

## Congestive Cardiac Failure (CCF)

A complex syndrome caused by a structural or functional abnormality in the cardiac muscle that impairs its ability to function as a pump and meet the metabolic needs of the body. Characterized by shortness of breath, fatigue and signs of fluid retention.

Decreased cardiac output triggers the baroreceptors in the LV, carotid sinus and the aortic arch . This leads to stimulation of the cardio-respiratory centre in the brains, increased ADH release (causing peripheral vasoconstriction and increases renal salt and water absorption) and increased sympathetic stimulation (activating renin - angiotension system, promoting more water retention and peripheral vasoconstriction). These lead to LV dilatation and hypertrophy (poor ejection fraction), increased peripheral vascular resistance (high afterload) and retention of fluid( high preload).

Most patients present with left heart failure which can progresses to right heart failure. The most common cause of right heart failure is left heart failure but it can also be caused by pulmonary hypertension (cor pulmonale) or disease that effect the  $RV > LF$  (like EMF). Heart failure can be either compensated (when the patient is stable) or decompensated (when the patient suddenly gets worse)

### Etiology of CHF

#### *Systolic Dysfunction (inability to expel blood)*

- Hypertension\*
- Ischemic heart disease
- Idiopathic cardiomyopathy (like HIV)\*
- Valvular disease\*
- Alcoholic cardiomyopathy
- Drug-associated cardiomyopathy
- Myocarditis

#### *Diastolic Dysfunction (abnormal filling)*

- Hypertension
- Fibrosis
- Ischemia
- Aging process
- Constrictive pericarditis (like TB)\*
- Restrictive pericarditis (like EMF)\*
- Hypertrophic cardiomyopathy

\* The most common causes in our setting

## The New York heart association (NYHA) functional classification

- Class I- no limitation in physical activity
- Class II- slight limitation of physical activity (fatigue, SOB)
- Class III- marked limitation of activity( comfortable at rest but slight exertion causes symptoms)
- Class IV- symptoms at rest

## Diagnosis

- History and clinical examination
- Echocardiography -wall thickness, cavity dimensions, ventricular function ( systolic and diastolic), can reveal underlying aetiology
- ECG-Commonly abnormal, Q waves, ST/T changes, LVH, arrhythmias and axis change
- Chest Xray - Cardiomegaly, pulmonary congestions ( upper lobe diversions, fluid in the fissures, Kerley B lines, pleural effusions)

## Framingham Criteria for CHF

- Validated CHF with 2 major criteria or 1 major and 2 minor
- Major : PND or orthopnea, Elevated JVP, Pulmonary rales, S3, Cardiomegaly on chest xray.
- Minor :peripheral edema, night time cough, dyspnea on exertion (DOE), pleural effusions,HR > 120, weight loss > 4.5 kg in 5 days with diuresis

## Causes of CHF exacerbation/decompensation: FAILURE

*F: forgot to take medication, ran out of medication*

*A: arrhythmias (especially atrial fibrillation)*

*I: ischemia / infarction / infection*

*L: lifestyle (poor diet)*

*U: up-regulation (high cardiac output states i.e. pregnancy, thyroid)*

*R: renal failure (fluid overload)*

*E: embolism / endocarditis*

## Treatment

- Counseling
  - Weight loss in obese patients, dietary sodium restriction (< 2 grams a day), fluid restriction, administration of oxygen if needed, exercise as tolerated for class I and II
- Vasodilator therapy: mainstay of chronic therapy; reduces mortality
  - ACE inhibitors (1<sup>st</sup> line) – but must follow renal function
  - Hydralazine (rarely used)
- Beta-blockers: for chronic therapy in patients with non-valvular CHF; not acute, decompensated heart failure; reduces mortality
  - Carvedilol (best), metoprolol, atenolol
- Digoxin
  - for Class II-III
  - improves symptoms, does not reduce mortality
- Diuretics
  - Loop diuretics (lasix) for diuresis are the primary treatment of decompensated heart failure but do not reduce mortality
  - Aldactone – useful in chronic therapy of patients with Class III-IV; reduces mortality but also greatly increases risk of hyperkalemia in patients who are also taking ACE inhibitors.



## Chest Pain

Chest pain is a common symptom and may be a manifestation of cardiovascular or noncardiovascular disease. Full characterization of the pain with regard to quality (squeezing, tightening, pressing, burning), quantity, frequency, location, duration, radiation, aggravating or alleviating factors and associated symptoms can help to distinguish the cause. All patients presenting to a hospital with severe or persistent chest pain should have a full set of vital signs, an ECG, and a CXR. ***\*\*The life-threatening causes that must be considered and ruled out in all patients with severe, persistent chest pain.***

## Cardiac Causes

### Angina/Myocardial infarction \*\*

- Substernal pressure +/- radiation to neck, jaw, Left arm
- Duration usually > 1 minute and < 12 hours for angina
- Associated with dyspnea, diaphoresis, nausea/vomiting
- Worsened with exertion, relieved with rest or nitroglycerin
- Infarction is same as angina except increased intensity and duration
- ECG: look for ST elevations or depressions, T wave inversions

### Pericarditis/Myocarditis \*\*

- Sharp pain radiation to trapezius
- Aggravated by respiration, relieved by sitting forward
- Listen for pericardial friction rub
- ECG: look for diffuse ST elevations and PR depressions

### Aortic Dissection \*\*

- Sudden onset of tearing chest pain, knife-life pain
- Radiation to back
- Usually severely hypertensive (can become hypotensive)
- Asymmetric blood pressure in arms and asymmetric pulses bilaterally
- Widened mediastinum on CXR, new aortic insufficiency murmur

## Pulmonary Causes

### Pneumonia \*\*

- A very common cause of chest pain in our settings
- Pleuritic in nature
- Associated with dyspnea, cough, fever, sputum production
- Presents with fever, tachycardia, crackles on physical exam
- CXR should show an infiltrate

### Pneumothorax \*\*

- Sharp, pleuritic pain +/- shortness of breath
- Unilateral hyperresonance and decreased breath sounds on one side
- Confirmed by CXR

### Pulmonary embolism \*\*

- Pleuritic, sudden onset
- Associated with tachypnea, tachycardia, hypoxemia
- ECG can show T wave inversions V1-V4, RAD, S1Q3T3
- Pulmonary hypertension
- Dyspnea, exertional pressure
- Hypoxemia, Loud P2 sound on heart exam, right sided S3 &S4

## **GI causes**

### **Esophageal reflux**

- Substernal burning, worsened with lying down
- Acid taste in mouth

### **Peptic ulcer disease**

- Epigastric pain
- Hematemesis or melena
- EGD with *H. pylori* test

### **Biliary disease**

- With RUQ pain, nausea/vomiting
- Aggravated by fatty foods
- Needs RUQ ultrasound, liver tests

### **Pancreatitis**

- Epigastric or back discomfort
- Increased amylase and lipase, has risk factors

## **Musculoskeletal and other Causes**

### **Costochondritis**

- Localized sharp or dull pain, tenderness to palpation

### **Herpes zoster**

- Intense unilateral pain often precedes rash, dermatomal rash and sensory findings

### **Cervical spine disease or arthritis**

- Precipitated by motion, lasts seconds to hours
- X-rays to confirm

# Ischemic Heart Disease (IHD)

## Definition

Due to insufficient oxygen supply to the heart. When the oxygen demands of the heart are greater than the amount of oxygen that can be delivered to the heart, ischemia occurs. This is usually caused by a narrowing of the coronary artery either due to plaque accumulation or vasoconstriction. If left untreated it can lead to an infarct (i.e. necrotic, dead tissue).

