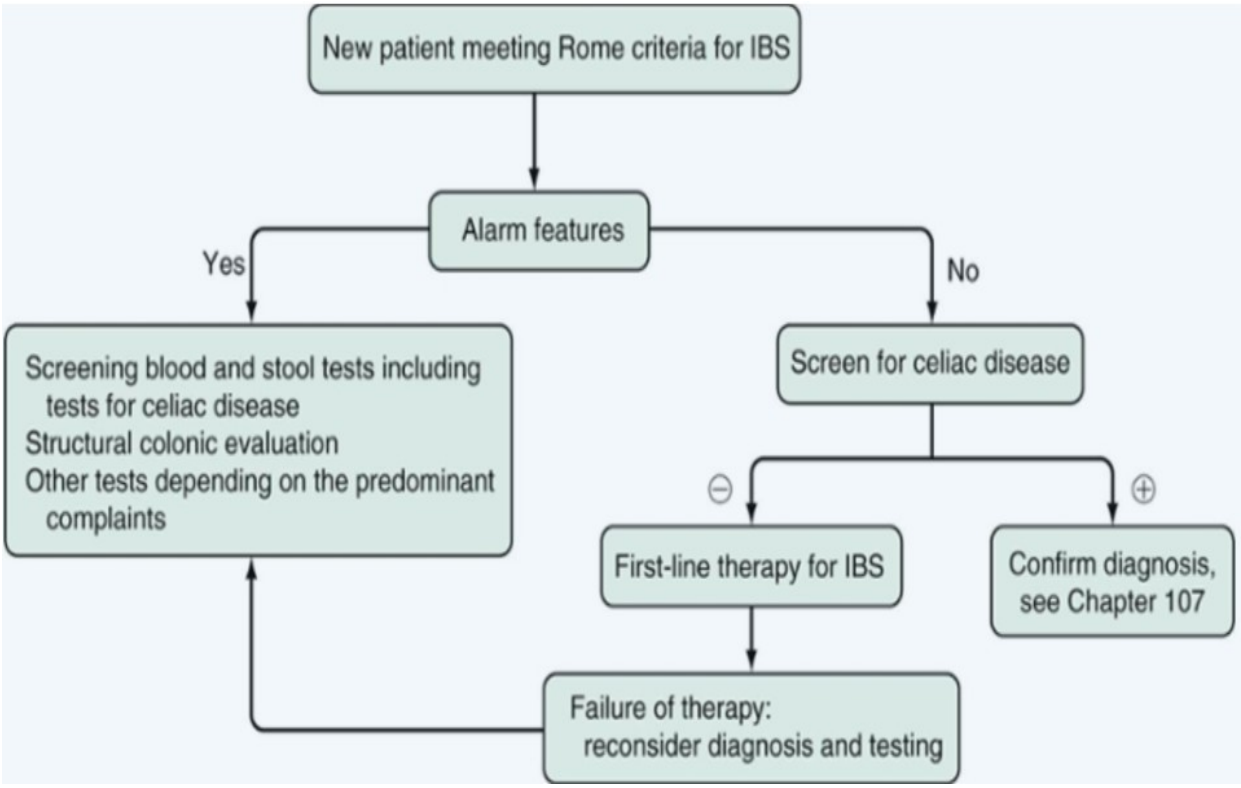
## Warning signs/symptoms

- Older age of onset >50 years.
- Upper GI bleeding.
- **Nocturnal** diarrhea.
- Worsening pain.
- **IDA**.
- Elevated **CRP**.
- Positive fecal lactoferrin & calprotectin.
- **Family hx** of early colon cancer or IBD

These require further investigation to **rule out** other etiologies.

## Supporting diagnostic features in IBS

- Symptoms **> 6 months**.
- Frequent consultation for non-GI problems.
- Previous medically **unexplained** symptoms.
- **Stress** worsen symptoms.




## Treatment

- 1st line treatment is **lifestyle modification** and **dietary changes** (High-fiber diet to increase bulk of the stool).
- **Hyoscyamine** or **dicyclomine** for abdominal pain (alternatively, TCAs or SSRI).
- Additional therapy for **diarrhea-predominant** IBS:
  - Rifaximin: non-absorbed antibiotic with modest effect in diarrhea-predominant IBS.
  - Eluxadoline: mu-opioid receptor agonist for diarrhea IBS; relieves pain/slows bowel.

Additional therapy for **constipation-predominant** IBS:
- Fiber.
- Polyethylene glycol (PEG): Osmotic laxative.
- Lubiprostone: use if PEG doesn't work.
- Linaclotide: use if PEG doesn't work.

# Peptic Ulcer Disease (PUD)

• **What is it?**
Gastric and duodenal ulcers.

• **What is the incidence in the United States?**
≈10% of the population will suffer from PUD during their lifetime!.

• **What are the possible consequences of PUD?**
Pain, hemorrhage, perforation, obstruction.

• **Which bacteria are associated with PUD?**
Helicobacter pylori.

## Gastric ulcer

- **Etiology**: **H.Pylori (80%),** NSAID, Smoking, ETOH, ZE= Gastrinoma MC In pancreas, duodenum, antrum (multiple ulcers+ Distal to duodenum, recurrent, resistant to treatment) may be associated with MEN-1.
- **Risk for gastric cancer (adenocarcinoma),** so we need biopsy esp. if ugly.
- **Most commonly in the lesser curvature.**
- **CLX: Epigastric pain** (gnawing or burning, worse with eat), **N, Early satiety, Dyspepsia, weight loss.**
- **DX: EGD+ BX,** Barium meal, **test for H.pylori** (BX with urease and culture/ stool antigen assay/ urea breath test).
- **TRX:** Lifestyle modification, Treat H.pylori (ABX), use of PPI.

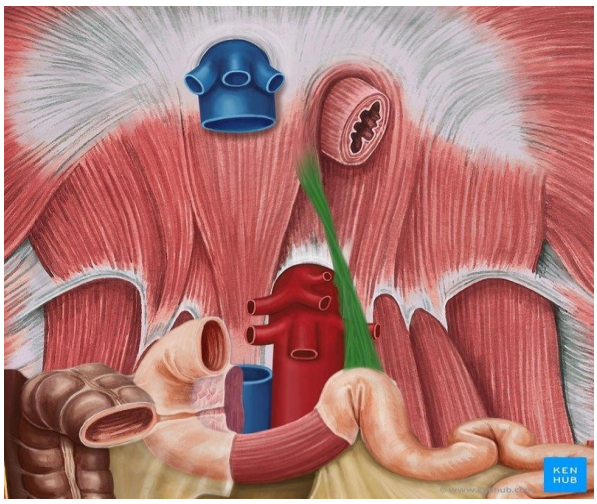
## Duodenal ulcer

- **Etiology: H.Pylori (95%), NSAID**, smoking, ZE= Gastrinoma.
- **No Risk for cancer.**
- **CLX:** Epigastric pain (gnawing or burning, **worse several hours after meal),** N, **weight gain.**
- **DX:** EGD+ BX, Barium meal, test for H.pylori.
- **TRX:** Lifestyle modification, Treat H.pylori, use of PPI (95% successful).
- **Surgery indications =** complications (Hemorrhage (posterior from gastrodeudenal artery), obstruction (Anterior), perforation).
- **Indications of surgery in Hemorrhage:**
  1. Bleeding > 6 units.
  2. Recurrent bleeding after endoscopic control.
- **Pyloro-deudenotomy and control the bleed.**
- **If perforated do (Grahams patch = closure by omentum).**

# UGIB

## Etiology

- **Definition:** Bleeding into the lumen of the proximal GI tract, proximal to the ligament of Treitz.



- **The common differential diagnosis of UGI bleeding?**
  1. PUD (Most common cause of significant UGI).
  2. Acute gastritis.
  3. Esophageal varices.
  4. Gastric ulcer.
  5. Mallory–Weiss tear.
  6. Tumor.

## Presentation (History and physical)

- **What are the signs/symptoms?**
  **Hematemesis, melena,** syncope, shock, fatigue, **coffee- ground emesis**, hematochezia, epigastric discomfort, epigastric tenderness, signs of hypovolemia, guaiac-positive stools.

- **Why is it possible to have hematochezia?**
  Blood is a cathartic, and hematochezia usually **indicates a vigorous rate of bleeding from the UGI source.**

- **History:**
- Use of: **NSAIDs, aspirin, anticoagulants, antiplatelet agents.**
- **Alcohol abuse,** previous GI bleed, **liver disease, coagulopathy.**

- **Physical:**
- **Tachycardia; orthostatic blood pressure changes suggest moderate to severe blood loss;** (hypotension may be late finding **hypotension suggests life-threatening blood loss** n healthy younger adult)
- **Rectal examination** is performed to assess stool color (melena versus hematochezia versus brown)
- Significant **abdominal tenderness** accompanied by signs of peritoneal irritation (eg, **involuntary guarding**) suggests perforation.

## Approach (Laboratory)

- **Which lab tests should be performed?**
  Chem-7, bilirubin, **LFTs, CBC, type and cross, PT/PTT,** amylase.

- **Why is BUN elevated?**
  Because of absorption of blood by the GI tract.

- **What about nasogastric lavage?**
  • It may be helpful if the source of bleeding is unclear or to clean the stomach before endoscopy.

## Approach

- **ABC:**
  1. **Airway patency.**
  2. **Breathing:** Provide a supplemental O2 if O2 sat < 94%.
  3. **Circulation.**

**What is the initial treatment?**

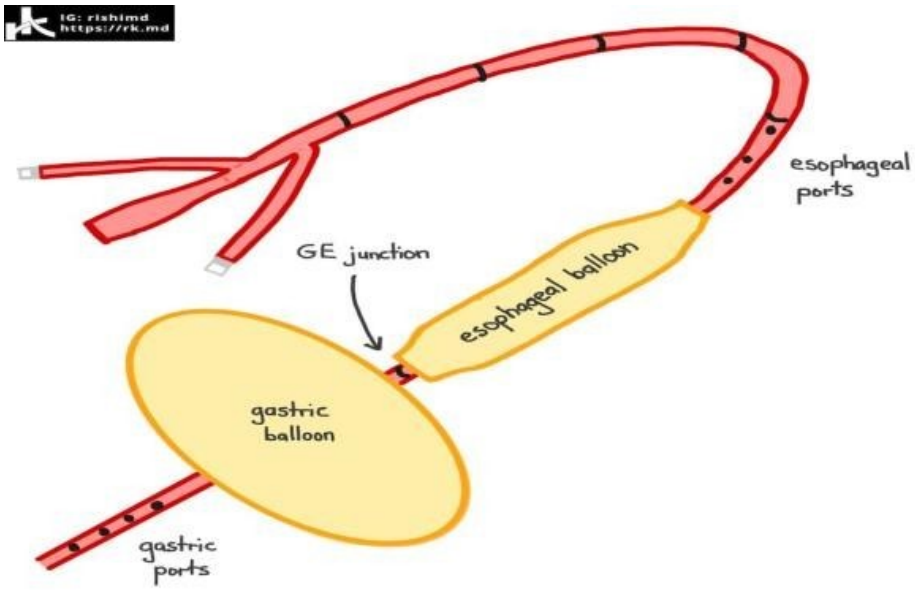
1. **IVFs** (16 G or larger peripheral IVS × 2), **Foley** catheter (monitor fluid status).
2. **NGT** suction (determine rate and amount of blood).
3. **EGD:** endoscopy (determine etiology/location of bleeding and possible treatment—coagulate bleeders).

- **Treat hypotension initially with rapid bolus isotonic crystalloid (e.g. 500-1000 NS) use smaller bolus if patient has compromise cardiac function.**
- **Transfusion:**
- **For severe, ongoing bleeding, immediately transfuse blood products in 1:1:1 ration of RBCs, plasma, and platelets, as for trauma patients.**
- **For hemodynamic instability despite crystalloid resuscitation, transfuse 1 to 2 units RBCs.**
- **For hemoglobin < 8 g/dL (80 g/L) in high-risk patients (eg, older adult, coronary artery disease), transfuse 1 unit RBCs and reassess the patient's clinical condition**
- **For hemoglobin < 7 g/dL (70 g/L) in low-risk patients, transfuse 1 units RBCs and reassess the patient's clinical condition**
- **Avoid over-transfusion with possible variceal bleeding**
- **Give plasma for coagulopathy or after transfusing four units of RBCs; give platelets for thrombocytopenia (platelets <50,000) or platelet dysfunction (eg, chronic aspirin therapy) or after transfusing four units of RBCs.**
- **Obtain immediate consultation with gastroenterologist; obtain surgical and interventional radiology consultation for any large-scale bleeding.**

- **What is the diagnostic test of choice with UGI bleeding?**
  EGD (>95% diagnosis rate).
- **What are the treatment options with the endoscope during an EGD?**
  Coagulation, injection of epinephrine (for vasoconstriction), injection of sclerosing agents (varices), variceal ligation (banding).

## Approach (Drugs)

- **Pharmacotherapy for all patients with suspected or known severe bleeding:**
- **Give a proton pump inhibitor:**
  1. **Evidence of active bleeding (eg, hematemesis, hemodynamic instability), give esomeprazole or pantoprazole, 80 mg IV**
  2. **No evidence of active bleeding, give esomeprazole or pantoprazole, 40 mg IV**
- **Endoscopy delayed beyond 12 hours, give second dose of esomeprazole or pantoprazole, 40 mg IV.**




- **Pharmacotherapy for known or suspected esophagogastric variceal bleeding and/or cirrhosis:**
- **Give somatostatin or an analogue (eg, octreotide 50 mcg IV bolus followed by 50 mcg/hour continuous IV infusion).**
- **Give an IV antibiotic (eg, ceftriaxone or fluoroquinolone.)**
- **Balloon tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using any of several devices (eg, Sengstaken-Blakemore tube, Minnesota tube); tracheal intubation is necessary if such a device is to be placed; ensure proper device placement prior to inflation to avoid esophageal rupture**

## Treatment

- **What are the indications for surgical intervention in UGI bleeding?**
  Refractory or recurrent bleeding and site known, >3 unit PRBCs to stabilize or >6 unit PRBCs overall.
- **What percentage of patients require surgery?**
  ≈10%.
- **What percentage of patients spontaneously stop bleeding?**
  ≈80% to 85%.

## Prognosis

- **What is the mortality of acute UGI bleeding?**
  Overall 10%, age 60 to 80 years 15%, age >80 years 25%.

- **What are the risk factors for death following UGI bleed?**
  1. Age >60 years.
  2. Shock.
  3. >5 units of PRBC transfusion.
  4. Concomitant health problems.

## Case scenario

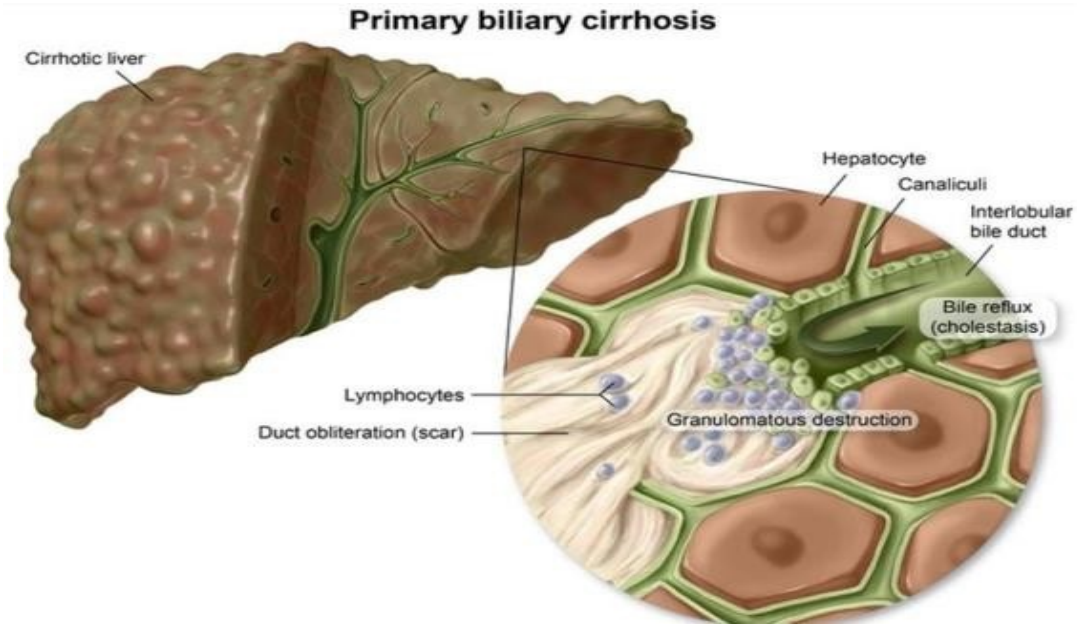
- **A 68-year old male** presents to the ED with **complaints of fatigue, stomach pain, and vomiting for 3 days.** The patient reports that his **emesis appears brown and granular, similar to coffee grounds, and his stool is black and tarry.** He has a past medical history of **arthritis, GERD**, and hypertension.

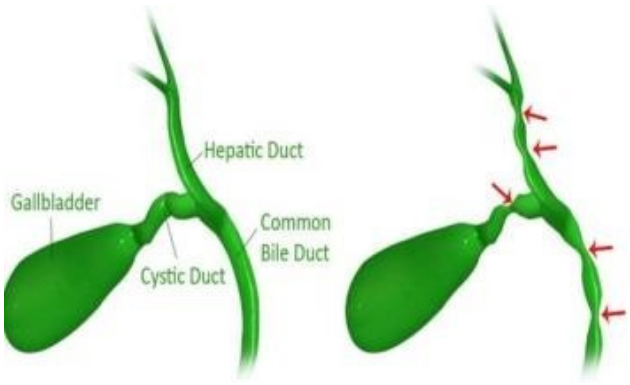
# Liver Cirrhosis

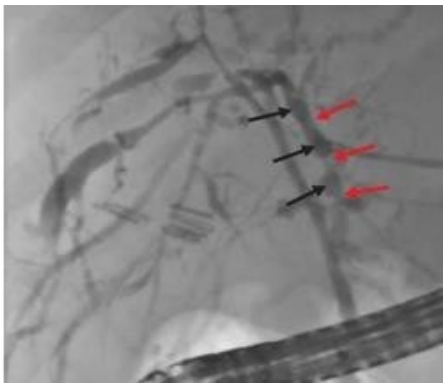
- Chronic liver disease characterized by **hepatic fibrosis,** distortion of **architecture,** and formation of **regenerative nodules.**
- The regenerative capacity of the liver is enormous; however, over a long time, fibrosis will develop.
- When **at least 70-80% of liver** function has been lost, the synthetic capacity of the liver is diminished.

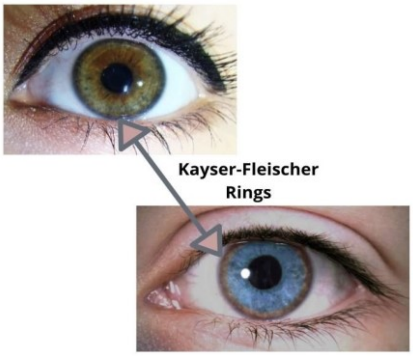
## Etiology

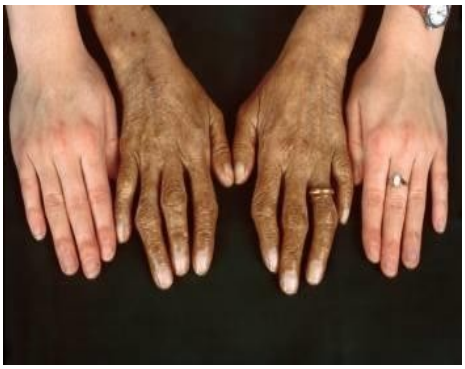
- **Alcohol** abuse.
- **Hepatitis C.**
- Non-alcoholic fatty liver disease **(NAFLD).**
- **Other** causes:
  - Primary biliary cirrhosis
  - Sclerosing cholangitis
  - Hemochromatosis, and Wilson disease.

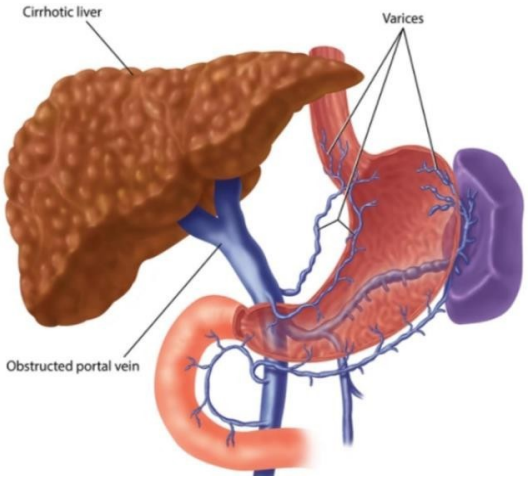


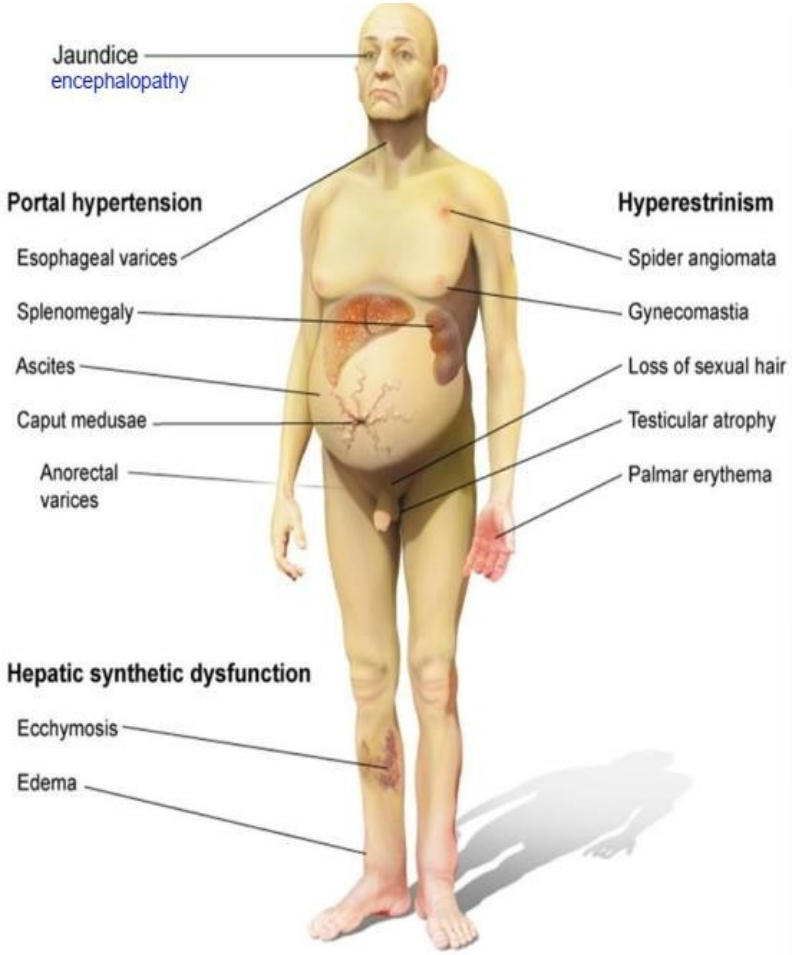




Kayser-Fleischer
Rings




Cirrhotic liver
Varices
Obstructed portal vein


Jaundice
encephalopathy
Portal hypertension
Esophageal varices
Splenomegaly
Ascites
Caput medusae
Anorectal
varices
Hyperestrinism
Spider angiomata
Gynecomastia
Loss of sexual hair
Testicular atrophy
Palmar erythema
Hepatic synthetic dysfunction
Ecchymosis
Edema

## Clinical picture

- **Nonspecific:** Anorexia, weight loss, fatigue.
- **Portal HTN:** Ascites, HSM, variceal bleeding.
- **Neurologic:** Hepatic encephalopathy, asterixis.
- **Skin:** Jaundice, palmar erythema, spider angioma, terry nails.
- **Heme:** Anemia, coagulopathy, thrombocytopenia.
- **Reproductive:** Testicular atrophy, gynecomastia.
- **Poor synthetic fx:** decrease albumin, Billuribin

## Esophageal varices

- In advanced disease, **portal blood** has an increasingly difficult time passing through the liver because the vasculature becomes compromised by the progressive **fibrosis**, causing portal hypertension.

| Esophagus | Risk of bleeding | Lt Gastric << Azygous |
|---|---|---|
| Rectum | Anorectal varices | Sup. >> Inf. Rectal |
| Umbilicus | Caput Medusae | Paraumbilical >> epigastric |

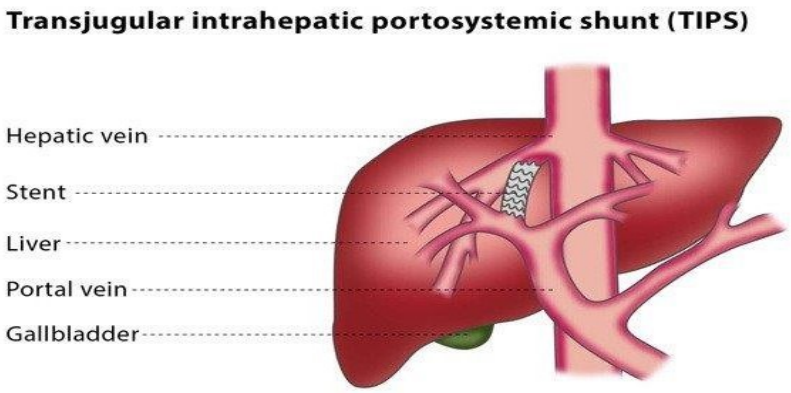
## Management

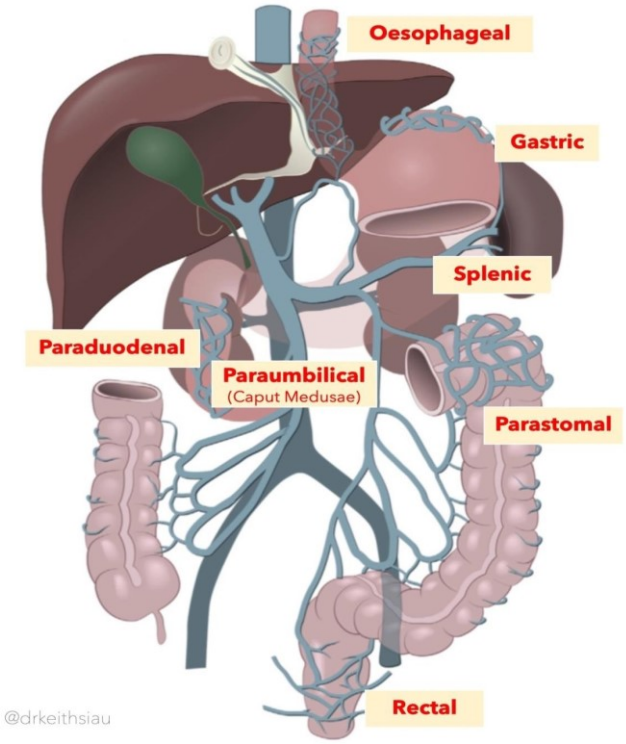
**Acute:**

- Resuscitation with **IVF**, **Antibiotic**, **Octreotide**.
- Urgent upper endoscopy (ligation or sclerotherapy).
- **TIPS** or surgery if refractory.

**Chronic:**

- Screening for varices (upper endoscopy).
- Beta-blocker (**Nadolol**) >> prophylaxis for lower risk.
- **Ligation** >> prophylaxis for higher risk.






## Ascites

Accumulation of fluid in the peritoneal cavity due to **portal HTN** (hydrostatic pressure) and **hypoalbuminemia** (oncotic pressure) leading to **abdominal distention and shifting dullness.**

**SAAG = (serum albumin) - (albumin level of ascitic fluid)**

1. When SAAG ≥ 1.1 and total protein < 2.5 g/dL, cirrhosis.
2. When SAAG ≥ 1.1 and total protein > 2.5 g/dL, heart failure.
3. When SAAG < 1.1 and total protein < 2.5 g/dL, there is nephrotic syndrome.
4. WhenSAAG < 1.1 and total protein > 2.5 g/dL, there is carcinoma.

## Management of Ascites

- Salt restriction.
- Combination (Furosemide – **Spironolactone**) therapy
- For tense ascites: large volume paracentesis.

## Spontaneous bacterial peritonitis

- An ascitic fluid infection **without** an obvious intraabdominal **surgical** etiology.
- SBP is most likely due to either **intestinal bacterial** translocation directly into the ascitic fluid or hematogenous spread to the liver and ascitic fluid.
- **E-coli** is the most common organism others organisims include Klebsiella, Strep, Staph.
- **Clinically:** Abdominal pain, **fever**, altered mental status

**Diagnosis: Paracentesis**

1. PMN **> 250/mm3**
2. SAAG >1.1
3. Positive ascites culture/ gram stain

**Management:**

– 3rd generation cephalosporin
– Albumin
– Prophylaxis **(Hx of SPB or Current GI bleeding)** by Ciproflxoacin or TMP-SMX

**TABLE 3-1 Child–Pugh Classification to Assess Severity of Liver Disease**

| | POINTS | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| Ascites | None | Mild–moderate | Severe |
| Bilirubin(mg/dL) | <2.0 | 2.0–3.0 | >3.0 |
| Encephalopathy | None | Mild–moderate | Severe |
| INR ratio | <1.7 | 1.7–2.3 | >2.3 |
| Albumin | >3.5 | 2.8–3.5 | <2.8 |

Class A—5 to 6 points total (least severe liver disease), 85% 2-year survival
Class B—7 to 9 points total (moderate–severe liver disease), 57% 2-year survival
Class C—10 to 15 points total (severe liver disease), 35% 2-year survival

## Management

1. Monitor & treat the **underlying cause to slow progression of disease** (Treat hepatitis, alcohol abstinence).
2. Treating the **complications.**
3. Monitor for **HCC (AFP+ US q6 months).**
4. Consider **liver transplantation.**

# Hepatitis & Serology

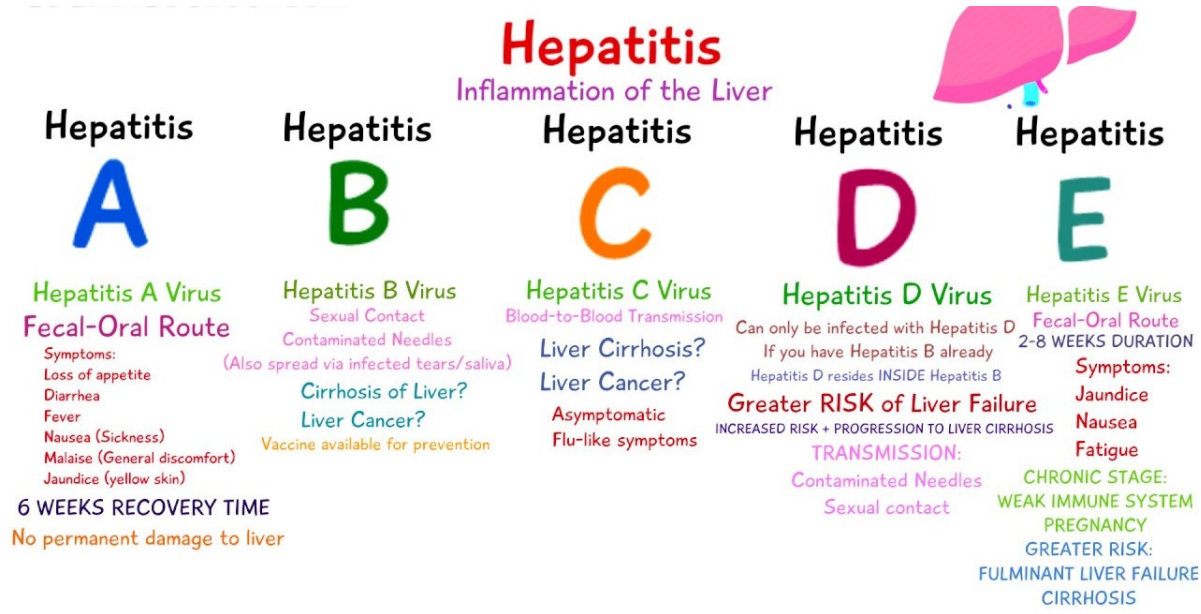
## Hepatitis

• **Definition**
Is an inflammation of the liver that is caused by a variety of **infectious viruses** and **noninfectious agents** leading to a range of health problems, some of which can be fatal .

– **Noninfectious causes of hepatitis:**
1- Heavy alcohol use.
2- Certain medications.
3- Toxins.
4- Autoimmune diseases
5- Non-alcoholic steatohepatitis (NASH).

## Viral Hepatitis

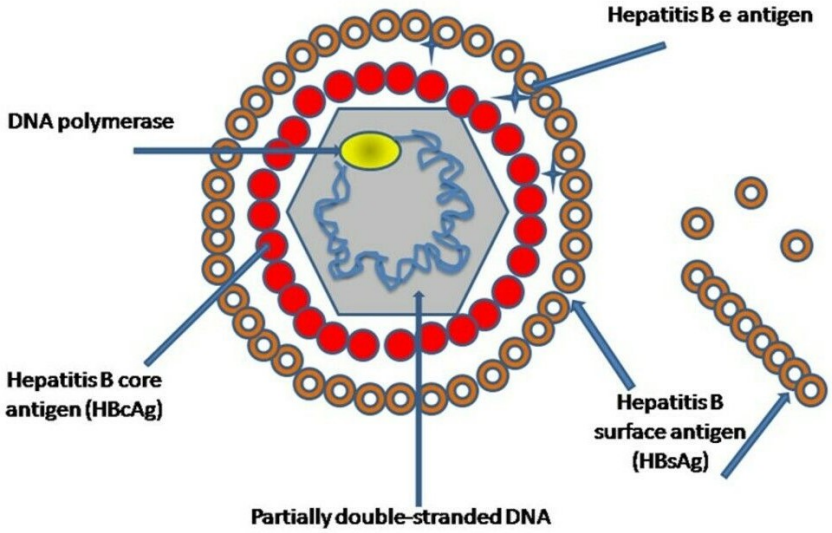
- **Hepatitis A and E are mainly spread by contaminated food and water (Fecal oral route).**
- **Hepatitis B is mainly sexually transmitted, but may also be passed from mother to baby during pregnancy or childbirth and spread through infected blood.**
- **Hepatitis C is commonly spread through infected blood such as may occur during needle sharing by intravenous drug users.**
- **Hepatitis D can only infect people already infected with hepatitis B**
- **Hepatitis A, B, and D are preventable with immunization.**
- **All Hepatitis Viruses are RNA Viruses except Hepatitis B.**



## Hepatitis B

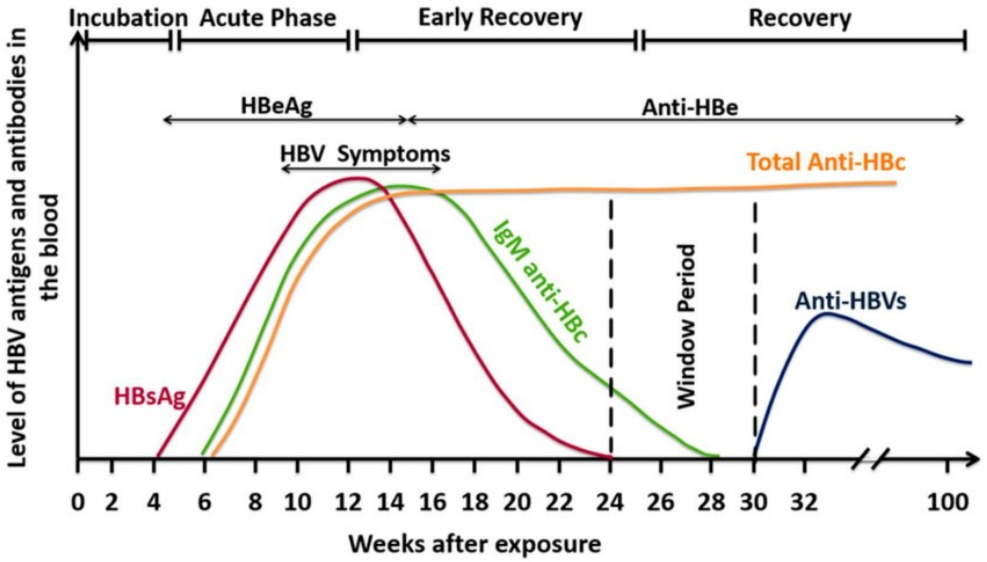
- Is an infectious disease caused by the hepatitis B virus (HBV) that affects the liver, it is a type of viral hepatitis. It can cause both **acute** and **chronic** infection.
- **Hepatitis B virus (HBV)** is a partially **double-stranded DNA virus**, a member of the **Hepadnaviridae** family of viruses.

### Hepatitis B virus




## Serology

### Hepatitis B Serology



# Interpretation of Tests for Acute Hepatitis B

| Anti-HBc IgM | Anti-HBc IgG | HBsAg | Anti-HBs | Interpretation |
|---|---|---|---|---|
| Positive | Negative | Positive | Negative | Acute HBV infection |
| Negative | Negative | Positive | Negative | Early acute HBV infection |
| Negative | Positive | Negative | Positive | Resolved acute HBV infection |
| Negative | Negative | Negative | Positive | Not infected<br>Prior vaccination for HBV |
| Negative | Negative | Negative | Negative | Not infected |
| Negative | Positive | Positive | Negative | Chronic HBV infection |

## Question

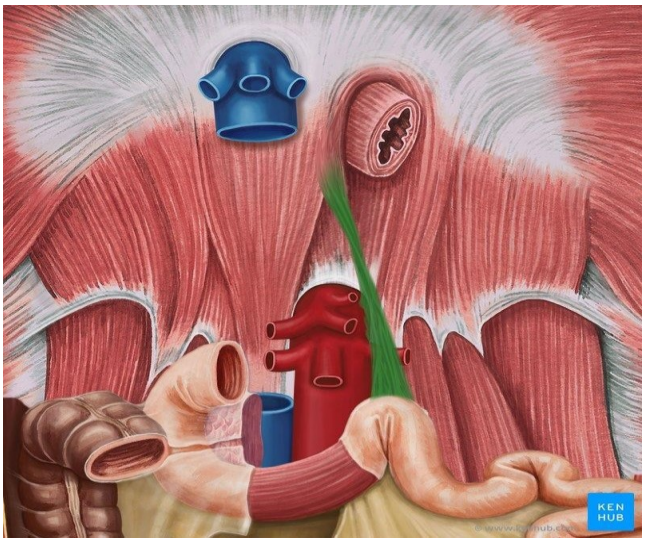
| | HBsAg | Anti-HBs | Anti-HBc |
|---|---|---|---|
| 1 | Negative | Positive | Positive |
| 2 | Positive | Negative | IgM Positive |

# LGIB

## Definition

• **Definition:** Bleeding into the lumen of the proximal GI tract, distal to the ligament of Treitz.

• **The common differential diagnosis of UGI bleeding?**
1. Anorectal disease.
2. Diverticulosis.
3. Colon cancer.
4. Vascular ectasia.
5. IBD.
6. Drug side effect (Aspirin, Clopidogrel, warfarin).




## Presentation

• **What are the symptoms?**
**Hematochezia (bright red blood per rectum [BRBPR])**, with or without abdominal pain, melena, anorexia, fatigue, syncope, shortness of breath, shock.

• **What are the signs?**
**BRBPR, positive hemoccult, abdominal tenderness, hypovolemic shock, orthostasis.**

• **Blood originating from the left colon tends to be bright red in color, whereas bleeding from the right colon usually appears dark or maroon colored and may be mixed with stool. Rarely, bleeding from the right side of the colon will present with melena.**

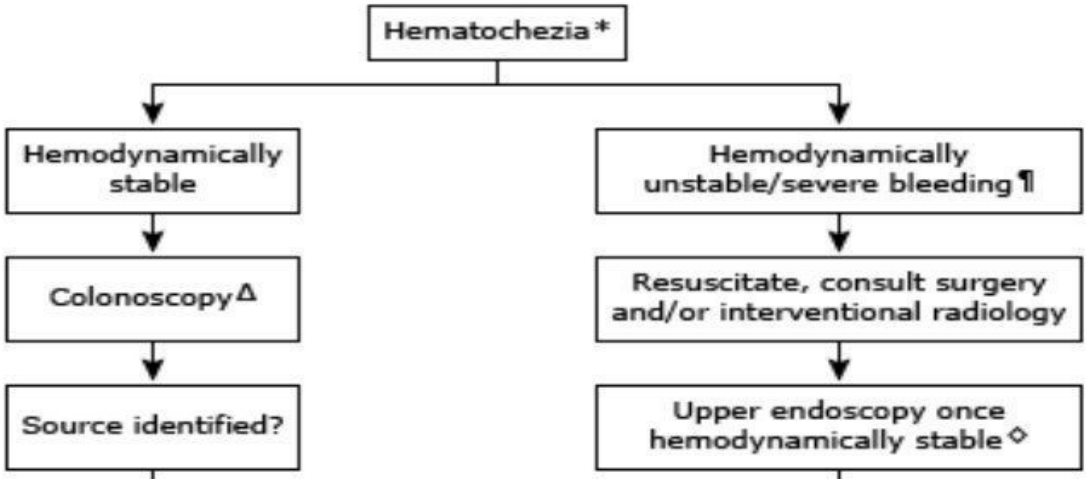
## Approach (Laboratory)

• **Which lab tests should be performed?**
Chem-7, bilirubin, LFTs, CBC, type and cross, PT/PTT.

• **Why is BUN elevated?**
Because of absorption of blood by the GI tract.

## Initial evaluation

- **The initial evaluation includes a history, physical examination, laboratory tests, and in some cases, upper endoscopy (algorithm 1).**
- **The goal of the evaluation is to assess the severity of the bleed, assess whether the bleeding may be coming from the upper GI tract, and determine if there are conditions present that may affect subsequent management.**




## Approach (Resuscitation)

- **ABC:**
  1. **Airway patency.**
  2. **Breathing:Provide a supplemental O2 if O2 sat< 94%.**
  3. **Circulation.**

- **What is the initial treatment?**
  1. **IVFs** (16 G or larger peripheral IVS × 2), **Foley** catheter (monitor fluid status).
  2. **NGT** suction (to rule out UGI bleeding; bile or blood must be seen; otherwise, perform EGD ).
  3. **Stop** any drugs could be the possible cause of bleeding.
  4. **Anoscopy/proctoscopic exam.**

## Approach (Diagnosis)

- **What must be ruled out in patients with lower GI bleeding? Upper GI bleeding!**

- **How to identify the source of bleeding in each of the following?**
  1. Mild to moderate = **Colonoscopy**.
  2. Severe = **A-gram.**
  3. Slow intermittent = **Radiolabeled RBC'S scan.**

## Treatment

- **What is the treatment if bleeding site is KNOWN and massive or recurrent lower GI bleeding continues?**
  **Segmental resection of the bowel**

- **What is the surgical treatment of massive lower GI bleeding WITHOUT localization?**
  **Exploratory laparotomy** with intraoperative enteroscopy and total abdominal colectomy as last resort.

- **What percentage of patients require surgery?**
  ≈10%.

- **What percentage of patients spontaneously stop bleeding?**
  ≈80% to 85%.

- **Does melena always signify active colonic bleeding?**
  **NO** - the colon is **very good at storing material and often will store melena/maroon stools and pass them days later** (follow patient, UO, HCT, and vital signs).

- **What is the therapeutic advantage of doing a colonoscopy?**
  Options of **injecting substance (epinephrine) or coagulating vessels** is an advantage with C-scope to control bleeding.

- **What is the therapeutic advantage of doing an A-gram?**
  Ability to **inject vasopressin** and/or **embolization**, with at least temporary control of bleeding in >85%.

