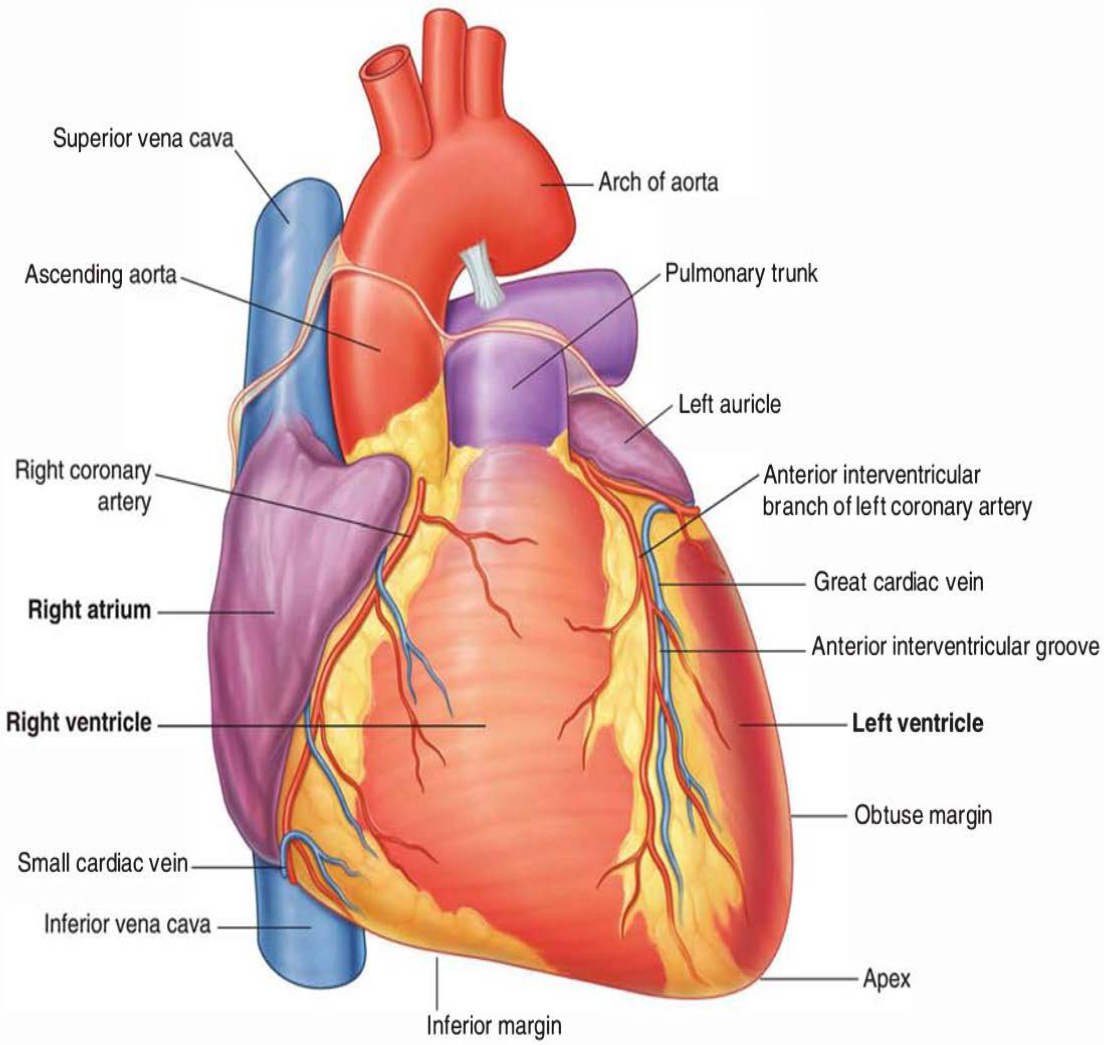