## Pathophysiology

Most commonly IHD is due to atherosclerotic plaque build-up. Over time this causes narrowing of the coronary arteries. This narrowing prevents adequate oxygen supply from reaching the heart and ischemia occurs. This narrowing can either completely occlude an artery causing a STEMI or it can partially occlude one or many arteries usually leading to either unstable angina or an NSTEMI.

## Predisposing conditions

Diabetes, HTN, and smoking are all risk factors for atherosclerotic plaque formation, which puts someone at increased risk for ischemic heart disease and a myocardial infarction.

## Epidemiology

IHD is much less common in East Africa than it is in the US and Europe. This is likely because the population of East Africa is younger (due to shorter life spans) as well as less diabetes mellitus, hypercholesterolemia and number of cigarettes smoked by smokers.

## Symptoms

Patients usually present complaining of chest pain and/or SOB. The chest pain is usually a squeezing pain that often radiates to arms or neck. It can be worsened by exercise and improves with rest or nitroglycerin. Other symptoms include: diaphoresis, nausea, vomiting, palpitations, or lightheadedness.

**\*Anytime a patient has SOB or chest pain, IHD or an MI need to be on the differential\***

Many times patients, especially women, present with atypical symptoms when they are having an MI. Abdominal pain is a common complaint for women having MIs.

**\*If a woman over 40yrs old comes in with risk factors for ischemic heart disease, include that on your differential even if her symptoms may not be typical\***

## Signs

On physical exam, look for signs of ischemia, heart failure, or atherosclerotic disease

- Signs of ischemia: S4, new MR
- Signs of heart failure: elevated JVP, crackles in lung bases, S3, hypotension, cool extremities
- Signs of atherosclerotic disease: carotid or femoral bruits, decreased distal pulses

## Diagnosis

Primarily made via ECG. Key changes on ECG include: ST elevation, ST depression, T wave inversion, or new LBBB. If patient is having a STEMI, the leads with the ST elevation tell you where the infarct has occurred.

- Lead I and aVL: lateral MI (left circumflex is affected)
- Leads II, III, and aVF: inferior MI (right coronary artery is affected)
- Leads V3-V6: anterior MI (LAD if affected)
- Leads V1-V2: septal MI ( either distal LAD, left circumflex or right coronary are affected)

## **Management**

This can be divided into care that needs to be given immediately and long term care that will continue after the patient leaves the hospital.

### **Immediate care**

Aspirin 300mg PO STAT, heparin (1000 IU/h), atenolol 50-100 mg PO STAT (goal PR of 60-70), isosorbide mononitrate (ISMN) 20mg PO STAT then BD, morphine prn for pain, O<sub>2</sub>, and simvastatin 40mg PO

### **Post-MI care**

Aspirin 75 PO OD, clopidogrel 75mg PO OD, atenolol 50-100 mg PO OD, simvastatin 40mg PO OD, captoril 25 mg PO BD, and you can add ISMN if needed

## Valvular Heart Disease

Valvular heart disease involves outflow obstruction or incompetence of one of four valves of heart. The distribution of disease varies greatly based on population and risk factors. The most valvular diseases are Rheumatic, Congenital or Degenerative. **Most common cause of valvular heart disease in Tanzania is Rheumatic heart disease.**

### Aortic Stenosis

- **Pathophysiology:** LV hypertrophies in order to overcome outflow obstruction but this compensatory change becomes maladaptive and leads to LV dilatation and CCF
- **Etiology:** 1) rheumatic, 2) calcification of normal valve in elderly, 3) calcification/fibrosis of congenital bicuspid valve
- **Symptoms:** Mnemonic- Aortic Stenosis Complication - Early symptoms of Angina, Serious later complication of Syncope and finally the late presentation of Congestive heart failure.
- **Exam:** AS is a harsh crescendo/decrecendo midsystolic (ejection) murmur. It is heard loudest at in the Aortic area (RUSB) with radiation to the carotids. It is heard best with the diaphragm when the patient sits forward. When loud enough it can be heard all over the precordium. The phase during systole which the murmur peaks can help to determine the severity of the disease. An Early- peaking murmur is usually a less stenotic valve where as a late peaking murmur is a more severe stenosis. This occurs because as the valve become more stenotic it takes the left ventricle longer time to generate enough pressure to overcome the stenosis. AS is associated with a narrow pulse pressure (due to lower cardiac outputs), Left ventricular hypertrophy (shifting of the apex beat) and pulsus parvus et tardus (weak and slow upstroke of the carotid pulse indicating flow limitations seen in AS). As the AS progresses the valve will become progressively become less mobile and stenotic causing the S2 to be quieter or even absent. If there is significant delay in the closing of the Aortic valve compared to the pulmonary valve there may be splitting of the S2
- **Investigations :** Severity graded by echocardiogram and symptoms
- **Management:** As symptoms develop prognosis becomes worse and presence of Syncope/CCF/Angina are poor predictors. Surgery is definitive treatment. Medical therapy to relieve symptom. Control HTN. Avoid vasodilators (nitrates) and beta-blockers in severe AS.

### Aortic Insufficiency/Regurgitation

- **Pathophysiology:** In diastole, blood flows from aorta into LV due to incompetence of aortic valve which increases End Diastolic Volume and Stroke Volume. These changes eventually lead to dilatation of the LV and CCF.
- **Etiology:** 1) rheumatic fever, 2) endocarditis, trauma, connective tissue disease, congenital bicuspid aortic valve, HTN
- **Symptoms:** angina / CCF
- **Exam:** High pitched early diastolic murmur. Starts immediately after the second heart sound and fades away in mid diastole. The murmur radiates from the aortic area to the left lower sternal edge, where is best heard. To Auscultate use the diaphragm of the stethoscope with the patient sitting forward in expiration. The Murmur is associated with a wide pulse pressure (fall in diastole pressure due to the regurgitating blood) and displacement of apex beat.
- **Treatment:** definitive is surgical. Medical therapy includes hydralazine, ACE inhibitors for severe disease and digoxin for CCF.