## Case scenario

- A **50-year old male** presents to the ED with **complaints of fatigue, dizziness, weight loss**. The patient reports that his **stool is mixed with blood**. He has a past medical history of hypertension, he is a heavy smoker.

# Chapter 5

# HLS

# Anemia

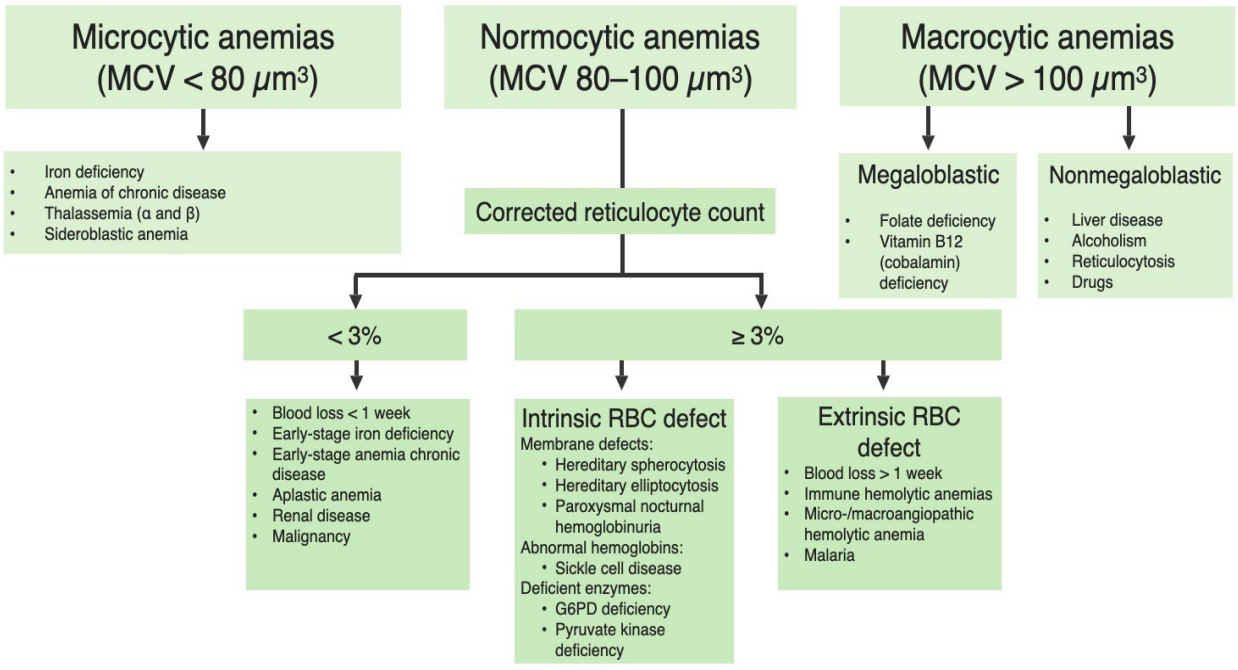
- **Anemia is a condition marked by the following :**
  - Hematocrit **<41%** in men or **<36%** in women.
  - Hemoglobin **<13.5 g/dl** in men or **<12 g/dl** in women**.**
    - Symptoms of anemia are generally based not on the etiology, but on the severity of disease .
    - **If Hematocrit:**
      1. >30%-35% → **No Sx.**
      2. 25%-30% → **Dyspnea (worse on exertion), fatigue.**
      3. 20%-25% → **Lightheadedness, angina.**
      4. Under 20%–25% → **Syncope, chest pain.**

## Diagnostic Tests

- Complete blood count (CBC) **is the best initial test** in the evaluation of anemia.
- If there is **low hematocrit or hemoglobin,** the first step is to determine the **MCV** .
- **Iron** studies, **Reticulocyte count, peripheral smear, red cell distribution width (RDW), Coombs test, vitamin B12, folate level**, and even a possible **bone marrow biopsy** may be necessary to determine **a specific etiology**.

## Treatment

- If anemia is **severe**, it is treated with **packed red blood cells**. Answering the question "At what hematocrit do I transfuse a patient?" depends on **the following factors :**
  - Is the patient **Symptomatic**? Then transfuse .
  - Is the **hematocrit very low** "means 25 to 30" **in an elderly patient** or one with heart disease? Then transfuse.

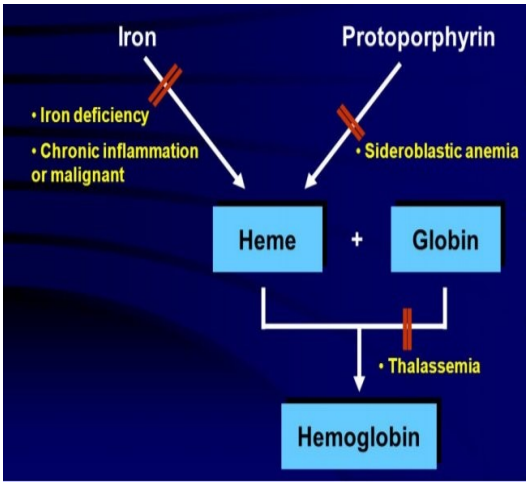


## Microcytic anemia

- **Definition/Etiology:-**

Microcytosis refers specifically to an MCV that is lower than normal, which is usually **below 80 fL.**

- **Hemoglobin** is made of **heme** and **globin**:
  heme is composed of **iron** and **protoporphyrin** .
  The most common causes are :
  1. Iron deficiency.
  2. Anemia of chronic disease.
  3. Thalassemia.
  4. Sideroblastic anemia.



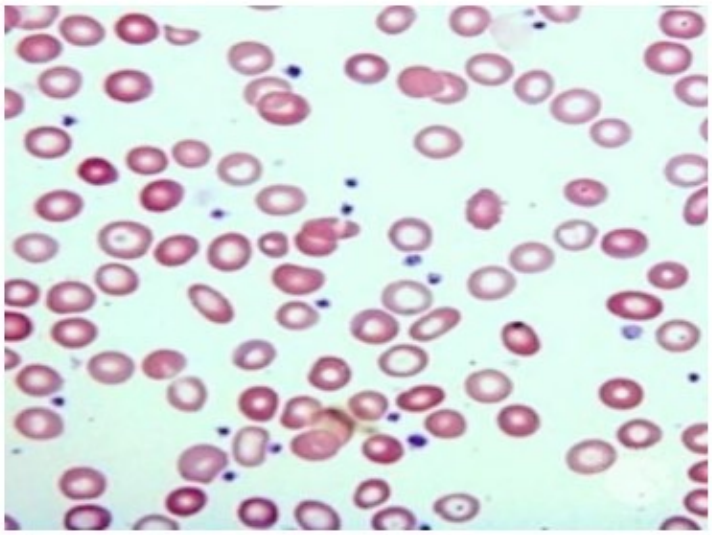

- Microcytic anemias generally have **a low** reticulocyte count.
- Most causes of microcytosis are **Production problems**. Production problems are nearly synonymous with low reticulocyte counts.
- Routine blood smear will not be effective in telling the difference between the types of microcytosis. All of them will be hypochromic. Best way to differentiate is iron study.

**Iron Indices in Microcytic Anemia Syndromes**

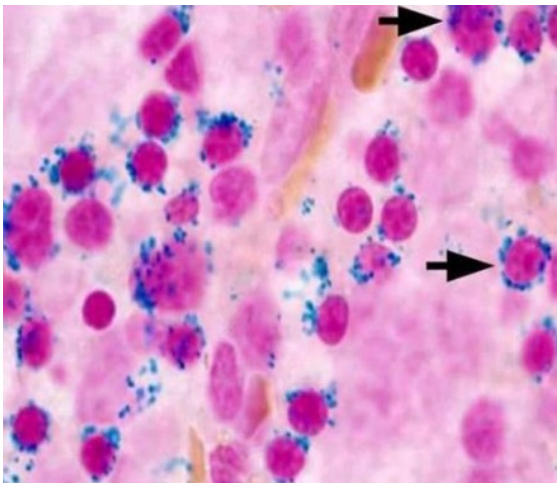
| Fe Panel | Iron Deficiency Anemia | Anemia of Chronic Disease | Sideroblastic Anemia | Thalassemia Minor |
|---|---|---|---|---|
| Serum Iron | Decreased | Decreased | Increased | Normal |
| Serum Ferritin | Decreased or Normal (early) | Increased | Increased | Normal |
| Transferrin/ TIBC | Increased | Decreased | Decreased | Normal |
| % Saturation | Decreased | N/ Decreased | Increased | Normal |

## Iron deficiency anemia

- **Most common** type of anemia .
- It is almost always caused by blood loss, most commonly GI or menstrual .
- Iron is consumed in heme (meat-derived) and non-heme (vegetable-derived) forms :
  - Absorption occurs in **the duodenum**.
  - **Transferrin** transports iron in the blood and delivers it to **liver** and **bone marrow** macrophages for storage .
  - Stored intracellular iron is bound to **ferritin**, which prevents iron from forming free radicals .
  - **Labs: (low** serum iron, low ferritin **< 10 ng/ml, low** transferrin saturation, **high** TIBC**)**.
  - Common in **menstruating female** and in **GI malignancy** as occult blood loss.



## Sideroblastic anemia



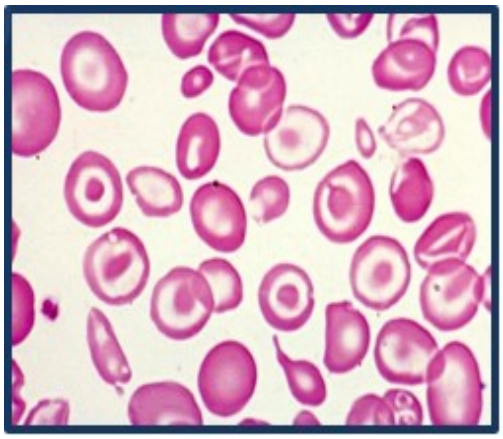
- Iron is transferred to erythroid precursors and enters the mitochondria to form heme. If protoporphyrin is deficient, **iron remains trapped in mitochondria**.
- Iron-laden mitochondria form **a ring around the nucleus of erythroid precursors**; these cells are called ringed sideroblasts (hence, the term sideroblastic anemia).
- Sideroblastic anemia is the **only microcytic anemia in which serum iron is elevated**.

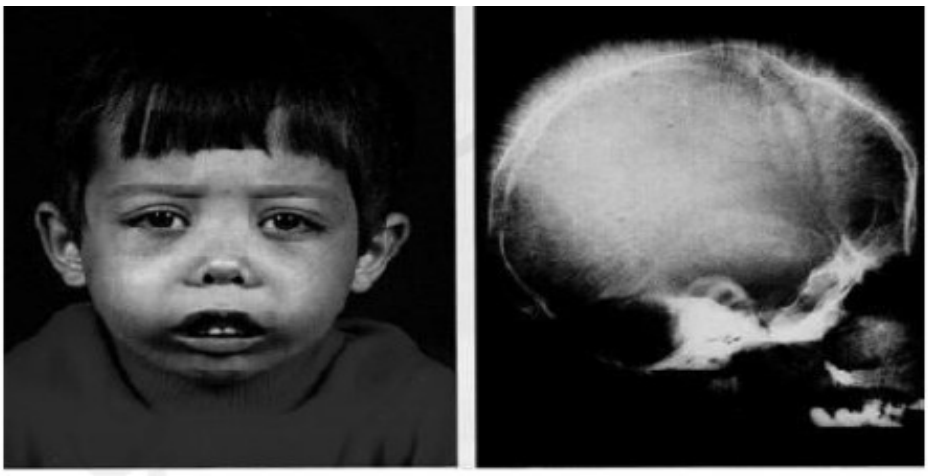
## Thalassemia

Anemia due to decreased synthesis of the **globin** chains of hemoglobin.

- ↓ Globin → ↓ hemoglobin → microcytic anemia.
- Divided into α- and β-thalassemia based on decreased production of alpha or beta globin chains both are inherited as **AR**.
- Normal types of hemoglobin are HbF (α2γ2) 1%, HbA (α2β2) >97%, and HbA2 (α2δ2) 2.5% .
  - Presentation: Minor (Asymptomatic) , Major (transfusion dependent)
  - In major forms , expansion of hematopoiesis into the skull (reactive bone formation **leads to 'crewcut ' appearance on x-ray**) and facial bones (**'chipmunk facies**), extra medullary hematopoiesis with hepatosplenomegaly.

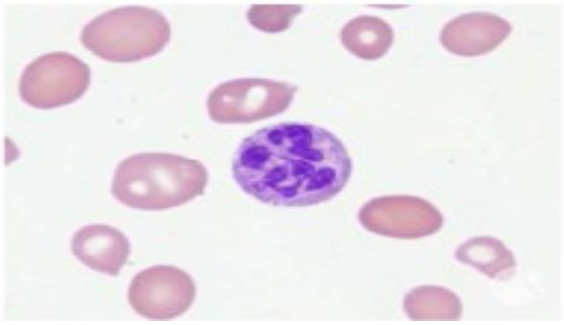
| Thalassemias | | | |
|---|---|---|---|
| | **Disorder (genotype)** | **Hb electrophoresis** | **Anemia severity** |
| **Alpha thalassemia** | Silent carrier (α α / α -) | Normal | Asymptomatic |
| | Trait (α α / - - **OR** α - / α -) | Normal | Mild symptoms |
| | Hb H disease (α - / - - ) | 5%-30% Hb H (adults) | Chronic hemolysis |
| | Major (fetal hydrops) ( - - / - - ) | • Hb Barts, Hb Portland & Hb H present<br>• Absent Hb A, Hb F & Hb A2 | Fatal in utero |
| | | | |
| **Beta thalassemia** | Trait ($\beta / \beta^{\circ}$) | Increased Hb A2 | Mild |
| | Intermediate ($\beta^{+} / \beta^{+}$, others) | Increased Hb F | Moderate |
| | Major ($\beta^{\circ} / \beta^{\circ}$) | Absent Hb A, only Hb A2 & Hb F present | Severe |

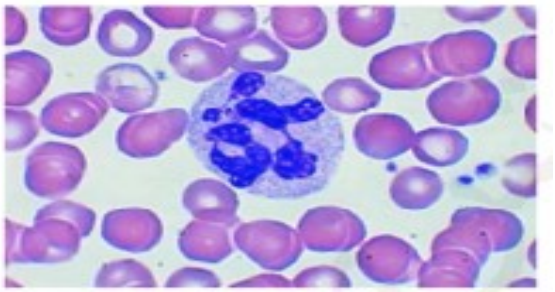




## Macrocytic anemia

- Although a macrocytic anemia could be from **B12 or folate deficiency, direct alcohol effect on the bone marrow,** or **liver disease.**
- The first step is a peripheral smear. This is to **detect hypersegmented neutrophils**.
- Once hypersegmented neutrophils are seen, then you would get B12 and folate levels (with a mean lobe count >5).
- **Megaloblastic anemia**
- Etiology:
  - **Vitamin B12 deficiency is caused by**:
    - Pernicious anemia. is the most common cause of vitamin B12 deficiency.
    - Pancreatic insufficiency (Pancreatic enzymes are needed to absorb B12).
    - Dietary deficiency (unusual and requires several years to produce disease). **Strict vegetarian.**
    - Crohn disease, celiac or any disease damaging the terminal ileum.
    - gastrectomy or gastric bypass for weight loss.
  - **Folate deficiency is caused by:**
    - Dietary deficiency (goat's milk has no folate and provides only limited iron and B12).
    - Drugs: **phenytoin**, **Trimethoprim** .and **methotrexate** use suggest folate deficiency.
- **Presentation:**
  B12 deficiency can give any neurological abnormality, but peripheral neuropathy is the most common .
- Diagnostic Tests: - B12 and folate deficiency are identical hematologically and on blood smear .
  - Laboratory abnormalities common to both B12 and folate deficiency are:
    - Megaloblastic anemia .
    - Decreased reticulocyte count (Red cells are destroyed as they leave the marrow due to ineffective erythropoiesis).
    - **Macroovalocytes .**
  - Both B12 and folate deficiency increase homocysteine levels, only B12 is associated with an increased MMA.
- **Tested** facts about macrocytic anemia :
  - The Schilling test is rarely used to determine the etiology of vitamin B12 deficiency .
  - Pernicious anemia is confirmed with **anti-intrinsic factor and anti-parietal cell antibodies .**
- 
- **Treatment**: - Replace what is deficient.





## Hemolytic anemia

- Caused by decreased RBC survival from increased destruction of the cells.
- The destruction may be inside the blood vessels (intravascular) or outside (extravascular), which generally means inside the spleen .
- Hemolytic anemia may be chronic (sickle cell disease, paroxysmal nocturnal hemoglobinuria, and hereditary spherocytosis) or acute (drug-induced hemolysis, autoimmune hemolysis, or glucose 6-phosphate dehydrogenase deficiency).
- All forms of hemolysis can lead to :
  - Sudden decrease in hematocrit .
  - Increased levels of LDH, indirect bilirubin, and reticulocytes.
  - Decreased serum haptoglobin (Haptoglobin binds circulating hemoglobin and reduces renal excretion of free hemoglobin, preventing tubular injury).
  - Hyperkalemia from cell breakdown .
  - Folate deficiency from increased cell production using it up; folate stores are limited .
  - Chronic hemolysis is associated with bilirubin gallstones

## Sickle cell anemia

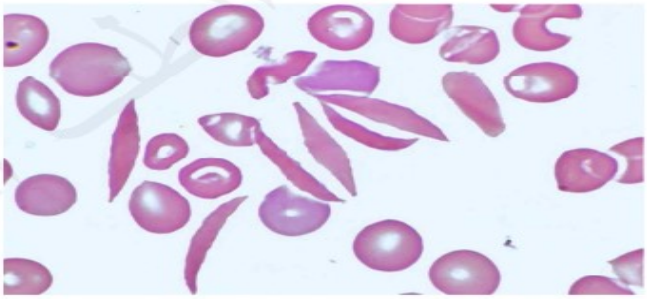
- **Autosomal recessive** mutation in β chain of hemoglobin; a single amino acid change replaces normal glutamic acid (hydrophilic) with valine (hydrophobic).
- HbS polymerizes when deoxygenated; polymers aggregate into needle-like structures, resulting in sickle cells :
  - Increased risk of sickling occurs with **hypoxemia, dehydration, and acidosis, infection and fever**.
  - HbF protects against sickling; high HbF at birth is protective for the first few months of life. Treatment with hydroxyurea increases levels of HbF.

**This boy presented with abdominal pain:**

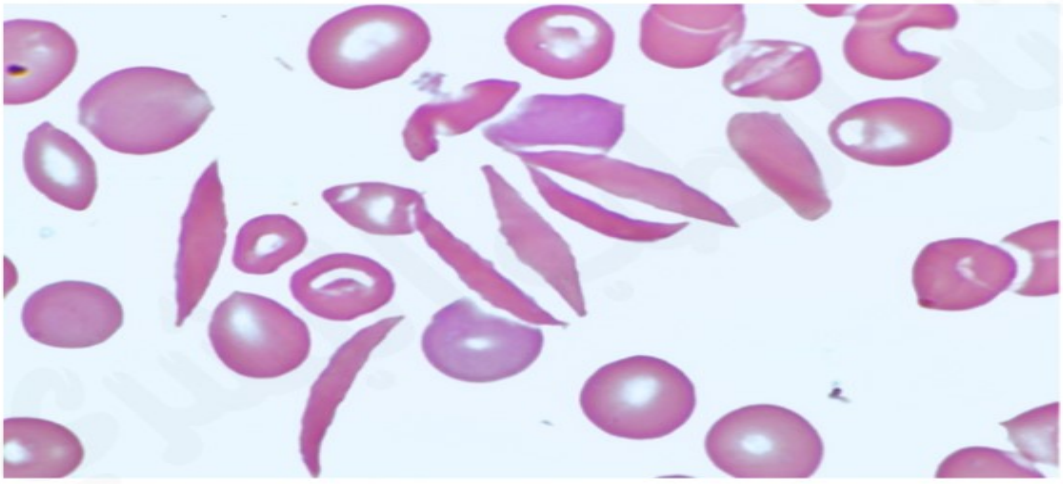
**Q1: What is your diagnosis?**
- Sickle cell disease

**Q2: What is the underlying cause of his abdominal pain?**
- Vaso-occlusive crises or infarction

**Q3: How to diagnose?**
- Hemoglobin electrophoresis



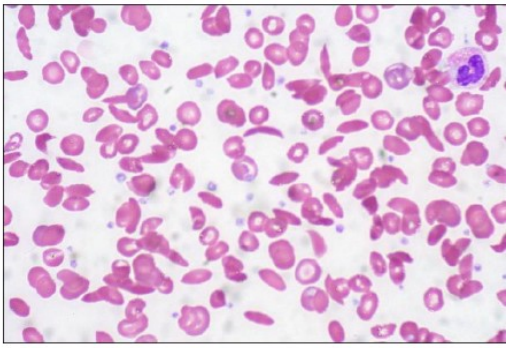
20. 16-year old boy with longstanding history of anemia presented with severe abdominal pain. Blood film as shown in the picture
    a. What is the diagnosis?
    b. What is the cause of his abdominal pain?



sickle cell anemia
vaso-occulsive pain

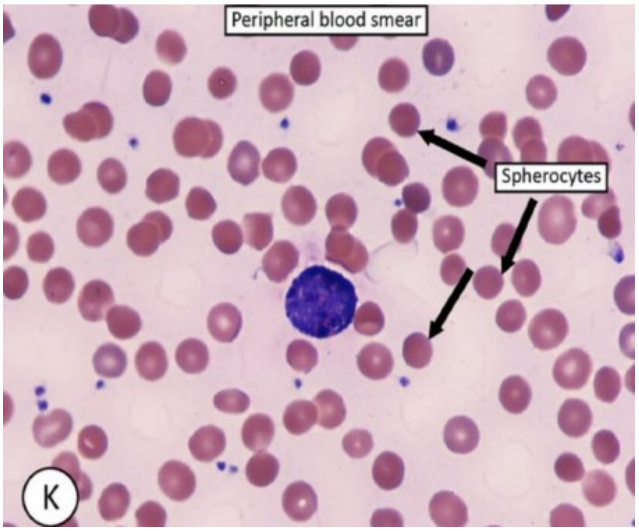
**Q12.21 YO male patient presented with dark urine & mild jaundice.What is the diagnosis?**

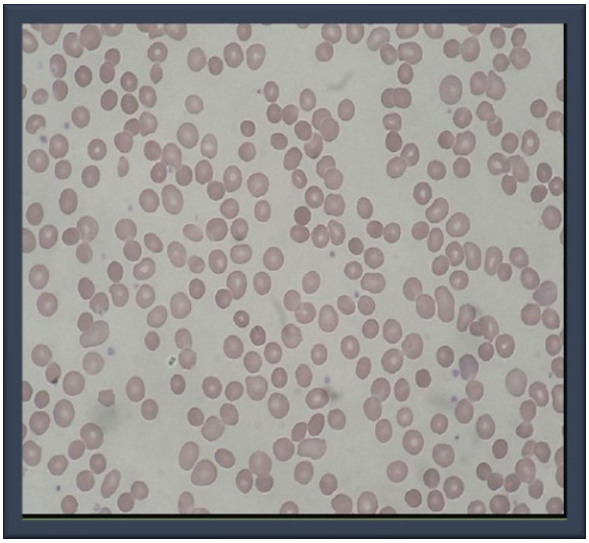
**Sickle cell anemia**

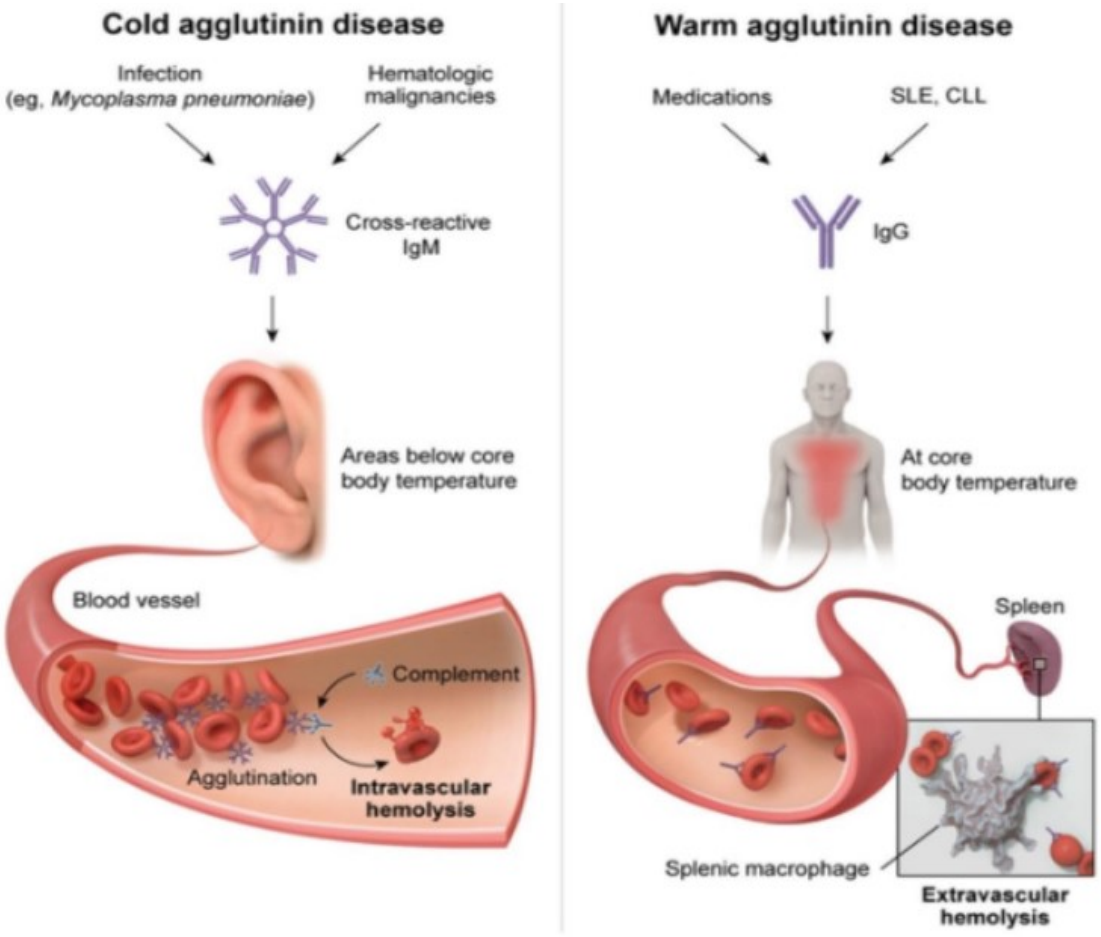


| Hereditary spherocytosis | |
|---|---|
| **Epidemiology** | • Usually autosomal dominant<br>• Northern European descent |
| **Clinical presentation** | • Hemolytic anemia<br>• Jaundice<br>• Splenomegaly |
| **Laboratory findings** | • ↑ MCHC<br>• Negative Coombs test<br>• Spherocytes on peripheral smear<br>• ↑ Osmotic fragility on acidified glycerol lysis test<br>• Abnormal eosin-5-maleimide binding test |
| **Treatment** | • Folic acid supplementation<br>• Blood transfusion<br>• Splenectomy |

**MCHC** = mean corpuscular hemoglobin concentration.

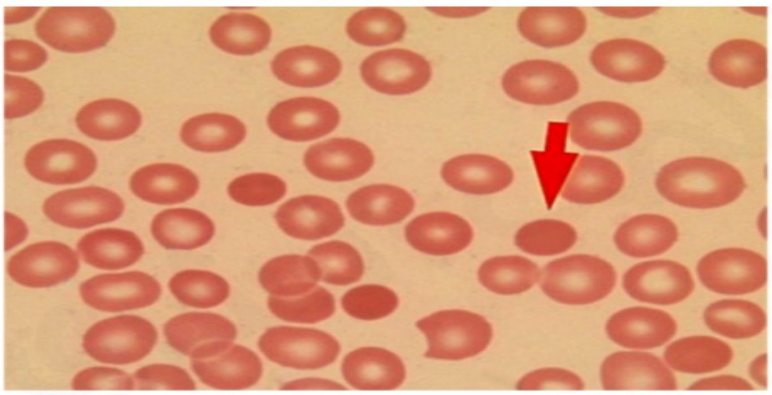







**CLL** = chronic lymphocytic leukemia; **SLE** = systemic lupus erythematosus.

23. A 30 y/o man presented with jaundice, his HB: 7MG/DL ,reticulocytes 7.5% , positive direct coombs test and this image finding on the blood film
a. what is the name of this cell ( red arrow)?
b. what is the most likely diagnosis?

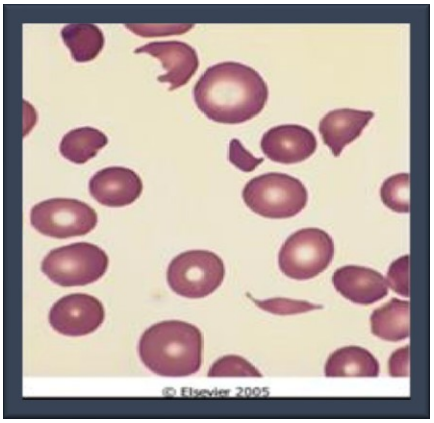


spherocyte
autoimmune hemolytic anemia

| Glucose-6-phosphate dehydrogenase deficiency | |
|---|---|
| **Epidemiology** | • Hemolytic anemia due to oxidative stress (infection, sulfa drugs, fava beans)<br>• X-linked: Asian, African, or Middle Eastern descent |
| **Manifestations** | • Pallor & fatigue<br>• Dark urine, jaundice & icterus<br>• Abdominal/back pain |
| **Laboratory findings** | • Hemolysis: ↓ hemoglobin, ↓ haptoglobin, ↑ bilirubin & LDH<br>• Peripheral smear: bite cells & Heinz bodies<br>• Negative Coombs test<br>• ↓ G6PD activity level (may be normal during attack) |
| **Management** | • Remove or treat responsible agent/condition<br>• Provide supportive care |

**LDH** = lactate dehydrogenase; **G6PD** = glucose-6-phosphate dehydrogenase.

**Q17: What's the hematological abnormality in this blood film?**
G6PD deficiency(x-linked, decrease NADPH which protect against oxidative stress).




**Most common type of stress?**
Infection

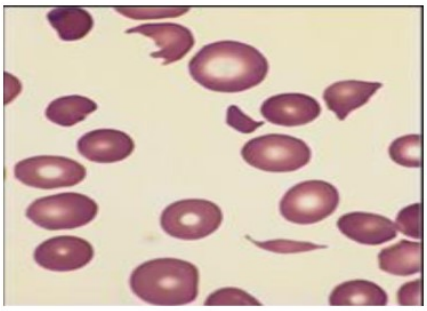
**Drugs that may cause oxidative stress?**
Primiquine, Aspirin , Isoniazid , Nitrofurantoin Sulfa drug **(PAINS)**

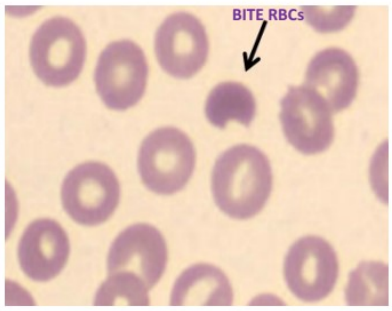
**Blood film show?**
Bite cells

Definitive test: G6PD level
Treatment? Hydration and transfusion if severe hemolysis

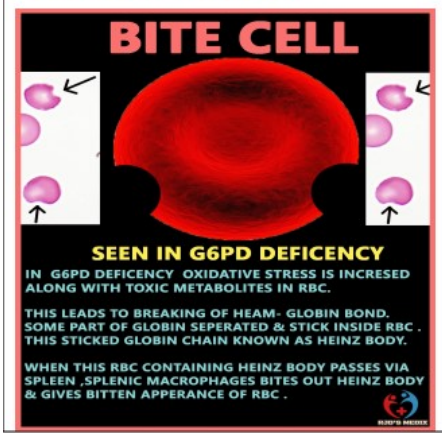
**Q16.What's the hematological abnormality in this blood film?**

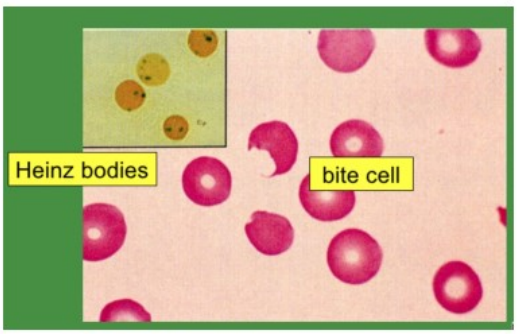
**G6PD deficiency**






**NOTE:-In G6PD def blood smear you can see Heinz bodies & bite cell**



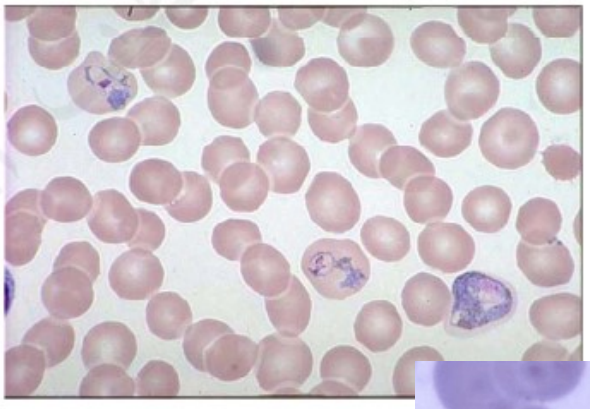

A patient presented with fever and a blood film is shown:

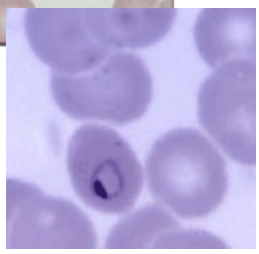
Q1: What do you see?

-

Q2: What is the diagnosis?

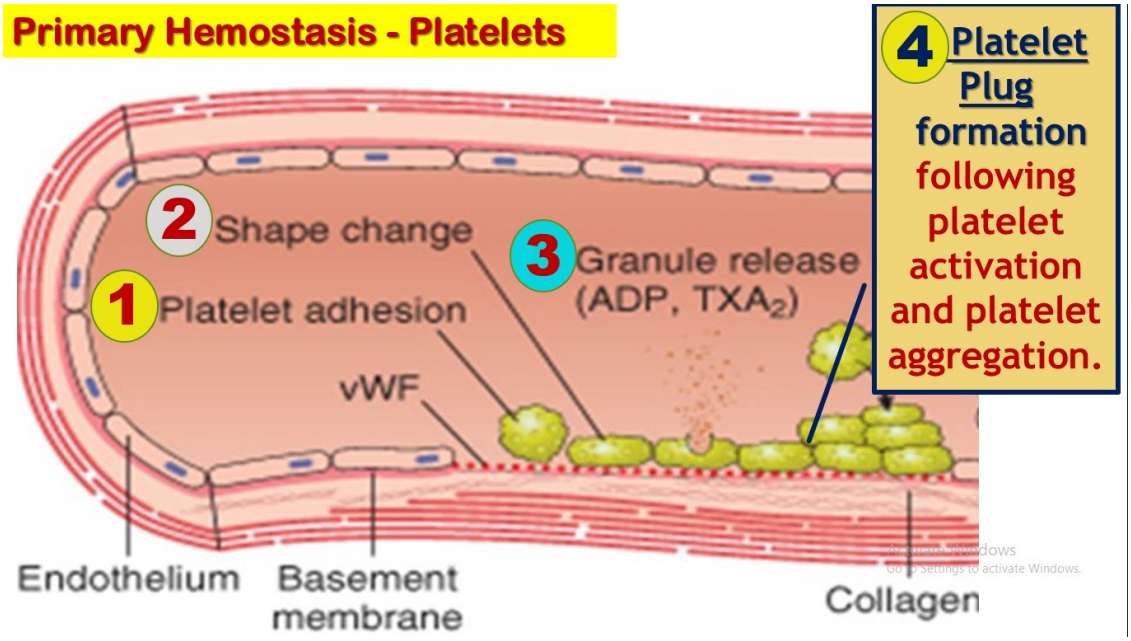
Malaria?!?!

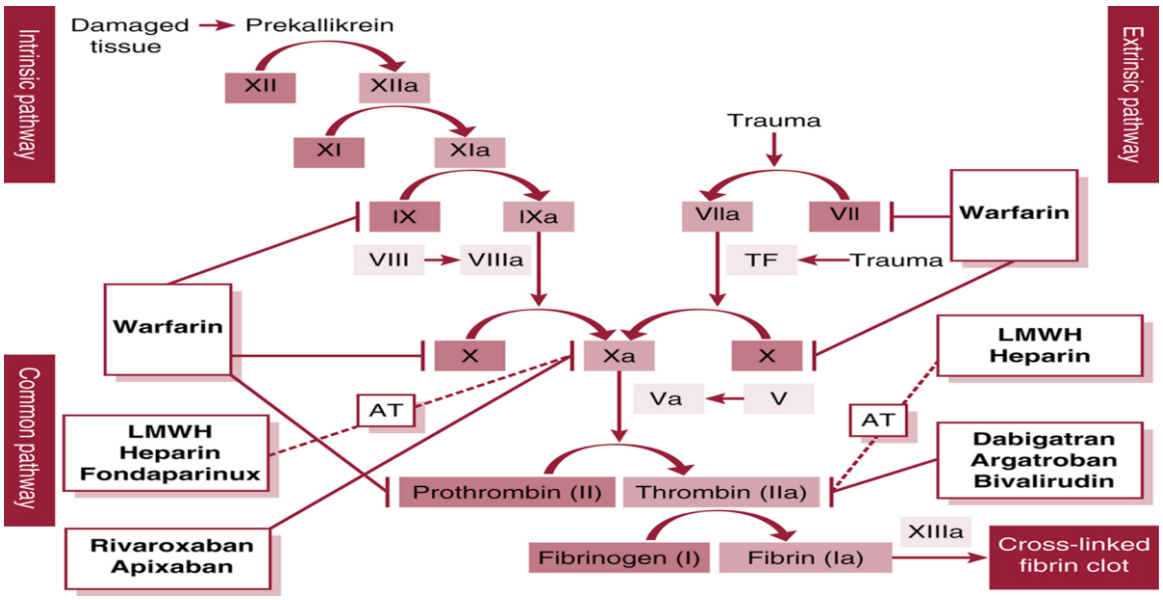




# Bleeding disorders

## Basic Physiology



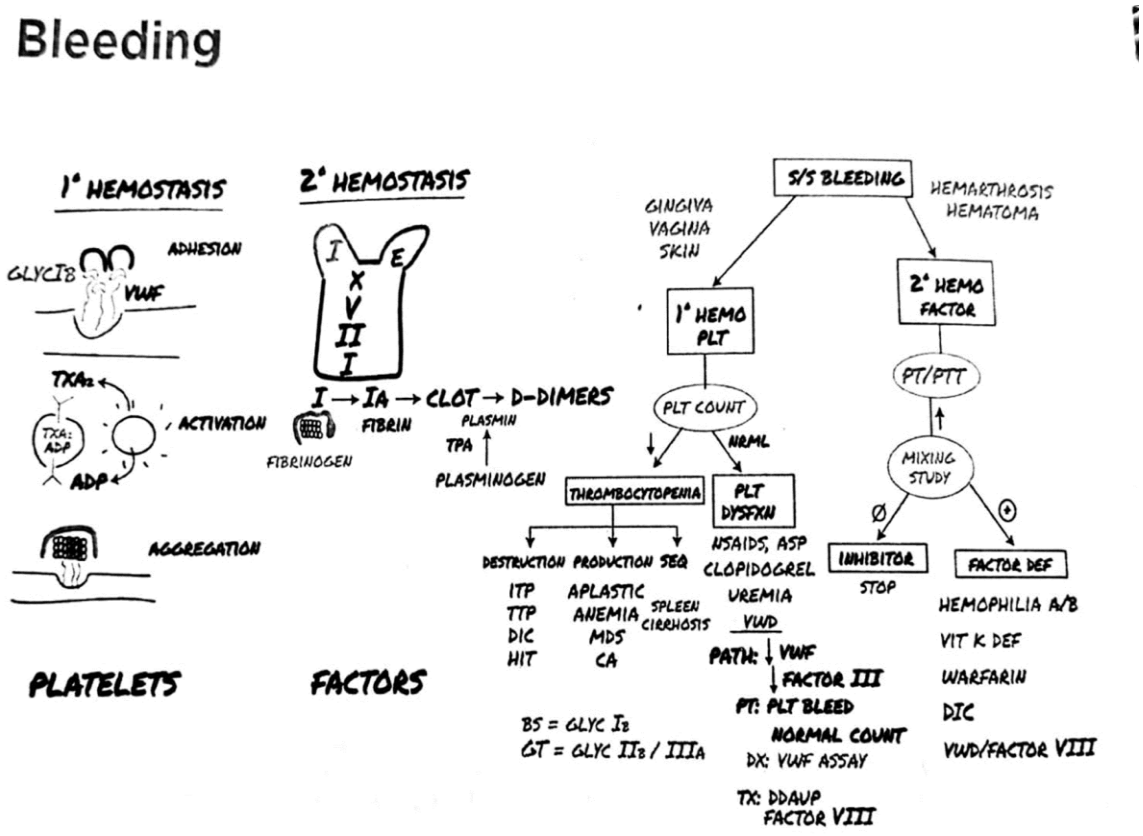



## Unfractionated Heparin vs LMWH

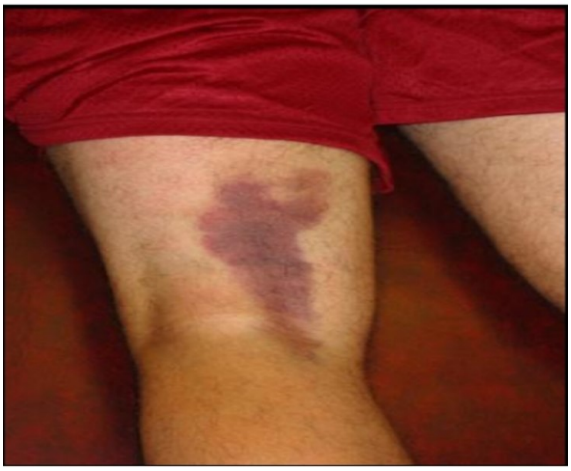
| Unfractionated Heparin | | Low Molecular Weight Heparin (LMWH) |
|---|---|---|
| Activates anti-thrombin III which forms a complex inhibiting clotting factors IIa and Xa, as well as IX, XI, XII | Mechanism of action | Examples include *enoxaparin, tinzaparin* *Fondaparinux* is a synthetic derivative of LMWH Activates anti-thrombin III which forms a complex inhibiting clotting factor Xa |
| Intravenous (IV) | Mode of administration | Subcutaneous (SC) |
| Shorter (~1 hour) | Half-life | Longer (~3-6 hours) Fondaparinux ~17-21 hours |
| Bleeding, osteoporosis, thrombocytopenia (HIT), hyperkalemia (due to hypoaldosteronism) | Side effects | Bleeding, osteoporosis, thrombocytopenia (HIT), hyperkalemia (due to hypoaldosteronism) |
| Rapidly reversible by protamine sulphate Useful in situations where rapid reversal required | Reversibility | Partially reversible by protamine sulphate |
| Useful in renal failure | Use in renal failure | Use with caution/avoid if GFR <30 due to increased risk of bleeding |
| Higher risk compared to LMWH | Risk of HIT | Lower risk of HIT No risk of HIT when using fondaparinux |

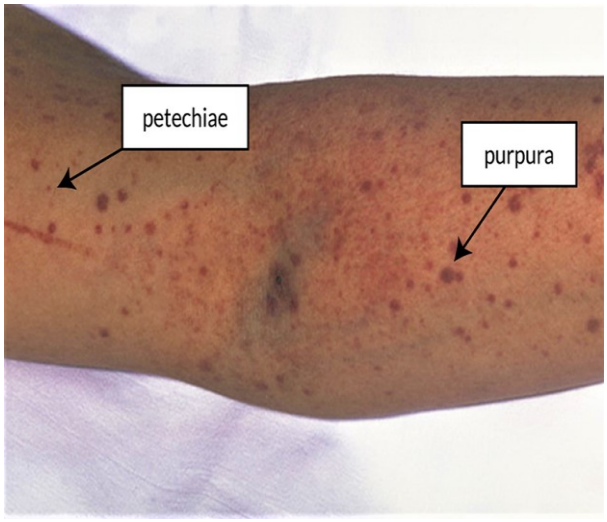
HIT – Heparin-induced thrombocytopenia

GRAM PROJECT

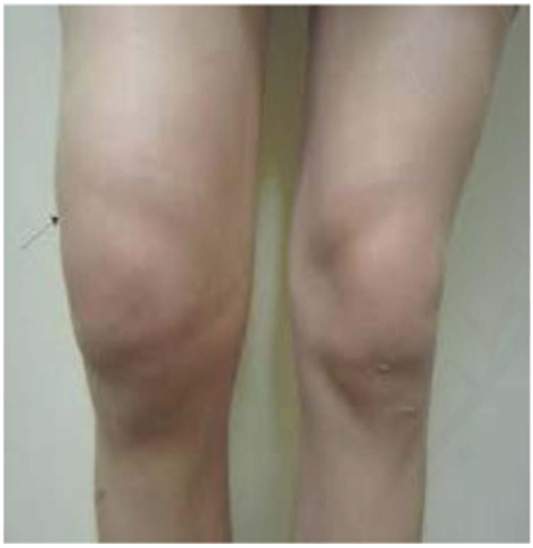


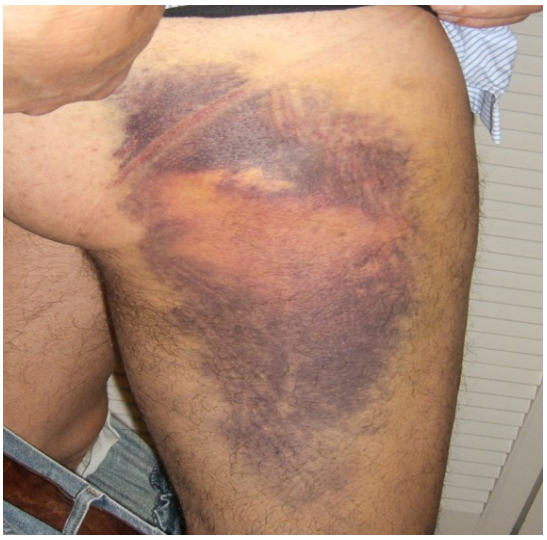
## Superficial bleeding






## Deep tissue bleeding





## Thrombocytopenia

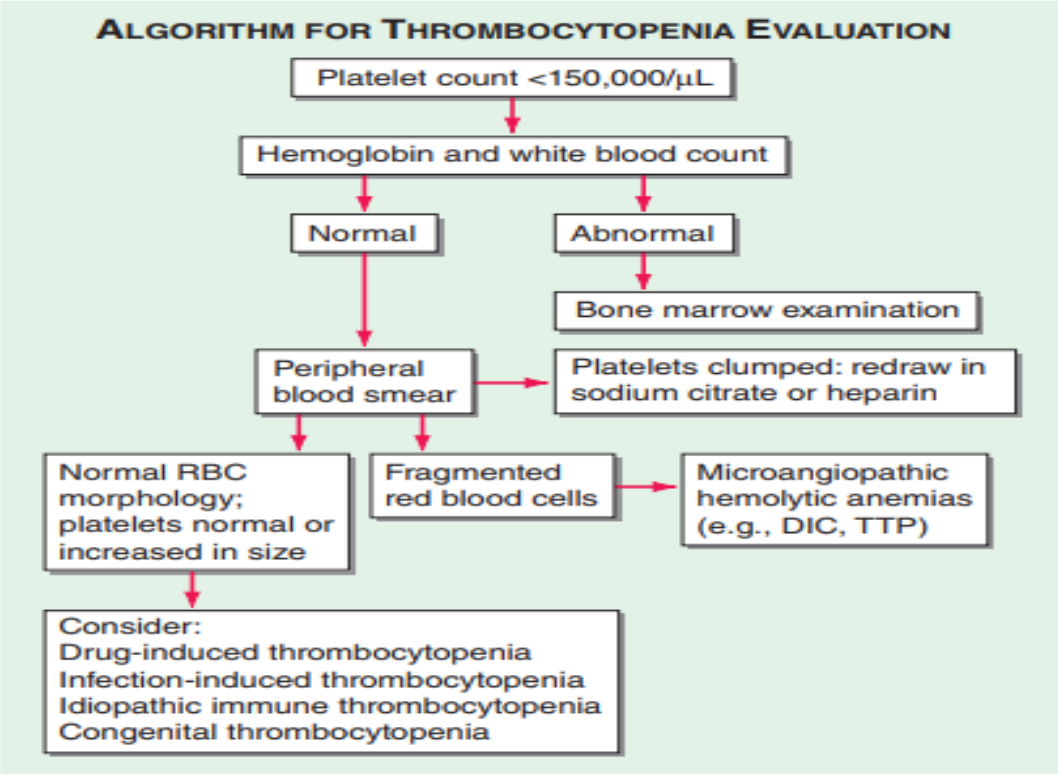
- **ITP: Immune thrombocytopenic purpura (ITP).**
  1. Pathology: **AB to platelet.**
  2. Etiology: **usually in females**, preceded by **URTI** Or **viral** infections (HIV) Or even SLE.
  3. Diagnosis: **DOE**.
  4. Treatment: Steroids, IVIG, Splenectomy, Rituximab.