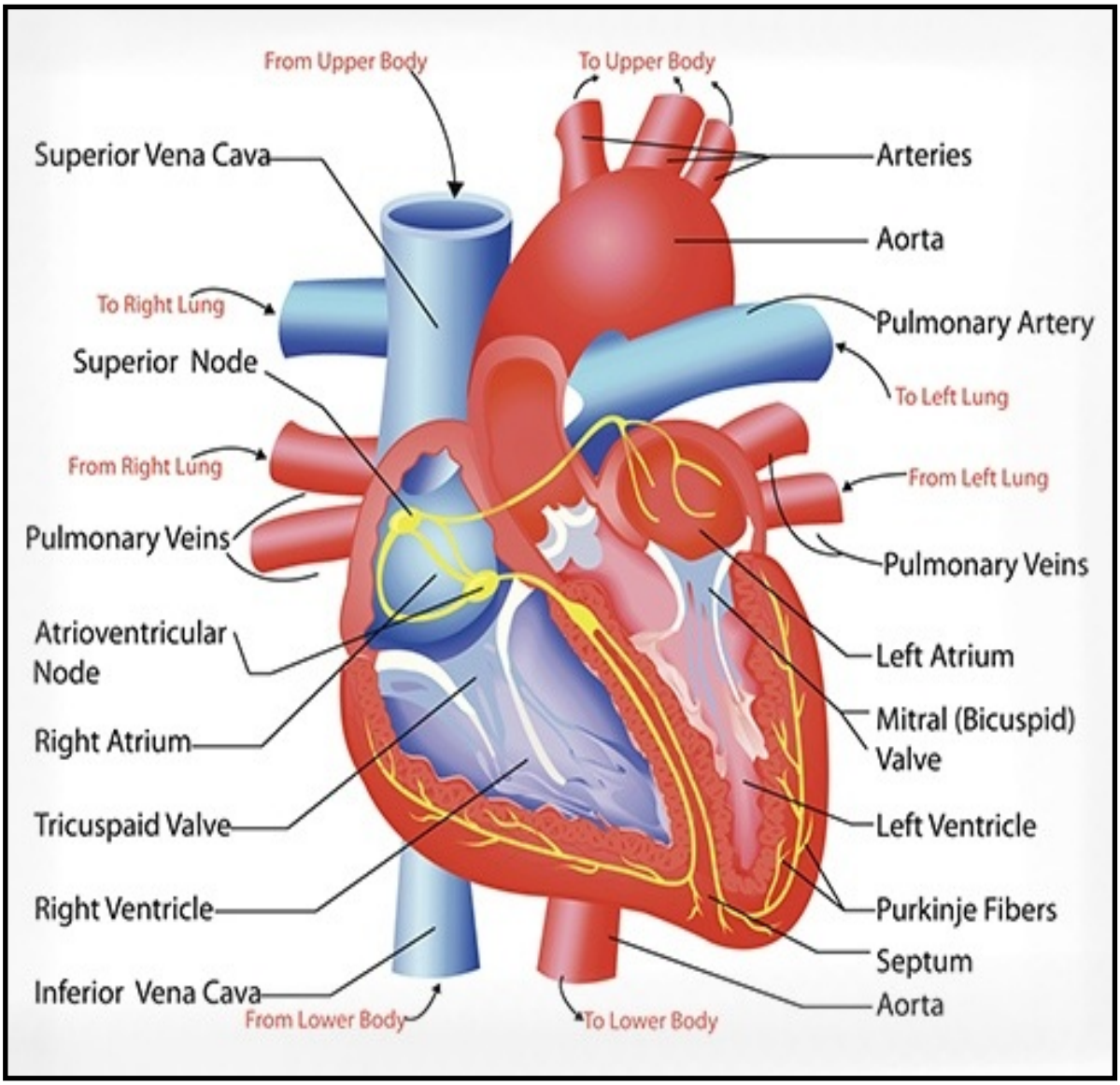
Cardiovascular System