## Mitral Stenosis

- **Pathophysiology:** Stenosis of the mitral valve results in outflow obstruction from the LA to LV and therefore high atrial pressures. These elevated pressures lead to atrial dilatation and often atrial fibrillation. Elevated atrial pressure causes elevated pulmonary pressures and pulmonary symptoms.
- **Etiology:** most common rheumatic heart disease, congenital
- **Symptoms:** SOB, palpitations, dyspnea on exertion, atrial fibrillation or features of CCF.
- **Exam:** Loud S1, low- pitch rumbling mid diastolic murmur. It is best heard at the apex beat with the bell of the stethoscope while the patient lying on the left side.
- **Treatment:** Surgery required if patient symptomatic. Can use beta blockers. Existence of atrial fibrillation OR prior systemic emboli require anticoagulation

## Mitral Regurgitation (RHD in 30% of cases, often secondary to ischemia)

- **Pathophysiology:** abnormal coaptation of mitral leaflets creates a regurgitant orifice and a resultant regurgitant volume creating a volume overload of the LA
- **Etiology:** 1) rheumatic heart disease, 2) endocarditis, ischemia, connective tissue
- **Symptoms:** pulmonary edema, progressive shortness of breath, fatigue
- **Exam:** high-pitched, blowing, pansystolic murmur at apex, radiates to axilla. Murmur increases with handgrip and decreases with valsalva. Brisk carotid upstroke
- **Treatment:** surgery in symptomatic severe cases , decrease afterload with ACE inhibitors, hydralazine. Decrease preload with diuretics

# Acute Rheumatic Fever and Rheumatic Heart Disease

## Acute Rheumatic Fever

### Definition

It is a nonsuppurative consequence of a pharyngeal infection by group A Beta hemolytic streptococcus (Strep pyogenes). It commonly occurs 2-3 weeks after throat symptoms in 3-6% of the cases due to immune cross reactivity between the bacteria and the connective tissue. This is disease of the poor, the overcrowded and the poorly housed. The presence of severe disease tends to reflect recurrent episodes of acute rheumatic fever. In most patients with carditis, if the recent attacks could be prevented they would eventually lose their murmur and the heart would return to normal or near normal.

Worldwide an estimated 10-20 million people get acute RF yearly. Rheumatic heart disease is the most common cause of valvular heart disease in the world. Most common in children ages 4-9 years old, but adults can get acute rheumatic fever also.

### Diagnosis:

Generally a clinical diagnosis with laboratory confirmation is needed.

### Clinical diagnosis:

*Jones Criteria:* 2 major criteria OR one major + 2 minor criteria **AND** evidence of recent streptococcal infection.

### Major Criteria

- Carditis: Occurs in about 50% of the cases and is the most serious manifestation of RF. It may affect only the endocardium, or it can affect all layers of the heart (pericardium to the endocardium). This acute presentation is different from the later sequelae of rheumatic heart disease (mitral stenosis). Congestive heart failure symptoms tend to represent advance disease.
- Arthritis: Occurs in 80% of the cases. Migratory polyarthritis usually of large joints. each affected joint inflamed for less than one week and typically over 6 joints involved
- Chorea: also called Sydenham chorea or St. Vitus dance is seen in 10% of the patients. It's abrupt, purposeless, nonrhythmic, involuntary movements, usually worse on one side. Chorea can occur up to 8 months after strep infection
- Subcutaneous nodules: Firm, painless, non inflamed, variable in size, symmetric when multiple and located over bony surfaces or near tendons, appear earlier in course of ARF and usually only in patients with carditis. This presentation is rare.
- Erythema marginatum: pink, evanescent, non-itchy rash on trunk and limbs, but not on face. Heat brings lesions out. Seen in < 5% of the cases.

### Minor criteria

- Fever
- Arthralgia
- Previous rheumatic fever or rheumatic heart disease

## Laboratory Diagnosis

- Increase titres of antistreptolysin O\*\* (ASO, most common antibody test used) or strep antibodies
- Positive throat culture for Group A beta-hemolytic strep
- Recent scarlet fever

\*\*Antibodies are better than culture because the culture is often negative. Antibody titer usually peaks at 4-5 weeks after pharyngitis. Cannot use titers as indicator of disease activity after initial illness.

### Treatment

- **Acute treatment:** Oral penicillin 500mg BD-TDS for 10 days, or Benzathine benzylpenicillin 1.2 million IM once. Use erythromycin 40mg/kg/day divided in 2-4 doses or a cephalosporin if PCN allergic. Treat even if no pharyngitis at the time of diagnosis; culture family contacts and treat if positive
- **Secondary prophylaxis:** Benzathine benzylpenicillin 1.2 million units every 2-4 weeks for approximately 5 years. If penicillin allergy use erythromycin 250mg PO BD. Lifelong prophylaxis is recommended for patients with carditis and residual heart disease.
- **Anti-inflammatory drugs:** These act to suppress the immune response. Aspirin 20-25mg/kg PO QDS 3-6 weeks if no cardiac involvement. For mild carditis 3 months and 6 months for severe carditis. In these severe cases prednisolone 0.5mg/kg QDS can be given for 2 weeks.
- **Heart failure:** as standard
- **Chorea:** Sodium Valproate 10mg/Kg PO BD or haloperidol for 3 months.

### Rheumatic heart disease

Rheumatic heart disease occurs 10-20 years after original attack. This is why the peak incidence of rheumatic heart disease is 15 – 30 years of age (since the peak age for acute rheumatic fever is at 4-9 years of age). Probably develops in over 50% of patients with initial carditis due to acute rheumatic fever. Severe rheumatic heart disease usually only occurs after multiple episodes of acute rheumatic fever. Recurrent episodes of inflammation lead to chronic fibrosis and then calcification of the valves. This is why antibiotic prophylaxis after a first episode of acute rheumatic fever is so important.

### Pathophysiology

Tiny nodules gather on the valve leaflets in acute rheumatic fever. Over time fibrin deposition occurs and valves thicken or fuse (fibrosis). Another proposed mechanism is acute inflammation causing adhesion of commissures and then degenerative sequelae. A subclinical inflammatory process caused by the stress of chronic turbulent flow due to the deformed valve contributes to the progression of stenosis. With time there is a gradual loss of valve area.

### Valve findings and when they occur

Mitral stenosis is most common finding, followed by aortic stenosis. Some studies suggest that over 70% of MS is caused by RHD. Even though stenosis occurs 10-20 years after infection symptoms may be delayed as late as 40 years. If antibiotic treatment is not adequate in ARF (not available vs. more virulent strains causing earlier adhesion of leaflets), onset of symptoms often occurs earlier.