- **HIT: Heparin induced thrombocytopenia.**
  1. Pathology: AB To platelets.
  2. S@S: **Heparin product, 7-14 days**.
  3. Diagnosis: HIT AB.
  4. Treatment: **Stop Heparin and start Argotraban, Bridge to Warfarin.**

- **DIC** (Disseminated intravascular coagulopathy).
  1. Pathology**: Fibrin clot.**
  2. Sign and symptoms: **Sepsis, shock, ICU** → Bleeding.
  3. Diagnosis: **CBC** (Decrease platelet), **Smear** (Schistocytes), **PT/PTT** (Increase), **Fibrinogen** (Decrease), **D-dimer (Increased).**
  4. Treatment: **supportive, and treat underlying cause.**

- **TTP:** Thrombotic Thrombocytopenic Purpura
  1. Pathology: **ADAMTAS-13 deficiency (Vwf metalloprotease).**
  2. Patient: **Fever**, Anemia, Thrombocytopenia, Renal failure, **Neuro SXS**.
  3. Diagnosis: CBC (Decrease platelet), **Smear** (Schistocytes), **PT/PTT** (normal), Fibrinogen ( normal), **D-dimer** (normal).
  4. **Treatment**: X change transfusion, Plasmaphereses, Never platelets.

- **HUS:** Hemolytic uremic syndrome.
  1. **Pathology:** Endothelial damage usually due to infection (**E.Coli O157:H7**).
  2. **Sign and symptoms:** Anemia, Renal failure, Thrombocytopenia.
  3. **Usually preceded by diarrhea, fatigue, pallor, bruising, petechia.**



## Hemophilia A

1. **Pathophysiology**: **Factor VIII deficiency** (either familial or denovo).
2. Familial Inherited by: **X- linked Recessive.**
3. Presented with: **Deep tissue bleeding (Joint/ Muscle), After surgical bleeding .**
4. Laboratory: **Prolonged PTT** but normal **(PT, platelets and bleeding time)**. With decreased factor VIII.
5. Treatment: **Recombinant VIII.**

## Chapter 6

# Endocrine system

# Diabetes Mellitus

## DM (Etiology)

### What is the definition of DM?

Diabetes mellitus (DM) is a **disorder of carbohydrate metabolism**, caused by **relative or absolute deficiency of insulin**, **hyperglycemia**, **and end-organ complications** (nephropathy, retinopathy, neuropathy, accelerated atherosclerosis).

### Types

**(Type 1) DM (insulin-dependent or juvenile onset):**

- **Accounts for 5-10% of diabetes worldwide.**
- **The age of onset is usually age <30 (Onset in childhood).**
- **There is an increased prevalence of autoantibodies to islet cells (Insulin dependent from an early age).**
- **Not related to obesity. Patients usually have a lean body build and are prone to ketosis owing to absent insulin production.**

**(Type 2) DM (non-insulin-dependent or maturity onset):**

- It is the **most common type of diabetes,** accounting for **90%** of cases.
- Age of onset is **usually age 40** (Onset in adulthood).
- Directly **related to obesity.**
- Defined as **insulin resistance.**

## DM (Clinical presentation)

- **Polyuria, polyphagia, and polydipsia** are the most common presentation .
- The **first event** may be an acute metabolic decompensation, resulting in **coma** (ketoacidosis for IDDM and hyperosmolar coma for NIDDM).
- Occasionally the initial expression of DM is a **degenerative complication like neuropathy.**

## DM (Diagnostic test)

- Diabetes is defined/diagnosed as:
  - **Two fasting blood glucose measurements ≥ 126 mg/dL.**
  - **Single glucose level above 200 mg/dL with above symptoms.**
  - **Hemoglobin A1c >6.5%** is a diagnostic criterion and is the **best test to follow response to therapy over the last several months.**
  - **Increased glucose level on oral glucose tolerance testing** (rarely required).

## Treatment (Non-pharmacological)

**1. Diet, Exercise, and Weight Loss:**

- Weight reduction of as little as **4-7%** body fat has an **enormous effect on peripheral insulin sensitivity.**
- Exercising muscle **needs no insulin** for glucose to enter .
- As many as 25% of diabetic patients can be kept off of medication with diet and exercise alone.
- **When diet and exercise do not keep the HbA1c <7%, medications are introduced.**

## DM (Medical Treatment)

**2. Oral Hypoglycemic Medication :**

- **Metformin is the drug of choice and along with lifestyle intervention should be used in all newly diagnosed patients**.
- The **goal** of therapy is **HgA1c <7%.**
- Metformin **should not be given to acutely ill patients** with **acute renal failure, liver failure, or sepsis** as these conditions **increase the risk of lactic acidosis.**
- In all cases, metformin is clearly the "best initial therapy" for type 2 diabetes. **After metformin, the choices are less clear.** If one drug is not sufficient, a second or third oral agent may be combined to keep the patient off insulin.

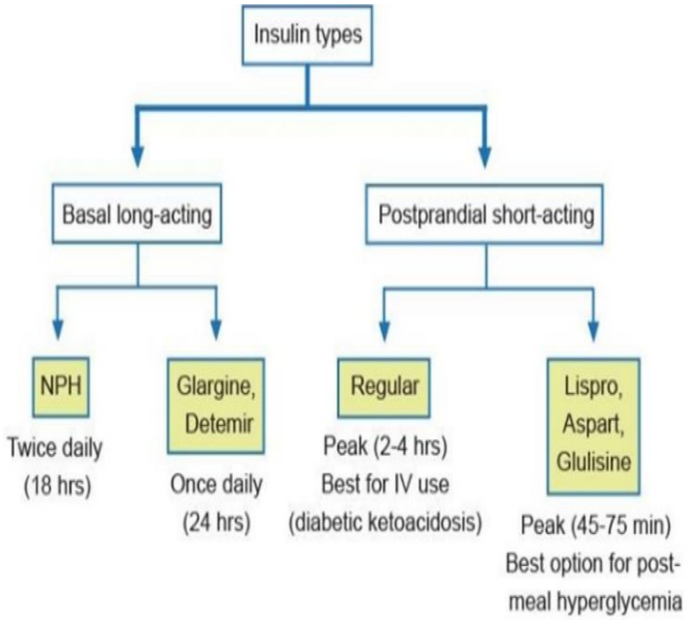
| Medication | ↓ A1c | Points to remember |
|---|---|---|
| Metformin (biguanide) | 1.0%-2.0% | • Initial therapeutic agent for most type 2 diabetics<br>• Weight neutral, low risk of hypoglycemia<br>• Lactic acidosis is a life-threatening complication |
| Sulfonylureas | 1.0%-2.0% | • Generally added in patients with metformin failure<br>• Weight gain & hypoglycemia are main side effects |
| Pioglitazone (TZDs) | 1.0%-1.5% | • Used if unable to tolerate metformin or sulfonylureas<br>• Side effects: weight gain, edema, CHF, bone fracture, bladder cancer<br>• Low risk of hypoglycemia when used alone or with metformin<br>• Can be used in renal insufficiency |
| DPP-IV inhibitors (eg, sitagliptin) | 0.5%-0.8% | • Low risk of hypoglycemia<br>• Weight neutral<br>• Can be used in renal insufficiency |
| GLP-1 receptor agonist (eg, exenatide) | 0.5%-1.0% | • Possible second agent for metformin failure, especially if weight loss is desired<br>• Low hypoglycemia risk when used alone or with metformin |

CHF = congestive heart failure; DPP = dipeptidyl peptidase-4; GLP-1 = glucagonlike peptide-1; IV = intravenous; TZDs = thiazolidinediones.

## Treatment

**3. Insulin :**

- **Only after therapy with multiple oral hypoglycemic fails should an insulin regimen be considered .**
- Diabetic patients often **need 2 types of insulin, a basal long-acting insulin and a postprandial short acting insulin.**
- When starting insulin, **divide 50% into long-acting and 50% into pre-meal short-acting.** This regimen is usually given as glargine insulin 1x/day injection along with 2–3×/day ultrashort-acting insulin such as lispro or aspart before meals.
- **The most common side effects of insulin are hypoglycemia and weight gain.**
- **Insulin is the medication of choice for the treatment of gestational diabetes mellitus (GDM).**




## DM (Complications)

**Acute complications:**

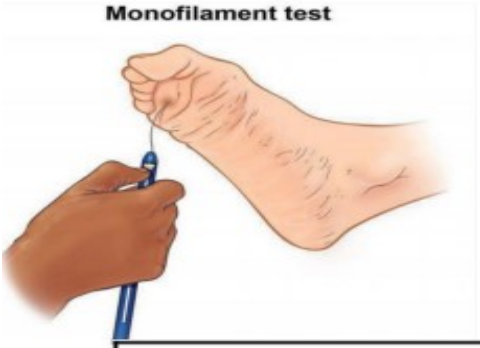
1. **Diabetic Ketoacidosis (DKA).**
2. **Hyperosmolar hyperglycemic state (HHS).**

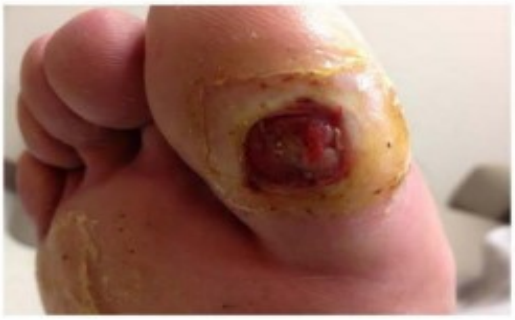
**Chronic complications:**

1. **Cardiovascular Complications:** The number 1 cause of death in patients with diabetes is cardiovascular disease .
   Lipid testing should be performed in patients with diabetes at least annually.
   Diabetes is considered the equivalent of coronary disease in terms of management of hyperlipidemia.
2. **Diabetic Nephropathy:** Patients with **DM should be screened annually** for **microalbuminuria** and started on an **ACE inhibitor or ARB** when it is present.

3. **Retinopathy:**
   - The only management for **non-proliferative retinopathy is tighter control of glucose.**
   - When **neovascularization and vitreous hemorrhages are present**, it is called **proliferative retinopathy**. This is treated with **laser photocoagulation**, which markedly retards the progression to blindness. **VEGF inhibitors** treat severe retinopathy.

4. **Neuropathy:**
   **A. Peripheral neuropathy (most common).**
   **B. Mononeuropathy.**
   **C. Autonomic neuropathy:** Orthostatic hypotension and Syncope, decreased sweating and dry feet, Diabetic gastroparesis (delayed gastric emptying), Impotence and retrograde ejaculation can occur.



| Diabetic foot ulcers | |
|---|---|
| **Risk factors** | • **Diabetic neuropathy** (loss of protective sensation, small muscle atrophy, abnormal vascular tone, decreased sweating with fissures)<br>• **Arterial insufficiency**<br>• End-stage renal disease in a patient on dialysis<br>• Smoking |
| **Location** | • Plantar surface, areas under pressure points (eg, bony prominences) |
| **Management** | • Mechanical offloading<br>• Debridement<br>• Wound dressings<br>• Antibiotics if infection |

## Metabolic syndrome

- **Metabolic syndrome is diagnosed when at least 3 of the 5 following criteria are met:**
  - **Abdominal obesity** (Men: Waist circumference **>40 inches**; Women: Waist circumference **>35 inches**).
  - **Fasting glucose >100 -110 mg/dL.**
  - **Blood pressure > 130/80 mm Hg.**
  - **Triglycerides >150 mg/dL.**
  - **HDL cholesterol (Men: <40 mg/dL; Women: <50 mg/dL.(**

# Diabeteic Ketoacidosis & Hyperosmolar Hyperglycemic State

## Acute Complications of Diabetes

- **Diabetic ketoacidosis) DKA (**and **hyperosmolar hyperglycemic state (HHS**) are the 2 most serious **acute complications of diabetes.**
- **Diabetic ketoacidosis) DKA (is more common** in those with Type 1 diabetes ,but **can definitely present** in those with Type 2 diabetes.
- **Hyperosmolar hyperglycemic state) HHS** (is a syndrome that occurs predominantly in patients **with type 2 diabetes** and is characterized by **severe hyperglycemia in the absence of significant ketosis.**

## Diabetic Ketoacidosis

- DKA may be **the initial presentation of DM.**

**Presentation:**

- Precipitated by **infections** ,**Severe stress** as myocardial infarction or Non compliance to insulin therapy.
- Patients with DKA clinically exhibit **nausea and vomiting ,severe abdominal pain ,dry mucous membranes and lethargy**.
- There is classically a fruity odor on their breath**) acetone odor.(**
- DKA patient has **both clinical) polyuria ,polydipsia ,volume depletion (and biochemical) hyperglycemia ,low bicarbonate ,high anion gap (signs.**
- Hyperglycemia is associated with **glycosuria that leads to obligatory water loss and subsequent marked dehydration**.
- **Ketoacidosis** → **metabolic acidosis** → ventilation becomes deep and rapid**) Kussmaul breathing).**
- Metabolic acidosis is typically accompanied by **hyperkalemia**.
- **Causes of hyperkalemia** are:
  - **Extracellular shift of potassium** in exchange to **hydrogen ion** ,with resultant intracellular potassium deficit.
  - **Impaired insulin-dependent** cell entry of the potassium ion.

## Diagnosis

- **Criteria for making a diagnosis of DKA** , **three things are necessary:**
  1. **Elevated blood glucose 500-250 mg/dl.**
  2. **Metabolic acidosis (low serum bicarbonate < 18, and low blood pH), and increased anion gap (sodium- [bicarbonate + chloride]).**
  3. **Detection of plasma ketones (increased serum levels of acetoacetate, acetone, and hydroxybutyrate).**

## Treatment

- Initial management is **rapid, intravenous administration of normal saline and regular insulin.**
- Essential measures in the management of DKA include the following:
  1. **Restoration of intravascular volume:** using 0.9% saline **(normal saline).**
  2. Correction of hyperglycemia: using **intravenous regular insulin.**
  3. **Correction of electrolyte abnormalities:**
     **Potassium** correction is very crucial.
  4. **Correct the underlying cause:**
     **Noncompliance** with medications, infection, pregnancy, or any serious illness.

## Hyperosmolar hyperglycemic state (HHS)

**Pathophysiology:**

Involved is **profound dehydration resulting from a sustained hyperglycemic diuresis.**

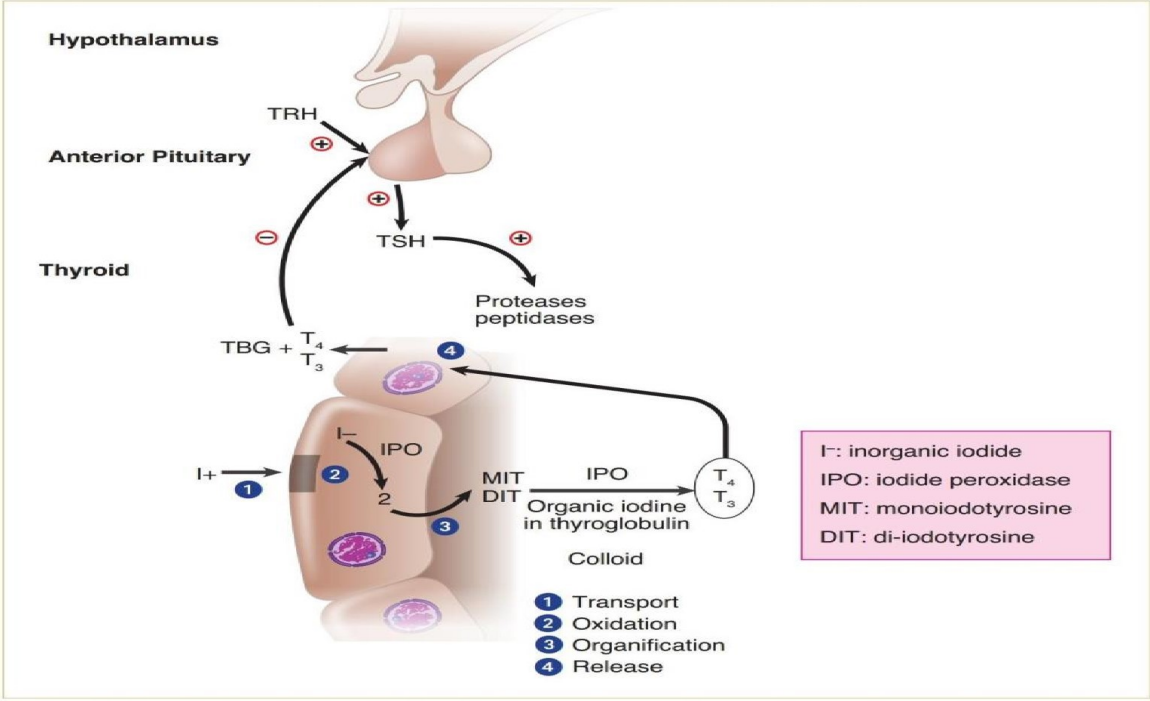
**Clinical findings:**
Weakness, polyuria, polydipsia, lethargy, confusion, convulsions, and coma.

**Diagnosis:**
1. **Severe hyperglycemia (frequently >1000 mg/d).**
2. Increased serum **osmolality (>320 mOsm/kg).**
3. Little or no ketonemia or acidosis present, **most patients have pH >7.3 and serum bicarbonate >20 mEq/L.**
4. Neurologic symptoms: **focal signs, lethargy, blurry vision, and obtundation.**

## Management of HHS

- Involves **high-volume fluid and electrolyte replacement, and insulin.**
- Fluid replacement with normal saline is the most important initial step in management of hyperosmolar hyperglycemic state.
- **Despite normal or elevated serum potassium.** Aggressive insulin therapy for HHS can lower serum potassium levels further and cause **severe hypokalemia.**

# Thyroid Disorders




## Hypothyroidism

(Primary hypothyrodisim)

- The most common etiology of hypothyroidism in areas where iodine is sufficientis Hashimoto's thyroiditis, an autoimmune process that destroys the cells of the thyroid and affects women more than men.
- Postablative surgery or radioactive iodine, heritable biosynthetic defects, andiodine deficiency.
- Drugs such as lithium and Amiodarone.
- Secondary hypothyroidism (pituitary induced )
- Tertiary hypothyroidism (hypothalamic induced )

## Presentation

Hypothyroidism is characterized by almost all bodily processes being slowed down (except menstrualflow, which is increased).

| Hypothyroidism |
|---|
| Bradycardia |
| Constipation |
| Weight gain |
| Fatigue, lethargy, coma |
| Decreased reflexes |
| Cold intolerance |
| Hypothermia (hair loss, edema) |

Hypothyroidism is an important cause of reversible changes in memory and mentation

## Diagnosis

All thyroid disorders are best tested first with a TSH. If the TSH level is suppressed, measure free T4levels. TSH levels are markedly elevated if the gland has failed

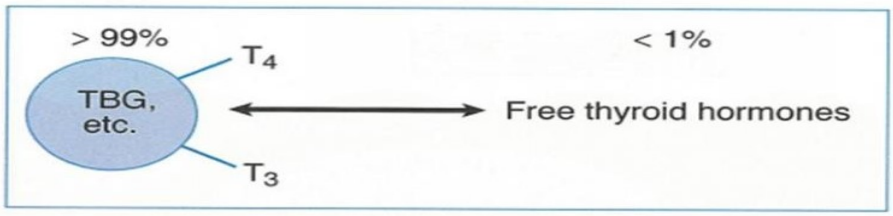
Small changes in thyroid hormone levels lead to marked changes in serum TSH level. In hypothyroidism,the TSH rise occurs well before a low thyroid hormone level is seen. Thus, **serum TSH is the most sensitive marker for diagnosis of hypothyroidism**

| Primary Hypothyroidism | 2° or 3° Hypothyroidism |
|---|---|
| ↑ TSH | ↓ TSH |
| ↓ Free $T_4$ | ↓ Free $T_4$ |

**Antithyroid peroxidase (anti-TPO) antibodies are present in >90% of patients with Hashimotothyroiditis**

## Treatment:

- **Replacing thyroid hormone with thyroxin (synthroid) is sufficient. T4 will be converted in the local tissues toT3 as needed**




| | Normal patient | Hypothyrodism patient |
|---|---|---|
| **Liver disease** | | |
| **OCP** | | |

## Hyperthyroidism

Primary hyperthyroidism can result from:

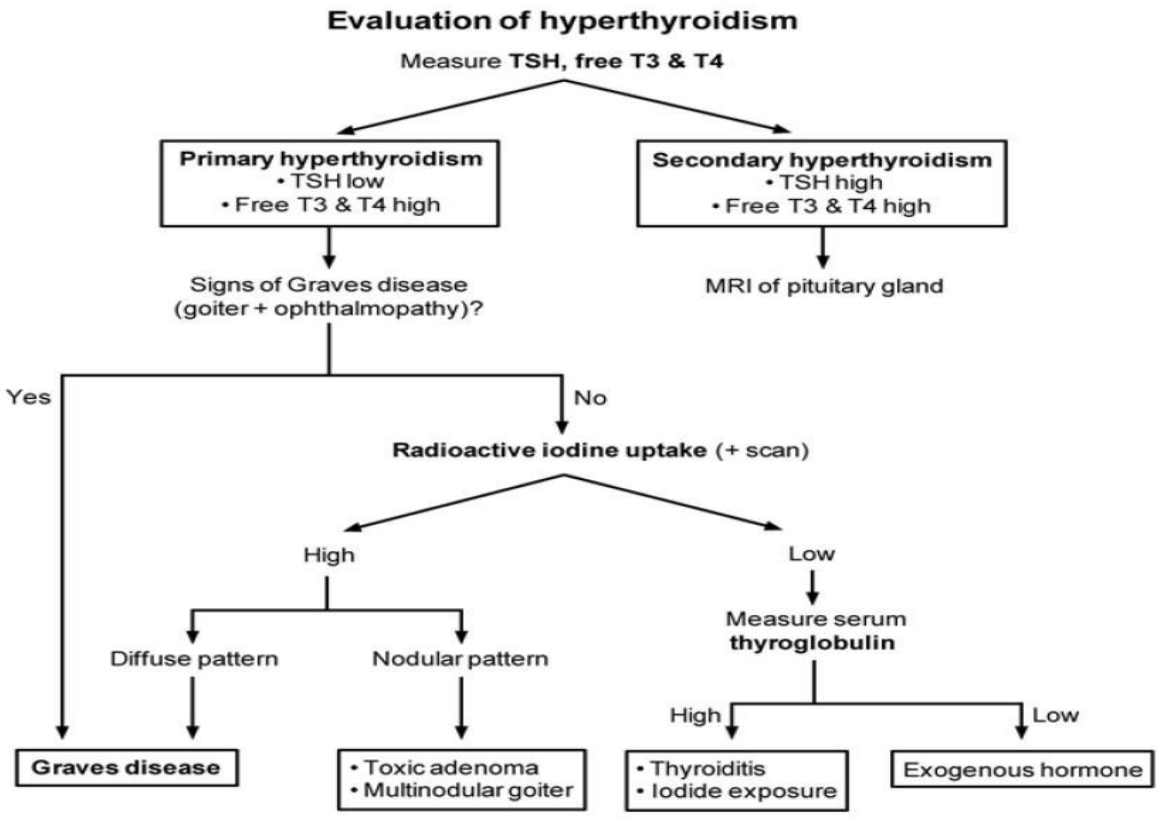
- **Overproduction of thyroid hormone (Graves disease, toxic adenoma, toxic nodular goiter).**
- **Release of preformed hormone (painless thyroiditis, subacute thyroiditis).**

**Secondary hyperthyroidism by pituitary adenoma**

## Presentation

| Hyperthyroidism |
| --- |
| Tachycardia, palpitations, arrhythmia (atrial fibrillation) |
| Diarrhea (hyperdefecation) |
| Weight loss |
| Anxiety, nervousness, restlessness |
| Hyperreflexia |
| Heat intolerance |
| Fever |

**The most common cause of hyperthyroidism is Graves' disease, which is caused by an autoantibody tothe TSH receptor and is characterized by a diffuse goiter and ocular abnormalities (proptosis, periorbital edema, ophthalmoplegia) , pretibial myxedema**




**Grave's disease** (toxic diffuse goiter) is an autoimmune problem in which autoantibody (IgG) is directed
against the thyroid receptor. It is referred to as the thyroid stimulating antibody (TSI).

- Women > men.
- Patients with Graves' disease develop lymphocytic infiltration of the orbital and pretibial connective tissue because of increased TSH receptor expression in these regions.
- Cytokines released by activated T-cells increase fibroblast proliferation and secretion of glycosaminoglycans, resulting in mucinous edema and tissue expansion , eventually leads tothe development of Graves' ophthalmopathy and pretibial myxedem

## Treatment:

- **Graves' disease can be treated with antithyroid drugs, radioactive iodine, or thyroidectomy**

Antithyroid drugs are used for patients with **mild disease who are likely to have a permanent remission**.They are also **used in preparation for treatment with radioactive iodine in patients with significant hyperthyroidism or who are at increased risk of complications** due to transient worsening of hyperthyroidism following RAI uptake

Radioactive iodine (RAI) treatment

**can raise titers of TRAB and worsen the ophthalmopathy**. Glucocorticoids and antithyroid drugs can beused to minimize the effects of RAI.

Mechanism of propylthiouracil (PTU) and methimazole (MMI): These agents **inhibit thyroperoxidase**The most common side effect of ATDs is allergic reaction (2% of patients).

**The most serious side effect is agranulocytosis (0.3% of patients)**, and all patients must be informed about it. Those developing sore throat and fever should stop the ATD and see a physician to check their white blood cell count. However, **routine monitoring of the white blood cell count is not needed due tothe rarity of the condition**

| Complications of Graves disease treatment | |
|---|---|
| **Treatment** | **Adverse effects** |
| **Antithyroid drugs (thionamides)** | • **Agranulocytosis**<br>• Methimazole: 1st-trimester teratogen, cholestasis<br>• Propylthiouracil: **Hepatic failure**, ANCA-associated vasculitis |
| **Radioiodine ablation** | • Permanent hypothyroidism<br>• Worsening of ophthalmopathy<br>• Possible radiation side effects |
| **Surgery** | • Permanent hypothyroidism<br>• Risk of recurrent laryngeal nerve damage<br>• Risk of hypoparathyroidism |

# Chapter 7
# Rhematology

# General Introduction

## Definition:

- It's a connective tissue disorder characterized by articular & extra- articular manifestations, most probably autoimmune.

## Etiology

- Still questionable.
- But, most probably autoimmune.
- Genetic factors may play a role (+ ve family history & HLA association) .
- Environmental factors play some role.
- In SLE, add : Hormonal factor also may play an important role . Drug induced lupus : Hydralazine, INH, penicillamine, phenytoin

## Clinical Picture

More in female

**Exceptions: Gout, Polyarteritis nodosa, Ankylosing spondylitis ...)**

► Rheumatoid Arthritis ( female :male = 3: 1 )

► SLE( female: male 9:1 )

I. General manifestations.
II. Articular manifestations.
III. Extra-articular manifestations.

**I- General manifestations:**

► Fever.
► Fatigue.
► weight loss

**II-Articular manifestations:**

► Distribution .
► Description .
► Deformity.

## Distribution

**1- Number of the affected Joints : mono or poly arthritis e.g.**

- RA, SLE, chronic gout ……polyarthropathy.
- Acute.gout, septic arthritis …… monoarthropatby.

**2- Symmetrical or asymmetrical**

All are asymmetrical except Rheumatoid Arthritis & SLE - both are symmetrical.

**3- Small Joints or large Joints : RA & SLE ➔ small Joints.**

**4- Peripheral** (upper & lower limbs) or central (vertebral column & sacroiliac): RA & SLE: peripheral> central.

**5- Erosive or non-erosive:**

- Non erosive : inflammation of synovial membrane only.
- Erosive : erode & destroy the cartilage e.g. RA, chronic gout, septic arthritis

## Description:

1- Hotness
2- Redness
3- Tenderness
4- Swollen
5- Limitation of movement.

**Deformity:** If the lesion is erosive only.

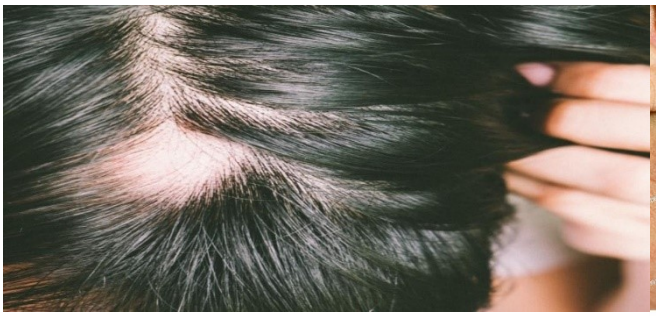
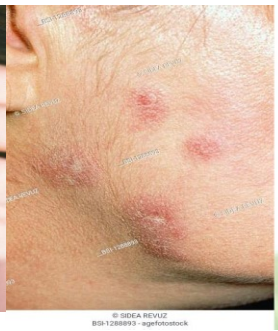
**Extra articular manifestations:**

- The same in any Rheumatologic disease, Except as regard to **skin & renal**

**Skin manifestations:**

**RA: Palmer erythema, SC nodule.**
**SLE:** (Malar rash, Butterfly rash, Discoid rash, Photosensitivity, Alopecia, Vacuities)

**Scleroderma: edema, Skin induraion then pigmentation.**
**In all diseases: pallor, vasculitis, Raynaud's phenomenon.**



**Renal manifestations:**

- RA: Amyloidosis.
- SLE: Glomerulonephritis ( Lupus nephritis)
- Scleroderma : Scleroderma renal crisis

**Cardiac manifestations:**

- Pericarditis.
- Myocarditis. Endocarditis (?)
- Systemic Hypertension
- Ischemic heart diseases.

**Chest manifestations.**

- Pleurisy.
- Interstitial pulmonary fibrosis.
- Pulmonary infarction
- Pulmonary Hypertension.
- Caplan's syndrome in RA.
- ARDS in SLE.

**CNS manifestations**

- Psychosis. Chorea.
- Neuropathy. Depression. Epilepsy.
- Myopathy.
- Cerebral vasculitis ~ stroke

**Eye manifestations:**
- Conjunctivitis.
- Scleritis.
- Anterior uveitis. ( NOT in rheumatoid arthritis)

**GIT manifestations:**
- Nausea & Vomiting.
- Esophagitis.
- Abdominal pain due to vasculitis.
- Gastritis & peptic ulcer.
- Pancreatitis .
- Hepatosplenomegaly.
- Dysphagia, malabsorption syndrome & constipation in scleroderma

**Blood manifestations:**
Anemia:
- Normocytic normochromic : autoimmune hemolytic anemia .
- Microcytic hypochromic :
  - Anemia of chronic disease.
  - Iron deficiency anemia : due to chronic blood loss

## Investigation

**1- x-ray:**
- Osteoporosis.

**In erosive diseases:**
► Narrow joint space.
► Deformity. (Mention)
X-ray chest: Pleural effusion.

**2- Aspiration of synovial fluid : Antibodies.**

| Disease | WBCs / $mm^3$ | Crystals / polarized light microscopy |
|---|---|---|
| Normal | < 200 | -ve |
| Non- inflammatory | < 2000 | -ve |
| Rheumatoid arthritis | 5000 - 50000 | -ve |
| Gout | 5000 - 50000 | ○ Needle shaped crystals of Na urate.<br>○ -ve birefringent. |
| Pseudogout | 5000 - 50000 | Rhomboid, +ve birefringent crystals of Ca pyrophosphate. |
| Septic arthritis | > 50000 | -ve |

3- Blood picture :

- Anemia: ↓↓ RBCs : normocytic or microcytic.
- WBCs: ↑↑ except in SLE & Felty's syndrome. MCQ
- ESR: ↑↑
- CRP: ↑↑ except in SLE . (CRP may ↑↑ in SLE with infection )
- SGOT & SGPT : ↑ , this is more likely to be drug induced rather than a result of the disease process.

## Serological tests

► **Nonspecific Antibodies:**
**Rhtumatology**
**(may be +ve in all rheumatologic diseases , inflammation or even in normal population)**

**I- Rheumatoid Factor (RF):**
- +ve in 70% of RA
- +ve in 25% of SLE & scleroderma.
- **Highest incidence of rheumatoid factor is found in: Sjogren's syndrome. ( 90% )**

**II- Lupus Erythematosis (LE):**
- +ve in 80% of SLE.
- +ve in 20% of RA.

**III- Antinuclear antibody (ANA): MCQ**
- +vein 95-100% of SLE ( most sensitive)
- +ve in 90% of scleroderma.
- +ve in 30% of RA

## Specific Antibodies:

- Anti CCP (Cyclic Citrullinated Peptide, NOT ContraCeptive Pills) Rheumatoid arthritis©.
- Anti-double stranded DNA( dsDNA) ¢ SLE. (80%)
- Anti-sm (Anti-smith Ab, NOT anti-smooth muscle Ab) ¢ specific to SLE.
- Anti Ro Anti La specific to SLE , Sjogren's syndrome.
- Anti-histone antibody: drug induced SLE e.g. hydralazine.
- SCL 70 antibody scleroderma.
- Anti- centromere CREST syndrome.
- Anti-RNP (ribonucleoprotein) : associated with mixed connective tissue disease.
- CANCA ( Anti-Neutrophil Cytoplasmic Ab) Wegener granulomatosis (90% )

► Complement:
Low complement levels indicate consumption as in active SLE.

## Treatment

**I. General:** Rest, Physiotherapy.

**II. Medical:**

- **Cortisone.**
- NSAIDs.
- DMARDs: Gold, Penicillamine .....
- Immunosuppressive drugs : (methotrexate , cyclophosphamide )

**III. Treatment of complications : e.g.**

- End stage kidney damage in a cases of SLE dialysis or kidney transplant.
- Renal crisis in a cases of scleroderma : ACE inhibitor is the drug of choice " Life saving

# Spetic Arthritis

## Bacterial septic arthritis

a) Non-gonococcal septic arthritis (staph aruas)
b) Gonococcal arthritis:

## Non-gonococcal septic arthritis

**Clinical picture:**

Flu-like symptoms: Fever, Headache, Malaise, Anorexia.

- Acute joint pain: usually single joint especially the knee. (80% of cases is monoarthritis)
- Signs of inflammation: hotness, redness, tenderness, swelling, limitation of movement.

## Gonococcal arthritis:

- Most common in young healthy adults ( females > males )
- Caused by N gonorrhea.
- Clinical features :
  1. Migratory polyarthritis.
  2. Tenosynovitis.
  3. Skin lesions, usually as a small macule or papule on the distal extremities.

## Investigation

- Blood : Leukocytosis , ESR , blood culture.
- X.ray : articular erosions may occur.
- Synovial fluid examination : +ve culture , j neutrophils.

The synovial fluid should be examined for crystals, because gout & pseudogout can resemble septic arthritis clinically.

## Treatment:

- Antibiotic (IV): according culture & sensitivity test.e.g. : Ox, Clox, Dicloxacillin or vancomycin for staph. For about 4 weeks.
- Analgesics.
- Drainage of the infected joint daily.

# Familial Mediterranean Fever

## Definition

FMF is an intermittent febrile disorder with inflammatory serositis, arthritis, and rash.

## Etiology

- **FMF is a hereditary inflammatory disorder. It is an autoinflammatory disease caused by mutations in the gene MEFV, localized in chromosome 16.**
- **Eastern Mediterranean persons ( especially Arabs, Turks, Greeks, Armenians, and Sephardic Jews) are most frequently affected**

## Clinical picture

- More than 80% of all patients have their first attacks before they are 20 years old.
- Initial attack is very rare after age 40.
- Most attacks involve fever.
- The attacks last from 6 hours to 3 days, arthritis may last for several weeks.
- Disease free intervals may last days or months.

**There are 7 types of attacks:**

1. Abdominal attacks affect the whole abdomen with signs of peritonitis, and acute abdominal pain like appendicitis. They occur in 95% of all patients and may lead to unnecessary laparotomy.
2. Joint attacks mainly occur in large joints, especially in the legs. Usually, only one joint is affected. (75%)
3. Chest attacks include pleurisy ( 40 % ) and pericarditis (rare).
4. Scrotal attacks due to inflammation of the tunica vaginalis occurs in up to 5% and may be mistaken for acute scrotum (i.e. testicular torsion).
5. Myalgia
6. Fever without any of the other symptoms listed above (25%).

## Complications

**Amyloidosis** with renal failure: amyloid protein is produced in very large quantities during attacks, and at a low rate between them, and accumulates mainly in **the kidney, as well as the heart & gastrointestinal tract.**

## Investigations:

- The diagnosis is clinically made on the basis of the history of typical attacks. No specific test is available to detect familial Mediterranean fever.
- A genetic test : to detect mutations in the MEFV gene.
- Markers of inflammation :
- Leukocytosis, elevated ESR & C-reactive protein levels.
- Monitoring the renal function is important especially in patients with a long history of attacks

## Treatment

- Symptomatic reliefs the goal of therapy e.g. NSAIDs
- **Colchicine as prophylaxis:** It reduces the frequency of attacks, protect against amyloidosis & stabilizes the proteinuria.
- If the symptoms are not controlled by colchicine, then alpha-interferon, infliximab may be recommended.
- Corticosteroids are ineffective
- NSAID
- Cholchicine

# Bechet disease

- It is a systemic vasculitis of unknown etiology which can involve large, medium and small arteries and veins.
- There is an association with HLA-B5

## Clinical picture

Highly variable with recurrence and remissions

► **Diagnostic criteria :**

**Recurrent painful oral ulcer (100%):** Initial criteria for diagnosis require the occurrence of at least 3 episodes of painful **oral aphthous ulcers/year.** To confirm the diagnosis, **at least two of the following must also be** demonstrated:

1. Recurrent painful genital ulcers that heal with scarring. (80%)
2. Recurrent anterior uveitis or retinal vasculitis (60%) : may lead to blindness.
3. Skin lesions (60%) : including erythema nodosum or acneiform lesions.
4. Positive pathergy skin test : defined as the formation of a sterile erythematous papule 2 mm in diameter or larger that appears 48 hours following a skin prick with a sharp sterile needle.

## Other features

**Arthritis:** non-erosive, asymmetrical oligoarthropathy, involves big joints.

Vasculopathy:

- Unlike most forms of vasculitis, both arteries and veins, of all sizes, are involved.

Venous thrombosis, Budd-chiari syndrome, IVC & SVC clot.

Aneurysms of pulmonary arteries may lead to massive lung hemorrhage

GIT: Ulcerations may occur anywhere in the GIT. The terminal ileum and cecum are common sites. It may be difficult to distinguish from inflammatory bowel disease (such as Crohn's disease).

CNS: aseptic meningitis, strokes, cerebellar ataxia, cranial nerve palsies, psychiatric disorders.

Cardiac: coronary vasculitis, pericarditis.

**Investigations:** Nonspecific, reflect inflammatory state (ESR, CRP)

**Treatment:** symptomatic treatment

NSAIDs for arthritis and thrombophlebitis.

Systemic anticoagulant is often used in patients with thrombosis. Mouth ulcers : oral washes, topical steroids.

Genital ulcers: topical steroids, colchicine

Anterior uveitis: systemic steroids, colchicine, cyclosporine.

# Gout

## Definition

Gout is a disease which is characterized by tissue deposition of Monosodium urate crystals due to hyperuricemia that results in

**Gouty arthritis.**

- Tophi

**Gout is the most common type of inflammatory monoarthritic**

## Ethology:

► increase Production of uric acid (Metabolic gout).
► decrease Excretion of uric acid (Renel Gout)

**(A) : increase Production:**

- lry: Enzyme defect :-l.--l.- HGPRT
- 2ry : leukemia, lymphoma, sarcoidosis, severe psoriasis.

**(B) : decrease Excretion:**

- lry: Idiopathic ( Isolated tubular defect).
- 0 2ry:

**Renal Failure**

Acidosis e.g. increased lactic acid production from alcohol, starvation.
- Hyperparathyroidism, Hypothyroidism.
Dehydration ➔Renal Flow.
Drugs: e.g. Diuretics, Pyrazinamide, Low dose aspirin.

## Acute gouty arthritis: 3D

**Precipitating factors:**

- Excess protein, Alcohol.
- Drugs e.g. thiazide, Lasix.
- Surgery, Trauma.
- Infection.
- Dehydration.

## Distribution:

- Monoarthritic.
- Usually in the first metatarsophalangeal joint (of the big toe).
- Other sites : the ankle, the dorsum of the foot, the knee & occasionally the joints of upper extremity
- Not erosive

## Description:

- Hotness, Redness, Tenderness, Swollen & limitation of movement.
- Acute attack is severely painful, it often develops overnight and reaches a peak within hours ( waking the patient in the early morning)
- Dramatic relief of pain with colchicine is suggestive.
- Slight fever & chills may be present

## Deformity: No

## Investigations:

**Uric acid (N= 2.5 - 7mg %): > 7 mg% in male > 6 mg% in female**

It is a biochemical hallmark of gout, but not by itself diagnostic for gout.
Minority of patients showing acute gout with normal serum uric acid, so, normal serum uric acid does not exclude the diagnosis of gout.

## X-ray:

- Soft tissue swelling around the affected joints.
- Punched out lesion & deformity in chronic gout
- Aspiration of synovial fluid ( Arthrocentesis ) :
  - Urate crystals by Polarized microscopy.
  - It is the single most useful diagnostic study in initial evaluation of monoarthritis.

## Treatment

- NSAID
- Cholchicine

# Chapter 8

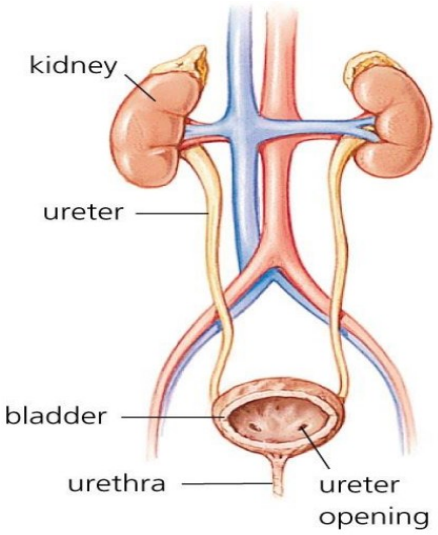
# Infectious

# Urinary Tract Infections

**Definition**

The presence of a "**significant number**" of bacteria in urine + **Symptoms**.

## Classification (Localization)




- **Lower U.T.I**
  - Urethritis
  - Cystitis
- **Upper U.T.**
  - Pyelonephritis

## Lower UTI

**Infection of the lower GU tract.**

**Risk factors:**

- Female gender. (short urethra)
- Sexual Activity. (honeymoon cystitis)
- Urinary catheterization.
- Diabetes.
- Pregnancy.
- Impaired bladder emptying.

## Clinical picture

- Common presenting symptoms include **dysuria, frequency, urgency,** and **suprapubic pain**.
- **Systemic signs** (high fever, chills) are usually **absent**.
- On exam, there is suprapubic tenderness but no flank tenderness.

## Causes

**E-coli** in >80% (most common)

**Enterobacteriaceae** such as Proteus, Klebsiella, enterococci.

**Staph. saprophyticus** in young women.

## Diagnosis

- **Urinalysis** (looking for WBCs, RBCs, protein, and bacteria; WBCs is the most important with more than 5-10 WBCs).
- Positive **leukocyte esterase** signifies significant **WBCs** and positive **nitrites** indicate the presence of Enterobacteriaceae which converts urinary nitrates to nitrites.
- Urine **culture** with >100,000 colonies of bacteria per mL of urine confirmatory but **not always necessary** with characteristic symptoms and a positive urinalysis.
- Sterile **pyuria** and **-ve culture** suggest urethritis.

## Upper UTI (Pyelonephritis)

- An acute patchy, most often unilateral, pyogenic infection of the kidney.
- **Causes** include: Ascending UTI, hematogenous spread to kidney.
- Predisposing factors include:
  - **Obstruction** due to strictures, tumors, calculi, BPH.
  - DM.
  - Pregnancy.
  - **Vesicoureteral reflux in infants.**
- **E. coli** is the most common pathogen; others include **Klebsiella, Proteus,** and **Enterococcus.**
- **Clinical findings** include **chills, fever, flank pain, nausea, vomiting, costovertebral angle tenderness,** increased frequency in urination, and dysuria.

## Complicated pyelonephritis

- Involves progression of the initial pyelonephritis to **renal corticomedullary abscess**, **perinephric abscess**, **papillary necrosis**.
- Patients can develop **sepsis** with **multiorgan failure**, **shock**, and **renal failure**.

## Treatment

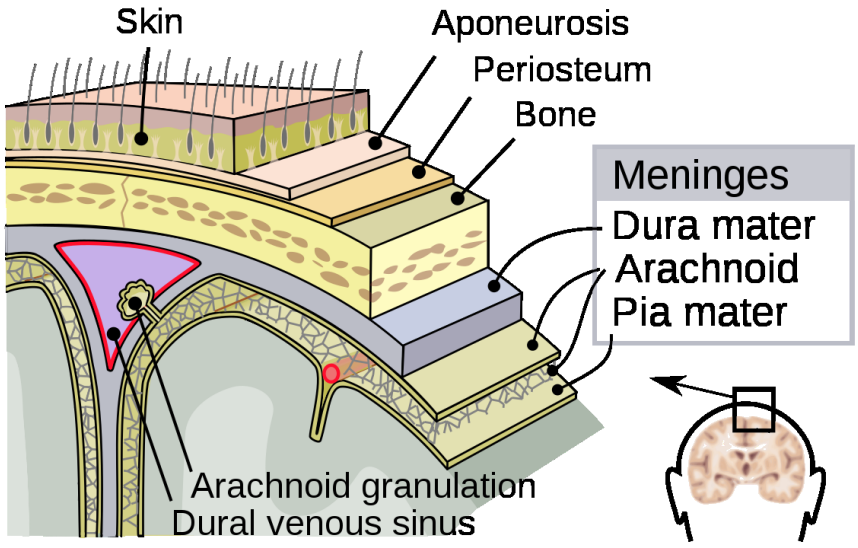
- **Uncomplicated cystitis:**
  TMP/SMX, nitrofurantoin.
- **Complicated cystitis:**
  **Oral fluoroquinolones,** but more severe cases may require intravenous broad-spectrum antibiotics (**ceftriaxone**) while awaiting culture results.
- Infections associated with factors that increase the risk of **antibiotic resistance** or **treatment failure**.
- Such factors include DM, CKD, immunocompromised state, or urinary tract obstruction; hospital-acquired infection; or infection associated with a procedure (cystoscopy) or indwelling foreign body (catheter, stent).
- **These patients should have urine culture prior to therapy.**
- **Stable patients** with **uncomplicated pyelonephritis:** can be treated with oral antibiotics (usually a fluoroquinolone)
- Unstable patients and those with complicated infection require intravenous antibiotics (**ceftriaxone**).

# Meningitis & Encephalitis

## Meningitis

Is an **infection or inflammation of the meninges**, which is the **connective tissue covering the central nervous system** (CNS).

- Most meningitis cases are caused by **Viruses** .




## Viral meningitis

- Is a usually self-limited inflammation. Almost 90% of cases are caused by **non-polio enteroviruses, such as echovirus and coxsackievirus.**
- Viral meningitis can present with **a viral prodrome of constitutional and upper respiratory symptoms with low-grade fever .**
- Focal neurologic signs are **not usually seen** .
- The cerebrospinal fluid (CSF) will show **pleocytosis with lymphocytic predominance** .
- CSF gram stain will **not** show any organisms .
- Treatment **is supportive; in most patients, symptoms resolve within 7-10 days.**

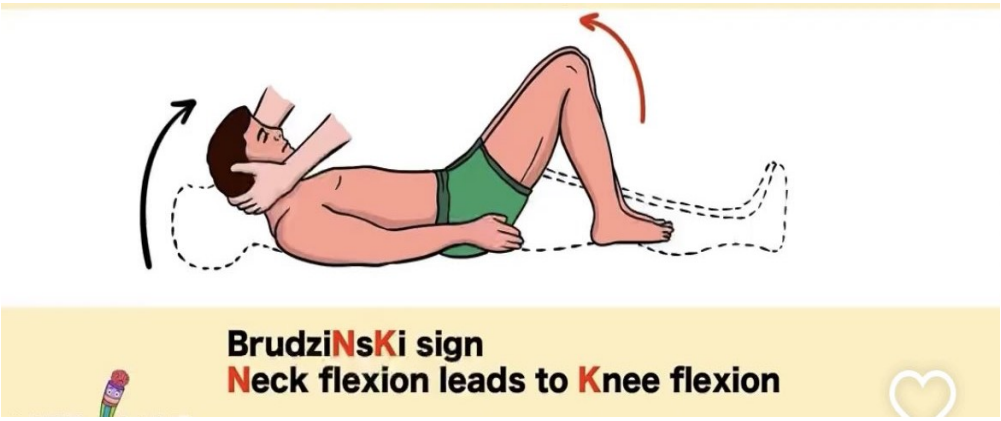
## Etiology

- **Bacterial causes of Meningitits:**
  1. **Streptococcus pneumoniae** → the most common cause.
  2. **Neisseria meningitidis** → causes life-threatening meningitis.
  3. **Hemophilus influenzae** (Rare).
  4. **Listeria monocytogenes** is more common in neonates.
  5. **Staphylococcus aureus** → **neurosurgery** because instrumentation and damage to the skin introduce the organism into the CNS.
  6. **Cryptococcus** → **HIV positive** and who have profound decreases in T-cell counts to levels.
  7. **Tuberculosis and syphilis** are also associated with meningitis

## Clinical Presentation

- All forms of meningitis present with **fever, photophobia, headache, nuchal rigidity** (neck stiffness, positive Kernig and Brudzinski signs), as well as nausea and vomiting.
- Most common Focal neurological deficit is **visual field and cranial nerve deficits**.
- The most common long-term neurologic deficit from bacterial meningitis **is damage to the 8th cranial nerve.**
- Rash is associated with several types of meningitis, **Petechial rash is suggestive of Neisseria** .

## Meningeal Signs





## Diagnosis

**- Lumbar puncture**

- Is essential for establishing the diagnosis.
- If lumbar puncture is **delayed >20-30** minutes for any reason, the best initial step is to **give an empiric dose of antibiotics.**

**- CT scan**

- CT scan of the head is **the best initial** diagnostic test if the patient has **papilledema, focal motor deficits, new onset seizures, severe abnormalities in mental status, or immunocompromised status** (HIV, immunosuppressive medications, post-transplantation).

**Cerebrospinal Fluid Interpretation**

| | Normal | Bacterial | Viral | Fungal | TB | Autoimmune |
|---|---|---|---|---|---|---|
| Appearance | Clear | Cloudy/Turbid | Clear | Clear/fibrin web | Clear/Fibrin web | Clear |
| Opening Pressure | 5-20 | Elevated | Normal or Slightly Elevated | Elevated | Variable | Normal |
| Leukocytes | <5 | >1,000 | 25-2,000 | 10-1,000 | 10-1,000 | 10-1,000 |
| Differential | 0 | Neutrophil predominant | Early- Neutrophil Late- Lymphocytic | Lymphocyte Predominant | Lymphocyte Predominant | Lymphocyte Predominant |
| Glucose | 45-85 | Low | Low/Normal | Low | Low | Normal |
| Protein | 15-45 | High | High | High | Very High | High |
| Cultures | Negative | Positive | Negative | Positive (Fungal) | Positive (AFB) | Negative |

## Treatment

- Initial treatment is **started without knowing the results** of culture.
- Empiric therapy of bacterial meningitis in adults **is best achieved with vancomycin** plus a third-generation **cephalosporin such as ceftriaxone**.
- Dexamethasone (corticosteroid) therapy for patients with bacterial meningitis **decreases mortality and rates of deafness.**

## Prophylaxis

- The close contacts of patients with Neisseria meningitidis should receive either **Rifampin or Ciprofloxacin .**
- **Ceftriaxone and azithromycin** are considered alternatives.
- Prophylaxis should be **given within 24 hours** of identification o the source case .
- It is given to the person who is considered a "**close contact**".
  **Like:** **Household contacts**, anyone with possible salivary contact.

## Encephalitis

▪ **Definition** :

Is **an infection of the brain**, **whether in the meninges or the brain parenchyma**.
**most cases are caused by viruses, with herpes simplex (usually type I [HSV-1]) the most common.**

▪ Any **bacterial, protozoal, or rickettsial** infection can cause encephalitis .
▪ **Varicella-zoster virus, CMV, enteroviruses** are significantly less common causes.

## Presentation

- Patients present with **fever and headache** but these findings are nonspecific.
- **Altered mental status with fever and headache is the primary clue to the diagnosis.**
- Patients may also have **nuchal rigidity and focal neurological abnormalities.**

## Diagnosis

**- Best initial test:**

- **CT scan of the head**
  (HSV has a predilection for involvement of the temporal lobes).

- **MRI** is abnormal in 90% .

**- Most accurate test :**

**PCR of the CSF** for herpes simplex (detecting viral DNA) is 95% to 99% sensitive and specific .

## Treatment

– Empiric treatment with **intravenous acyclovir** should be started while **awaiting PCR results.**
– Acyclovir-resistant herpes is treated with **foscarnet.**

## Case Scenario

- 24 YO female, presented with headache, fever & deterioration in level of consciousness, brain CT was free, the L.P s (values shows WBCs count 1000 predominantly Neutrophil, Glucose 30 mg/dl). What is the most likely diagnosis?

# Infective Endocarditis

## IE (Etiology)

- **Definition:** Endocarditis is an infection of the valve of the heart leading to a fever and a murmur.

- **Risk factors:**
  1- Artificial heart valves.
  2- Damaged heart valves diseases.
  3- A history of endocarditis.
  4- A history of illegal IV drug use.
  5- Poor dental health/ Dental procedure.

## Microorganisms

- **Viridans group streptococci** (Streptococcus sanguinis) are the most common cause of IE following **dental procedures.**
- **Staphylococcus epidermidis: prosthetic valve.**
- **Enterococcus: GI or GU procedures.**
- **Staphylococcus aureus** is the responsible pathogen for more than half of IE cases in **IVDU**.
- **S. gallolyticus (S bovis biotype 1)**, all such patients should have further evaluation with **colonoscopy** to look for underlying occult malignancy **(colon cancer)**.

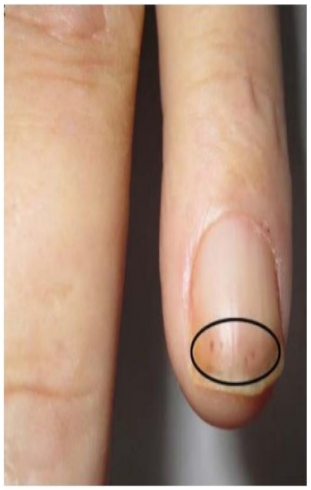
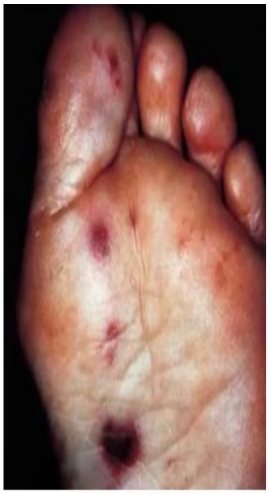
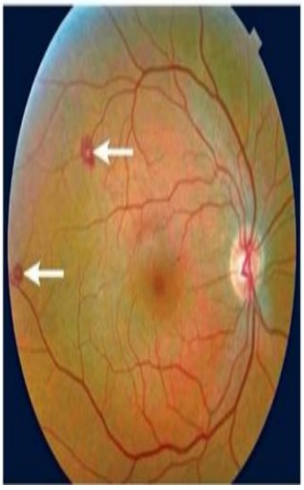
## Endocarditis types

1. **Acute endocarditis**
   a) Most commonly caused by **Staphylococcus aureus** (highly virulent).
   b) Occurs on a **normal** heart valve.
   c) If untreated, fatal in **less than 6 weeks**.

2. **Subacute endocarditis**
   a) Caused by less virulent organisms, such as **Streptococcus viridans** and **Enterococcus**.
   b) Occurs on **damaged** heart valves.
   c) If **untreated**, takes much **longer than 6 weeks** to cause death.

## Clinical presentation

- Fever.
- New murmur or change in a murmur.
- Complications of endocarditis:
  - **Splinter** hemorrhages.
  - **Janeway lesions** (flat and painless).
  - **Osler nodes** (raised and painful).
  - **Roth spots** in the eyes.
  - Brain (mycotic aneurysm).
  - Kidney (hematuria, glomerulonephritis).
  - Conjunctival petechial.
  - Splenomegaly.
  - Septic emboli to the lungs.

## IE (Diagnosis)

- It is recommended that a minimum of 3 blood cultures be obtained from separate venipuncture sites (not from a vascular catheter) over several hours prior to initiating antibiotic therapy.
- HACEK difficult to culture that cause endocarditis:
- Haemophilus aphrophilus.
  - Haemophilus parainfluenzae.
  - Actinobacillus. - Cardiobacterium.
  - Eikenella. - Kingella.
- The most common causes of culture negative endocarditis are **Coxiella and Bartonella.**

| Infective endocarditis – modified Duke criteria | |
|---|---|
| **Diagnostic criteria for IE** | **Major criteria**<br>• Blood culture positive for typical microorganism (eg, *Streptococcus viridans*, *Staphylococcus aureus*, *Enterococcus*)<br>• Echocardiogram showing valvular vegetation<br><br>**Minor criteria**<br>• Predisposing cardiac lesion<br>• Intravenous drug use<br>• Temperature >38 C<br>• Embolic phenomena<br>• Immunologic phenomena (eg, glomerulonephritis)<br>• Positive blood culture not meeting above criteria<br><br>**Definite IE**<br>2 major **OR** 1 major + 3 minor criteria<br><br>**Possible IE**<br>1 major + 1 minor **OR** 3 minor criteria |
| **Clinical findings (frequency)** | • Fever (>90%)<br>• Heart murmur (85%)<br>• Petechiae (≤50%)<br>• Subungual splinter hemorrhages (<50%)<br>• Osler nodes, Janeway lesions (<50%)<br>• Neurologic phenomena (embolic) (≤40%)<br>• Splenomegaly (≤30%)<br>• Roth spots (retinal hemorrhage) (<5%) |

## IE (Treatment)

- **Treatment:**
  - **The best initial empiric** therapy is **vancomycin and gentamicin** .
  - Add **rifampin for prosthetic valve endocarditis with Staphylococcus**.
  - **When culture results are available,** treat as indicated in the table "Treatment of Endocarditis."
  - **Treatment of Resistant Organisms:** Add an **aminoglycoside and extend the duration** of treatment.
  - **Treatment of Culture Negative Endocarditis:** Use **ceftriaxone** for the HACEK group of organisms.

## Treatment

**The criteria for surgery (valve replacement) in infective endocarditis:**

- **CHF** from ruptured valve or chordae tendineae.
- **Prosthetic valves.**
- Fungal endocarditis.
- Abscess.
- AV block.
- Recurrent emboli while on antibiotics.

- **The single strongest indication for surgery is acute valve rupture and CHF.**

| Organism | Treatment |
|---|---|
| Viridans streptococci | Ceftriaxone for 4 weeks |
| Staphylococcus aureus (sensitive) | Oxacillin, nafcillin, or cefazolin |
| Fungal | Amphotericin and valve replacement |
| Staphylococcus epidermidis or resistant Staphylococcus | Vancomycin |
| Enterococci | Ampicillin and gentamicin |

## IE (Prophylaxis)

- **Two features are needed to establish the need for prophylaxis:**

**A. Significant cardiac defect:**

- **Prosthetic valve.**
- **Previous endocarditis.**
- **Cardiac transplant recipient with valvulopathy.**
- **Unrepaired cyanotic heart disease.**

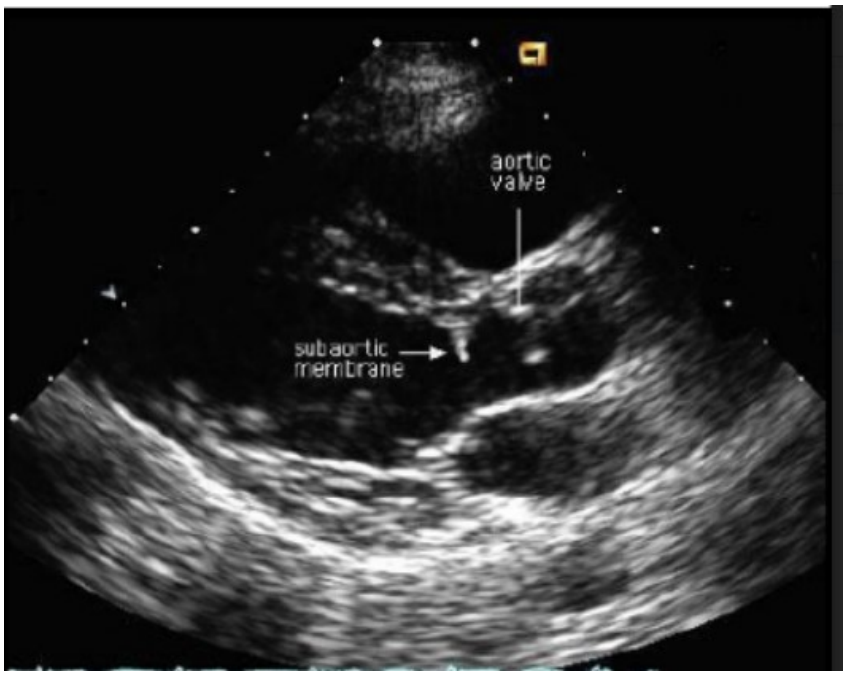
**B. Risk of bacteremia:**

- **Dental work with blood.**
- **Respiratory tract surgery that produces bacteremia.**

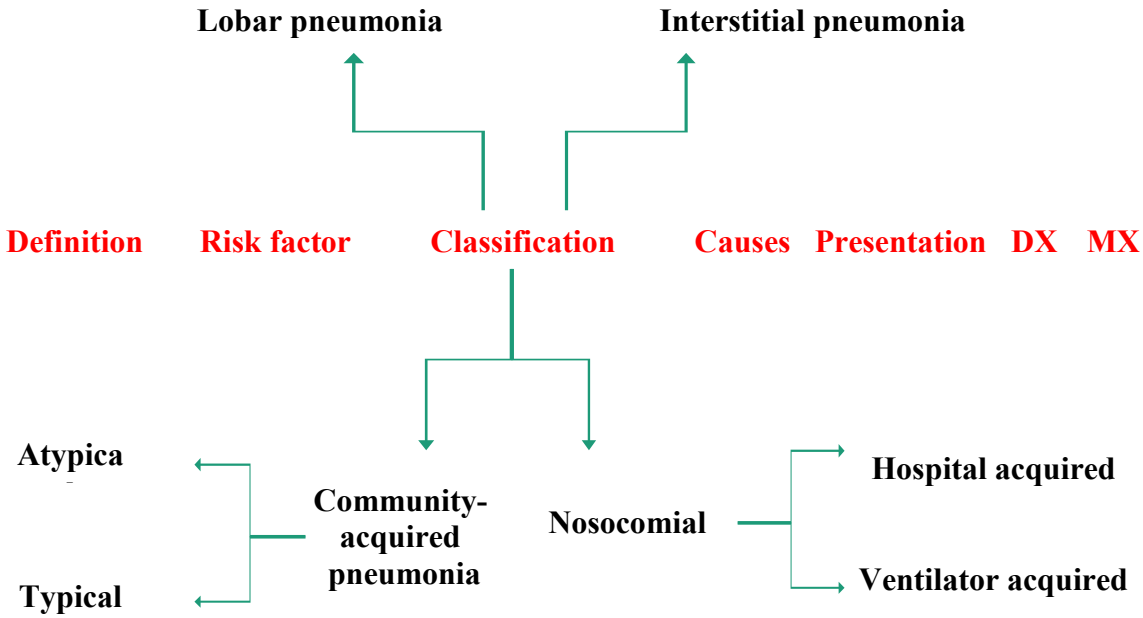
- **The best initial management is amoxicillin prior to the procedure. If the patient is penicillin allergic, then azithromycin, or clarithromycin, clindamycin.**
- **Endoscopic and genitourinary procedures do not need prophylaxis.**

## Clinical Case Scenario

- **32 Y.O Pt with a HX of IV drug abuse & renal dialysis, was presented with fever, malaise & endurance fatigue. Chest auscultation has revealed pan-systolic murmur. An ECHO showed the following, what is your spot DX?**



# Pneumonia




## Definition

**Pneumonia is an infection of the lung parenchyma**

**It is the 6th leading cause of death in the United States.**

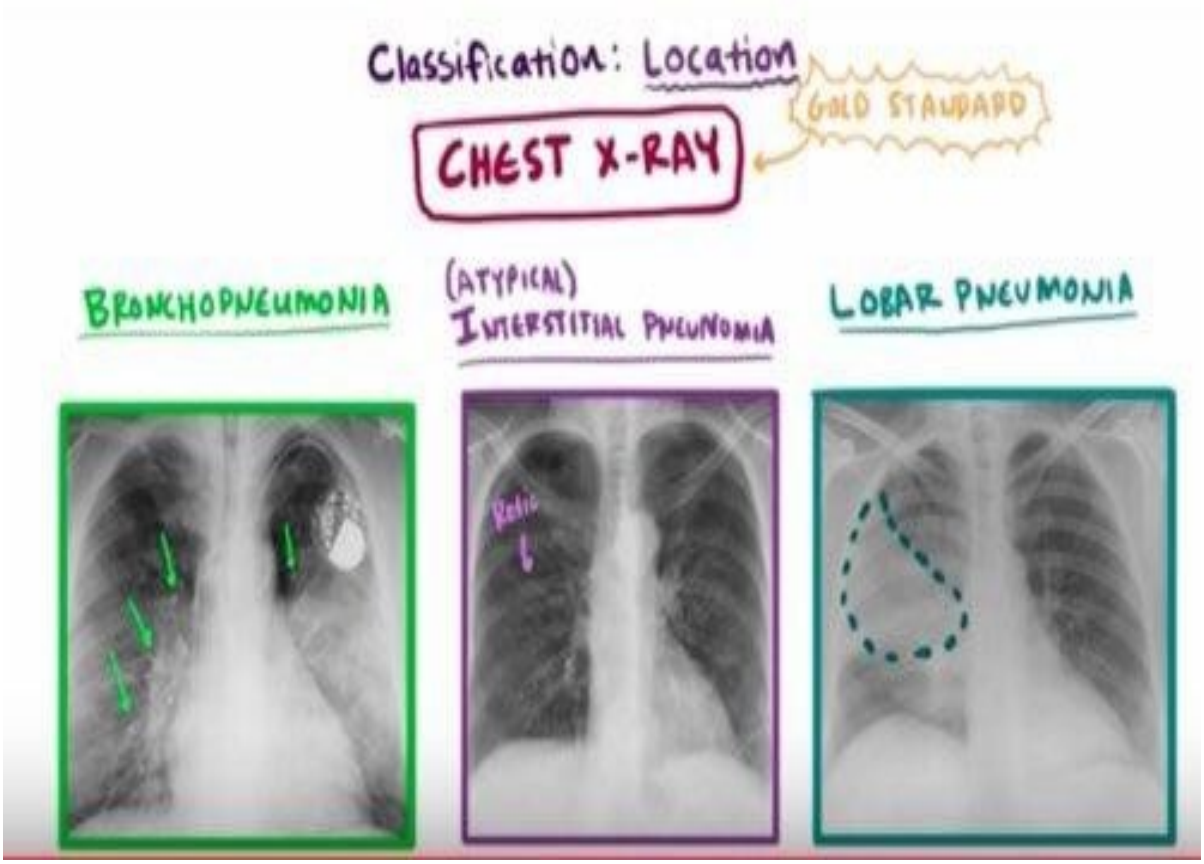
## Risk factors

- Cigarette smoking
- Diabetes
- Alcoholism
- Malnutrition
- Obstruction of the bronchi from tumors
- Any immunosuppression condition
- AGE >65

## CLINICAL Classification

1. **Community acquired pneumonia**
   a) Typical pneumonia
   b) Atypical pneumonia

2. **Nosocomial pneumonia**
   a) Hospital acquired pneumonia
   b) Ventilator acquired pneumoniaCommunity acquired pneumonia




## Community acquired pneumonia

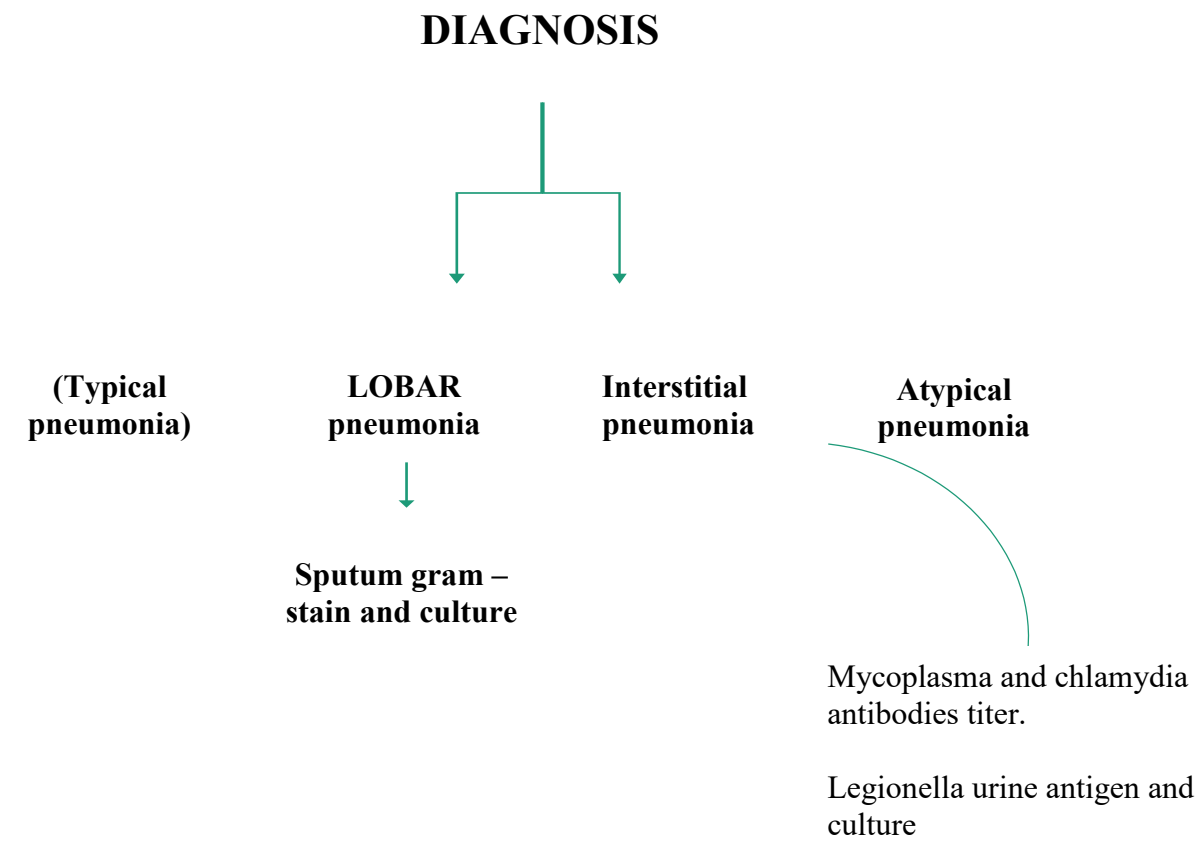
**Typical pneumonia**
**Presentation is typical: productive cough , pleuritic chest pain, dyspnea**

**Causes: " سحسك "**
1-Strep. Pneumonia (MC) RUSTY SPUTUM
2-Hemophilus influenza
3-staph.aerus purulent sputum
4-Klebsiella current jelly sputum

**ATYPICAL Pneumonia ( walking pneumonia )**
**Presentation is ATYPICAL ( mainly constitutional symptoms ) plus dry cough**
**causes" كمل "**
1. Chlamydia
2. Mycoplasma pneumonia (MC)
3. Legionella
4. VIRUSES

## Legionella

| Overview of *Legionella* pneumonia | |
|---|---|
| **Clinical features** | • High fever with relative bradycardia<br>• Headache & confusion<br>• Watery diarrhea |
| **Laboratory findings** | • Hyponatremia<br>• Sputum Gram stain showing many neutrophils, but few or no organisms |
| **Diagnosis** | • *Legionella* urine antigen test |
| **Treatment** | • Respiratory fluoroquinolones or newer macrolides |



## DIAGNOSIS

- **The most important initial test for any type of pneumonia is the chest x-ray.**
- **The most important initial clue to the diagnosis is according to radiological classification**
- **S. pneumoniae (and other causes of "typical" pneumonia) usually appear as a lobar pneumonia**
- **Interstitial infiltrates are associated with viral, Mycoplasma, Chlamydia, Legionella pneumoniae.**

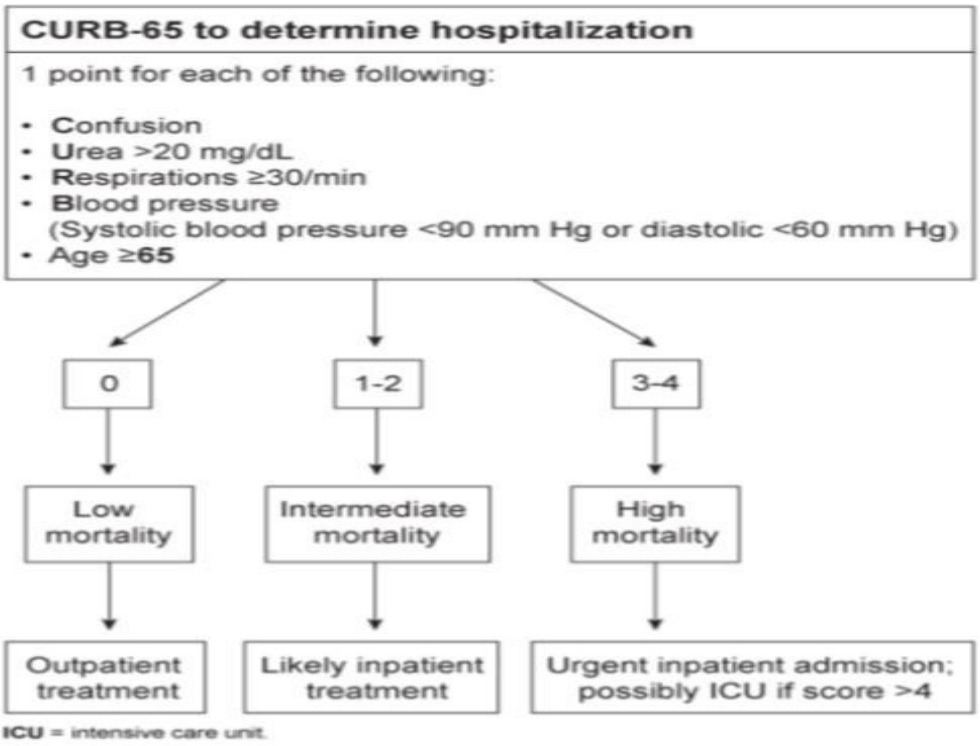
**- Sputum should be obtained for both Gram stain as well as culture. Sputum culture is the most specific diagnostic test for lobar pneumonia, such as with S. pneumoniae, Staphylococcus, Klebsiella, and Haemophilus.**

**The other organisms (viral, Mycoplasma, Chlamydia, Coxiella, etc.), the so-called "atypical" organisms, will not show up on a Gram stain or regular bacterial culture for various reasons**

- **Organism-specific diagnostic methods are as follows:**
- **Mycoplasma AND Chlamydia pneumoniae : All of these are diagnosed with specific serologic antibody titers.**
- **Legionella: Specialized culture media with charcoal yeast extract and urine antigen tests.**

### TREATMENT

- **Treatment depends on whether the patient has a mild disease that can be treated as an outpatient or a more severe illness that must be treated with IV antibiotics as a hospitalized inpatient.**
- **Patients with CAP are often risk stratified using the pneumonia severity index or CURB-65 criteria to help guide treatment and treatment location (home, medical floor, intensive care unit) decisions**



| Empiric treatment of CAP | |
|---|---|
| Outpatient | • Macrolide or doxycycline (healthy)<br>• Fluoroquinolone* or beta-lactam + macrolide (comorbidities) |
| Inpatient (non-ICU) | • Fluoroquinolone* (IV)<br>• Beta-lactam + macrolide (IV) |
| Inpatient (ICU) | • Beta-lactam + macrolide (IV)<br>• Beta-lactam + fluoroquinolone* (IV) |

*Respiratory fluoroquinolones (eg, levofloxacin, moxifloxacin) are required.
CAP = community-acquired pneumonia; ICU = intensive care unit; IV = intravenous.

## Nosocomial pneumonia

- **Hospital acquired pneumonia: onset >72 hr after hospitalization**
- **Ventilator acquired pneumonia: Onset >48 hr after ventilation**

**Causes**: gram negative( pseudomonas ) and MRSA

**MX:** in hospital coverage of MRSA : VANCOMICNE coverage of pseudomonas : Piperacilin -Tazobactam - Cefepime - Gentamicine .