### When does the patient need an intervention?

Symptoms drive the need for intervention. Can do closed or open commisurotomy, percutaneous balloon valvulotomy, or valve replacement. Mitral valve replacement should occur in symptomatic patients (NYHA Class III-IV) with severe mitral stenosis

### **Complications of rheumatic heart disease**

- Congestive heart failure: Mortality is related to patient's functional status.
- Atrial fibrillation: occurs in over 45% of mitral stenosis patients
- Pulmonary HTN: mean survival without surgery 2.4 years
- Thromboembolic events: mostly occur in patients with atrial fibrillation, but can happen in normal sinus rhythm in patients with mitral stenosis
- Bacterial endocarditis

## Infective Endocarditis (IE)

Infection of the endothelium of the heart, usually but not always limited to the valves. Either subacute (often due to *Strep viridans*) or acute (less common, often due to *Staph aureus*).

**Pathophysiology:** Infection of the valves with bacteria (or rarely fungi) causes injury to the valve and valvular regurgitation. The bacteria on the valve can form a mass or vegetation. Parts of this can embolize. Immune complexes form with the bacteria. Infection of the endothelium causes 1) persistent bacteremia, 2) valvular disfigurement (with vegetations/regurgitation), 3) septic emboli and 4) immune complex phenomenon.

### Predisposing conditions

- **Abnormal valve:** prior endocarditis, h/o rheumatic heart disease, valvular heart disease, congenital heart disease, prosthetic valves. Low virulence organisms such as *Strep viridans*.
- **Abnormal risk of bacteremia (valves may or may not damaged):** poor dentition, tooth extraction (or other GI/GU procedures breaking mucosal barriers like endoscopy), IV drug use, hemodialysis (organism tend to be more virulent such as *Strep. Pneumoniae* or *Staph Aureus*). In our settings post-partum pelvic infection and an acute pyomyositis are important sources.

### Symptoms

- Persistent bacteremia causes fevers, weight loss, night sweats, fatigue. (Nonspecific symptoms).
- Valvular disfigurement can cause symptoms of CCF.
- Septic emboli may cause stroke, renal or renal infarcts, infected joints and pulmonary emboli.
- Immune complex phenomenon can cause arthritis or glomerulonephritis.
- Remember: **Fever + regurgitant murmur = IE until proven otherwise**

### Signs

- General - Finger clubbing, Splenomegaly, pallor ( anaemia)
- New regurgitant murmurs due to valvular disfigurement- AR and MR are most common.
- Septic emboli : Strokes, acute limb ischaemia, embolic abscesses or mycotic aneurysms, Janeway lesions - nontender, hyperpigmented macules on palms or soles, subconjunctival hemorrhage.
- Vasculitic events ( Due to immune complexes formed)
- Splinter hemorrhages in nailbed
- Roth spots (retinal hemorrhage + pale center)
- Osler's Nodes (tender nodules on tips of fingers and toes)
- Glomerulonephritis

### Diagnostic studies

- 3 sets of blood cultures from different sites, ideally >1 hour apart, should be drawn before starting antibiotics! In some resource limited settings, obtaining 2 sets of blood cultures may be more feasible.
- Echocardiogram- Floating vegetations seen on affected valves
- FBP with differential, ESR, rheumatoid factor, creatinine, urinalysis
- EKG (to assess for conduction abnormalities)

- Urinalysis to detect renal failure ( Microscopic haematuria and proteinuria)

## Modified Duke Criteria

### Major

- Sustained bacteremia by organism known to cause endocarditis (at least 2 cultures positive)
- Endocardial involvement documented by either vegetation or new valvular regurgitation seen on echocardiogram

**Definitive:** 2 major OR 1 major + 3 minor OR 5 minor

**Possible:** 1 major +1 minor OR 3 minor

### Minor

- Fever >38.0 C
- Predisposition (like rheumatic heart disease)
- Embolic phenomena (arterial emboli, septic pulmonary infarct, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhage, Janeway lesion)
- Immunologic phenomena (glomerulonephritis, Osler's nodes, Roth spots, + rheumatoid factor)
- Blood cultures that don't meet criteria (only 1 positive)

## Microbiology

- Native valve endocarditis, *Strep viridans*, *Staph aureus* and *Enterococcus* most common
- The organisms that cause endocarditis are different in:
  - IV drug abusers – *Staph aureus* most common
  - Prosthetic valve endocarditis - *Staph epidermidis* most common <6mo after surgery
  - Immunosuppression – fungi more common

## Treatment

- Empiric antibiotics, adjust according to organism and sensitivities, continue for at least 6 weeks after last positive blood culture
- Start with penicillin 1.2g IV 4hrly + gentamicin 60mg BD
- If acute onset add in staph Aureus cover (like Flucloxacillin)
- Surgery may (rarely) be necessary if refractory CCF, persistent/refractory bacteraemia, invasive infection, prosthetic valve, fungal infection

## Endocarditis Prophylaxis:

Patients with h/o rheumatic heart disease, previous endocarditis, unrepaired congenital heart disease and valve replacement when undergoing dental procedures.

Antibiotics used: amoxicillin 3g PO 60 min prior to procedure OR Clindamycin 600mg if PCN allergy or > 1 course of penicillin in the last month use of antibiotics.

## **Arrhythmias (emphasis on Atrial Fibrillation)**

**Definition:** Abnormality in cardiac conduction that can manifest as either change in rate or rhythm

### **Types and etiologies**

**Bradyarrhythmia:** any rhythm that results in a ventricular rate of less than 60 beats per minute.

**Sinus bradycardia:** sinus rate of less than 60 beats/minute. Has normal P wave configuration consistent with origin in sinus node area. Etiology: increased vagal tone, hypothyroidism, ischemia, medication such as digoxin, beta blockers, calcium channel blockers

### **AV Block**

- 1<sup>st</sup> degree: conduction delay within AV node, with prolonged PR interval on ECG > 200 msec. Etiology: medication, CHF, ischemia, electrolyte abnormalities. No therapy needed.
- 2<sup>nd</sup> degree Type I (Wenckebach): progressive PR interval prolongation before a blocked or dropped beat. Etiology: medication, electrolyte abnormalities, ischemia. If symptomatic, can give atropine.
- 2<sup>nd</sup> degree Type II: abrupt AV conduction block without evidence of PR prolongation. No change in PR interval and then sudden dropped beat. Etiology: ischemia, conduction system disease. Need pacemaker.
- 3<sup>rd</sup> degree: dissociation of atrial beats and ventricular beats. Atrial impulses fail to conduct to the ventricle. And ventricle is beating on its own with a slower rate. Etiology: medication toxicity, ischemia, infiltrative disease (sarcoid, amyloid), Lyme disease, Chagas disease. Need pacemaker.