## Recurrent pneumonia

**Different lung regions: immunodeficiency**

**Same lung region: airway obstruction (lung cancer)**

**Recurrent right middle or right lower lobe pneumonia: Aspiration**

## Aspiration

**2 TYPES**

1. **Aspiration pneumonia : aspiration of oropharyngeal normal flora (Anaerobic ,staph aureus, klebsilla).**

2. **Aspiration pneumonitis : aspiration of gastric acid**

## Risk factor of Aspiration

• Decrease consciousness
• seizure
• Alcoholics
• dysphagia

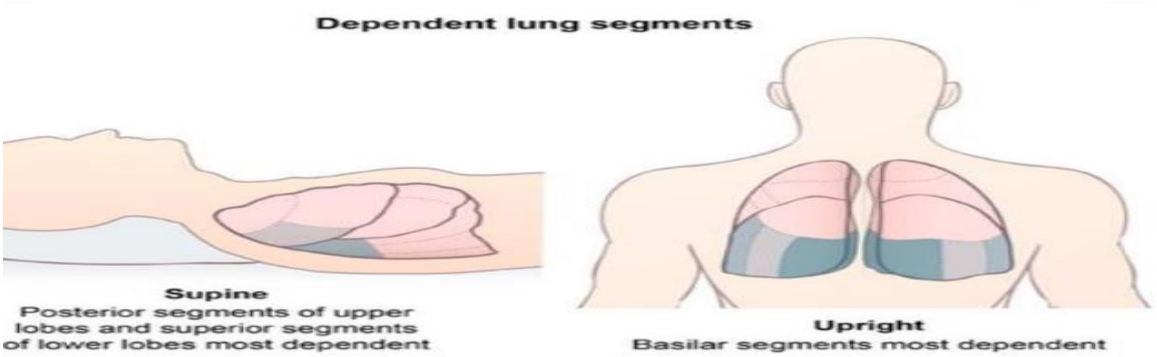
## Diagnosis

**Chest x-ray**

**According to position**

**Posterior segment of upper lobe and superior segment of lower lobe….. Supine position**

**right lower lobe …..Erect position**




## Management of aspiration

- **Aspiration pneumonia : broad spectrum antibiotic Piperacillin- tazobactam**
- **Aspiration pneumonitis : 02 supplement and suction**

## Lung abscess

- **Lung abscess is necrosis of the pulmonary parenchyma caused by microbial infection.**
- **90% involve anaerobes . The most commonly implicated anaerobes are Peptostreptococcus, Prevotella, and Fusobacterium species, which are oral anaerobes found in the gingival crevices.**
- **45% only anaerobic, 45% mixed with aerobes, 10% aerobes only.**
- **Aerobic bacteria, most frequently involved are S. aureus, E. coli, Klebsiella, and Pseudomonas.**
- **90% of cases have a clear association with periodontal disease with predisposition to aspiration (altered sensorium, seizures, dysphagia).**

## Presentation

- Patients present with the usual symptoms of pulmonary infection, such as fever, cough, sputum production, and chest pain, and foul-smelling sputum (60-70% of cases)

## Diagnosis and management

- **Chest x-ray in an abscess will often show a thick- walled cavitary lesion.**
- **In the absence of specific microbiologic diagnosis, clindamycin is good empiric coverage for the "above the diaphragm" anaerobes most often found.**

# Tuberculosis

## Tuberculosis (TB) is an infection with Mycobacterium tuberculosis.

- TB is spread exclusively by person-to-person transmission by means of respiratory droplet infection.
- Impairment of T-cell-mediated cellular immunity is the most significant defect associated with reactivation. This is why steroid use, organ transplantation, leukemia, lymphoma, and HIV are such important risk factors

## Presentation:

– Patients present with cough, sputum, fever, and an abnormal lung exam.
– Weight loss is common because of the chronicity of the infection.
– Night sweats may occur.
– TB can occur outside the lungs. **Miliary TB is caused by the hematogenous spread of Mycobacterium tuberculosis.**

It may arise during primary infection or with reactivation.
**- Presentation depends on site involved. Any part of the body can be involved**

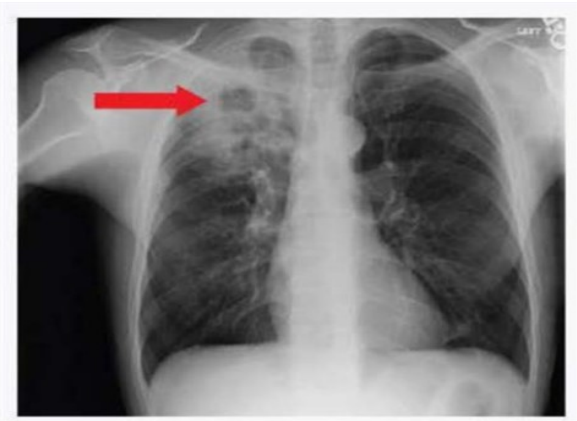
## Diagnosis:

– Chest x-ray is the best initial test. Apical involvement with infiltrates and sometimes cavitation is the most common finding

**Reactivation TB typically occurs at the site of latent infection (apical lobes).**

– **Sputum examination with specific staining for acid-fast bacilli (AFB) allows specific diagnosis. AFB stain has limited sensitivity, and you need 3 negative smears to reach >90% sensitivity. AFB-positive sputum staining is usually the trigger to start therapy for TB.**

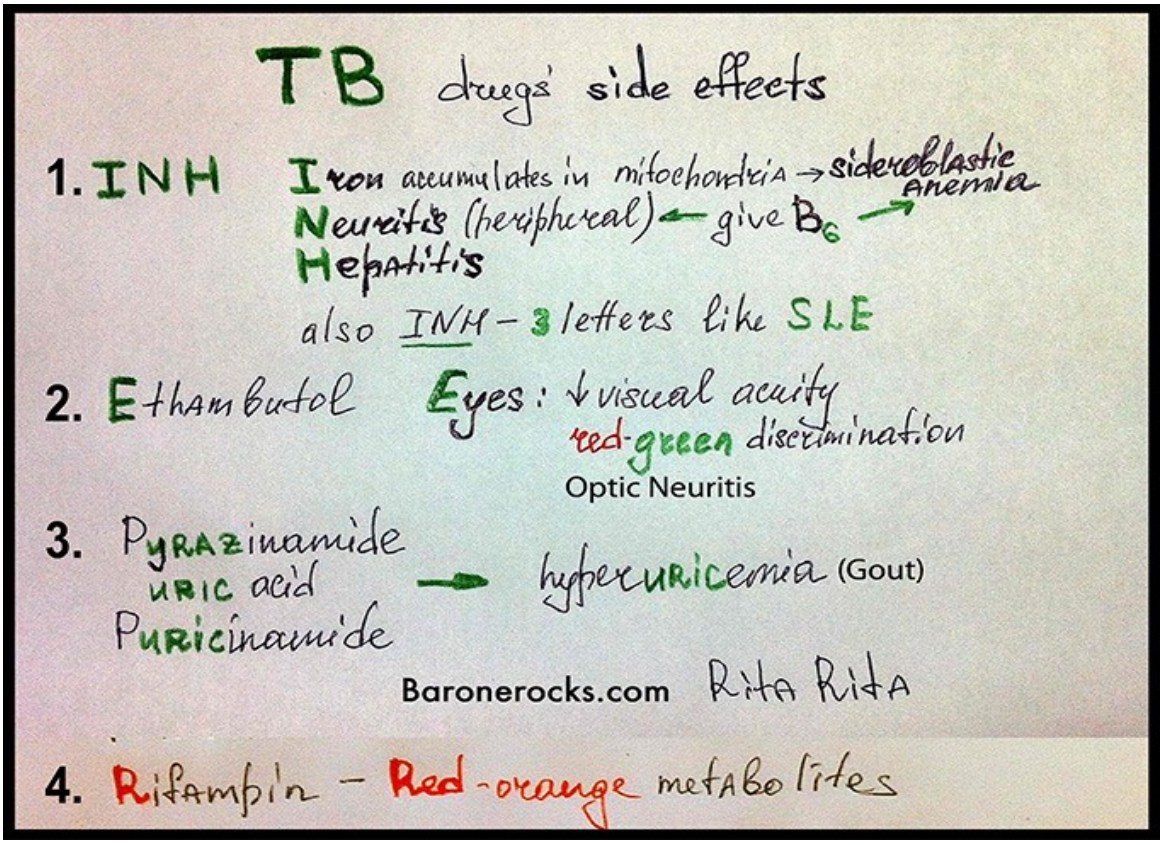
**Culture is the most specific test, but because it takes 4-6 weeks to grow it is not often available to guide initial therapy. The culture is also necessary in order to do sensitivity testing.**



## Treatment:

- Initial therapy of TB before the results of sensitivity testing are known consists of 4-drug therapy with isoniazid (INH), rifampin (Rif), pyrazinamide (PZA), and ethambutol (ETB).
- All 4 drugs are continued for the first 2 months or until sensitivity testing is known. PZA and ETB are
  then discontinued, and therapy continues with INH and rifampin for another 4 months.
  This makes routine therapy last for a total of 6 months

## ANTI TB SIDE EFFECTS



# Chapter 9

# Investigations

# Complete Blood Count (CBC)

- A complete blood count (CBC) is **a blood test used to evaluate overall health and detect a wide range of disorders, including anemia, infection and leukemia.**
- The CBC indicates the **counts of white blood cells, red blood cells** and **platelets**, the concentration of **hemoglobin**, and the **hematocrit.**

## Normal CBC

**Routine Hematology**

| Test | Result | Unit | Refrence value |
|---|---|---|---|
| CBC | | | |
| W.B.C | 6.5 | x1000/mm³ | 4.0 - 10 |
| R.B.C | 5.01 | Mill/mm³ | M: 4.5 - 6.3; F: 4.2 - 5.4 |
| Hb | 15.4 | gm/dl | M: 14 - 18; F: 12 - 16 |
| Hct | 45.2 | % | M: 39 - 52; F: 36 - 46 |
| M.C.V | 90.2 | fL | 77 - 97 |
| M.C.H | 31 | Pgm | 26 - 32 |
| M.C.H.C | 34 | % | 32 - 36 |
| Platelet | 209 | x1000/mm³ | 140 - 440 |

## How to read CBC?

**RBCs (Red Blood Cells)**

1. **Look At Hemoglobin (if low → Anemia).**

2. **Look at MCV (to detect type of anemia).**

If **less** than **80** → **Microcytic anemia e.g: IDA**
If **more** than **100** → **Macrocytic Anemia e.g: B12 def** .
If it was **between 80 - 100** → **Normocytic anemia e.g : hemolysis or blood loss**.

| | |
|---|---|
| WBC | 5.5 |
| | % |
| NE | 54.7 |
| LY | 34.1 |
| MO | 7.5 |
| EO | 3.0 |
| BA | 0.7 |
| RBC | 4.28 |
| HGB | 9.7 |
| HCT | 29.9 |
| MCV | 69.7 |
| MCH | 22.6 |
| MCHC | 32.4 |
| RDW | 18.4 |
| PLT | 331 |
| MPV | 8.8 |

| | |
|---|---|
| WBC | 12.1 |
| | % |
| NE | 71.1 |
| LY | 15.9 |
| MO | 3.3 |
| EO | 0.5 |
| BA | 8.7 |
| RBC | 2.69 |
| HGB | 10.6 |
| HCT | 31.6 |
| MCV | 117.6 |
| MCH | 39.6 |
| MCHC | 33.7 |
| RDW | 14.1 |
| PLT | 578 |
| MPV | 7.2 |

**3. look at WBCs (White Blood Cells) & its differential**

- A white blood cell (WBC) count measures **the number of white blood cells. Normally (4000 – 11000)**
- WBC differential determines **the percentage of each type of white blood cell present in your blood.**
- A WBC count can also be called a **leukocyte count.**
- WBCs Number **Elevated** called **Leukocytosis**.
- WBCs Number **less** than normal called **Leukopenia.**

| | | |
|---|---|---|
| WBC | 6.8 | |
| | % | # |
| NE | 52.6 | 3.6 |
| LY | 36.7 | 2.5 |
| MO | 7.8 | 0.5 |
| EO | 2.5 | 0.2 |
| BA | 0.4 | 0.0 |
| RBC | 5.29 | |
| HGB | 16.2 | |
| HCT | 47.0 | |
| MCV | 88.8 | |
| MCH | 30.7 | |
| MCHC | 34.5 | |
| RDW | 12.5 | |
| PLT | 179 | |
| MPV | 8.4 | |

## LEUKOCYTOSIS

**Neutrophilia**

- Represents either a **reactive phenomenon (leukemoid reaction)** or a **myeloid malignancy**.
- A leukemoid reaction often is associated with **infection, inflammation, malignancy**.

**Eosinophilia**

- Caused by **parasite infestation**, **drugs**, comorbid conditions such as **asthma and other allergic conditions.**

**Basophilia**

- Peripheral blood basophilia is an extremely rare condition that suggests **chronic basophilic leukemia.**

**Lymphocytosis**

- Represents Reactive T-cell lymphocytosis (eg, from viral infection) or leukemia

## Leukopenia

**Neutropenia**

- Neutropenia is clinically most relevant when it is severe (ANC, $< 0.5 \times 10^9$/ L) because of the associated risk of infection.
- acquired neutropenia is **drug therapy**; the most commonly implicated agents (Carbamazepine, Carbimazole ) .
- **Infection** is another common cause of neutropenia, and the major culprits are **viruses and sepsis**.

## 4- Look at Platelets count

- Platelets count measures **the number of Platelets in the blood normally (150 – 450 x 10^3).**
- A Platelet count can also be called a **Thrombocyte count**.
- Platelets Number **Elevated** called **Thrombocytosis.**
- Platelets Number **Less** than 150000 called **Thrombocytopenia.**

| | | |
|---|---|---|
| WBC | 6.8 | |
| | % | # |
| NE | 52.6 | 3.6 |
| LY | 36.7 | 2.5 |
| MO | 7.8 | 0.5 |
| EO | 2.5 | 0.2 |
| BA | 0.4 | 0.0 |
| RBC | 5.29 | |
| HGB | 16.2 | |
| HCT | 47.0 | |
| MCV | 88.8 | |
| MCH | 30.7 | |
| MCHC | 34.5 | |
| RDW | 12.5 | |
| PLT | 179 | |
| MPV | 8.4 | |

## Thrombocytopenia

- Another important point to consider before **starting a costly search for disease is the fact that healthy women may experience mild to moderate thrombocytopenia** (platelets, 75-150 × $10^9$/L) during pregnancy, and such incidental thrombocytopenia of pregnancy requires no further investigation.
- Consider the possibility of **thrombotic thrombocytopenic purpura/hemolytic uremic syndrome.**

## Thrombocytosis

- Thrombocytosis may represent either **a myeloid malignancy** (primary thrombocytosis [PT]) or a secondary process related to various clinical conditions **including IDA, surgical asplenia, infection, chronic inflammation, hemolysis, tissue damage, and nonmyeloid malignancy** (reactive thrombocytosis [RT]

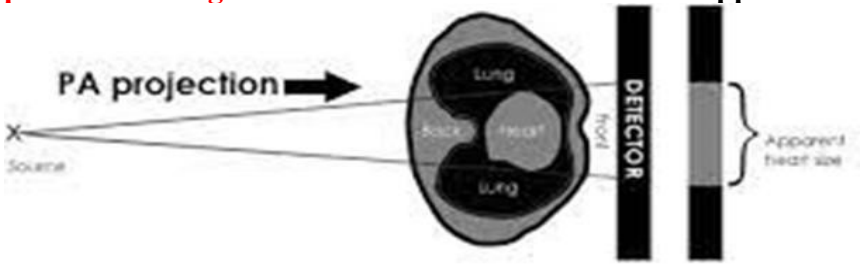
# Chest x-ray

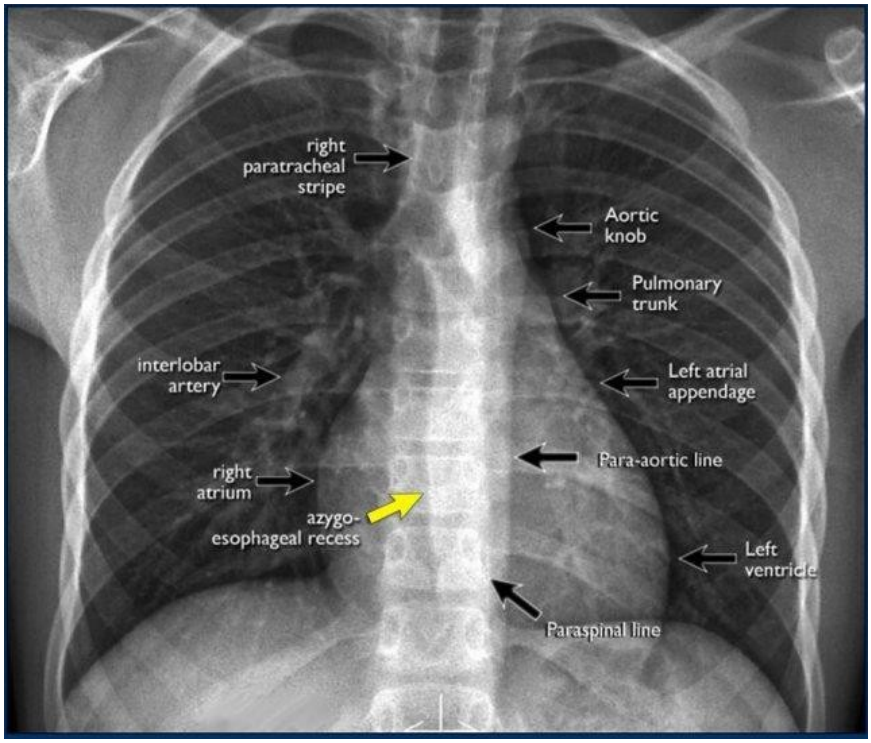
• The chest x-ray is the **most commonly performed diagnosticx-ray examination.**

• A chest x-ray makes images of **the heart, lungs, airways,blood vessels and the bones of the spine and chest**.

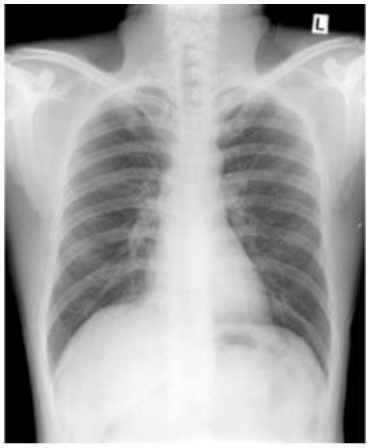
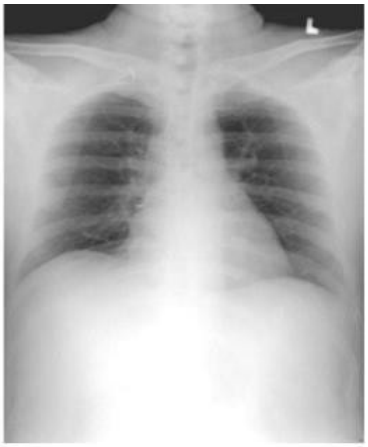
## Routine plain chest radiography

1- **Postero-anterior view (PA)**
2- **Lateral view.**
   - The term PA refers to the direction of the x-ray beam which traversesthe **patient from posterior to anterior.**
   - PA chest radiography is preferred to AP, why?
     • **Less magnification** of the heart.
     • **More lung fields** are visualized.
     • The PA **projects the scapula away from the lung fields**.
     • The **apices of the lungs are closer** to the film in the PA and **appear more clear.**



## Chest X ray view

**3- Antero-posterior view (AP)**
- Very ill patients who **are unable to stand.**
- Infants and small children.

**4- Inspiration-expiration films**
- Suspected **bronchial foreign body aspiration**.
- Suspected **small pneumothorax.**

**Before reading the x-ray film, the following should be checked:**

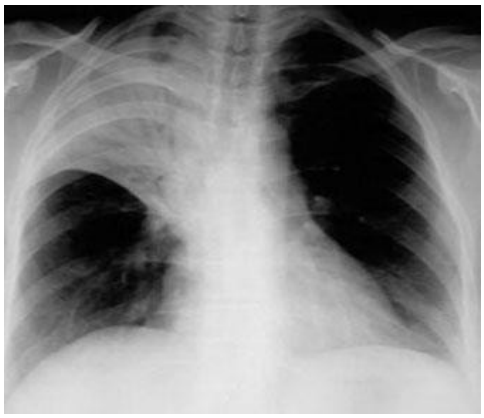
**1- Request form**
- Name, age, sex and date.

**2- Technical factors:**
- Markers. (Right / Left)
- Centering (patient position).
- Degree of inspiration.
- Exposure (penetration).

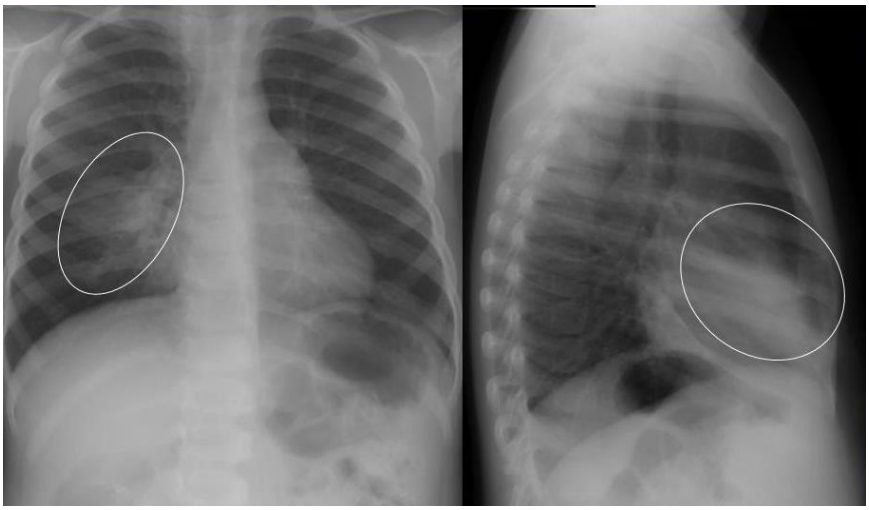
## PNEUMONIA

- Is an **inflammation of the lung**, which can be caused by a variety of micro-organisms, including bacterias, viruses, andfungi.
  A. **Lobar pneumonia:** inflammation confined to a lobe of thelung.
  B. **Bronchopneumonia**: refers to bilateral multifocal areas ofconsolidation.

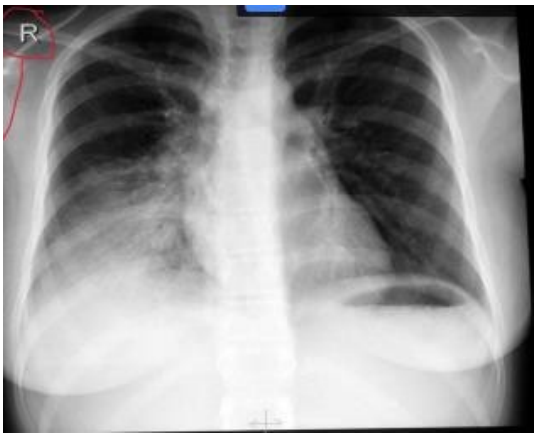
### Right Upper Zone Pneumonia

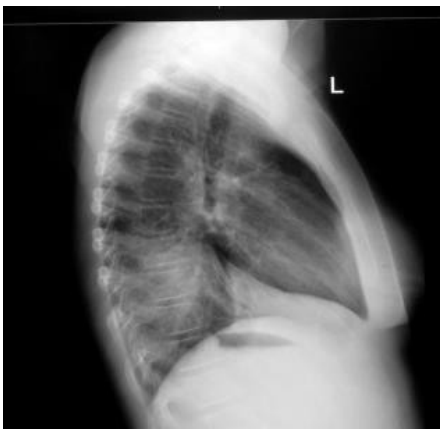


## Right Middle Zone Pneumonia

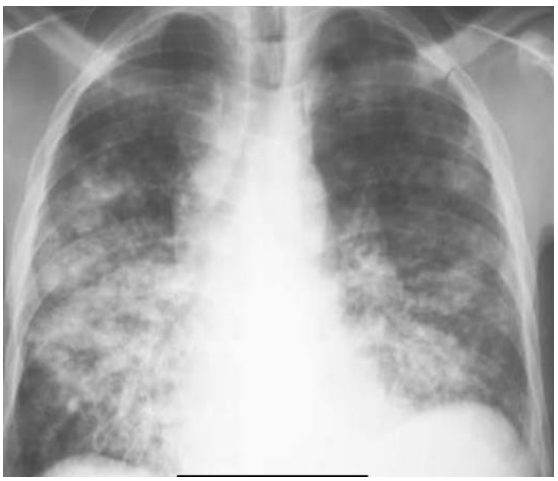


## Right Lower Zone Pneumonia



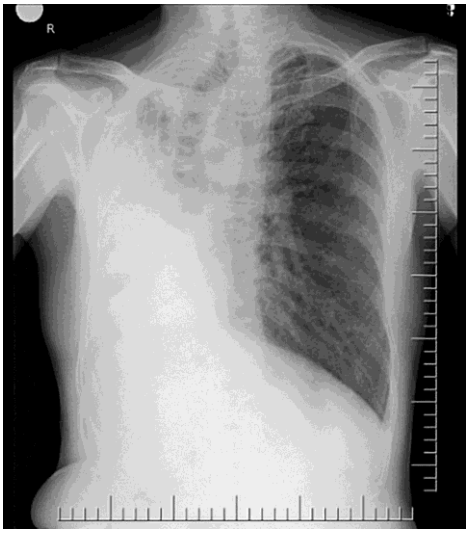




## Bronchopneumonia



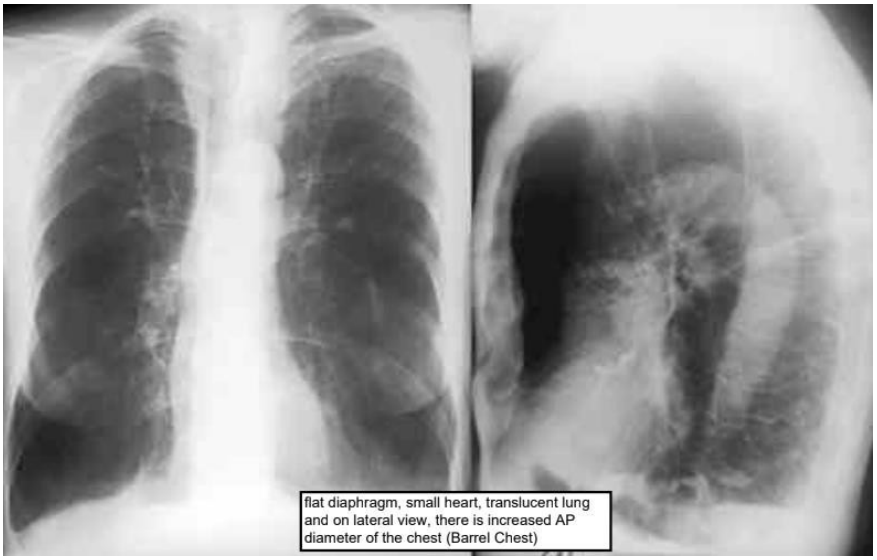
## Pulmonary Collapse

- Pulmonary collapse or atelectasis refers to **a decrease in volume of a lung, lobe or segment.**
- Obstruction to flow of air is **the most common cause of collapse.**
- Air in the alveoli **is absorbed** and because **no further air enters the alveoli distal to the obstruction**, the lung tissue **collapses and becomes more opaque**.
- Emphysema
- Is an **increase in the size of the air spaces distal to the terminal bronchioles**, with **dilatation or destruction** of their walls?




## Emphysema

**1-** The lung appear **more translucent** with reduction in size and number of the **small vascular markings.**
**2-** The **diaphragms** are **low and flat**.
**3-** The **heart** shadow is **long and narrow.**
**4-** The **postero-anterior diameter** of the chest is increased in thelateral view resulting in **barrel chest**.



## Pleural effusion

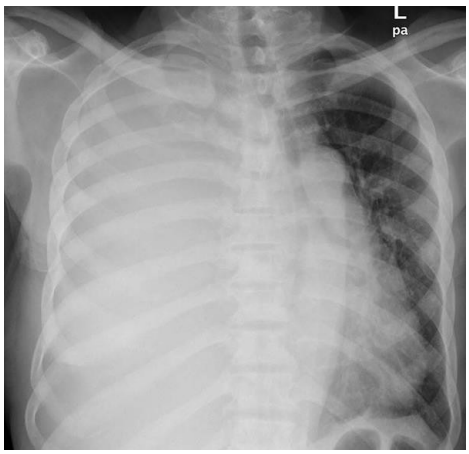
Is fluid collection in the **space between the parietal and visceral layers of the pleura**, usually contains **serous fluid**, but may have differing contents.

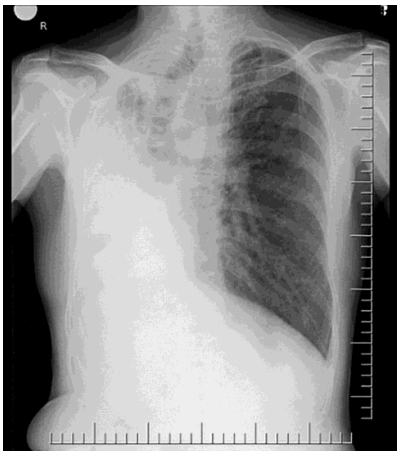
- **Haemothorax**: blood, usually following trauma.
- **Empyema**: purulent fluid (pus).
- **Hydropneumothorax**: fluid and air.

Radiological features of pleural effusion ona chest x- ray:

1- **Homogeneous opacification.**
2- Loss of the **diaphragm outline.**
3- **No visible** pulmonary or bronchialmarkings.
4- **Concave upper border** which appearhigher laterally.
5- **blunting or obliteration of thecostophrenic angle**.

**Pleural Effusions** **VS.** **Lung collapse**





### Diagnostic Criteria for Pleural Effusion

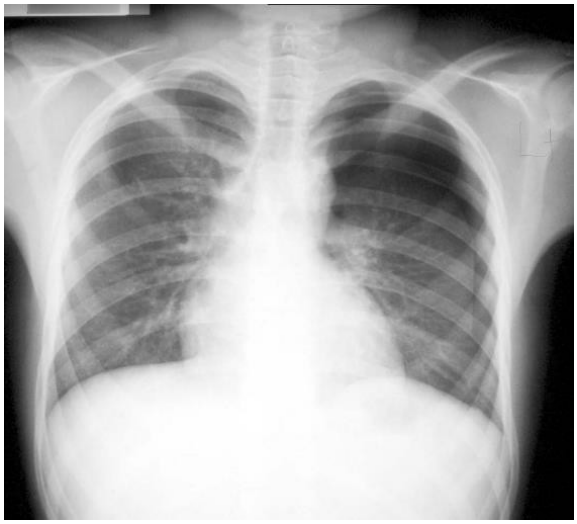
| | TRANSUDATIVE | EXUDATIVE |
|---|---|---|
| MECHANISM | ↑ Capillary hydrostatic pressure<br>↓ Capillary oncotic pressure | ↑ Capillary permeability |
| FLUID PROTEIN / SERUM PROTEIN | ⩽ 0.5 | > 0.5 |
| FLUID LDH / SERUM LDH | ⩽ 0.6 | > 0.6 |
| LDH | ⩽ ⅔ the upper limit of normal serum LDH | > ⅔ the upper limit of normal serum LDH |
| COMMON CAUSES | HEART FAILURE<br>CIRRHOSIS<br>NEPHROTIC SYNDROME | INFECTION<br>MALIGNANCY<br>PULMONARY EMBOLISM<br>AUTOIMMUNE |

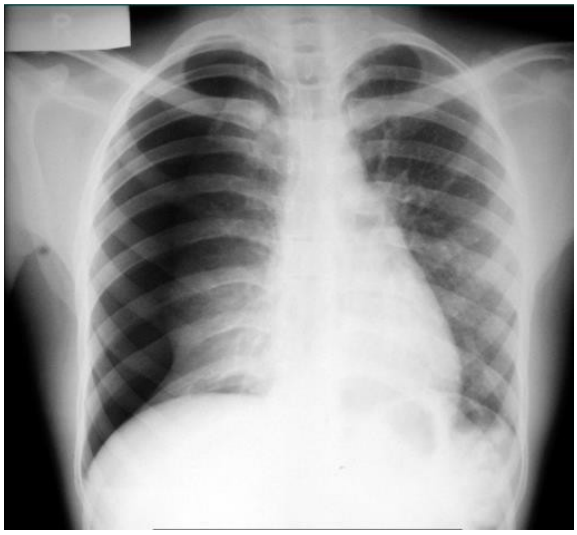
## Pneumothorax

- Is the presence of **free air in the pleural space, by a tear in either theparietal or visceral pleura.**
- The most common cause is **chest injury**, but the most common cause of**spontaneous** pneumothorax is **rupture of sub-pleural emphysematous bullae (bleb).**

**Radiological features of pneumothorax:**

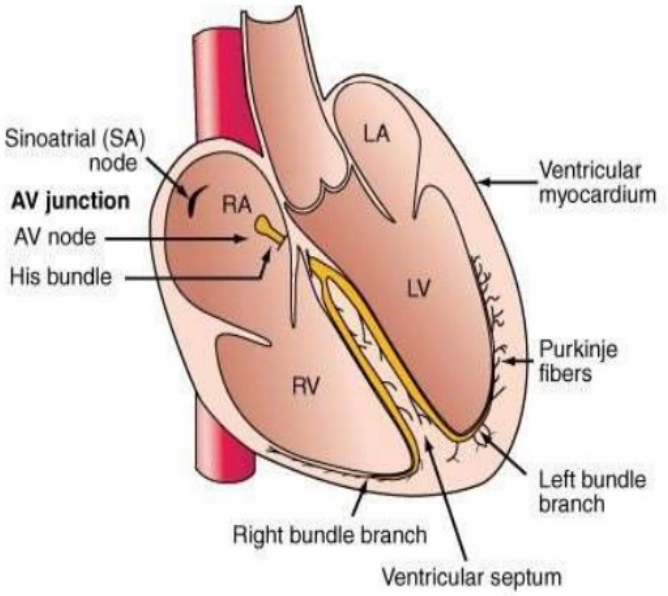
1- **Lung edge**: a thin white line at the lung margin, represent the visceral pleura.
2- **Absent lung markings** between **the lung edge and chest wall.**
3- **Mediastinal shift:** occur when **a tension pneumothorax develops**.

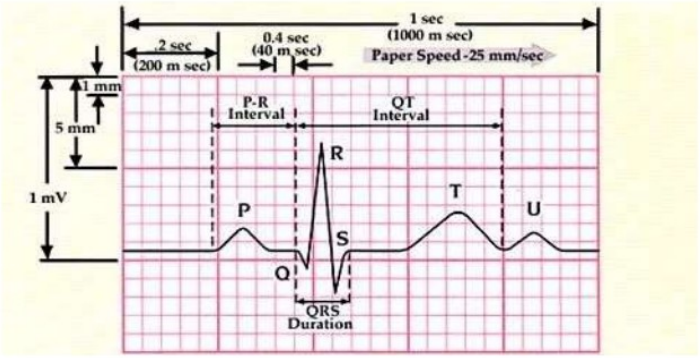




# General Principles in ECG

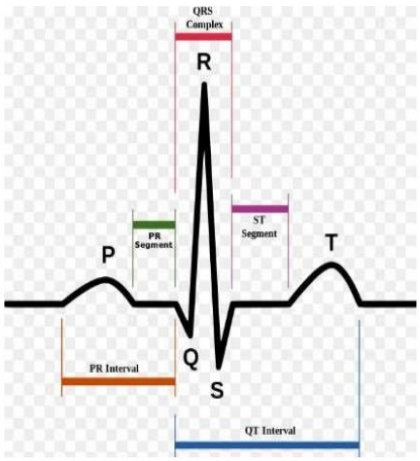
## Cardiac Conduction System




- Y axis=voltage, 1 mm (small box) = 0.1 mV
- X axis=time, 1 mm (small box) = 0.04 seconds

Normal ECG trace

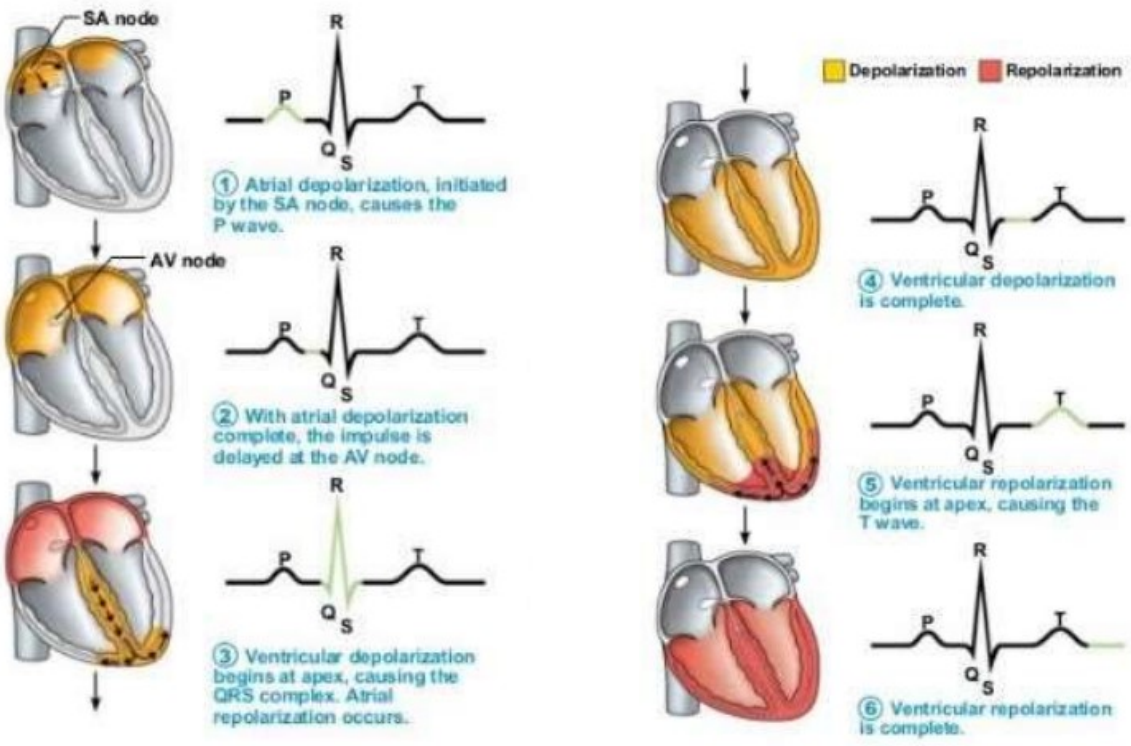


# ECG (ELECTROCARDIOGRAPHY)

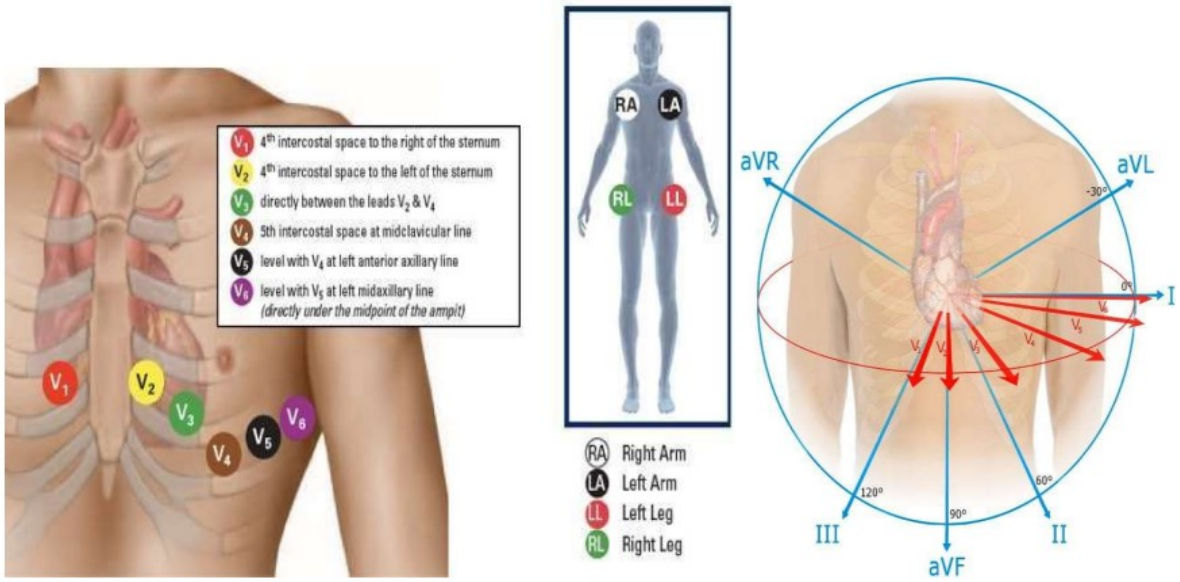
1. Each cycle of cardiac contraction and relaxation is initiated by spontaneous depolarization of **the SA node**. This event is **not seen** on the ECG.
2. The **P wave records** atrial depolarization and contraction. The first part of the **P wave reflects right atrial activity**; the second part **reflects left atrial activity**.
3. There **is a brief pause** when the electrical current reaches the **AV** node and the EKG falls silent **(the PR segment).**
4. The **wave** of depolarization then spreads **along the ventricular conducting system** (**bundle of His**, **bundle branches**, and **Purkinje fibers**) and out into the ventricular myocardium. The first part of the ventricles to be depolarized is the **interventricular septum.** Ventricular **depolarization** generates the **QRS complex.**
5. The T wave **records ventricular repolarization**. Atrial repolarization is not seen.

## Summary

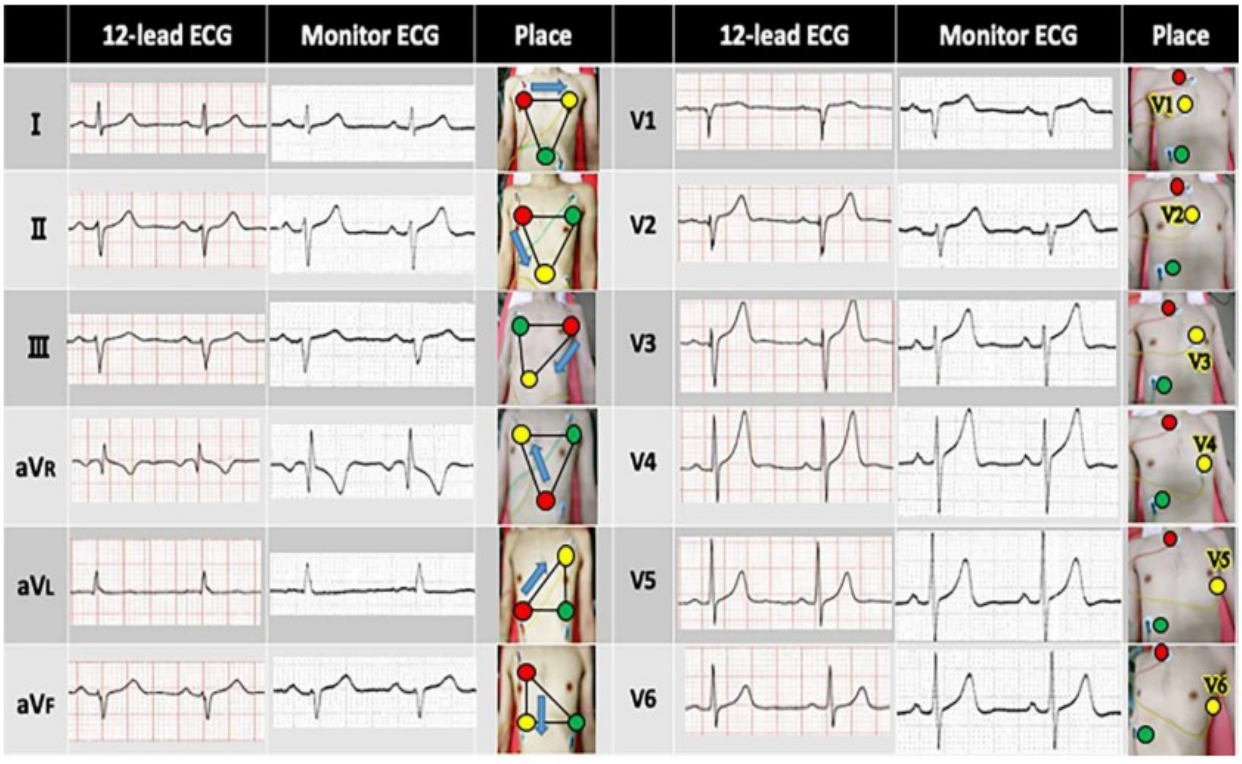
- **P wave** relates to **atrial depolarization** (normal time length **2.5 small squares** on ECG trace).
- **QRS complex** relates to **ventricular depolarization** (normal time length **0.12sec = 3** small squares on ECG trace).
- **T wave** relates to **ventricular repolarization** (no strict criteria for width but need to look at ST segment for changes – myocardial ischemia or infarction).
- **PR interval** (measured from beginning of P wave to beginning of QRS complex) should be between **0.12-0.20** sec (equivalent to 3-5 small squares). Represents **time taken for atrial depolarization and pass message to ventricles** (involves **SA node, atrial tissue and AV node).**



# ECG Leads & Its Places




# Leads with Each specific ECG



## Stepwise Approach to looking at an ECG

**1) Check RATE – normal, fast (tachycardia) or slow (bradycardia)?**

**2) Check Rhythm – sinus or not?**
Sinus rhythm has **a P wave followed by a QRS complex and every QRS complex has a preceding P wave.**

**3) Check Axis – normal or not?**
**If the QRS in Leads I and aVF are positive, the axis is normal.**

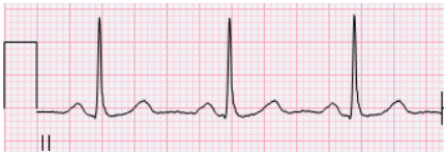
**4) Check Intervals – long or short?**
**- PR interval: prolonged in heart blocks, short in Wolff Parkinson White (WPW).**
**- QRS interval: prolonged and wide - ventricular, bundle branch block.**
**- QT interval: prolonged with certain drugs – potentially dangerous.**

**5) Check for Ischemia or Infarction?**
**ST segment depression or elevation, Q waves or T wave inversion.**

## 1- RATE

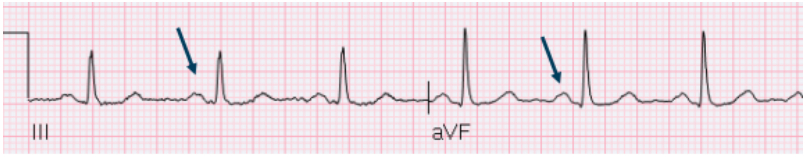
**Normal, fast (tachycardia) or slow (bradycardia).**

**- If R-R interval regular , Dividing 300 by the number of big boxes between R waves.**



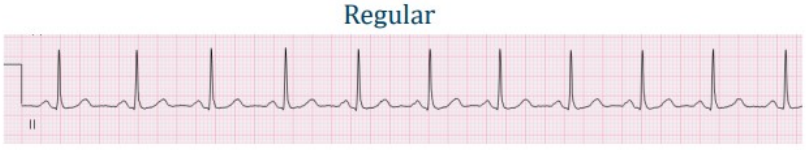

## 2- Rhythm

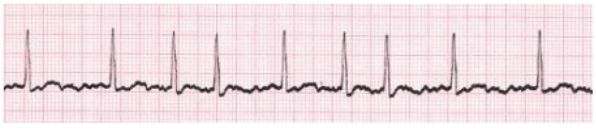
**A- Sinus rhythm** has **a P wave followed by a QRS complex and every QRS complex has a preceding P wave.**




**B- Regular or Irregular**

**• Distance between QRS complexes (R-R intervals)**



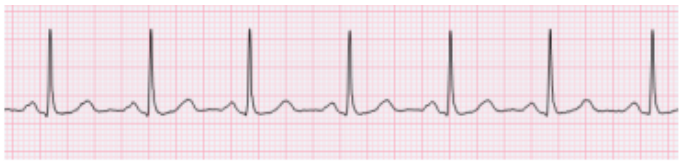



### Rhythm

**A- P waves present, regular rhythm**

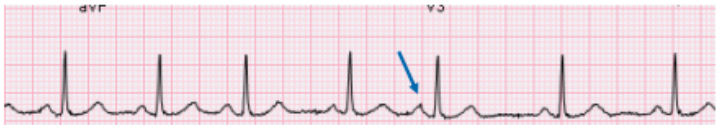
• Sinus rhythm

• Rare: atrial tachycardia, atrial rhythm

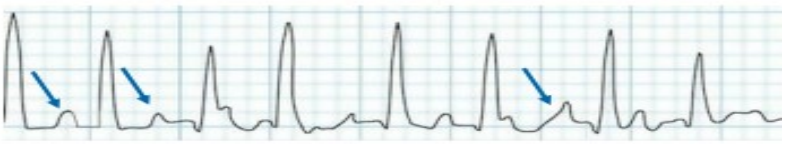


**B- P waves present, irregular rhythm**

• Sinus rhythm with PACs



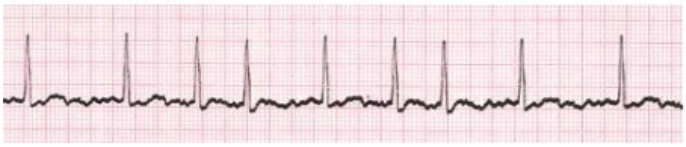
• Multifocal atrial tachycardia



**C- No p waves, irregular rhythm**

• Atrial fibrillation – irregularly irregular

• Atrial flutter with variable block



## Atrial Fibrillation

• **ECG findings** : **"Undulating baseline" f wave** , no discernible **p-waves**, **irregular R-R** interval

• **Causes**
- **Heart disease**: CAD, MI, HTN, mitral regurge, Pericarditis, rheumatic disease.
- **Pulmonary disease :** including PE
- **Endocrine** : Hyperthyroidism, hypothyroidism or Pheo
- Excessive **alcohol** intake "holiday heart syndrome"

• **Treatment:**

**AV nodal blockers** or **synchronized cardioversion** if **unstable**. **CHA2DS2-VASc** score to determine **anticoagulation.**

## Clinical Management of Atrial Fibrillation

- **Urgent referral / admission?**
  - New onset AF **(within last 48 hours)** may be candidate for **cardioversion (if below age of 60 years) .**
  - **Unstable and needs stabilization** (low or high BP,severe Pneumonia) .
- **Rate control**

  Start **Bisoprolol 2.5mg** and titrate up every 2/52 to target pulse (60-80bpm)
- **Anti-coagulation?**

  Meet criteria through CHADS2 or **CHA2DS2VASc score**

  Start **warfarin OR NOAC?**

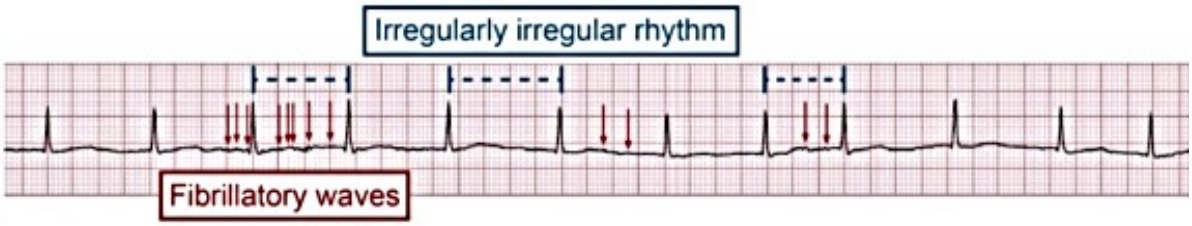



Table 1| **CHADS2 and CHA2DS2-VASc scores for calculating annual risk of stroke**[23]

| Item | $CHADS_2$ score | $CHA_2DS_2$-VASc |
|---|---|---|
| Congestive heart failure | 1 | 1 |
| Hypertension | 1 | 1 |
| Age ≥75 years | 1 | 2 |
| Diabetes | 1 | 1 |
| Stroke, transient ischaemic attack, or thromboembolism | 2 | 2 |
| Vascular disease (myocardial infarction, peripheral arterial disease) | — | 1 |
| Age 65-74 years | — | 1 |
| Sex (female) | — | 1 |
| Maximum score | 6 | 9 |

- When CHADS score is 1 or less, use **aspirin.**
- When CHADS score is 2 or more, use a **NOAC or warfarin.**

## Case Scenario

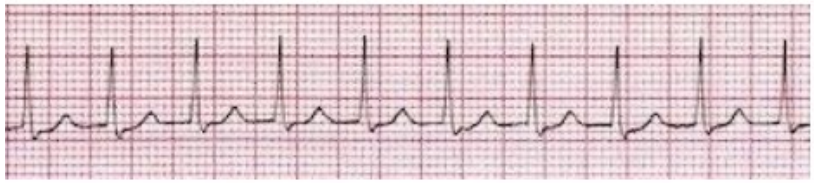
76 year old Mohammed Salah starts with new palpitations 1 month ago. He is a known hypertensive and last BP reading was 170/100 mmHg and he admits he is not compliant with his medications. He had a CVA in 2012 and takes regular Aspirin. He also has Chronic Hepatitis C and his last LFTs showed raised ALT and AST (over 3 times normal levels).

- What is his CHADS2 score?
- Would you anticoagulated this man?

## Rhythm

**D- No p waves, regular rhythm**

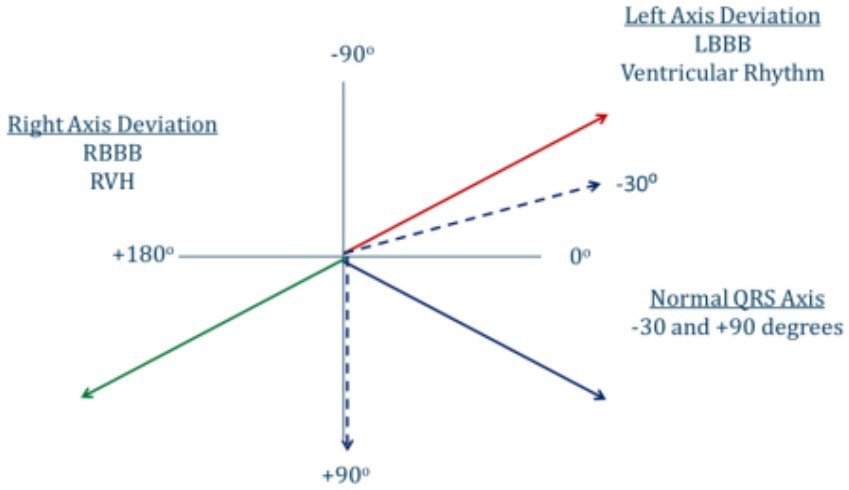
• Supraventricular tachycardias (SVTs)



• Ventricular tachycardia

## 3- Axis

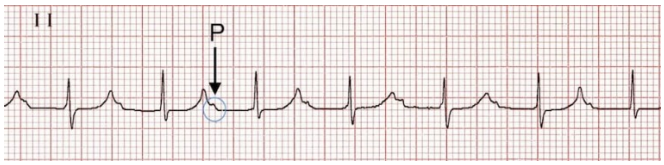
**Check Axis – normal or not ?**
**If the QRS in Leads I and aVF are positive, the axis is normal.**



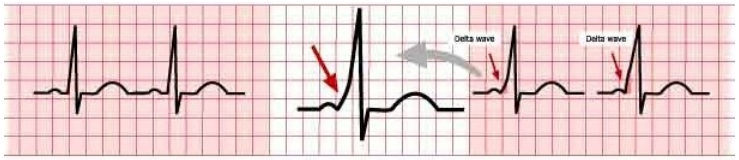

## 4- Look at Intervals

**1- PR interval:**

**- Prolonged in heart blocks.**



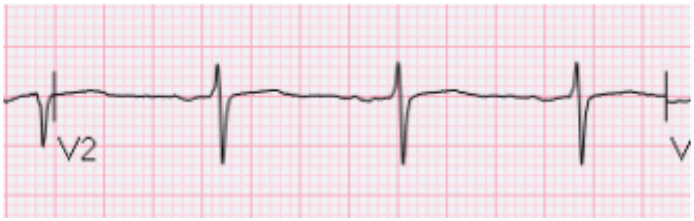

**- Short in Wolff Parkinson White (WPW)**



**2- QRS Interval:** Wide or narrow

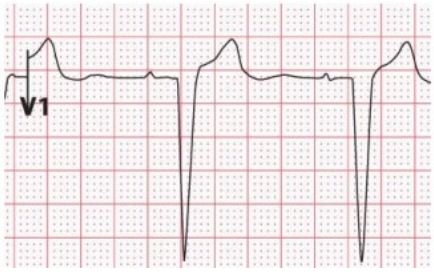
**A- Narrow QRS (< 120 ms; 3 small boxes) NORMAL**

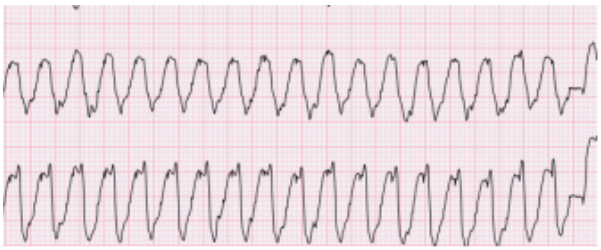
- His-Purkinje system works
- No bundle branch blocks present




**B- Wide QRS**

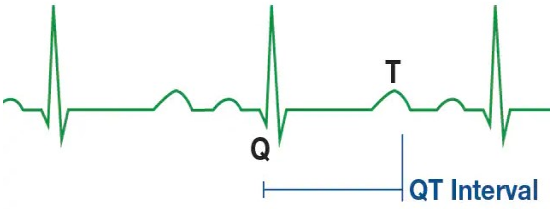
- Most likely a bundle branch block
- Ventricular rhythm (i.e. tachycardia)



**3- QT Interval: (normal < 1/2 R-R interval)**

- **Prolonged** with ↓ **Ca .**
- **Shortened** with ↑ **Ca .**
- Can lead to (VT) **torsades de pointes.**




## 5- Check for Ischaemia or Infarction?

- **ST segments**

**1- T wave abnormalities**

• Inverted: ischemia • Peaked: Early ischemia, hyperkalemia (↑K) • Flat/U waves: hypokalemia (↓K)

**2- ST Segment**

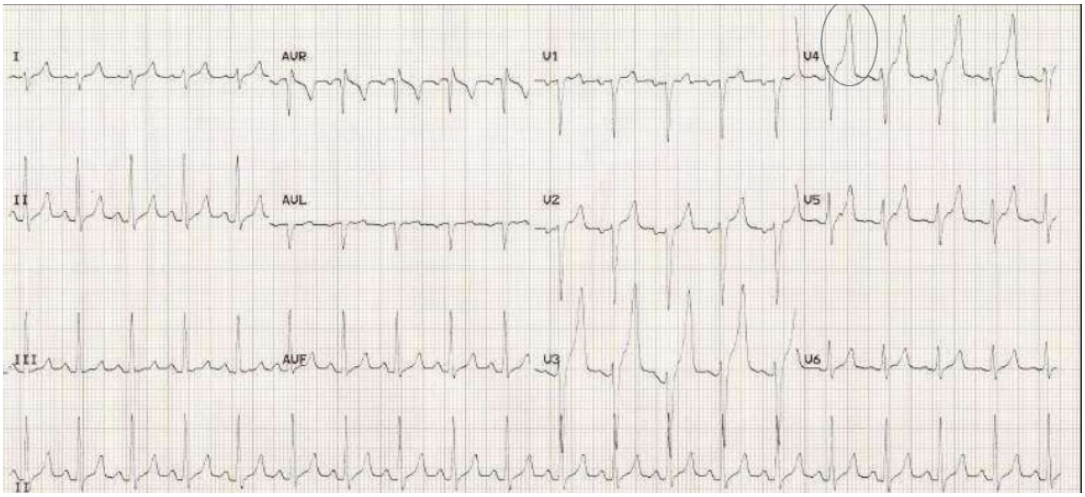
**ST depression**

• Subendocardial ischemia • Common in UA/NSTEMI

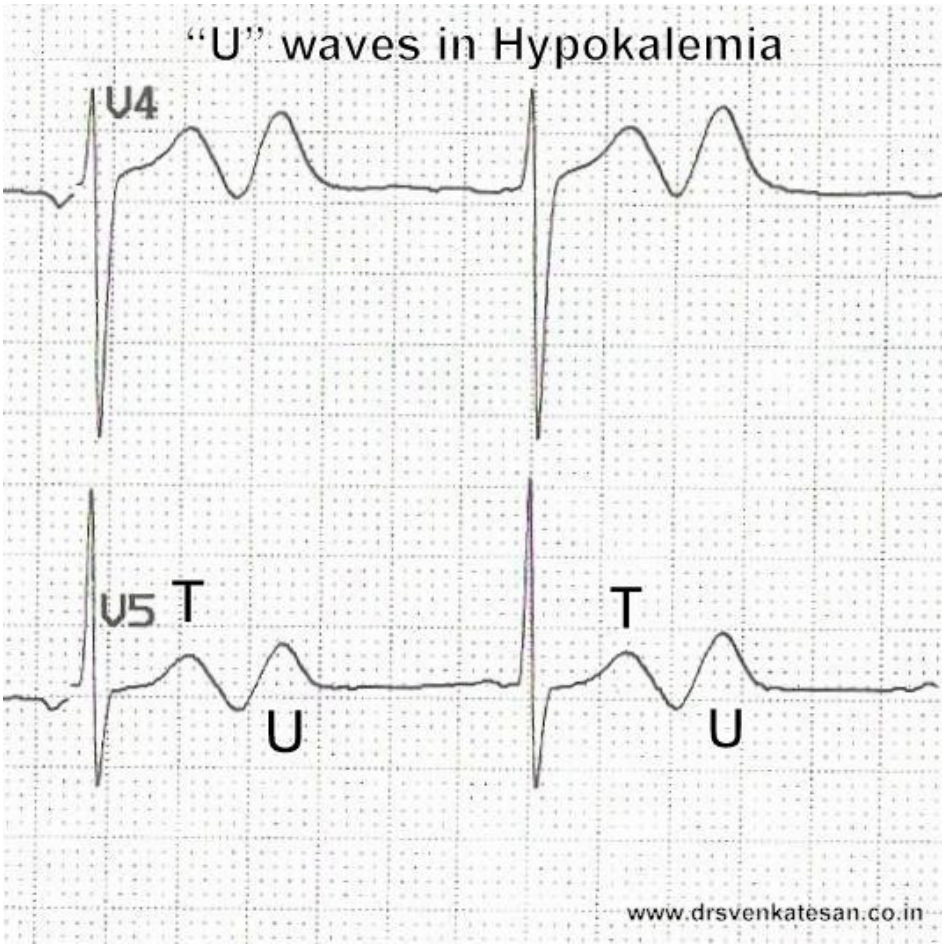
**ST elevation**

• Transmural ischemia • STEMI

## Peaked T wave: hyperkalemia (↑K)

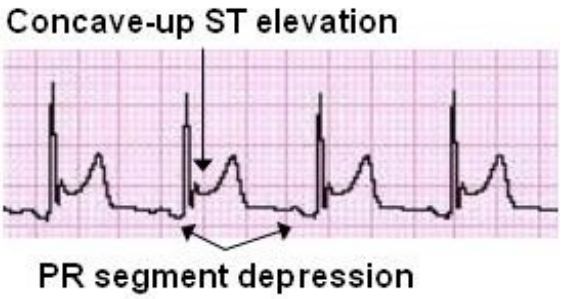


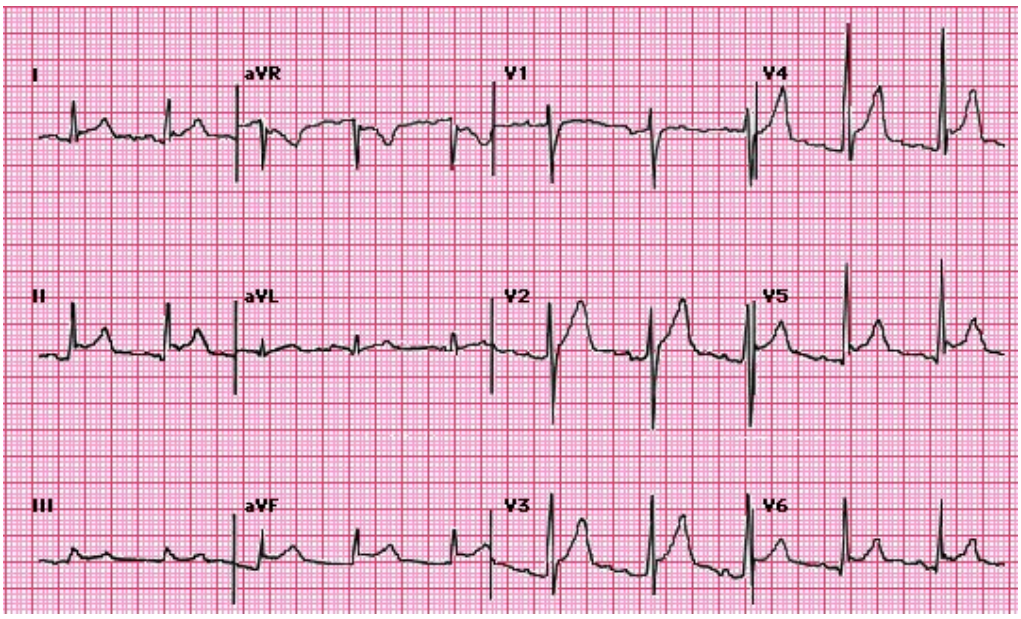

## Flat/U waves: hypokalemia (↓K)



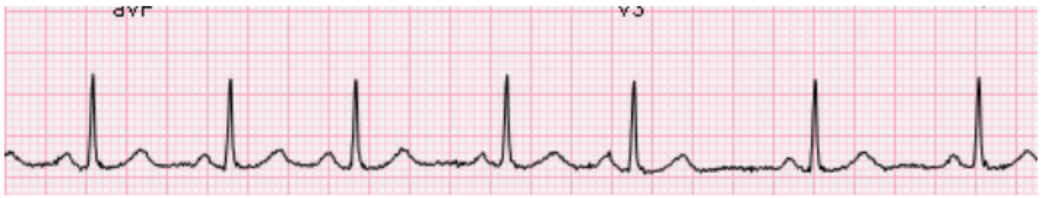
## Acute Pericarditis

- Typical ECG findings include **diffuse concave-upward ST-segment elevation and, occasionally, PR-segment depression.**

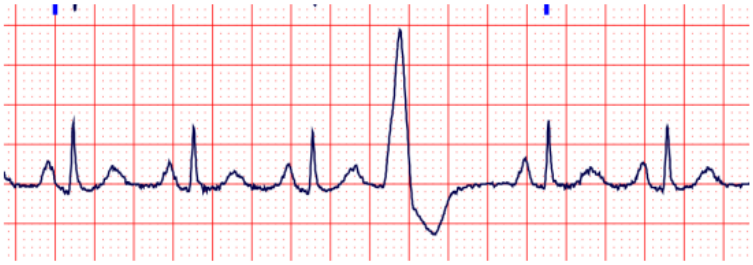



## PAC Premature Atrial Contraction

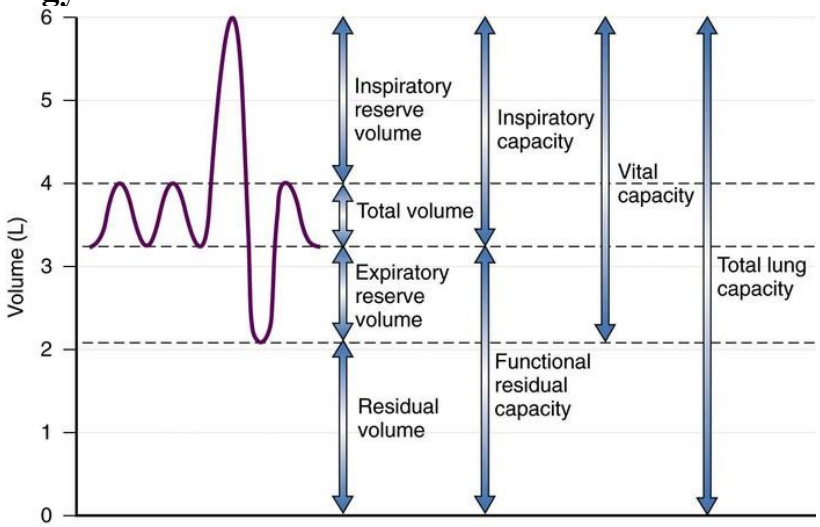


## PVC (Premature Ventricular Contraction)



# Pulmonary Function Test (PFT)

## Basic Physiology




## Lung volumes

1. **Tidal volume (TV)**
   - In/out air with each quiet breath
2. **Expiratory reserve volume (ERV)**
   - Extra air pushed out with force beyond TV
   - RV remains in lungs
3. **Inspiratory reserve volume (IRV)**
   - Extra air can be drawn in with force beyond TV
   - Lungs filled to capacity
4. **Residual volume (RV)**
   - Air that can't be blown out no matter how hard you try

## Lung Capacities

**Capacity = sum of two volumes**

**1. Total lung capacity**
- Sum of all volumes
- RV + ERV+ IRV + TV

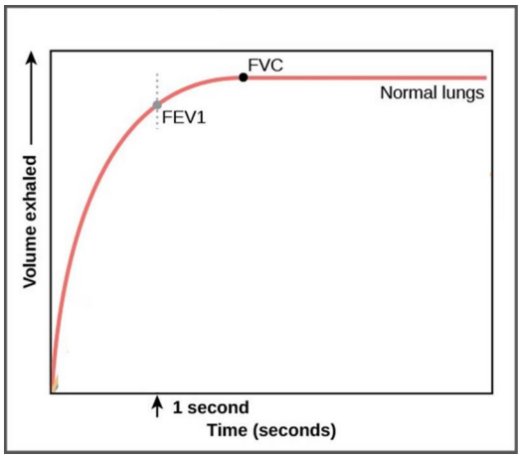
**2. Inspiratory capacity**
- Most air you can inspire
- TV + IRV

**3. Forced vital capacity (FVC)**
- Most you can exhale
- TV + IRV + ERV

**4. Functional residual capacity**
- RV plus ER

## Pulmonary Function Testing

- Must meet criteria for **adequate test**
- **Sharp peak in flow curve**
- **Expiratory duration more than six seconds**
- Inadequate test should be repeated




## Purpose of PFT

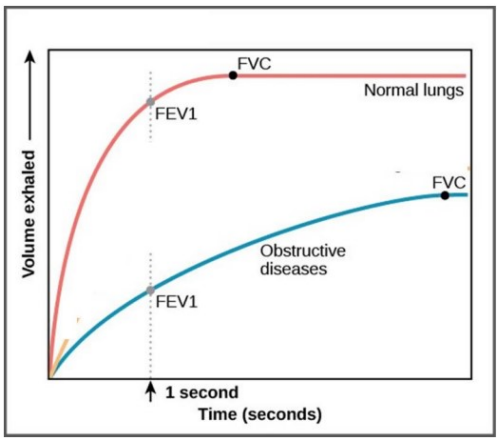
- Assessment of various aspect of **pulmonary physiology**.
- **Detect & quantify the respiratory disease**.
- **Evaluation of the disease & its response to therapy.**
- Provide valuable clinical information.

## Contraindications

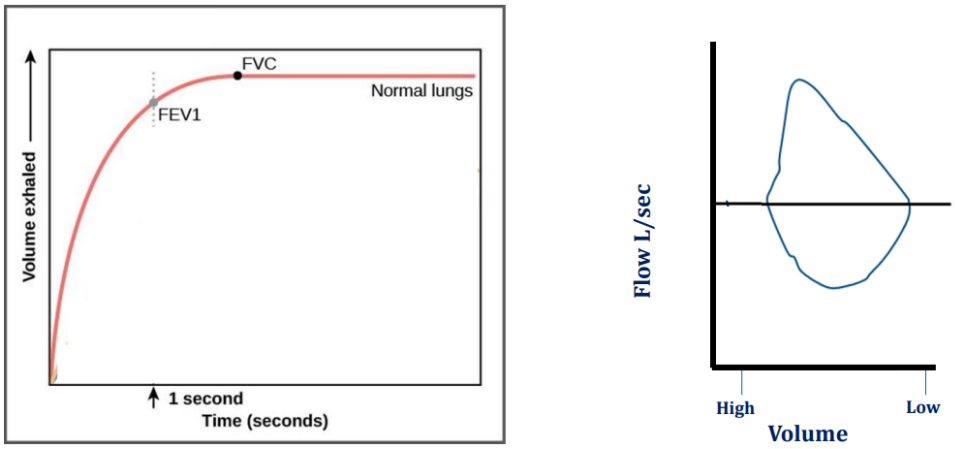
1. **Active hemoptysis**.
2. **Pneumothorax**.
3. Unstable cardiovascular stats.
4. **Cerebral/ Thoracic/ Abdominal aneurysm**.
5. **Recent eye surgery**.
6. **Recent CVA or pulmonary emboli**.
7. Respiratory distress.

## Obstructive Lung Diseases (Asthma, COPD)

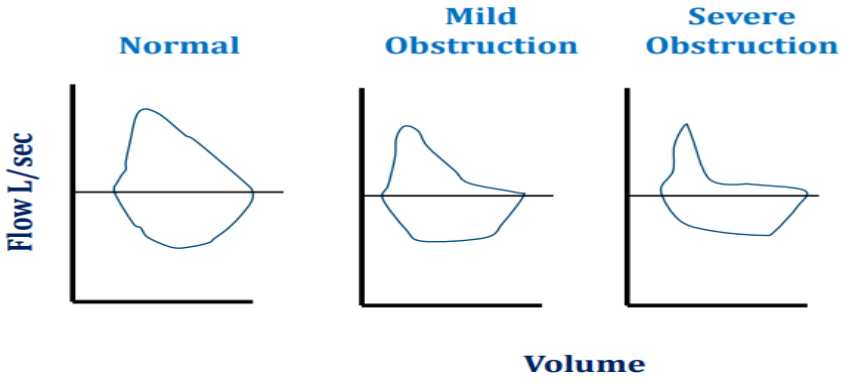
- **Reduced FEV1** (slow flow out).
- **Reduced FVC** (less air out).
- **Reduced FEV1/FVC** (**hallmark**).
- **Asthma**: **reversible obstruction**.
- ↑ FEV1 with bronchodilators.
- **COPD**: **partial/no change** bronchodilators.
- **Increased volumes from air trapping:**
  1. Total lung capacity.
  2. Functional residual capacity.
  3. Residual volume.



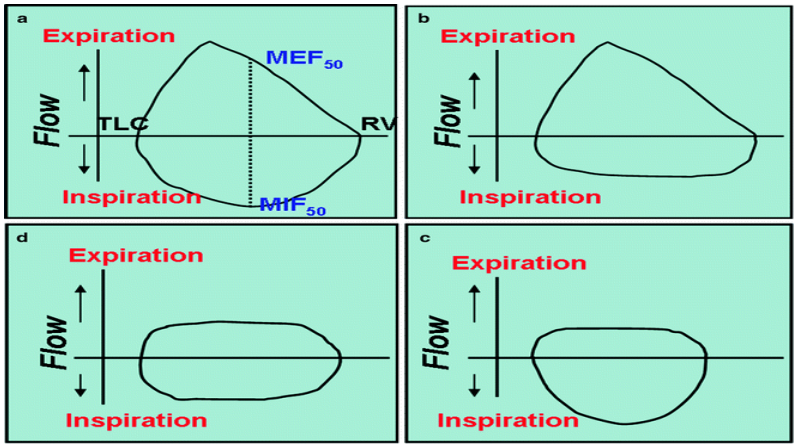
## Flow Volume Loops (Normal)




## Flow Volume Loops (Obstructive)




## Other form of obstruction



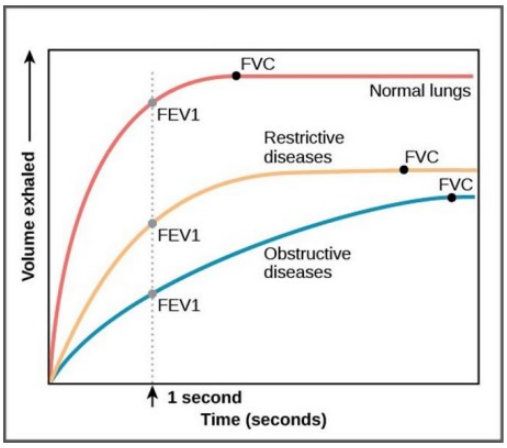

**Vocal cord paralysis**

**Goiter, Tracheal stenosis.**

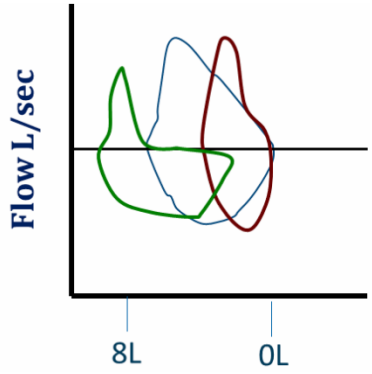
**Airway tumor (Unilateral).**

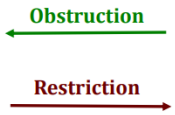
## Restrictive Lung Diseases

- Disorders where **air cannot get in → less air out**
- **Reduced FEV1** (less air in/out)
- **Reduced FVC** (less air in/out)
- **Normal** (> 80%) **FEV1/FVC** (hallmark)
- **Reduced volumes:**
  1. Total lung capacity
  2. Functional residual capacity
  3. Residual volume




## Flow Volume Loops




## Restrictive Lung Diseases

- **Restrictive Lung Disease**
- **Causes :**
  1. Poor breathing mechanics
  2. Interstitial lung diseases

## DLCO

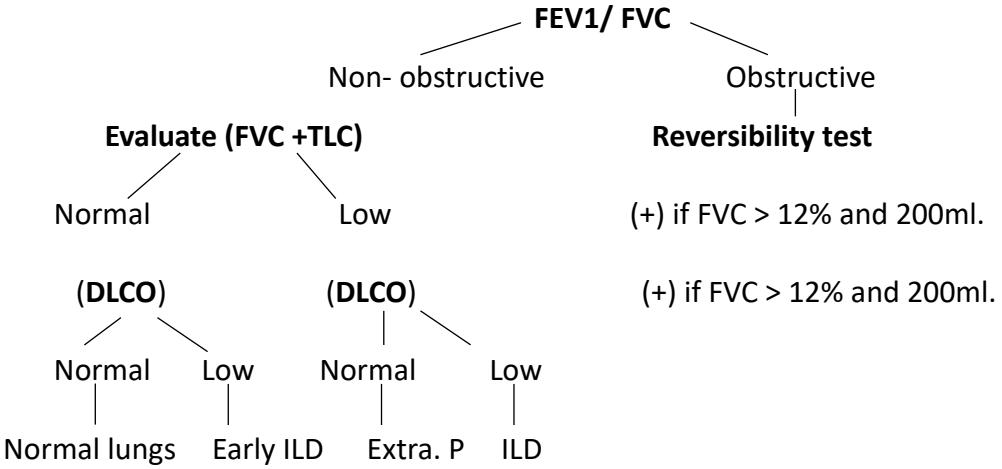
**Diffusing capacity of carbon monoxide:**

- Measures **ability of lungs to transfer gas.**
- Patient inhales small amount (not dangerous) CO.
- Machine measures CO exhaled.
- Normal = **75–140% predicted**.
- **Severe disease < 40% predicted.**

## Low DLCO Conditions

- **Interstitial lung disease**
- **Emphysema**
- **Abnormal vasculature**
  1. Pulmonary hypertension.
  2. Pulmonary embolism.
- **Prior lung resection**
- **Anemia**
  1. Corrects when adjusted for Hgb level.

## Interpretation



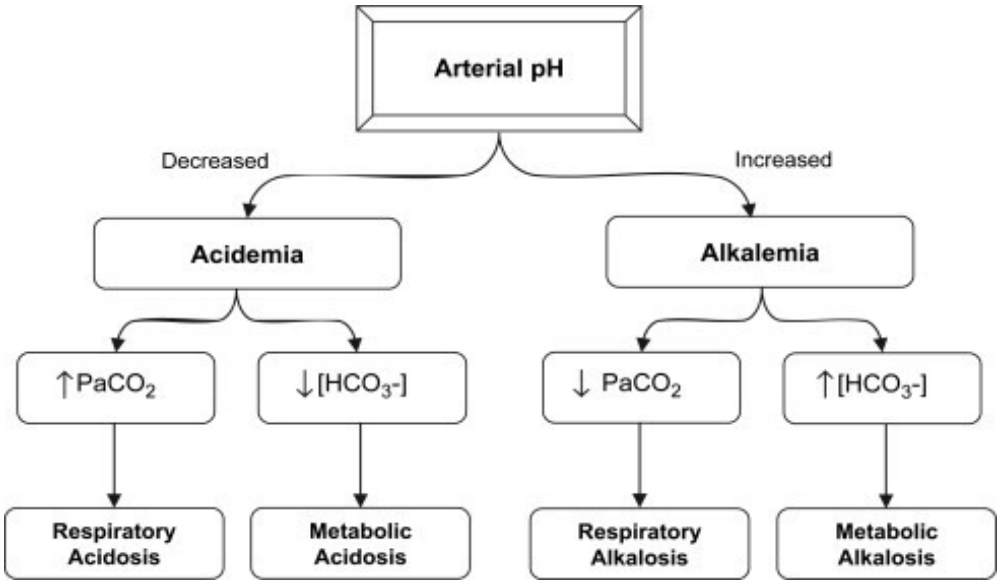

### Q24.What's the Dx. depending on this pulmonary function test?

Age: 59 Height (cm): 172 Weight (kg): 92.0 BMI: 31.10 Gender: male

| | Ref | Pre Meas | Pre %Ref | Post Meas | Post % Chg | CI | LLN |
|---|---|---|---|---|---|---|---|
| $FEV_1$ (L) | 3.11 | **2.00 | **64 | 2.85 | 42 | 1.00 | |
| FVC (L) | 4.35 | 3.40 | 78 | 4.10 | 21 | 1.36 | |
| $FEV_1$/FVC % | 72 | 59 | | 69 | | | |
| PEF (L/sec) | 8.17 | 4.45 | 54 | 6.81 | 53 | 3.87 | |
| FEF25-75 (L/sec) | 4.06 | **1.23 | **30 | 2.24 | 82 | 2.67 | |
| FET100% (sec) | | 7.46 | | 10.62 | 42 | | |
| $FEV_6$ | 4.22 | 3.40 | 81 | 3.97 | 17 | | 3.34 |
| $FEV_1/FEV_6$ | 79 | 59 | | 72 | | | 70 |

| | Pre-Bronchodilator (BD) | | | Post- BD | |
|---|---|---|---|---|---|
| Test | Actual | Predicted | % Predicted | Actual | % Change |
| FVC (L) | 1.73 | 4.37 | 40 | 1.79 | 4 |
| $FEV_1$ (L) | 1.57 | 3.65 | 43 | 1.58 | 0 |
| $FEV_1$/FVC (%) | 91 | 84 | | 88 | -3 |
| RV (L) | 1.01 | 1.98 | 51 | | |
| TLC (L) | 2.68 | 6.12 | 44 | | |
| RV/TLC (%) | 38 | 30 | | | |
| DLCO corr | 5.13 | 32.19 | 16 | | |

# Arterial Blood Gases




## Acid-Base Principles

• Normal arterial pH: 7.35 to 7.45
• Tightly controlled
• **Lungs**: excrete carbon dioxide
• **Kidneys**: excrete acid & produce bicarbonate

## Arterial Blood Gas

• Normal HCO3- = 22 – 26 mEq/L
• Normal pCO2 = 35 – 45 mmHg
• Normal pH = 7.35-7.45

## Acid-Base Disorders

• **Metabolic Disorders**
• Excess or insufficient HCO3-
• Metabolic acidosis (↓ HCO3-)
• Metabolic alkalosis (↑ HCO3-)
• **Respiratory disorders**
• Excess or insufficient CO2
• Respiratory acidosis (↑ CO2)
• Respiratory alkalosis (↓ CO2)

## Acid-Base Problems

1. Check the **pH**
   - pH < 7.35 = acidosis
   - pH > 7.45 = alkalosis

2. Check the **HCO3-** and **pCO2**
   - Increased or decreased?
   - HCO3- : normal 22-26 mEq/L
   - pCO2 from ABG: normal 35-45mmHg

3. Determine acid-base disorder
   - Acidosis + ↓ HCO3- = metabolic acidosis
   - Acidosis + ↑pCO2 = respiratory acidosis
   - Alkalosis + ↑HCO3- = metabolic alkalosis
   - Alkalosis+ ↓ pCO2= respiratory alkalosis

4. Calculate anion gap (metabolic acidosis)

## Compensatory Changes

- Respiratory disorders → abnormal CO2
- Compensation: HCO3- (renal)
- Metabolic disorders → Abnormal HCO3-
- Compensation CO2 (respiratory)

| Acid-Base Disorder | Primary Abnormality | Compensation |
|---|---|---|
| Metabolic Acidosis | ↓ $HCO_3^-$ | ↓ $CO_2$ |
| Metabolic Alkalosis | ↑ $HCO_3^-$ | ↑ $CO_2$ |
| Respiratory Acidosis | ↑ $CO_2$ | ↑ $HCO_3^-$ |
| Respiratory Alkalosis | ↓ $CO_2$ | ↓ $HCO_3^-$ |

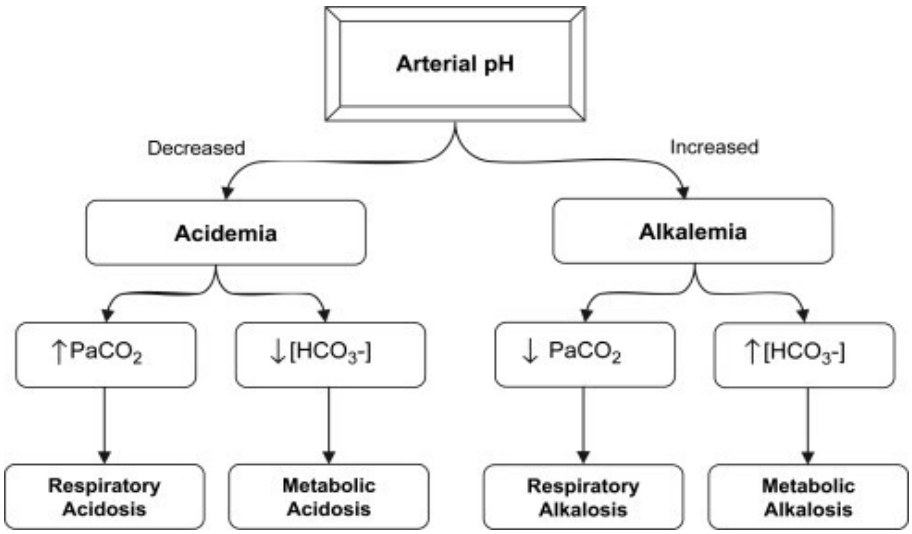
## Examples

**Example 1**
pH = 7.30 (acidosis)
HCO3- = low
pCO2 = low
Metabolic acidosis with respiratory compensation

**Example 2**
pH = 7.30 (acidosis)
HCO3 - = high
pCO2 = high
Respiratory acidosis with metabolic compensation

## Compensation Timeframe

- Respiratory compensation to metabolic disorders Occurs in **minutes.**
  Rapid change in respiratory rate.
- Metabolic compensation to respiratory disorders Chronic, significant compensation in **days** from kidneys.



## Respiratory Acidosis "Hypoventilation"

- Airway obstruction.
- Acute & chronic lung disease.
- Opioids, sedatives.
- Weakening of respiratory muscle

## Respiratory Alkalosis "Hyperventilation"

- Panic attack.
- Hypoxemia (high altitude).
- Salicylates (early)
- Pulmonary embolism

## Metabolic Alkalosis "H+ loss or HCO3- excess"

- Loop diuretics.
- Vomiting.
- Antacid use.
- Hyperaldosteronism

## Metabolic Acidosis

Check anion gap: Na+ - (Cl- + HCO3-)
Normal: 10-12

## Increased Anion gap

- Methanol.
- Uremia.
- DKA.
- Propylene glycol.
- Iron or INH.
- Lactic acidosis.
- Ethylene glycol.
- Salicylates.

## Normal Anion Gap "loss HCO3- or increase H+"

- Addison disease.
- Spironolactone.
- Saline infusion.
- Diarrhea.
- Acetazolamide.
- RTA.

# Urine Analysis

**SPECIMEN VALIDITY TESTING**

| REPORTED PRESCRIPTION | TEST OUTCOME | MEASURED RESULT | UNITS | REFERENCE RANGE |
|---|---|---|---|---|
| pH | Normal | 6 | pH | 5 - 9 |
| Specific Gravity | Normal | 1.015 | | 1.000 - 1.060 |
| Glucose | Normal | 0 | mg/dL | <20 (Max > 500) |
| Protein | Normal | 0 | mg/dL | <20 (Max > 500) |
| Bilirubin | Normal | 0 | mg/dL | <1.80 (Max > 4.00) |
| Urobilinogen | Normal | 0 | mg/dL | <1.60 (Max > 4.00) |
| Blood | Normal | 0 | mg/dL | <0.02 (Max > 1.00) |
| Ketone | Normal | 0 | mg/dL | <3 (Max > 80) |
| Nitrite | Normal | Negative | mg/dL | 0 - 1 (Max > 1) |
| Leukocytes | Normal | 0 | WBCs/uL | 15 - 40 (Max > 500) |
| Ascorbic Acid | Abnormal | 0 | mg/dL | <17 (Max > 40) |
| Clarity | Normal | Clear | | Clear/ Slightly-Cloudy/ Cloudy/ Turbid |
| Color | Normal | Yellow | | Yellow/ Amber/ Red/ Blue/ Colorless/ Straw |

## Urine analysis components:

**The urinalysis measures chemical reactions associated with:**

- Protein.
- White cells (direct microscopic examination) or leukocyte esterase (dipstick).
- Red cells.
- Specific gravity and PH.
- Nitrites.

**Urinalysis is two parts:**

- Dipstick.
- Microscopic analysis.