**Tachyarrhythmia:** any rhythm with a rate in excess of 100 beats per minute

- **Narrow Complex Tachycardia or Supraventricular** (narrow QRS < 120 msec)
  - Sinus Tachycardia. Etiology: pain, fever, hypovolemia, hypoxia, anemia, anxiety, thyroid disease; rate not greater than 220-age
  - AV nodal reentrant tachycardia (AVNRT): reentrant circuit using AV node and accessory pathway, rate can be > 150.
  - Atrial flutter: macro-reentry usually within right atrium (atrial rate is 300 and usually conducts 2:1 for HR = 150)
  - Atrial fibrillation: see below for more
- **Wide Complex Tachycardia** (wide QRS > 120 msec)
  - Ventricular tachycardia: monomorphic (QRS all the same size) or polymorphic Etiology: ischemia, cardiomyopathy, structurally abnormal heart, prior MI

### **Atrial Fibrillation**

**Definition:** Most common arrhythmia for which patients seek treatment. This is an irregularly irregular rhythm in which the atria depolarize chaotically and are not able to properly contract. The ventricular response to an irregular atrial beat is also irregular and sometimes rapid (i.e. rapid ventricular response).

### **Types**

- *Valvular* atrial fibrillation: usually associated with rheumatic heart disease due to MS or MR with left atrial enlargement; \*the most common type in our setting\*
- *Isolated* atrial fibrillation: secondary to another illness (hyperthyroidism, PNA, PE, etc.)
- *Lone* atrial fibrillation: age < 65, no history of stroke or HTN, no structural heart disease
- *Paroxysmal* atrial fibrillation: intermittent (less than 24 hours)
- *Persistent* atrial fibrillation: lasts > 7 days or requires cardioversion
- *Chronic* atrial fibrillation: atrial fibrillation is the predominant rhythm
- \*paroxysmal, persistent, and chronic afib have the same risk of stroke

## **Pathophysiology**

Commonly originates from ectopic pacemakers in atria around the pulmonary veins. The loss of atrial contraction then leads to heart failure. This loss of atrial contraction also leads to stasis and clots in left atrium which further leads to thromboemboli (like stroke)

## **Causes and Risk factors of Atrial Fibrillation:**

### ***Acute atrial fibrillation:***

- Cardiac: heart failure, hypertensive crisis, ischemia, myocarditis
- Pulmonary: acute pulmonary disease or hypoxia (PNA), pulmonary embolus
- Metabolic: high catecholamine states (stress), infection, post-op, pheochromocytoma,
- Thyrotoxicosis
- Drugs: alcohol, stimulants

### ***Chronic atrial fibrillation:***

- Age, hypertension, ischemia, valvular disease\*, cardiomyopathy, hyperthyroidism, obesity

**History:** Ask about prior symptoms and onset, history of rheumatic heart disease, symptoms of thyroid disease, alcohol abuse, and prior digoxin use. Symptoms include fatigue, syncope, chest pain, palpitations. Severe symptoms include acute pulmonary edema. Many patients have no symptoms at all. Most symptoms are related to rapid ventricular rate. Look for

**Signs:** There are 2 important signs of atrial fibrillation are: Irregularly irregular pulse. A pulse deficit of > 10 (The “pulse deficit” = heart rate – pulse rate). Make sure to do thorough cardiac and pulmonary exams

## **Evaluation**

- ECG
- CXR
- Echocardiogram to look for valvular disease, presence of thrombus, left ventricular function
- Thyroid function tests (TSH), creatinine

## **Treatment**

- If patient is hemodynamically unstable, consider electrical cardioversion in the ICU (consider heparin drip if doing cardioversion)
- If low TSH and symptoms of hyperthyroidism, do complete thyroid workup (T3/T4, thyroid ultrasound) and treat atrial fibrillation as below
- \*Rate Control (goal heart rate 60-80) – best treatment for most patients

- Beta blockers (atenolol, propranolol)
  - Calcium channel blockers (verapamil)
  - Digoxin for heart failure patients if blood pressure is low or if severe valvular heart disease is present (but beware of renal dysfunction)
- Rhythm Control – used only for severely symptomatic patients
  - Amiodarone
- \*Anticoagulation to reduce risk of stroke. Give in patients with valvular heart disease, prior stroke, or any two of the following (older > 65, hypertension, diabetes, or congestive cardiac failure). Consider simultaneous peptic ulcer disease prophylaxis.
  - Warfarin (goal INR 2.5)
  - Aspirin (if monitoring of INR is not feasible) – technically an antiplatelet drug
- \*Thromboembolism prevention: Keeping the INR 2-3 with warfarin reduces risk of stroke by 66% in patients with above risk factors. Always monitor for risk of bleeding.

# **Introduction to Hypertension**

## **Definition**

HTN is simply defined as a persistently abnormal elevation in blood pressure, < 140/90mmHg. HTN is not diagnosed unless BP is elevated on multiple occasions (at least 2-3) or if the patient is complications of HTN (as with patients admitted with hypertensive emergency). We treat HTN because it is a major risk factor for stroke, MI, CCF, CKD, retinopathy and peripheral vascular disease. The risk of hypertensive complications increases continuously throughout the BP range.

## **Physiology of HTN**

HTN is caused by a combination of cardiac output, peripheral vascular resistance and sodium retention (regulated by the renin-angiotensin system). The latter 2 factors are more important. All treatment of HTN targets these factors.

## **Epidemiology**

HTN is a growing problem in sub-Saharan Africa. Early studies indicated HTN was rare in Africa but several recent studies have shown that the prevalence of HTN is now 5-15% (higher in urban areas). One from Tanzania indicated that HTN occurs in 22% of males and 18% of females in Dar es Salaam and 13% of both men and women in rural areas (Edwards et al., 2000)! The average blood pressure in this group was higher than studies from America and Europe!

## **Types of HTN**

- Essential (Primary) HTN – most common (95%) and due to a combination of genetic, environmental factors (salt intake, weight, exercise etc) and age. Usually develops after the age of 30 but can develop earlier.
- Secondary HTN – HTN due to other causes. All patients < 30yo with HTN and those with HTN not sufficiently controlled on 3 drugs should be assessed for these conditions.
  - Renal – most common; can be related to CKD or renal artery stenosis
  - Cushing's syndrome – hypercortisolemia
  - Conn's syndrome - hyperaldosteronemia
  - Coarctation of the Aorta
  - Pheochromocytoma – catecholamine producing tumor
  - Hyperthyroidism or hypothyroidism.

## **Degrees of HTN**

- Mild (Grade 1) = 140-160/90-99mmHg
- Moderate (Grade 2) = 160-180/100-109mmHg
- Severe (Grade 3) = > 180/110mmHg
- Hypertensive Urgency – severe HTN but no end organ damage
- Hypertensive Emergency – severe HTN with end organ damage
  - Usually does not occur unless sudden increase in DBP to < 130mmHg. Was called malignant HTN in the past.
  - Signs of end organ damage can include encephalopathy (confusion with severe HA), blurry vision/retinal hemorrhage, angina, pulmonary edema, aortic dissection and acute kidney injury

## Symptoms/Signs

Most patients with HTN are asymptomatic! Symptoms and signs develop only with complications of HTN or in cases of secondary HTN. The only reliable sign of HTN is the blood pressure.