The dipstick gives some quantitative values as well. This means it is not just positive or negative, but can give an **approximation** of the quantity of the protein, white cells, and red cells. This can be described either as a direct number (300 mg protein) or a scale: 0/ 1+/ 2+/ 3+/ 4+.

| The urine dipstick for proteinuria (albumin) | |
|---|---|
| Trace | Between 15 and 30 mg/dL |
| 1+ | Between 30 and 100 mg/dL |
| 2+ | Between 100 and 300 mg/dL |
| 3+ | Between 300 and 1000 mg/dL |
| 4+ | >1000 mg/dL |

## Protein:

1. **It is normal to excrete a very tiny amount of protein. The tubules secrete slight amounts of protein normally known as Tamm-Horsfall protein.**
2. **This should be less than 30 to 50 mg per 24 hours.**
3. **The urine dipstick detects albumin but no other protein, such as immunoglobulin light chains.**
4. **Bence-Jones protein in myeloma is not detectable on a dipstick use immuno-electrophoresis.**

## Protein (Albuminuria):

- **Microalbuminuria = 30-300 mg/24 hours.**
- **The presence of tiny amounts of protein that are too small to detect on the UA is called microalbuminuria.**
- **This is very important to detect in diabetic patients.**
- **Long-term microalbuminuria leads to worsening renal function in a diabetic patient and should be treated.**
- **Greater amounts of protein can be associated with either tubular disease or glomerular disease.**
- **Severe proteinuria means glomerular damage.**

**In terms of proteinuria, the problem with using the scale of "trace" . It does not give an average or total amount of protein excreted over 24 hours because renal function itself varies during the day based on bodily position and physical activity.**

**Assuming constant protein excretion throughout the day, 1+ protein is about one gram excreted per 24 hours, 2+ protein is about 2 grams per 24 hours, and so on.**
**The 2 methods to assess the total amount of protein in a day are:**
**- Single protein to creatinine ratio.**
**- 24-hour urine collection.**

**The P/Cr (g/d) ratio can be superior in accuracy to a 24-hour urine collection.**

## White Blood Cells:

- **Normal urinalysis has <5 WBCs per high power field.**
- **White blood cells detect inflammation, infection, or allergic interstitial nephritis.**
- **Dipsticks detect the presence of leukocyte esterase and nitrite in the urine of patients with suspected UTI.**
- **You cannot distinguish neutrophils from eosinophils on a UA.**
- **Neutrophils indicate infection/ Eosinophils indicate allergic or acute interstitial nephritis.**
- **Microscopic examination gives a precise numerical count of the number of white cells present.**
- **Wright and Hansel stains detect eosinophils in the urine. They are the answer for allergic interstitial nephritis.**

## Bacteriuria:

- **By itself, the isolated finding of bacteria in the urine is of very limited significance. The most important exception is in pregnant women, whom you should screen for bacteria and treat. About 30% of pregnant women with bacteriuria progress to pyelonephritis.**

## RBC'S:

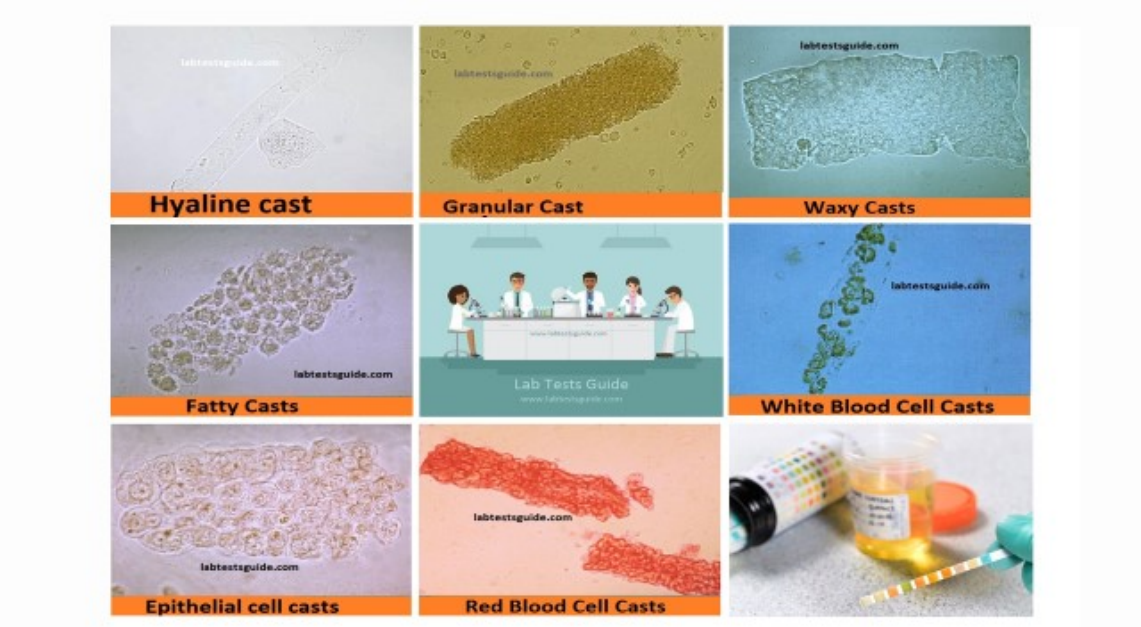
- **Normal urinalysis has <5 RBCs per high power field.**
- **False positive tests for hematuria on dipstick are caused by hemoglobin or myoglobin in the urine.**
- **Hemoglobin and myoglobin make the dipstick positive for blood, but no red cells are seen on microscopic examination of the urine.**
- **When "dysmorphic" red cells are described, the correct answer is glomerulonephritis.**

## Casts:

**These are microscopic collections of material clogging up the tubules and being excreted in the urine.**

| Type of cast | Association |
|---|---|
| **Red cell** | Glomerulonephritis |
| **White cell** | Pyelonephritis |
| **Eosinophil** | Acute (allergic) interstitial nephritis |
| **Hyaline** | Dehydration concentrates the urine and the normal Tamm- Horsfall protein precipitates or concentrates into a cast. |
| **Broad, waxy** | Chronic renal disease |
| **Granular "muddy-brown"** | Acute tubular necrosis; they are collections of dead tubular cells |

**Casts are very useful if found, but they are often not present.**



## Chapter 10

# History Check lists

# General History Taking

**Introduce yourself, take permission**

**Patient profile**

**(name , age , occupation , marital status , address )**

**Chief complaint (what + when)**

**-How can I help you today?**

**-What has brought you along to see me today?**

**Duration**

**-When did you first feel unwell ?**

**History of presenting illness "HOPI"**

**(Analysis , Assoicaated Sx & System related Questions)**

May be systemic wise or by the broad categories of disease

**IF PAIN SOCRATES:**

Site:
Onset (sudden or gradual, first time)
Character
Radiation
Associated symptoms
Timing (Time of each episode)
a) Course
b) Pattern
Exacerbating and Relieving factors
Severity

**IF NOT, OPERATS:**

Onset (sudden or gradual) Previous Episodes
Exacerbating and Relieving factors
Associated symptoms
Timing (Time of each episode)
a) Course
b) Pattern
Severity

**Past medical and surgical history**

- What illnesses have you seen a doctor about in the past?
- Have you been in hospital before or attended a clinic? Any Blood transfusion?
- Have you had any operations? If yes, 4Ws (What, Why, When, Where) + COMPLICATIONS. + Trauma Hx

**Drug Hx and allergy history**

- What drugs are you taking? What is the dose? Why does you take it (indication)? Are you compliance?
- Do you take any over the counter drug? Vitamins? Or herbal remedies?
- Any known allergy ?

**Family Hx**

If there is Family Hx of ANY disease (according to the presenting symptoms). 'Are there any illnesses that run in your family?"

**Social Hx:** Smoking history (# of pack years), alcohol, travel history, occupation , home environment, contact with sick people, Sexual history .

# System enqui ry “Review of systems

## 2.10 Systematic enquiry: cardinal symptoms

### General health

- Wellbeing
- Appetite
- Weight change
- Energy
- Sleep
- Mood

### Cardiovascular system

- Chest pain on exertion (angina)
- Breathlessness:
  - Lying flat (orthopnoea)
  - At night (paroxysmal nocturnal dyspnoea)
  - On minimal exertion – record how much
- Palpitation
- Pain in legs on walking (claudication)
- Ankle swelling

### Respiratory system

- Shortness of breath (exercise tolerance)
- Cough
- Wheeze
- Sputum production (colour, amount)
- Blood in sputum (haemoptysis)
- Chest pain (due to inspiration or coughing)

### Gastrointestinal system

- Mouth (oral ulcers, dental problems)
- Difficulty swallowing (dysphagia – distinguish from pain on swallowing, i.e. odynophagia)
- Vomiting blood (haematemesis)
- Indigestion
- Heartburn
- Abdominal pain
- Change in colour of stools (pale, dark, tarry black, fresh blood)

### Genitourinary system

- Pain passing urine (dysuria)
- Frequency passing urine (at night: nocturia)
- Blood in urine (haematuria)
- Libido
- Incontinence (stress and urge)
- Sexual partners – unprotected intercourse

### Men

If appropriate:

- Prostatic symptoms, including difficulty starting (hesitancy):
  - Poor stream or flow
  - Terminal dribbling
- Urethral discharge
- Erectile difficulties

### Women

- Last menstrual period (consider pregnancy)
- Timing and regularity of periods
- Length of periods
- Abnormal bleeding
- Vaginal discharge
- Contraception

If appropriate:

- Pain during intercourse (dyspareunia)

### Nervous system

- Headaches
- Dizziness (vertigo or lightheadedness)
- Faints
- Fits
- Altered sensation
- Weakness
- Visual disturbance
- Hearing problems (deafness, tinnitus)
- Memory and concentration changes

### Musculoskeletal system

- Joint pain, stiffness or swelling
- Mobility
- Falls

### Endocrine system

- Heat or cold intolerance
- Change in sweating
- Excessive thirst (polydipsia)

### Other

- Bleeding or bruising
- Skin rash

# Chest pain

Introduce yourself , take permission
Patient profile (name , age , occupation , marital status)
Chief complaint + duration

**Analysis of the Chief Complaint**

**Site:**
**a)** Retrosternal → ACS, Angina, Pericarditis
**b)** Lateral → PE, Pneumonia, Shingles

**Onset**
a) Sudden → ACS, PE
b) Gradual → Angina, Pneumonia

**Character**
**a)** Heaviness → ACS, Angina
**b)** Stabbing → PE, Pneumonia, Pericarditis
**c)** Tearing → Aortic dissection

**(DDX: ACS, Angina, PE, Pneumonia, Pericarditis, Shingles, Trauma, GERD)**

****Investigations:**
1. **ACS + Angina → ECG and cardiac enzymes**
2. **Pneumonia → CXR, ESR, CRP**
3. **PE → CT-angiogram, D-dimer**
4. **GERD → 24-hour monitoring.**

**Radiation**
**a)** Left shoulder, neck and teeth → ACS, Angina
**b)** Back → Aortic dissection

**Associated symptoms (finish the CC analysis then ask about them ↓)**
**Timing** (Course and pattern)
a) Intermittent or episodic, how much it lasts →ACS, Angina
b) Persistent for more than 30 minutes → MI

**Exacerbating:**
a) Exertion, Emotion, Cold, After meals→ ACS, Angina
b) Movement, respiration and cough, lying supine → PE, Pneumonia, Pericarditis

**Relieving:**
a) Rest AND NTG → Angina b) eating → GERD, ACS.
b) **Leaning forward,** Sitting up, Analgesics, NSAIDS → Pericarditis

**Severity 1. Very severe (ACS, Aortic dissection) 2. Mild (esophageal).**

## Associated symptoms

I. CVS: Sweating, Nausea, vomiting and impending death → MI
a) SOB
b) Orthopnea
c) PND
d) Ankle swelling, Palpitation, Syncope.

II. RS: Fever & chills, contact with sick patient → Pneumonia
a) Cough and sputum → Pneumonia
b) Hemoptysis, leg pain and swelling → PE
c) Cyanosis → PE

III. GI
Heart burn, regurgitation, Hematemesis and melena → GERD, Esophagitis

IV. MSS
a) Skin rash → Shingles
b) Joint pain → SLE

V. Depression: Mood and loss of interest.

**Risk Factors** (always ask about smoking and alcohol)
I. ACS → HTN, DM, Hyperlipidemia, Family history, Smoking
II. Viral etiologies may be preceded by flu-like respiratory or GI symptoms → Percardiaitis
III. Trauma → Pneumothorax
IV. PE (DVT) → Recent travel, Surgery, Immobility, Pregnancy, OCP, Previous DVTs

**Review of systems**

**Past medical and surgical** HTN, hyperlipidemia, DM, previous caths and stents, recent infections, previousheart surgeries

**Drug Hx** NSAIDs, B-blockers, Thyroxine, Cocaine AND **Vaccine Hx** if Pneumonia

**Allergies:** Drug..etc

**Family Hx** Family Hx of heart disease or premature CAD (♂<55 , ♀<65)

**Social Hx**: Smoking history (# of pack years), alcohol, travel history

**Chest Pain**

**1- Intermittent (Angina Vs. Esophageal spasm)**

**2- Acute**

1. Acute coronary syndrome
2. Aortic dissection
3. Pericarditis
4. Esophageal Spasm
5. Pneumothorax
6. Musculoskeletal pain

**Premature CAD**

- **In the patient**
  CAD < 55 years in female, < 45 years in male

- **In the family**
  First degree relative
  CAD < 65 years in female, < 55 years in male

**4.3 Cardiovascular causes of chest pain and their characteristics**

| | Angina | Myocardial infarction | Aortic dissection | Pericardial pain | Oesophageal pain |
|---|---|---|---|---|---|
| Site | Retrosternal | Retrosternal | Interscapular/retrosternal | Retrosternal or left-sided | Retrosternal or epigastric |
| Onset | Progressive increase in intensity over 1–2 minutes | Rapid over a few minutes | Very sudden | Gradual; postural change may suddenly aggravate | Over 1–2 minutes; can be sudden (spasm) |
| Character | Constricting, heavy | Constricting, heavy | Tearing or ripping | Sharp, 'stabbing', pleuritic | Gripping, tight or burning |
| Radiation | Sometimes arm(s), neck, epigastrium | Often to arm(s), neck, jaw, sometimes epigastrium | Back, between shoulders | Left shoulder or back | Often to back, sometimes to arms |
| Associated features | Breathlessness | Sweating, nausea, vomiting, breathlessness, feeling of impending death (angor animi) | Sweating, syncope, focal neurological signs, signs of limb ischaemia, mesenteric ischaemia | Flu-like prodrome, breathlessness, fever | Heartburn, acid reflux |
| Timing | Intermittent, with episodes lasting 2–10 minutes | Acute presentation; prolonged duration | Acute presentation; prolonged duration | Acute presentation; variable duration | Intermittent, often at night-time; variable duration |
| Exacerbating/ relieving factors | Triggered by emotion, exertion, especially if cold, windy<br>Relieved by rest, nitrates | 'Stress' and exercise rare triggers, usually spontaneous<br>Not relieved by rest or nitrates | Spontaneous<br>No manœuvres relieve pain | Sitting up/lying down may affect intensity<br>NSAIDs help | Lying flat/some foods may trigger<br>Not relieved by rest; nitrates sometimes relieve |
| Severity | Mild to moderate | Usually severe | Very severe | Can be severe | Usually mild but oesophageal spasm can mimic myocardial infarction |
| Cause | Coronary atherosclerosis, aortic stenosis, hypertrophic cardiomyopathy | Plaque rupture and coronary artery occlusion | Thoracic aortic dissection rupture | Pericarditis (usually viral, also post myocardial infarction) | Oesophageal spasm, reflux, hiatus hernia |

NSAIDs, *non-steroidal anti-inflammatory drugs.*

# Palpitation

Introduce yourself , take permission
Patient profile (name, age, occupation, marital status)
Chief complaint + duration

**Analysis of the Chief Complaint (OPCERATS)**
**Onset** (sudden or gradual)
**Progression get worse or better with time**
**Character: (regular or irregular) (tachycardia or bradycardia).Exacerbating,**
**Relieving:**
-Stress, Exercise, caffeine, alcohol, smoking

**Timing (Course/ pattern)** IF Lasts for a few minutes or Constant
**Severity (loss of consciousness, dizziness)**

## Associated symptoms

I. **CVS:** (HF OR IHD)
Chest pain, Orthopnea, PND, lower limb edema, SOB, Palpitation, intermittent claudication.
II. **SVT, Afib:** Polyuria, light headedness, chest tightness.
III. **Ventricular arrhythmia:** Presyncope, and syncope.
IV. **Hyperthyroidism:** heat intolerance, weight loss, diarrhea
V. **Infection and sepsis** → Fever.
VI. **Anemia:** Fatigue, Pallor or Jaundice, Weakness.
VII. **Psychological:** Anxiety (nervousness, insomnia, tachypnea).
VIII. **Pheochromocytoma** (episodic headache + sweating ).

**Review of systems**

**Past medical and surgical**

-IHD (Previous MI)

-Valvular heart disease (Mitral stenosis) → Atrial fibrillation

Previous admission.

Previous surgeries.

**Drug Hx** (Thyroxine, B-agonists , Decongestants , Anti-depressants)

**Family Hx** Family hx of heart disease or sudden death

**Social Hx**: Smoking history (# of pack years), alcohol, travel history, diet (caffeine .. etc.).

**(DDX: Atrial fibrillation, Hyperthyroidism, Phechromocytoma, Anxiety, Anemia)**

****Investigations:**

1. **CBC.**
2. **ECG.**
3. Echocardiogram.
4. Thyroid function test.
5. Urine metanephrins.

**4.6 Descriptions of arrhythmias**

| | Extrasystoles | Sinus tachycardia | Supraventricular tachycardia | Atrial fibrillation | Ventricular tachycardia |
|---|---|---|---|---|---|
| Site | - | - | - | - | - |
| Onset | Sudden | Gradual | Sudden, with 'jump' | Sudden | Sudden |
| Character | 'Jump', missed beat or flutter | Regular, fast, 'pounding' | Regular, fast | Irregular, usually fast; slower in elderly | Regular, fast |
| Radiation | - | - | - | - | - |
| Associated features | Nil | Anxiety | Polyuria, lightheadedness, chest tightness | Polyuria, breathlessness Syncope uncommon | Presyncope, syncope, chest tightness |
| Timing | Brief | A few minutes | Minutes to hours | Variable | Variable |
| Exacerbating/ relieving factors | Fatigue, caffeine, alcohol may trigger Often relieved by walking (increases sinus rate) | Exercise or anxiety may trigger | Usually at rest, trivial movements, e.g. bending, may trigger Vagal manœuvres may relieve | Exercise or alcohol may trigger; often spontaneous | Exercise may trigger; often spontaneous |
| Severity | Mild (usually) | Mild to moderate | Moderate to severe | Very variable, may be asymptomatic | Often severe |

# Shortness of Breath

**Introduce yourself , take permission**

**Patient profile** (name , age , occupation , marital status, adress)

**Chief complaint + duration**

## HOPI: Analysis of the Chief Complaint (OPERATS)

**Onset (Sudden or gradual) Instantaneous, hours, insidious. Previous Episodes**

**Exacerbating, Relieving:**

a) **Rest over night?? (COPD, HF, Asthma).**
b) **Exercise and the relation if it is present? (Limit exercise or at end of it)**
c) **Cough sputum**
d) **Cold air**

**Timing ( course, pattern):**

**Episodic with free interval (asthma)/Constant Get worse or better with time?**

**Severity effect on life: How can you walk? And the things that makes you SOB.**

## Associated symptoms

I. **Constitutional**
Fever, weight loss, night sweat, loss of appetite.

II. **CVS:** Chest pain, Orthopnea, PND, Ankle swelling, SOB, Palpitation, intermittent claudication.

III. **RS:**
a) Cough and sputum → Pneumonia
b) Hemoptysis → Pneumonia, PE
c) Cyanosis → PE
d) Wheeze

IV. GI: Nausea, vomiting, Heart burn, regurgitation, Abdominal pain, Jaundice.

V. MSS: Skin rash, Joint pain, Muscle wasting, lymphadenopathy.

VI. **Psychological:** anxiety, perioral and digital paresthesia, light headedness, can't get enough air in.

VII. **Anemia:** Pallor, dizziness , fatigue.

**Past medical and surgical**: **Hx of respiratory and cardiac disease** (HTN/ HF/ Hyperlipidemia/ Arrhythmias),
DM, Stroke, previous DVT, **Hx of blood transfusion . Any surgeries or trauma or any source of immobility.**

**Drug Hx** what he is taking (**Aspirin , B-Blocker, CCB, inhaler** ) , any recent change , adherence to medications)

**Family Hx asthma , atopy , hay fever , eczema, Lung cancer , IHD**

**Social Hx**: **Smoking** history (# of pack years), alcohol**, travel history (Recently), recreational drugs, contact with sick patient, house ventilation, pets.**

**Review of systems:** (**GU**, search for **malignancies** ….etc.).

**DDX: All respiratory and cardiac diseases, Anemia, Psychogenic) + (MSS chest trauma and costocondritis, neurogenic myasthenia gravis GBS, GIT liver ds and pancreatitis).**
**But most common cases in hospital are (Decompensated HF (Acute pulmonary edema), Acute exacerbation or asthma or COPD, PE, Pneumonia, Anemia).**

****Investigations:**
1. **CXR → Pneumonia, Pulmonary edema, Asthma, COPD**
2. **Spirometry → Asthma, COPD, RLD**
3. **CT-angiography and D-dimer → PE**
4. **CBC → Anemia**

**7.6 Breathlessness: modes of onset, duration and progression**

| | |
|---|---|
| **Minutes** | |
| • Pulmonary thromboembolism<br>• Pneumothorax | • Asthma<br>• Inhaled foreign body<br>• Acute left ventricular failure |
| **Hours to days** | |
| • Pneumonia<br>• Asthma | • Exacerbation of COPD |
| **Weeks to months** | |
| • Anaemia<br>• Pleural effusion | • Respiratory neuromuscular disorders |
| **Months to years** | |
| • COPD<br>• Pulmonary fibrosis | • Pulmonary tuberculosis |

| Grade | Degree of breathlessness related to activities |
|---|---|
| 1 | Not troubled by breathlessness except on strenuous exercise |
| 2 | Shortness of breath when hurrying on the level or walking up a slight hill |
| 3 | Walks slower than most people on the level, stops after a mile or so, or stops after 15 minutes walking at own pace |
| 4 | Stops for breath after walking about 100 yds or after a few minutes on level ground |
| 5 | Too breathless to leave the house, or breathless when undressing |

## 7.5 Causes of breathlessness

### Non-cardiorespiratory

- Anaemia
- Metabolic acidosis
- Obesity
- Psychogenic
- Neurogenic

### Cardiac

- Left ventricular failure
- Mitral valve disease
- Cardiomyopathy
- Constrictive pericarditis
- Pericardial effusion

### Respiratory

**Airways**
- Laryngeal tumour
- Foreign body
- Asthma
- COPD
- Bronchiectasis
- Lung cancer
- Bronchiolitis
- Cystic fibrosis

**Parenchyma**
- Pulmonary fibrosis
- Alveolitis
- Sarcoidosis
- Tuberculosis
- Pneumonia
- Diffuse infections, e.g. *Pneumocystis jiroveci* pneumonia
- Tumour (metastatic, lymphangitis)

**Pulmonary circulation**
- Pulmonary thromboembolism
- Pulmonary vasculitis
- Primary pulmonary hypertension

**Pleural**
- Pneumothorax
- Effusion
- Diffuse pleural fibrosis

**Chest wall**
- Kyphoscoliosis
- Ankylosing spondylitis

**Neuromuscular**
- Myasthenia gravis
- Neuropathies
- Muscular dystrophies
- Guillain–Barré syndrome

# Cough , Sputum & Hemoptysis

**Introduce yourself , take permission**

**Patient profile** (name , age , occupation , marital status, address)

**Chief complaint + duration** (acute < 3 weeks , chronic > 8 weeks)

## HOPI: Analysis of the Chief Complaint (FCBCA + OPERATS)

**Frequency**

**Content** (dry or productive) **Bloody** (hematemesis ?!!) **Color/Consistency of sputum**

**Amount (in cups)**

**Onset** (sudden or gradual) **Previous Episodes** (first time)**Exacerbating, Relieving:**

a) Rest over night

b) Exercise/ Cold air

c) Swallowing

d) Pollens, Dust, fumes.

**Associated symptoms (finish the CC analysis then ask about them ↓)Timing (course, pattern):**

Get worse or better with time?

Constant/ Episodic with free interval (asthma)

**Severity**

## Associated symptoms

I. **Constitutional**

Fever, weight loss, night sweat, loss of appetite.

II. **CVS:** Chest pain, Orthopnea, PND, Ankle swelling, SOB, Palpitation, intermittent claudication.

III. **RS:**

A) Nasal congestion/ Sore throat.

B) Change in voice/ swallowing.

C) Cyanosis → PE.

D) Wheeze → asthma or Foreign body aspiration.

IV. **GI**

Nausea, vomiting, Heart burn, regurgitation, Abdominal pain → (GERD).

**Past medical and surgical**: Hx of respiratory and cardiac disease or other diseases, history of previous admission, history of blood transfusion, previous surgeries and trauma.

**Drug Hx** (what he is taking (ACEI, Aspirin , B blocker, inhaler ), any recent change , adherence to medications)

**Family Hx** asthma , atopy , hay fever , eczema ,TB, Lung cancer , CHF

**Social Hx**: Smoking history (# of pack years), Pets, ventilated house, alcohol, travel history, contact with sick people nor elderly people/ prisoners.

**Review of systems**

**DDX: All respiratory (OLD, RLD) and cardiac diseases, GERD, Side effect of drug)**

****Investigations:**

1. CXR → Pneumonia, Pulmonary edema, Asthma, COPD
2. Spirometry → Asthma, COPD, RLD
3. CT-angiography And D-dimer → PE
4. CBC → Pneumonia
5. 24 Hour esophageal PH **monitoring** → GERD.

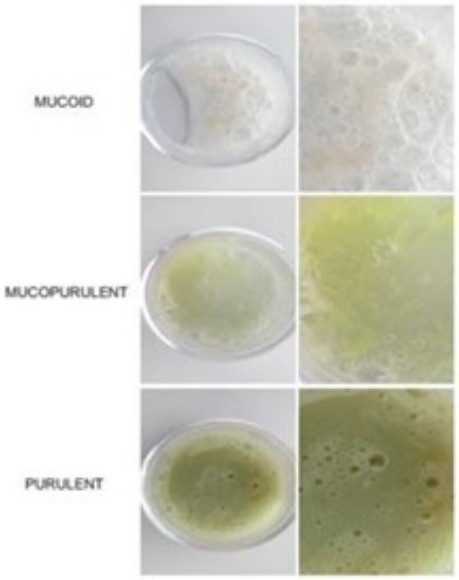
| | Normal chest X-ray | Abnormal chest X-ray |
|---|---|---|
| **Acute cough** (<3 weeks) | Viral respiratory tract infection<br>Bacterial infection (acute bronchitis)<br>Inhaled foreign body<br>Inhalation of irritant dusts/fumes | Pneumonia<br>Inhaled foreign body<br>Acute hypersensitivity pneumonitis |
| **Chronic cough** (>8 weeks) | Gastro-oesophageal reflux disease<br>Asthma<br>Postviral bronchial hyperreactivity<br>Rhinitis/sinusitis<br>Cigarette smoking<br>Drugs, especially angiotensin-converting enzyme inhibitors<br>Irritant dusts/fumes | Lung tumour<br>Tuberculosis<br>Interstitial lung disease<br>Bronchiectasis |

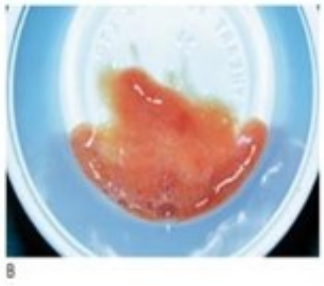
**1-Acute cough: URTIs, llergic Rhinitis, Pneumonia**
**2- Chronic cough: Chronic bronchitis, Asthma, Postnasal drip**

# Color

- Clear (mucoid): COPD/bronchiectasis without current infection/rhinitis.
- Yellow (mucopurulent): acute lower respiratory tract infection/asthma.
- Green (purulent): current infection – acute disease or exacerbation of chronic disease, such as COPD.
- Red/brown (rusty): pneumococcal pneumonia.
  Try to distinguish between rusty and frank red blood.
- Pink (serous/frothy): acute pulmonary edema.

In bronchiectasis, the color of sputum may be used to guide the need for antibiotic treatment.



Color of sputum :
1-Rusty → S.pneumonia
2 – Red Current jelly → Klebsiella
3- frothy pink → P.edema
4- Greenish → Pneumonia

## 7.4 Causes of haemoptysis

| | |
|---|---|
| **Tumour** | |
| **Malignant** | **Benign** |
| • Lung cancer<br>• Endobronchial metastases | • Bronchial carcinoid |
| **Infection** | |
| • Bronchiectasis<br>• Tuberculosis<br>• Lung abscess | • Mycetoma<br>• Cystic fibrosis |
| **Vascular** | |
| • Pulmonary infarction<br>• Vasculitis<br>• Polyangiitis<br>• Trauma<br>• Inhaled foreign body<br>• Chest trauma<br>• Cardiac<br>• Mitral valve disease<br>• Haematological<br>• Blood dyscrasias | • Arteriovenous malformation<br>• Goodpasture's syndrome<br>• Iatrogenic<br>• Bronchoscopic biopsy<br>• Transthoracic lung biopsy<br>• Bronchoscopic diathermy<br>• Acute left ventricular failure<br>• Anticoagulation |

# Massive Haemoptysis:

more than 20ml/one time, OR more than 200ml/24hrs.

Larger volumes of hemoptysis suggest:

**hemoptysis (Frank blood / blood stained) → Pneumonia/ CA/ TB / PE**

- lung cancer eroding a pulmonary vessel
- bronchiectasis (such as in cystic fibrosis)
- Cavitatory disease (such as bleeding into an aspergilloma).
- Pulmonary vasculitis
- Pulmonary arteriovenous malformation.

# DM follow up

**Patient profile** (name , age , occupation , marital status, address)

**Chief complaint + duration**

## Analysis of the Chief Complaint

How **long do you have DM**? At **which age** you have been diagnosed?

What was the **first chief complaint** (Weight loss/ screening/ recurrent skin infections/polyphagia/ polydipsia)? What was your blood glucose test measure?

Is your blood sugar **controlled**? / Do you measure it regularly? / How much is the reading?

**Lab results** (if the patient is educated) what is the type? And their results? And if they are not onthe required level ask about the **compliance** with diet and medications or any stressful conditions?
**on insulin or oral** hypoglycemic drugs? / any drug complications? / Do you require insulinfrom the start? Exercise and diet (What the type of diet you follow up)? Obesity and BMI?

**Severity** (**Life affection**)
Have you been able to work? How did your family cope with your problem?

**Complications**

I. **Retinopathy** → Decreased or loss of vision
II. **Nephropathy** → Polyuria, Anuria, Frothy urine, Uremia (Nausea & Vomiting / Abdominal pain)
III. **Neuropathy** → Paresthesia of limbs, Urinary incontinence
IV. **Atherosclerosis** → MI (Chest pain / SOB), CVA (Headache / Paralysis), PVD(Foot ulcers / Intermittent claudication), HX: of MI or CVA
V. **Hypoglycemia** → Hunger, Tremor, Palpitation, Sweating, Pallor, Irritability, Confusion, Seizures. Do you know if that happens what to do? (Check the sugar levels/ take an oralglucose/ go to hospital).
VI. **DKA** → Nausea & Vomiting, Polyuria, Polydipsia, Anorexia, Kussmaul breathing, Tachycardia,Dehydration. Have you had it before? And ask about admission to hospital?
VII. **Other** → Hair loss, Easy bruising, delayed wound healing, Sexual Dysfunction.

**And if you do any testing to monitor** these complications regularly? (**LDL/ HBA1C/ Scr/ Foundoscopy**).

**Review of systems**

**Past medical and surgical:** Any chronic illnesses, and any previous surgery

**Drug Hx & Allergy**

**Family Hx:** I. Same condition II. Chronic illnesses

**Social Hx:** Smoking history (# of pack years), alcohol, travel history, drug abuse.

# Weight Loss

**Introduce yourself , take permission**
**Patient profile** (name , age , occupation , marital status, address)
**Chief complaint + duration**

## Analysis of the Chief Complaint (HOPI)

a) Are you following **diet** program?
b) How **many Kg** you lost? Time?
c) Last time you **weigh yourself**, How much?
d) How is **appetite**? How is diet? is there any **problem that prevents eating** (teeth pain/odynophagia)?
**e)** Increase **in Physical activity.**
g) **Previous episode**.

## Associated symptoms

Malignancy → Fever, Night sweating, Palpable Lump anywhere in body.

**GI causes :**

A) **Malabsorption** → Abdominal pain, Abdominal distention, Diarrhea.
B) **IBD** → Lower abdominal pain, Nausea & Vomiting, Constipation, Bloody Diarrhea .
C) **PUD (GU)** → Epigastric pain related to food, Bloating, Melena, Upper GI bleeding.

**Endocrine causes :**

A) Hyperthyroidism → Heat intolerance, Palpitation, Increased Appetite.
B) DM → Polyuria, Polydipsia, Polyphagia, Recurrent infections.
C) Addison's disease → Hyperpigmentation, Orthostatic hypotension, Fatigue.

**Depression & Anxiety:** loss of interest , change in mood , poor memory , insomnia . RS

**Sx:** Cough, SOB, Hemoptysis (TB)

**Cardiac Sx:** Orthopnea, PNDs, Ankle swelling.

**Renal disease:** Oliguria, Altered mental status.

**Liver failure& hepatitis:** jaundice, edema, bleeding tendency , abdominal distention.

**Anemia Sx:** Pallor, Fatigue, Dizziness

**CTD:** Arthritis, Skin rash, morning stiffness.

**Review of systems**

**Past medical and surgical**

- Previous surgeries
- Chronic illnesses (DM, HTN, Hyperlipidemia). Hx of recurrent infection (HIV)

**Drug Hx & Allergy:** → Thyroxin , laxatives , Metformin

**Family Hx:** Same condition , Chronic illness

**Social Hx:** Smoking history (# of pack years), alcohol, Drug abuse, travel history.

**DDx: Malabsorption Diseases, Hyperthyroidism, DM, Malignancy, Addison's disease, IBD, PUD , TB , Renal failure, Depression )**.

****Investigations:**
1. **Malabsorption** → Lower GI endoscopy.
2. **Hyperthyroidism** → TFT.
3. **DM** → Fasting blood glucose level, OGTT, HgbA1C.
4. **Addison's disease** → ACTH stimulation test.
5. **PUD** → Upper GI endoscopy.
6. **TB** → BPD, Interferon Gamma, ziehl neelsen stain (culture), Chest Xray

# Fatigue / Pallor (Anemia)

**Introduce yourself , take permission**
**Patient profile (name , age , occupation , marital status)**
**Chief complaint + duration (fatigue)**

## Analysis of the Chief Complaint (OPPERATS)

**Onset** (sudden or gradual, progression, first time)**Progression get worse or better with time**
**Previous Episodes**
**Exacerbating , Relieving:**

a) Rest over night
b) Exercise

**Timing** (Episodic, Constant)
**Severity effect on life.**

## Associated symptoms

I. Constitutional
Fever, wt loss, night sweat, loss of appetite.

II. ANEMIA Sx :
Pallor , exertional dyspnea and if:

1- Hemolysis: jaundice, dark urine.
2- B12 def. : neurological sx , other autoimmune diseases.
3- Thalassemia: change in facial features, delayed puberty.
4- Sickle cell: Recurrent hand or leg swelling, recurrent abdominal pain.
5- Diet : Pica , Strict vegetarian , Fava beans ingestion dyspnea on exertion, postural (orthostatic) lightheadedness

III. RS + CVS: Chest pain, Orthopnea , PND , Ankle swelling , SOB, Palpitation, Cough.

IV. Blood loss :

- GI : hematemesis , melena , hematochezia
- Trauma
- Bleeding disorders : Epistaxis , Gum bleed , hemarthrosis.
- GU : hematuria , menorrhagia .

**Review of systems**
**Past medical and surgical**
Preivous admission , blood transfusion ,any chronic diseases (esp. renal)
**Drug Hx** PAINS ( Primiquine , Aspirin , Isonizid , Nirtrofurantoin ,Sulfa Drugs), Penicillin's , Methyl dopa
**Family Hx** Spleenectomy , hemolysis , bleeding disorders
**Social Hx**: Smoking history (# of pack years), alcohol, travel history

****Investigations:**

1. **CBC → look for Hgb and MCV.**
2. **Blood film**
3. **Iron study**
4. **Hgb electrophersis.**

# Dysphagia

## (Etiology)

• **Subjective sensation of <u>difficulty or abnormal swallowing</u>.**

• **Tow main classes:**

1. **Oropharyngeal:**
   **Difficulty in initiating a swallow, associated with chocking, coughing, aspiration and globus sensation.**

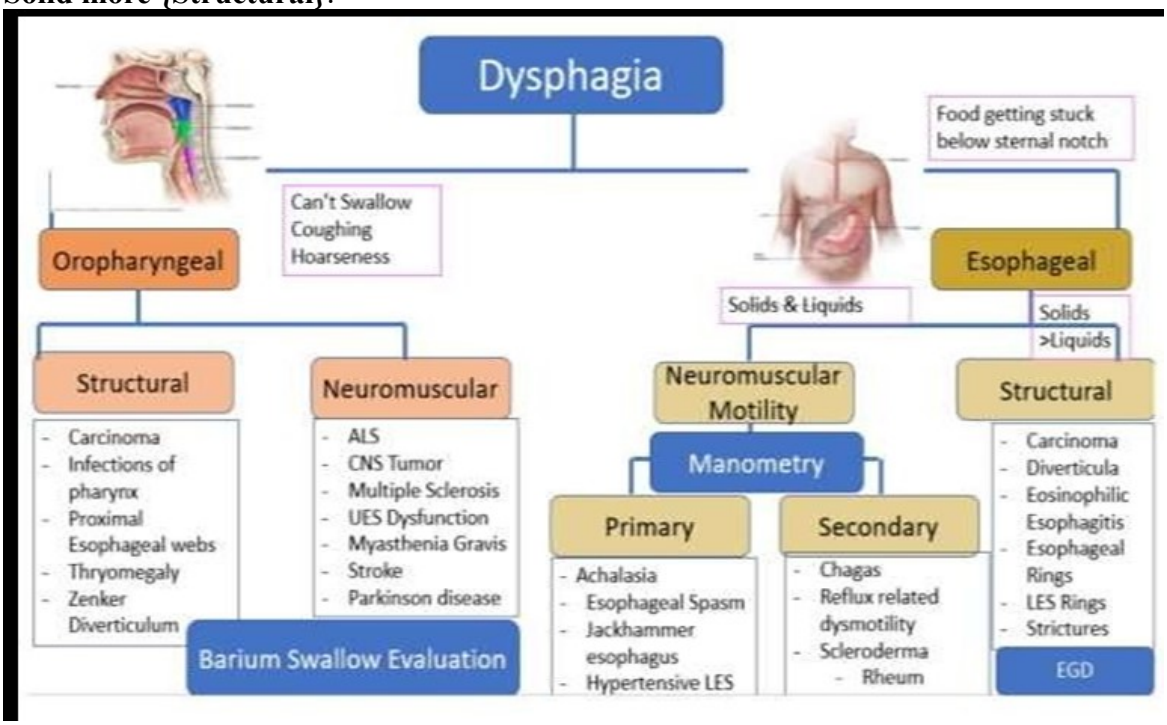
   **Usually due to Varity of neurological, structural, infectious, and iatrogenic cause.**

2. **Esophageal:**
   - **Motality disorders.**
   - **Structural lesions.**
   - **Esophagitis.**

## Dysphagia (Clinical presentation)

1. **Oropharyngeal dysphagia:**
   **Difficulty in initiating a swallow, associated with chocking, coughing, aspiration and globus sensation.**

2. **Esophageal dysphagia:**
   **Difficulty in swallowing seconds after initial swallow, associated with Sensation of food in esophagus.**
   **Solid and liquid {Motality disorders}.**
   **Solid more {Structural}.**



## (Work-up)

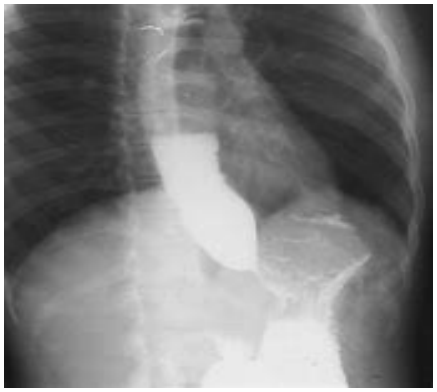
• **Esophageal dysphagia:**

1. **Upper Endoscopy (generally first done to rule out structural lesions).**
2. **Barium swallow.**
3. **Manometry.**

• **Oropharyngeal dysphagia:**

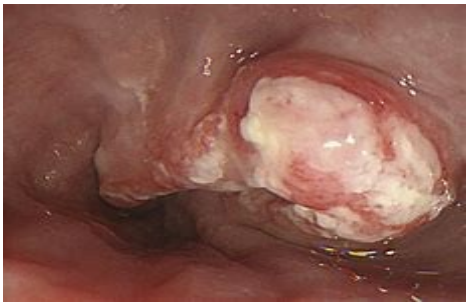
1. **Modified barium swallow (Investigations oral, pharyngeal, and esophageal).**

## Achalasia



- **Failure of LES to relax:**
- **(idiopathic/ autoimmune/ Chagas/ malignancy).**
- **Diagnosis:**
- **(EGD/ Manometry/ Barium swallow (rate-tail)).**
- **Manometry:**
  **Incomplete LES relaxation, Elevated resting pressure (>45 mmHg), Aperistalsis of esophageal body.**
- **Management:**
  **(Drugs (CCB/NITRATE), Botox, Pneumatic dilation, Surgical myotomy (95% in first year).**

## Esophageal cancer



- **Two types:**
  **1. Squamous cell carcinoma** (upper 2/3, Smoking/ ETOH/ Achalasia/ Strictures/ dietary(N-nitroso), 50%, metastasis to cervical and mediastinal LN).

  **2. Adenocarcinoma:** (Distal 1/3, Barret's/ LONGSTANDING GERD/ ETOH, 50%, Metastasis to celiac and gastric LN).

- **CLX:** Progressive dysphagia (Solids then liquids) + Constitutional symptoms + RS symptoms + Horsiness.
- **DX:** EGD with BX, CT(staging), PET.
- **TRX:** Surgery + Chemo-radiotherapy.
- **Poor prognosis because of late presentation**. (5-YR = 5%-30%)

**Introduce yourself , take permission**
**Patient profile (name , age , occupation , marital status, address)**
**Chief complaint + duration (odynophagia?!!!)**

## Analysis of the Chief Complaint

**Site:** At what level does the food stick
**Onset:** (sudden or gradual)
**Character** Fluids, Solids or both (at the same time!), Stage the dysphagia occurs: initiating swallowing, after initiation swallowing?
**Associated symptoms (finish the CC analysis then ask about them ↓) Timing** (Progression, episodic (intermittent) or continuous)
**Severity** (Is there complete obstruction, regurgitation?)

## Associated symptoms

**Constitutional:**
- Weight loss
- Loss of appetite
- Night sweat.
- fever

**URTI:** Cough, nasal congestion, sore throat.
**Neurological:** vision problem, tremor, Recurrent choking (previous strokes).
**GI:** Nausea/vomiting, Regurgitation, heart burn, Bloating/abdominal swelling, Early satiety, Jaundice/ RUQP/ Steatorrhea, Bowel habit, Melena and Hematochezia.
**Scleroderma:** Skin tightness and discoloration (Raynaud Phenomenon).
**Myasthenia gravis:** Ptosis, diplopia, fatigue
**Pharyngeal pouch** (zenker diverticulum) Neck bulge , gurgle on drinking or halitosis ?

**Review of systems**
**Past medical +Blood transfusion and surgical +Trauma.**
• Stroke • Thyroid problems (Goiter) • PUD and GERD •Scleroderma •Iron deficiency.
• Previous admission.
• Previous surgeries.
**Drug HX:** → • NSAIDs • Bisphosphonates/Doxycycline •Use of antacids (related to GERD and PUD).
**Family HX:** Esophageal cancer, neuromuscular diseases , any chronic illnesses
**Social HX**: • Alcohol (peptic ulcer disease, gastritis) • Smoking • Illicit drug use • Diet: spicy foods )
peptic ulcer disease )

# Epigastric pain

**Introduce yourself, take permission**

**Patient profile (name, age , occupation , address , marital status)**

**Chief complaint + duration**

**Analysis of the Chief Complaint (SOCRATES)**

**Site**

**Onset** (sudden or gradual, progression, first time)

**Character**
- Squeezing
- Sharp/stabbing
- Burning/pricking
- Dull

**Radiation**
- To back
- To Right shoulder, scapula
- Up to chest
- Diffuse

**Associated symptoms (finish the CC analysis then ask about them ↓)**

**Timing** (episodic or continuous)

**Exacerbating:**
- Eating or fasting.
- Increased by swallowing.
- Fatty foods.
- Acidic/spicy foods/coffee.
- Does it increase by movement or breathing?

**Relieving:**
- Eating or fating
- Certain position (lying on one side, or leaning forward)
- Bowel motion.
- Drugs

**Severity** (from 0-10).

**Associated symptoms**

- **GI symptoms**:
- Dysphagia, Regurgitation, heart burn, hoarseness of voice.
- Dyspepsia , N+V
- Bloating/abdominal swelling (generalized/localized)
- Early satiety
- Jaundice/ RUQP/ Steatorrhea, urine & stool changes, itching
- Bowel habit, diarrhea/constipation
- Flatulence
- Melena and Hematochezia

- **Heart symptoms:** Chest pain, sweating, SOB, PND, orthopnea, ankle swelling,.

- **Respiratory symptoms:** Cough, SOB, wheeze.

**General**

- Fever, weight loss, loss of appetite, night sweat.

**Risk Factors (always ask about smoking and alcohol)**

I. PUD → Smoking, NSAIDS, Alcohol
II. Hepatitis → Alcohol, blood Transfusion, HBV infection, DM, contact with patient having Hepatitis
III. MI → Smoking, HTN, DM, Hyperlipidemia, Family Hx
IV. Cholecystitis→ Family Hx of gall bladder stones

**Review of systems**

**Past medical and surgical**

- Previous surgeries.
- Hepatitis, or history of blood transfusions, sexual intercourse, contact with jaundiced patient.

**Drug Hx:** NSAIDs, Steroids ,antacids, anticoagulant

**Family Hx:** Ask about relevant conditions related to the history (Gastric cancer, PUD … etc.), andany chronic diseases.

**Social Hx**: Smoking history (# of pack years), alcohol, travel history

# Vomiting

Introduce yourself , take permission

Patient profile (name , age , occupation , marital status)

Chief complaint + duration (May help in assessing the dehydration risk)

## Analysis of the Chief Complaint (FCBCAM/ OPPERATS)

**Onset: -** Sudden (Gastroenteritis/ Bowel obstruction/ appendicitis) - Insidious onset of vomiting (consider pregnancy, bulimia, brain tumor .. etc.).

**Progression** (Getting worse or better)

**Previous episodes**

**Exacerbating and relieving factors.**

**Time:** Constant or episodic with free interval.

**Severity:** Assess the dehydration symptoms (Feeling thirst/ dry mucous membrane/ oliguria/ alteredmental status).

**Frequency** (How many times)/ Forceful (Projectile vomiting) = Gastric outlet obstruction/ IICP.

**Content** (Food or not and if it is digested) Undigested food = may be due to motility disorders (likeachalasia) or a structural disorder (like a pharyngeal pouch).

**Bloody** (Hematemesis or coffee ground appearance) Bright red = may be fresh blood caused by a Mallory Weis tear or esophageal varices / Coffee ground = may indicate an upper GI bleed such as in ableeding peptic ulcer.

**Color** (Green/yellow vomit = may be bilious and caused by small bowel obstruction)

**Amount** (In cups).

**Meal relation** (Before or after meal/ not related).

## Associated symptoms

**GI Sx :**• Dysphagia • Dyspepsia • Regurgitation, heart burn , hoarseness of voice • Bloating/abdominal swelling (generalized/localized) •Early satiety • Jaundice/ RUQP/ Steatorrhea • Abdominal distension • Bowel habit,diarrhea/constipation • Flatulence •Melena and Hematochezia • Fevers, weight loss, loss of appetite, night sweat.

Fever and diarrhea (may indicated gastroenteritis)

Abdominal pain (e.g. cholecystitis, pancreatitis, appendicitis, bowel obstruction, renal colic etc)

**Cardiac Sx:** • Chest pain, palpitation, sweating, pallor (myocardial infarction)

**RENAL Sx :** Urgency, frequency, hesitancy, flank or loin pain, fever, intermittency, dribbling (UTI/Stones)

**CNS Sx:** Headache, vertigo, visual change, vertigo (migraine, raised intracranial pressure, stroke, innerear dysfunction) • Early morning headache, altered mental status, seizures, focal neurological symptoms, photophobia, phonophobia (IICP/ meningitis)

**(DKA)** Polyuria, polydipsia, acetone breathing

**(Malignancy)** Weight loss,anroxeia , night sweating .

**Risk Factors (always ask about smoking and alcohol)**

I. PUD → Smoking, NSAIDS, Alcohol

II. Hepatitis → Alcohol, blood Transfusion, HBV infection, DM, contact with patient having Hepatitis

III. MI → Smoking, HTN, DM, Hyperlipidemia, Family Hx

IV. Cholecystitis→ Family Hx of gall bladder stones.

V. DKA→ Family history of DM.

VI. Gastroenteritis: Recent travel, Recent takeaway/eaten out (food poisoning e.g. Campylobacter),Re-cooked rice (Bacillus cereus infection), family member with the same symptoms

**Review of systems**

**Past medical and surgical**

- Previous surgeries.
- Hepatitis, or history of blood transfusions, sexual intercourse, contact with jaundiced patient.

**Drug Hx:** Chemotherapy, antibiotics, oral contraceptive.

**Family Hx:** Ask about relevant conditions related to the history (Gastric cancer, PUD … etc.), and any
chronic diseases.

**Social Hx**: Smoking history (# of pack years), alcohol, travel history, drug abuse.

GASTROINTESTINAL CAUSES: Gastroenteritis, Bowel obstruction, Appendicitis, Peptic ulcer, Renal colic,Pancreatitis.

NEUROLOGICAL CAUSES: Migraine, Raised intracranial pressure, Inner ear pathology. ENDOCRINE CAUSES: Pregnancy, Addison's disease, Renal failure, Diabetic ketoacidosis.PSYCHIATRIC CAUSES: Bulimia, Anorexia.

MEDICATION SIDE EFFECTS.

# Abdominal Distention

**Introduce yourself , take permission**

**Patient profile (name , age , occupation , marital status)**

**Chief complaint + duration**

## Analysis of the Chief Complaint

I. **Onset** (duration, progression, first time)

II. **Character:**

a) Painful?

b) Swelling on other site (Leg/ genital/ eye)?

**III. Exacerbating & Relieving factors** (eg. Food).

**Associated symptoms**

I. **CVS:** a) SOB b) Orthopnea c) PND d) Ankle swelling e) Palpitations → Heart Failure

II. **GI**

a) Nausea & Vomiting → Intestinal Obstruction, Cirrhosis
b) UGI bleeding → Cirrhosis (↑ Bleeding tendency)
c) Diarrhea
d) Constipation
e) Jaundice → Cirrhosis

**III. UGS** → Renal Failure

a) Renal Pain
b) Urine (amount/color/frequency)
c) Edema around the eyes

**Risk Factors (always ask about smoking and alcohol)**

I. **HF** → Previous MI, HTN, DM, Smoking, Vulvular heart disease
II. **Cirrhosis** → Alcohol, Hx of hepatitis, Hx of blood Transfusion
III. **RF** → DM, Polycystic kidney disease, HTN.

**Review of systems**

**Past medical and surgical:** Chronic illnesses (DM, HTN, Hyperlipidemia), Blood disorders, Previous
surgeries.

**Drug Hx:** Steroids, IV Fluids

**Family Hx:** Ask about relevant conditions related to the history, and any chronic diseases.

**Social Hx:** Smoking history (# of pack years), alcohol, travel history, drug abuse.

# Hematemisis

**Introduce yourself, take permission**
**Patient profile (name, age, occupation, address, marital status)**
**Chief complaint + duration**
**Analysis of the Chief Complaint**

## Severity:

**Onset:**
- Sudden acute
- Chronic
- Insidious onset of vomiting

**Progression** (Getting worse or better)

**Previous episodes**

**Character:**

**Smell**

**Color** (Fresh bright red, dark color "coffee grounds").

**Amount** (In cups).

**Associated bleeding from other sites**

**Time:** Constant or episodic.

**Exacerbating and relieving factors:**
- NSAIDs → PUD
- Food → GU
- Trauma to abdomen → Esophageal perforation
- Alcohol, Vomiting/retching → Mallory-Weiss tear

## Associated symptoms

**I. GI:**

a) Heartburn and regurgitation
b) Dysphagia and odynophagia.
c) Dyspepsia
d) Abdominal Pain → Epigastric → PUD
e) Abdominal Distention
f) Jaundice / change in urine & stool color / itching/ limb swelling→ Cirrhosis
g) Diarrhea or constipation
h) Hematochezia/ anal pain or anal lump.

**II. Blood disorders:** Bleeding from other site, ecchymosis, purpura, petechial, hematuria.
**III. Constitutional symptoms:** Fever, Weight loss, Anorexia, Night sweat.

## Risk Factors (always ask about smoking and alcohol)

I. **PUD** → Smoking, NSAIDS, Alcohol
II. **Bleeding disorders** → Drugs {Anti-coagulants (Heparin or Warfarin) / NSAIDS (Aspirin).
III. **Cirrhosis** → Alcohol, Blood transfusion, HBV infection, sexual intercourse, easy bruising, leg swelling.
IV. **Mallory –Weiss** >> binge drinking

## Review of systems

- **Past medical and surgical**: GERD, PUD, liver problems, coagulopathy, IBD, Colorectal cancer,previous GI surgery, AAA repair (Aorto-enteric fistula).
- **Drug Hx:** NSAIDs, steroid, aspirin, warfarin
- **Family Hx:** Ask about relevant conditions related to the history (Gastric cancer, PUD, colon cancer… etc.), and any chronic diseases.

# Constipation

**Introduce yourself, take permission**
**Patient profile (name, age, occupation , address , marital status)**
**Chief complaint + duration**

## Analysis of the Chief Complaint

**Onset** (sudden or gradual, progression, first time) = **OPP**
**Frequency:** Times per day
**Consistency:** (Sausage shape, separate hard lumps like nuts)
**Blood**
**Caliber**: large caliber, narrow or pencil thin tools
**Amount (small/large)**
**Mucous**
**Pain**
**Melena**
**Associated symptoms (finish the CC analysis then ask about them ↓)**

## Associated symptoms

**Constitutional**:
- Weight Loss
- Anorexia
- fever
- night sweat

Anal pain or itching → Hemorrhoid, Perianal fissure

**GI: From above to down**
a) Mouth ulcers → IBD
b) Nausea & Vomiting → Intestinal obstruction
c) Abdominal pain >> Intestinal obstruction, IBD
d) Abdominal distention → IBS, Intestinal obstruction
e) Alternating diarrhea → IBS

**MSS**
Skin rash, Joint Pain, Eye Symptoms

**Hypothyroidism:** Cold intolerance, Weight Gain, fatigue.

**DM:** Polyuria, Polydipsia, Polyphagia

**Hypercalcemia:** Renal stones, bone pain, polyuria, abdominal pain.

**Dehydration:** feeling thirst, dark urine, oliguria

**Risk factors**
I. IBD → Family History
II. Colon CA → Low fiber diet, family History
III. Intestinal obstruction (Adhesions) → Previous surgeries

## Review of systems

## Past medical and surgical

- Previous attacks
- Previous surgeries → Intestinal obstruction (Adhesions)
- Chronic illness
- (DM, HTN, Hyperlipidemia) , History of trauma (spinal cord)

**Drug Hx:** →Iron and Ca supplement, opioids, thiazides, Antacids
**Family Hx:** Ask about relevant conditions related (IBD, Colon Cancer) and any chronic diseases.
**Social Hx**: Smoking history (# of pack years), alcohol, travel history, Diet and water intake

# Diarrhea

**Introduce yourself , take permission**
**Patient profile (name , age , occupation , marital status)**
**Chief complaint + duration**

**Analysis of the Chief Complaint (FCBCAO)**
**Frequency how many times a day? Character (mucus (greasy) or watery)**
**Blood (fresh blood ( hematochezia) , melena )Color (fatty . pale)**
**Amount**
**Odor (foul smelling)**
**Onset** (duration, sudden or gradual, progression, first time) = **OPP**
**Associated symptoms (finish the CC analysis then ask about them ↓)Timing** (episodic (at night) or continuous)
**Exacerbating:**
- Dietary factors, fatty foods (gallstones), (Gastric ulcer disease).
**Relieving:**
Bowel motion (defecation)Drugs.
**Severity** → dehydration symptoms ( thirst . oliguria , dark urine, Dry mucous membranes )

## Associated symptoms

•**GI symptoms**
a) Mouth ulcers → IBD, Celiac Disease
b) Nausea & Vomiting → GE, PUD (if bloody vomit)
c) Abdominal pain → GE, IBD (Crohn's), Celiac disease, CA
d) Abdominal distention + Alternating constipation → IBS
•**Constitutional**
Fever , wt loss , night sweat , loss of appetite
•**MSS** Skin rash , Joint Pain , Eye Symptoms.

## Risk Factors (always ask about smoking and alcohol)

I. GE → Eating anything spoiled
II. Bacillary dysentery / ameba → Recent travel to endemic area
III. IBD → Family hx
IV. Colon CA → Low fiber diet, family hx
V. Celiac → Family hx and hx of allergy

## Review of systems

**Past medical and surgical**
• Previous surgeries.
• Previous attacks
• Chronic illnesses (DM, HTN, Hyperlipidemia) ,contact with jaundiced patient.

**Drug Hx:** → Antibiotics, NSAID, Laxatives

**Family Hx:** Ask about relevant conditions related to the history (Gastric cancer, PUD … etc.), and any chronic diseases.

**Social Hx**: Smoking history (# of pack years), alcohol, travel history

**(DDX: Gastroenteritis, Bacillary dysentery or Ameba, IBD, Colon CA, PUD, IBS, Celiac disease) Investigations:**

1. Stool Culture → Infectious Colitis
2. Endoscope → Colon CA, IBD, Celiac disease.

# Jaundice

**Introduce yourself, take permission**
**Patient profile (name, age , occupation , address , marital status)**
**Chief complaint + duration**

## Analysis of the Chief Complaint

I. **Site**
   a) Eyes (Sclera)
   b) Skin

II. **Onset** ( sudden or gradual, progression, first time) OPP

III. **Associated symptoms (finish the CC analysis then ask about them ↓)**

IV. **Exacerbating and relieving factors** (Drugs, exercise, fasting, and certain foods like fava beans).

V. **Time**: Intermittent (e.g. Gilbert's syndrome), continuous.

## Associated symptoms

I. **Prehepatic: Hemolytic Anemia** → Fatigue, Dizziness, Pallor, SOB.
II. **Hepatic:**
   a) **Hepatitis** → Fever, RUQ pain, Nausea & VomitingAutoimmune →Arthralgia, vitiligo, skin rashes
   b) **Cirrhosis** → Ascites, Limb swelling, Bleeding tendency , Hematemesis , Anal lump
III. **Post hepatic: - Obstructive Jaundice**→ Itching , Dark urine and pale stool
   **- Constitutional (Periampullary tumor):**
   - Weight Loss - Anorexia - nigh sweat - steatorrhea – DM
IV. **GI Sx:** from above to below.

## Risk factors: (always ask about smoking and alcohol)

1. **Pre-hepatic:** Hx of blood diseases (Thalassemia / G6PD)**, Drugs** → PAINS (Primiqune , Aspirin , Isonizid ,Nitrofurantoin, Sulfa drugs)
2. **Hepatic:** , Hx of hepatitis , Hx of blood Transfusion, or contact w/ jaundiced patient
3. **Post-hepatic:** Hx of gallstones, Hx of cholecystitis, **Hx of** IBD **(Crohn's)**

**Past medical and surgical**

- Previous surgeries
- Chronic illnesses (DM, HTN, Hyperlipidemia)

**Drug Hx:** → PAINS (Primiqune , Aspirin , Isonizid ,Nitrofurantoin, Sulfa drugs) , OCPs

**Family Hx:** Hx of blood diseases (Thalassemia / G6PD), Hepatitis and liver failure. And anychronic diseases.

**Social Hx**: Smoking history (# of pack years), alcohol, Drug abuse, travel history, Sexual history

# Leg swelling

**Introduce yourself , take permission**

**Patient profile (name , age , occupation , marital status, address)**

**Chief complaint + duration**

## Analysis of the Chief Complaint

**Site**

a) Extent of swelling

b) Unilateral or bilateral, Other sites: Periorbital? Abdomen? Genitalia? Back? Hands?

**Onset** (sudden or gradual)

Do they progress with activity or throughout the day? Or with lying down?

**Character** (with)

a) Redness

b) Hotness

c) Tenderness

d) itching

**Associated symptoms (finish the CC analysis then ask about them ↓)Exacerbating, Relieving.**

**Severity:** loss of the limb function.

**Associated symptoms**

**I. Unilateral Swelling**

a) **DVT**:
Limb → Redness, Hotness, Tenderness
PE Symptoms → Chest pain, SOB, Hemoptysis.
Risk factors → recent travel, surgery, immobility, pregnancy, OCP, previous DVTs.

b) **Cellulitis** → Fever & Chills, Brown areas, Rapid progression, Ulcers.

c) **Venous Obstruction:** HX of pelvic tumor, AV fistula.

d) **Trauma**.

e) **Joint disease:** Pain, hotness, redness, skin rash, decreased range of movement.

**II. Bilateral Swelling**

a) **HF** → Cough, Orthopnea, PND.

b) **Liver cirrhosis** → Bleeding tendency, Abdominal distention.

c) **Renal failure** → Frequency, Nocturia, Urine (color/smell/ amount)

d) **Hypoproteinemia** → Nutrition, Malabsorption

e) **Hypothyroidism** → Weight gain, Cold intolerance, Lethargy and Fatigue

**Review of systems**

**Past medical and surgical**

Chronic illnesses (DM, HTN, Hyperlipidemia) , AllergyPast surgeries and admissions.

**Drug Hx** NSAIDs, steroids, Ca+2 Ch. Blockers (Nifedipine, Amlodipine)

**Family Hx** Ask about relevant conditions related to the history (thrombophilia , cancers)

**Social Hx**: Smoking history (# of pack years), alcohol, travel history

****Investigations:**

1. Doppler U/S and D-dimer → DVT
2. Liver function test (LFT) → Liver cirrhosis
3. Kidney function test (KFT) → Renal failure
4. Thyroid function test (TFT) → Hypothyroidism
5. CBC → Cellulitis

# Joint Pain

**((DDx: RA, SLE, Scleroderma, Inflammatory myopathy, Spondyloarthropathies, Gout, Enteropathic Arthritis, Septic arthritis, FMF, Behcet's disease))**

**Introduce yourself , take permission**
**Patient profile (name , age , occupation , marital status…)**
**Chief complaint + duration**
**Analysis of the Chief Complaint (SOCRATES)**
**Site**
a) Which joints? o Small / o Large
b) How many joints affected?o One/Multiple
c) Symmetrical joint involvement? Yes / No **Onset (sudden or gradual, progression, first time)**Acute, severely painful (septic, gout, )
**Character**
a) Migratory b) Redness, swelling c) Joint deformities
**Radiation**
**Associated symptoms (finish the CC analysis then ask about them ↓)**
**Timing**
a) Continuous or intermittent
b) Day or Night
c) Morning Stiffness
**Exacerbating & Relieving factors**
- **Exacerbating:** a) Movement / b) Cold weather
- **Relieving:** a) Rest / b) Movementc) Drugs

**Severity →** affect movement and daily activities?

**Associated symptoms**
**Constitutional**
a) Fever → FMF, Septic Arthritis b) Weight loss c) Anorexia d) Fatigue
**MSS**
a) Skin rash (Inflammatory myopathy)
b) Skin Nodules
c) Muscle weakness
d) Skin thickening
e) Back pain

**CVS :** a) Chest pain /b) SOB
**RS**
a) cough
b) shortness of breath
c) pleuritic pain
**GUS**
a) haematuria, ankle swelling (nephritis)
b) Genital Ulcers

**GI**

a) Mouth Ulcers
b) Dysphagia
c) Vomiting
d) Abdominal pain
e) diarrhoea (Reiter's syndrome), bloody diarrhea (inflammatory bowel disease)

**Eye Symptoms**

**Review of systems**

**Past medical and surgical**

- Autoimmune conditions + Trauma
- Previous admissions.
- Previous surgeries.

**Drug Hx:** Thiazides (increase uric acid) , Long-term steroids (osteoporosis) • NSAIDs (gout) • Over-the-counter medication

**Family Hx:** Ask about relevant conditions related to the history , and any chronic diseases.

**Social Hx**: Smoking history (# of pack years), alcohol, travel history

## **Investigations:

1. RA → RF/anti-CCP/ESR
2. SLE → ANA/anti-smith AB/ant-ds DNA AB
3. Scleroderma → ANA/anti-centromere AB
4. Inflammatory myopathies → creatinine phosphokinase/aldose
5. Gout → synovial fluid analysis (urate crystals)

# Red Urine

**Introduce yourself , take permission**

**Patient profile (name , age , occupation , marital status)**

**Chief complaint + duration**

## Analysis of the Chief Complaint

**I. Onset** (duration, sudden or gradual, progression, first time) = OPP

**II. Character**

**a) Color**

o Bright Red →
o Dark brown →
0 Tea (Cola) color >>

**b) Part of stream**

o Initial →
o Total →
o Terminal →

**c) Clots →**

**d) Smell**

o Foul → UTI

**e) Amount**

**f) With Pain**

o Yes →
o No

**III. Timing**

a) Continuous or intermittent

b) Times per day

## Associated symptoms

**I. General**

a) Fever & Chills → Pyelonephritis

b) Weight loss → Malignancy

**II. UGS**

a) Flank pain → Kidney Stones

b) Dysuria → Urethritis

c) Frequency, Urgency, Nocturia

d) Straining, Poor stream

**III. GI**

a) Nausea & Vomiting → Pyelonephritis, Obstructive Jaundice

b) Abdominal pain

c) Jaundice

d) Pale stool

**IV. CVS**

a) Chest pain → Nephritic syndrome secondary to SLE

b) Palpitations → Pyelonephritis

c) Ankle edema → Nephritic syndrome

**V. MSS**

a) Skin rash (malar rash)

b) Joint Pain

c) Raynaud phenomena

d) Muscle pain or trauma → Rhabdomyolysis

**Risk Factors (always ask about smoking and alcohol)**

**I. Kidney Stones** → Family Hx of stones, Diet.

**II. Hemolytic Anemia** → G6PD deficiency, Family Hx

**III. Nephritic Syndrome** (due to PSGN) → Sore throat in the last 10 days

**IV. Rhabdomyolysis** → Strenuous exercise

**V. Food** → Dyes, Beetroot

**VI. Malignancy** → Age > 50.

## Review of systems

**Past medical and surgical:** Chronic illnesses (DM, HTN, Hyperlipidemia), Blood disorders, Previous surgeries.

**Drug Hx:** Rifampicin, Cyclophosphamide, Penicillin, NSAIDs, Aminoglycosides, Aspirin, Anticoagulants.

**Family Hx:** Ask about relevant conditions related to the history (Red urine), and any chronic diseases.(Bleeding disorders, Blood disorders)

**Social Hx:** Smoking history (# of pack years), alcohol, travel history, drug abuse.

## Chapter 11

# Physical Examination

# Cardiovascular physical examination

**H: Hello "Introduce yourself , take permission & Confirm patient identity"**

**E: Explain What are going to do & Exposure " above the waist"**

**L: Light**

**P: Privacy " ask for chaperone" & Position " At 45 degree"**

## GENERAL examination

**Hands: Periperal cyanosis , Capillary refill , Clubbing, Splinter hemorrhage , Tar staining, Osler node , Janeway lesion , Temperature , then take PR ( Check for Radio-Radial delay and Radio-Femoral delay , RR , measure BP and tremor.**

**Eyes: Xanthelasma , Corneal arcus , Conjunctival pallor and petechial hemorrhage.**

**Face: Malar flush, toungue for central cyanosis.**

**Neck: JVP** **Lower Limbs : Ankle edema**

## PRECORDIUM Examination

**Inspection(from 2 Sites)**

**From the foot of the bed & from Right Side of the patient**

1- Symmetry of the chest
2- Chest deformities
3- Attached devices & drains
4- Breathing pattern

1- Visible Scars (Sternotomy)
2- Superficial masses or swelling
3- Visible Pulsation

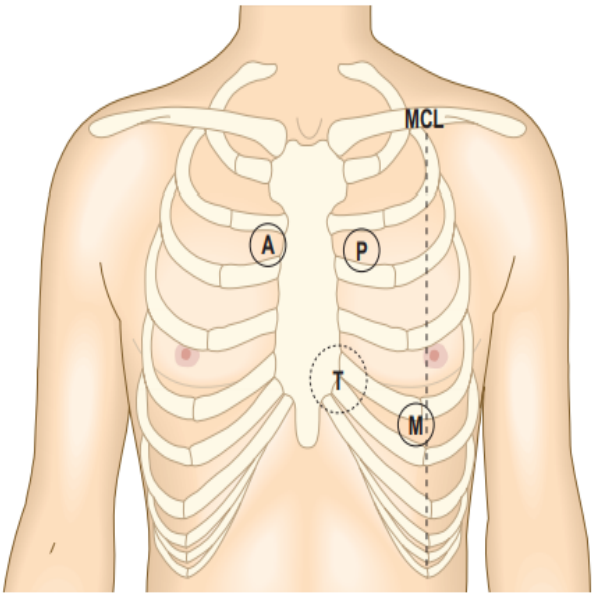
**Palpation (Is There Any Pain ?)**

1- **Heart : Apex beat (lt. Sided heaves) & Rt. Sided (left parasternal) heave.**
2- **Thrill : The tactile equivalent of a murmur ( Palpable vibration ).**

**Auscultation:**

1. **Heart Sound Vs. Added Sound**
2. **Murmur on ALL areas Of 4 Valves with bell and diaphragm.**
   **With 2 maneuvers for AR & MS**
   **- Don't forget to Auscultate Lung bases.**

**Thank the patient and clean your hands**



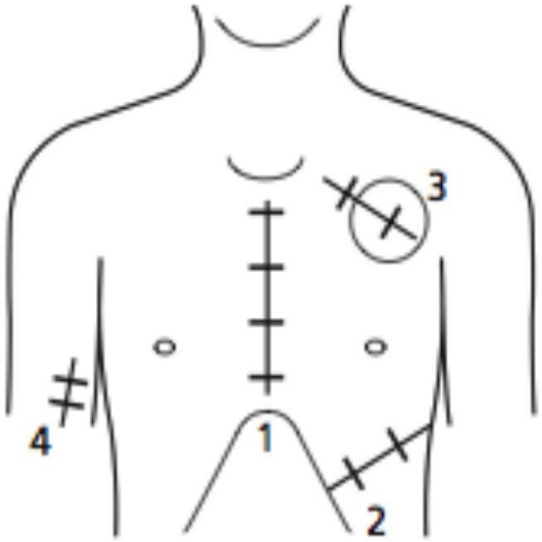
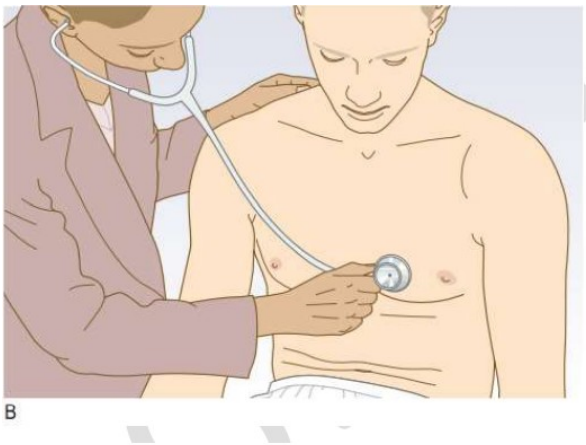
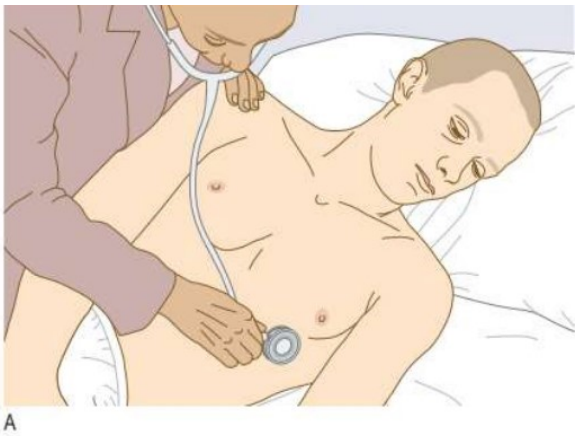




**Fig. 4.17 Sites for auscultation.** Sites at which murmurs from the relevant valves are usually, but not preferentially, heard. *A*, aortic; *M*, mitral; *MCL*, mid-clavicular line; *P*, pulmonary; *T*, tricuspid.



B

**For Aortic Regurgitation**



A

**For Mitral Stenosis**

# Respiratory physical examination.

**H: Hello "Introduce yourself , take permission & Confirm patient identity"**

**E: Explain What are going to do & Exposure " above the waist"**

**L: Light**

**P: Privacy " ask for chaperone" & Position " At 45 degree"**

## General examination

**Hands: Clubbing , Tar staining , Muscle wasting then take PR RR, tremor.**

**Eyes: Ptosis , Miosis , Conjunctival pallor .**

**Face: Plethoric face , toungue for central cyanosis.**

**Neck: JVP , Lymphnodes & Trachea.** **Lower Limbs : Ankle edema**

## Chest Examination

**Inspection(from 2 Sites)**

**From the foot of the bed & from Right Side of the patient**

1. **Symmetry of the chest**
2. **Chest deformities**
3. **Accessory muscle use**
4. **Attached devices & drains**
5. **5- Breathing pattern**

1. **Visible Scars (Thoracotomy)**
2. **Superficial masses or swelling**
3. **Dilated veins**
4. **You Should inspect Axilla**

## Palpation (Is There Any Pain?)

1. **Trachea : For tracheal deviation + Cricosternal distance**
2. **Heart : Apex beat & Rt. Sided (left parasternal) heave.**
3. **Chest expansion .**
4. **Tactile Vocal fremitus.**

**Percussion (Compare right with left, from TOP to bottom, then axilla).**

**Auscultation (Deep breaths; compare right with left, from top with bottom, then axillae) :**

**1-Breathing Sound Vs. Added Sound**

**2-Vocal resonance.**

**Thank the patient and clean your hands**

| Common causes of tracheal deviation | | |
|---|---|---|
| **Away from the side of the lesion** | **Towards to the side of the lesion** | **Upper mediastinal mass** |
| Tension pneumothorax | Upper lobe consolidation | Retrosternal Goiter |
| Massive pleural effusion | Upper lobe fibrosis | Lung cancer |
| | Pneumoectomy | Lymphoma |

| Tactile vocal fremitus / Vocal Resonance | |
|---|---|
| **Increased** | **Decreased** |
| **-Consolidation**<br>**-Dense pulmonary fibrosis**<br>**- Lobar collapse with patent major bronchi**<br>**- Lung mass** | **- Pleural effusion/ Heamothorax**<br>**- Obesity**<br>**- Pneumothorax**<br>**- Collapsed lung with obstructed major bronchi** |

## Percussion notes

| Resonant | Hyperresonant | Dull | Stony dull |
|---|---|---|---|
| • Normal lung | • Pneumothorax | • Pulmonary consolidation<br>• Pulmonary collapse<br>• Severe pulmonary fibrosis | • Pleural effusion<br>• Haemothorax |

# Abdominal physical examination

**H: Hello "Introduce yourself , take permission & Confirm patient identity"**
**E: Explain What are going to do & Exposure " xiphisternum to the symphysis pubis,"**
**L: Light**
**P: Privacy " ask for chaperone" & Position " SUPINE (lying flat) "**

## GENERAL examination

**Hands: Clubbing, Koilonychia (spoon-shaped nails) and signs of chronic liver disease, including leuconychia (white nails), Flapping Tremor , Dupuytren's Contracture and palmar erythema .**
**Eyes: Conjunctival pallor , Scleral Jaundice and Red eye .**
**Face: Mouth for IDA (angular cheilitis , atrophic glossitis) ,B12 Def. (beefy raw tounge) and Aphthous ulcer , Parotid enlargement .**
**Neck: for lymph nodes (Scalene LNs).**
**Chest : Gynecomastia , Hair Distribution & Spider Naevi.**

## Abdominal Examination

**Inspection(from 2 Sites)**

**From the foot of the bed & from Right Side of the patient**

4- Symmetry of the Abdomen
5- Umbilicus (central & inverted)
6- Abdominal Respiration
7- Attached devices & drains

4- Visible Scars
5- Superficial masses or swelling
6- Visible Dilated veins
7- Skin bruising

**Palpation (Is There Any Pain?** **If so; leave that area to the last.**

1- ***Superficial Palpation:*** **a.Gain patient's confidence. b.Superficial Masses & Superficial Tenderness.**
2- ***Deep Palpation*****: a.Deep Masses. b.Deep Tenderness.**
3- ***Palpation for Organomegaly*****: - Liver, Spleen & Kidneys.**
   - **A. Hepatomegaly: start from RIF move your hand vertically with each inspiration.**
     **Liver SPAN by Percussion starting from Right 5th intercostal space till dullness appears.**
   - **B. Splenomegaly: start from RIF move your hand obliquely with each inspiration.**
   - **C. Kidney: Ballottement test & Renal angle tenderness.**

## Percussion (Percuss all over 9 regions)

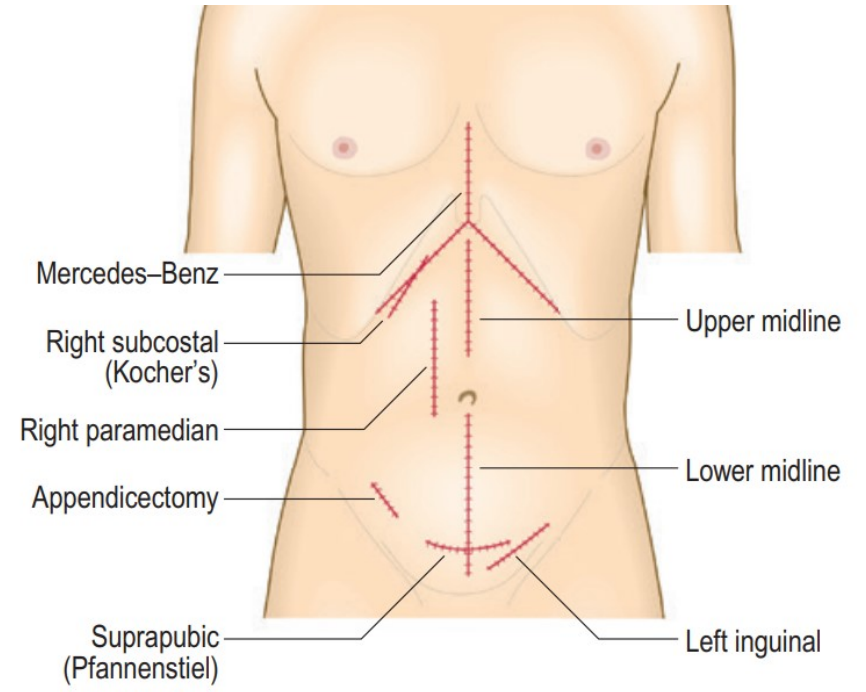
- **Normally it should be tympanic**
- **Over mass or fluid (dull)**
- **Percuss for Ascites (Shifting dullness "mild to moderate" & Transmitted Thrills).**

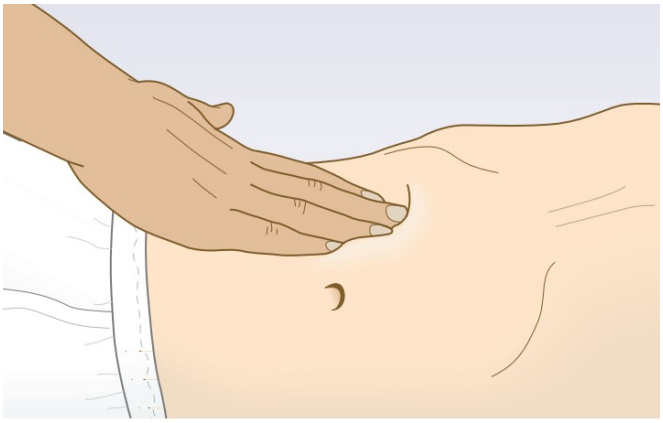
## Auscultation:

- **Auscultate for bowel sounds " at iliocecal valve" & for bruit over renal & iliac arteries.**

**Mention that you have to do DRE & hernial orifices exam.**

**Thank the patient and Clean your hands**



Palpation of the liver.

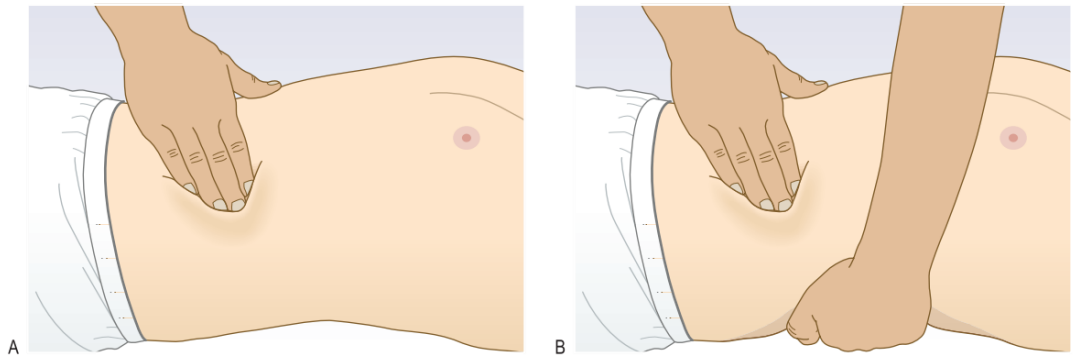


**Fig. 6.16 Palpation of the spleen.** A Initial palpation for the splenic edge moving diagonally from the umbilicus to the left hypochondrium. B If the spleen is impalpable by the method shown in A, use your left hand to pull the ribcage forward and elevate the spleen, making it more likely to be palpable by your right hand.

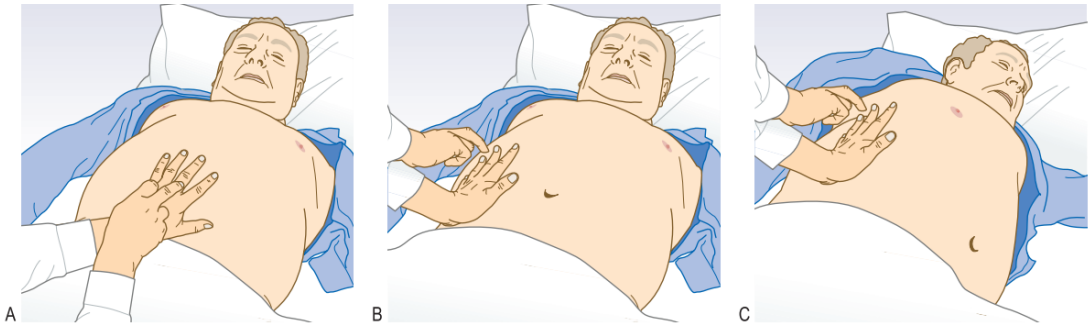
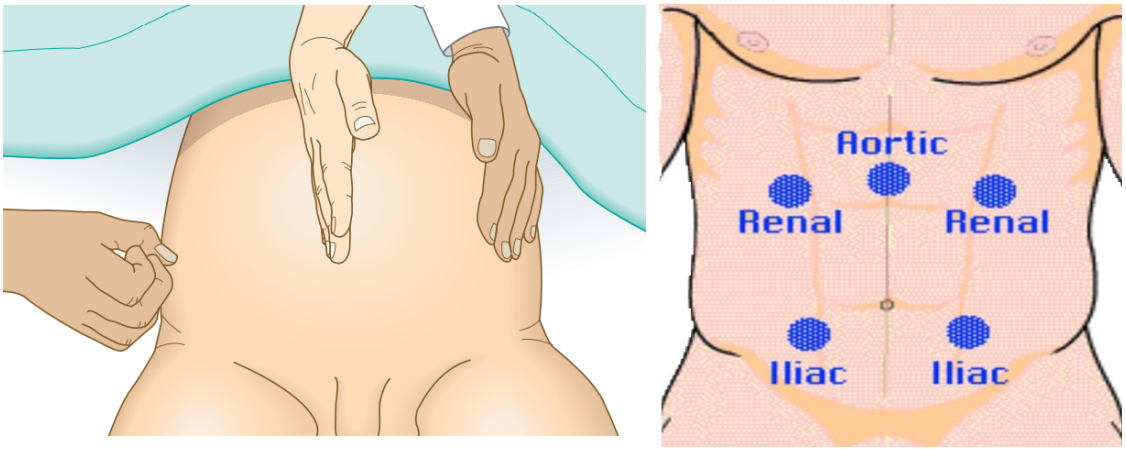


**Fig. 6.17 Percussing for ascites.** A and B Percuss towards the flank from resonant to dull. C Then ask the patient to roll on to their other side. In ascites the note then becomes resonant.



# Thyroid Examination

**H: Hello "Introduce yourself , take permission & Confirm patient identity"**
**E: Explain What are going to do & Exposure " (NIPPLES & Above)"**
**L: Light**
**P: Privacy " ask for chaperone" & Position " Sitting "**

## GENERAL examination

**Hands: thyroid acropachy , Sweaty hand , fine Tremor , palmar erythema and pulse .**
**Eyes: exophthalmos , lid retraction, lid lag and Ophthalmoplegia (eye movement).**
**Face: dry coarse hair, periorbital puffiness or loss of lateral 1/3 of eyebrows .**
**Lower limb: pretibial myxedema.**

## Neck Examination

### Inspection

**From the front with the patient slightly extending his neck.**

1. Symmetry
2. Swelling
3. Scars
4. Ask the patient to swallow and to protrude his tongue

### Palpation (Is There Any Pain?

**Palpation from Front**

-Tracheal deviation

-Tenderness

- Any masses

**Palpation from behind**

- Palpate the 2 lobes of the thyroid
- Ask the patient to swallow while palpation
- Cervical and supraclavicular LNs

### Percussion (Percuss over the sternum if dull → Retrosternal goiter)

### Auscultation

**Over the neck for thyroid bruit**

**Thank the patient and clean your hands**

# Lower limb examination

**H: Hello "Introduce yourself , take permission & Confirm patient identity"**
**E: Explain What are going to do & Exposure " (from the groin and below but mid-thigh is accepted)"**
**L: Light**
**P: Privacy " ask for chaperone" & Position " Supine , Lying flat "**

## Inspection(from 2 Sites)

**From the foot of the bed** **&** **from Right Side of the patient**

1. Symmetry of the legs
2. Deformities
3. Attached devices & drains
4. Abnormal position

1. Hair
2. Skin lesions (ulcers, scars).
3. Dilated veins
4. Redness (change in color).

- **Elevate the leg looking for pressure ulcers or hidden abnormality.**
- **Examine the nails and between toes.**

## Palpation (Is There Any Pain?)

3- **Tenderness, Temperature**
4- **Pulses: Dorsalis pedis, Posterior tibial, Popliteal, Femoral arteries)**
5- **Pitting edema**
6- **Inguinal LN Palpation**

## Leg Circumference (both legs)

- **Identify anatomical landmarks (Tibial tuberosity & medial malleolus)**
- **Attempt actual measurement.**

## Mention that you should do burger test.

## Auscultation

**Using the bell over the major arteries**

**Thank the patient and clean your hands**