Measuring the BP – **The blood pressure cuff must be large enough so that the bladder of the cuff encircles the arm + 30%!** If the cuff is too small the blood pressure will be falsely elevated.

## Diagnosis

HTN is diagnosed if BP is elevated on 3 separate occasions. Once the diagnosis of HTN has been made the following steps tests should be ordered:

- Cr, electrolytes, RBG, cholesterol, ECG, fundoscopy in all patients
- TSH, Renal US (with dopplers), urinary catecholamines/VMA/cortisol, serum renin/aldosterone, CXR, Echo if looking for cause of secondary HTN

## Other important concepts

“Burnt out” HTN – Occurs in patients who have had severe, long standing HTN but have now progressed to CCF (usually with dilated ventricles) with decreased systolic function and a blood pressure that is now normal or low.

# Treatment of Hypertension

## Treatment of Chronic HTN

### When to Treat?

- See new WHO guidelines for Prevention of Cardiovascular Disease!
- In general, any patient with severe (Grade 3) HTN and/or signs of complications (stroke, CKD, CAD, CCF, retinopathy etc) should be started on antihypertensive treatment immediately
- Patients with mild to moderate (Grade 1-2) HTN should be given 3 months to see if they respond to behavioral modification first. If BP remains  $>140/90$  they should then be started on antihypertensives.

### Counseling

- lose weight ( $>5\text{kg}$ ) if overweight by BMI  $> 25$
- reduce salt intake – no added salt in cooking or at table
- increase physical activity
- Smoking cessation!
- Reduce alcohol intake ( $<3$  units/day)

Patient should also be counseled that, if they start antihypertensives, they will likely need to take medications every day for life to prevent complications. They need to take the medication even if they feel well. If they have side effects they should come directly to see the doctor and not stop the medications until they are seen.

### Which drug to start with?

- For most patient, bendrofluazide 5mg PO OD is the best first drug as it is cheap, easy to take and very effective in Africans.
  - Use with caution in patients with DM and gout as bendrofluazide can cause hyperglycemia and hyperuricemia.
- CCBs (like Nifedipine or amlodipine) are all very effective in Africans and is a good first antihypertensive if you want to lower the BP rapidly (as in hypertensive urgency)
- For patients with DM or CCF and a normal or stable creatinine, ACE inhibitors (like captopril or lisinopril) are the best first antihypertensive.
- In patient with CAD, beta blockers are the best first antihypertensive as they reduce the risk of death from CAD
- Of note, most antihypertensives take 2-4 weeks to reach maximal effect so it is good to wait 1 month before increasing the dose of a medicine or adding another one.

### What to do if the first drug doesn't work?

- 2/3 of patient with hypertension will require at least 2 drugs to control their hypertension and 1/3 will require 3 drugs
- Always titrate your first drug to its maximum dose first before adding another drug.
- Monitor for side effects
  - ACE inhibitors – monitor creatinine
  - Thiazide diuretics – monitor electrolytes
  - Beta blockers – monitor heart rate

- If the BP remains elevated despite maximal dose of a first drug, add another drug and then titrate this to its maximal dose. Whatever you start with, either thiazides or CCB are good second drugs in most African patients.

### **What is the goal BP?**

- In most patients the goal BP is < 140/90
- In patients with DM or CKD we use a goal BP of < 130/85

## **Treatment of Hypertensive Urgency and Emergency**

In any patient with BP > 220/120 (“Very Severe Hypertension”), assess for signs of end organ damage and consider admission to the hospital. Hypertensive emergency usually does not occur unless DBP > 130. Keep in mind that urgency is much more common than emergency

### **Signs of End Organ Damage**

- Hypertensive Encephalopathy (confusion, headache)
- Acute retinal hemorrhage (sudden onset of blurry vision, massive hemorrhage on ophthalmoscopy)
- Myocardial ischemia or infarction (chest pain, ECG changes)
- Pulmonary Edema (shortness of breath, CXR with pulmonary edema)
- Acute Kidney Injury (recent onset of oliguria or anuria, elevated creatinine, blood on UA)

### **Treatment Goals**

If Hypertensive Urgency (no signs of end organ damage)

- Aim to lower MAP by 25% over 2-3 days using oral medications
- Start with Nifedipine 20mg BD and add other meds as necessary

If Hypertensive Emergency (+ signs of end organ damage)

- Aim to lower MAP by 25% over 1-2 hours using IV medications
- Currently we are using IV Hydralazine drips titrated to goal BP
- Labetalol drips (+ other meds) are better when available
- Once the BP improves, patients can be transitioned to oral medications

## **Shock & Hypotension**

**Hypotension** is state of **low blood pressure** with systolic blood pressure  $<90$  mmHg (relative hypotension with  $>30$  mmHg below baseline, or mean arterial pressure  $<65$ ).

**Shock** is a physiologic state characterized by a significant reduction of systemic tissue perfusion (“**decreased perfusion**”), resulting in decreased oxygen delivery to the tissues & organ injury (e.g. brain, kidneys, liver, etc)

### **Types of Shock:**

1. Hypovolemic\* – due to loss of fluid or blood
2. Cardiogenic\* – due to decreased cardiac contractility
3. Septic\* - due to infection
4. Anaphylactic – due to allergic reaction
5. Obstructive – due to decreased blood flow to the left ventricle as in cardiac tamponade or PE
6. Endocrine - Adrenal Insufficiency, pituitary failure etc

\* The 3 most common types of shock

### **Etiology & Pathophysiology**

- Shock is due to inadequate blood pressure.
- Low blood pressure is due to inadequate cardiac output or low peripheral resistance.
- Low cardiac output is caused by a problem with heart rate or stroke volume
- Heart rate abnormalities: too fast (tachycardia), too slow (bradycardia).
- Stroke Volume abnormalities: failure to receive (preload), failure to eject (contractility & afterload), inadequate volume
- Low peripheral vascular resistance is due to inappropriate vasodilatation.

### **Clinical presentation: Cardinal findings in all shock presentations**

- Hypotension – Absolute (systolic blood pressure  $<90$  mmHg) or relative (a drop in systolic blood pressure  $>40$  mmHg), which is why a patient may be in shock with a high or normal blood pressure.
- Oliguria – From shunting of renal flow to other vital organs, intravascular volume depletion, or both.
- Change in mental status – A continuum from agitation → confusion/delirium → obtundation/coma.
- Cool, clammy skin – Compensatory peripheral vasoconstriction redirects blood from the periphery to the vital organs (heart, brain, splanchnic). However, in early distributive shock or terminal shock the skin may be flushed or hyperemic skin due to failure of compensatory vasoconstriction
- Metabolic acidosis – Increased lactate production from anaerobic metabolism when shock progresses to circulatory failure and tissue hypoxia, along with decreased clearance of lactate by the liver, kidneys, and skeletal muscle.

Once the diagnosis of shock has been made, the most important next step is to quickly determine the type of shock. In low-resource settings, this is usually done based on history and physical examination. Some basic investigations may be helpful.

## History

Look for underlying causes, try to classify type of shock. Often limited due to confusion or obtundation.

- History of bleeding? Trauma? Hematemesis? Hematochezia? Melena? If so => hypovolemic shock
- History of coronary artery disease? Valvular heart disease? CCF? If so => cardiogenic shock
- History of fever? Pneumonia? Meningitis? => septic shock

**Remember that patients may have more than 1 type of shock, for example hypovolemic + septic.**

## Examination

- General: Ill-appearing? Pale? Confused? Lethargic? Unresponsive? Temperature  $>38^{\circ}\text{C}$  or  $<36^{\circ}\text{C}$ ?
- Neurologic: Meningismus? Focal neurologic deficits?
- Cardiac: Tachycardic (HR $>100$ )? Hypotensive (SBP $<100$ )? Dizziness? Orthostatic hypotension? S3? Murmurs? Extremities warm/well-perfused or cold/blue? Capillary refill brisk or slow? Chest pain?
- Pulmonary: Evidence of pneumonia? Using accessory muscles of respiration? RR $>30$ ? Oxygen saturation  $<95\%$ ?
- Gastrointestinal: Abdominal pain? Tense/rigid abdomen (may indicate perforated viscus, hemorrhage, peritonitis)? Rebound? Guarding? Melena or blood on rectal exam?
- Renal: Flank tenderness (pyelonephritis)? Urine output  $<0.5 \text{ mL/kg/h}$  or  $<30 \text{ mL/h}$ ?

## Diagnostic studies

- Basic chemistry tests (Na, K, Cl, HCO<sub>3</sub>), Renal function tests, Liver enzymes
- Glucose, arterial blood gas/lactate (if possible)
- Full blood picture (leukocytes & bandemia in sepsis)
- Type and cross
- Septic work up (CXR, blood culture, sputum microscopy culture & sensitivity, urine dipstick, microscopy, culture & sensitivity)
- ECG, cardiac enzymes & echocardiogram.
- Ultrasound – focused assessment with sonography for trauma (FAST)

## Management

***In shock, the management depends entirely on the type of shock!!! Shock is ALWAYS an emergency and must be treated rapidly.***

Direct at most likely underlying cause; see other topics (GI bleeding, fever, meningitis, pneumonia, MI, etc). Usually need:

- \*\*2 large IV cannulas – 18 gauge (green) or greater\*\*
- \*\*Aggressive colloid (NS or LR) fluid resuscitation for hypotension – especially in hypovolemic + septic shock (may require  $>10\text{L}!!!$ )\*\*
- \*\*Packed red blood cells if bleeding/hypovolemic shock\*\*
- Foley catheter and monitoring of urine output
- Frequent monitoring of vital signs

- Intubation if in respiratory distress
- Stop any antihypertensives or diuretics. If pressors (dopamine, epinephrine, neosynephrine, etc), if shock is present despite aggressive fluid resuscitation and pressors are available
- If septic: search for source & start empiric antibiotics based on likely type of infection. Give IV fluids
- If hypovolemic: IV fluids, check electrolytes, fix underlying condition (e.g. diabetic ketoacidosis), send for type & crossmatch for urgent transfusion if hemorrhage (hemorrhage is the most common cause of hypovolemic shock).
- If cardiogenic: IV fluids may be harmful, IV lasix and or dopamine may help, fix the underlying problem (e.g. valve replacement).
- If outflow obstruction suspected: IV fluids, urgent ECG, CXR to confirm, thrombolysis for PE, chest tube for tension pneumothorax, percardiocentesis for tamponade.
- If anaphylactic: IV fluids, subcutaneous epinephrine 0.3ml 1:1000 solution if severe, antihistamines, & corticosteroids may help.
- If adrenal insufficiency: give hydrocortisone 100mg TDS x 5/7

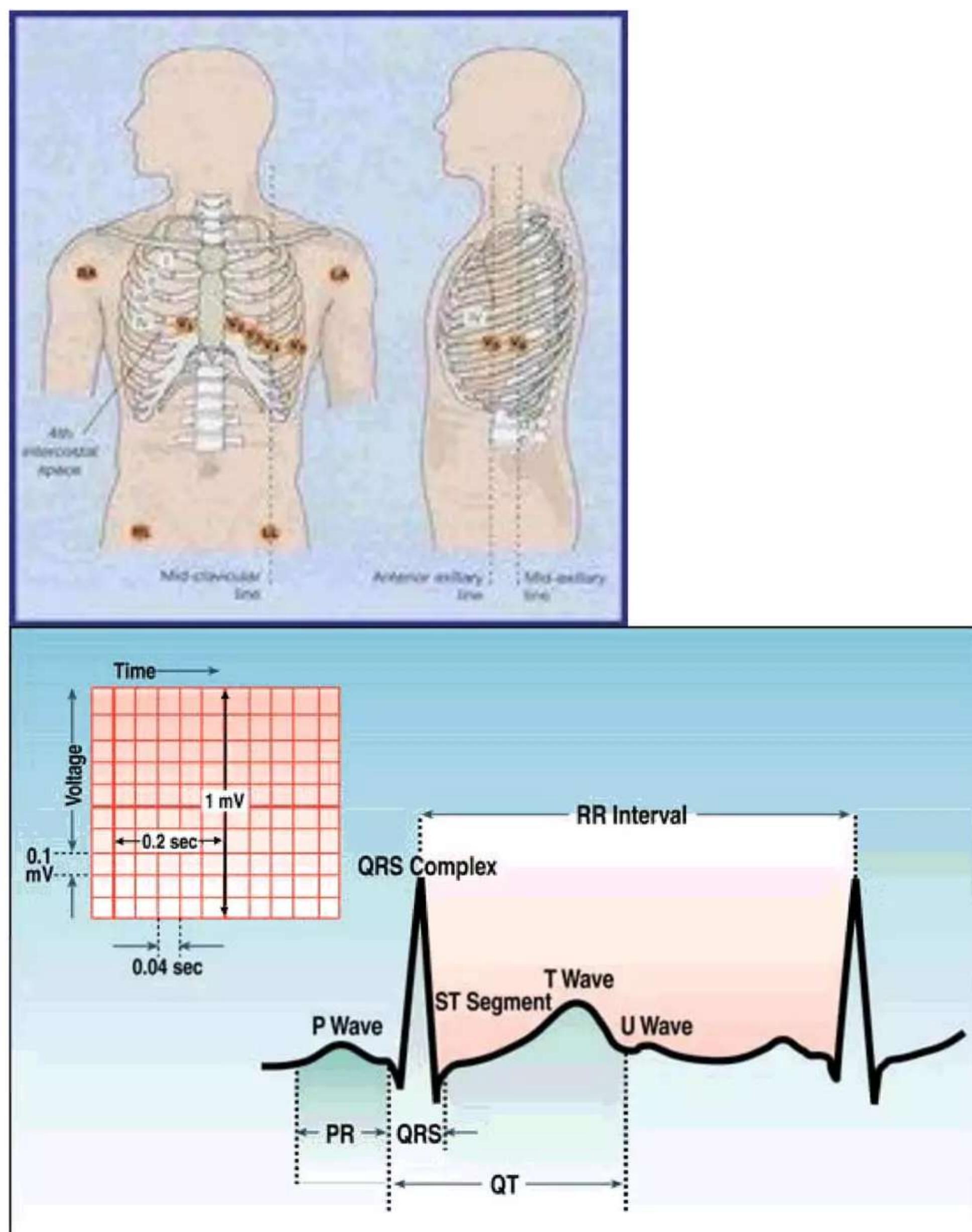
# Introduction to Electrocardiograms (ECG/EKG)

## Indications

- Any patient with severe or persistent chest pain should have an EKG to rule out myocardial ischemia or infarction.
- Any patient with irregular cardiac rhythms or abnormal rate should have EKG to determine the type of arrhythmia.
- EKGs are also useful in patients with CCF, patients with unexplained shortness of breath (like pulmonary embolism) etc.

## How to Perform

Place leads on the patient as seen in diagram:



## Basic EKG Interpretation:

\*\*You Must have a systemic approach (FOLLOW SAME PROCESS EVERY TIME).

- 1. RATE
- 2. RHYTHM
- 3. AXIS
- 4. INTERVALS
- 5. CHAMBERS

Some Basic Measurements  
--one small box is 1mV  
--one small box is 40ms (0.04 sec)

- **6. ST CHANGES AND Q WAVES**

### 1. Rate

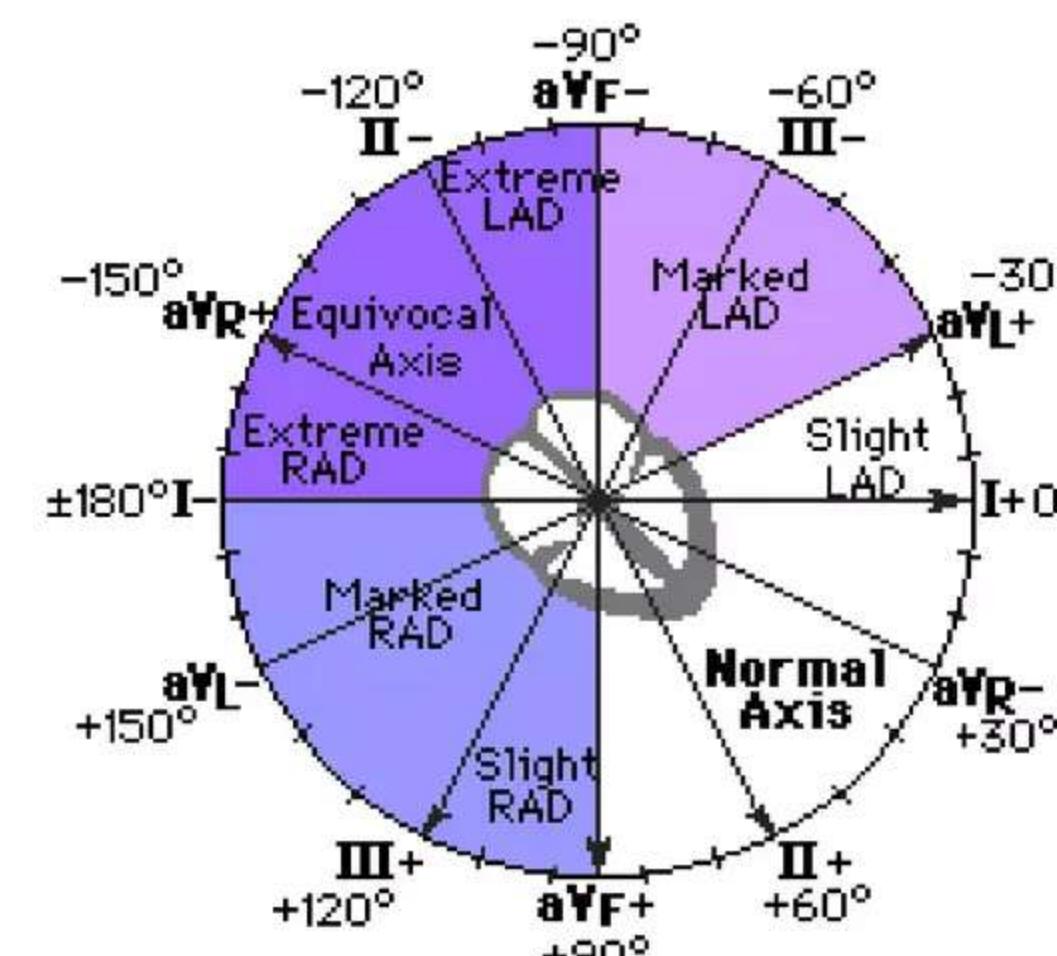
- normal is 60-100
- bradycardia is less than 60
- tachycardia is greater than 100
- Calculate Rate by looking at R waves
- Memorize: 300—150—100—75—60—50
- 300 divided by total number of big boxes

### 2. Rhythm

- Is the patient in sinus rhythm or not?
- Is the R-R interval regular or irregular?
- Is there a P for every QRS and a QRS for every P?
- Is the P upright in II and downward deflection in V1?

### 3. Axis

- What is the direction of the depolarization of the ventricle?
- Figure this out by looking at the QRS complex in the limb leads or the frontal plane.
- Normal is -30 to 90.
- Right Axis is greater than 90.
- Left Axis is less than -30.
- Positive complex in I and II means axis is in the normal range.



### 4. Intervals

- What is the PR interval – normal is less than 200ms (0.2 sec) one big box
- What is the QRS interval – normal is less than 100ms
  - incomplete block is 100-120ms
  - complete block is greater than 120ms
- What is the QT interval – this is rate dependent but should be less than  $\frac{1}{2}$  of RR interval.

### 5. Chambers

- What size are the atria?
  - LAE – look for biphasic P in V1 or notched P in II
  - RAE – look for tall P in II ( $>2.5\text{mV}$ )
- What size are the ventricles?
  - RVH – look for large R in V1 ( $R > S$  in V1)
  - LVH – many different criteria
    - S in V1 + R in V5  $> 35\text{mV}$  OR
    - R in aVL + S in V3  $> 28$  in men and  $> 20$  in women OR
    - R in aVL  $\geq 11\text{mV}$

### 6. Look for ST Segments and Q Waves