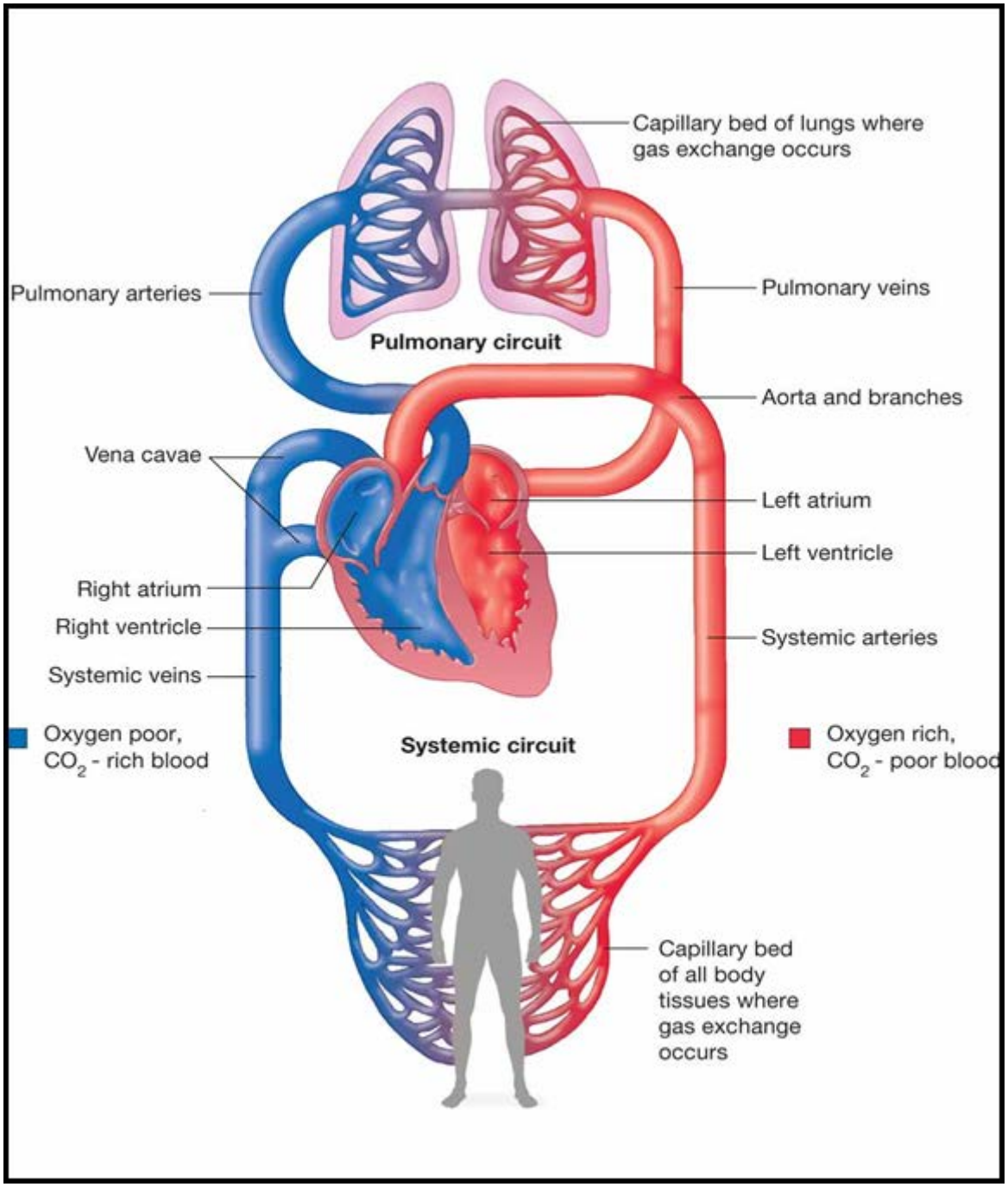


DR. MOHAMMED ELAGOURI
MRCP-UK

Blood circulation



Cardiovascular System



Symptomatology

Acute Dyspnea

Causes:

1. Acute Pulmonary Edema
2. Acute exacerbation of Bronchial Asthma
3. Acute exacerbation of COPD
4. Pulmonary Embolism
5. Pneumonia
6. Pneumothorax

Acute Pulmonary Edema:

- Dyspnea
- Orthopnea
- PND (Paroxysmal Nocturnal Dyspnea)

  After 1-2 hours of sleep patient awake with:
  - Dyspnea
  - Cough + frothy sputum (blood tinged)
  - Sweating
  - Anxiety

Dyspnea of hour's duration

***Ask about Chest Pain** (IHD) as a cause

Acute Exacerbation of Asthma:

- Paroxysmal Attacks
- Between attacks patient completely well
- The Attack is:
  - Acute Dyspnea
  - Wheeze
  - Cough
  - Chest tightness

***Ask about:** Precipitating Factors

- **General:** smoke, dust, perfumes, cold ….etc.
- **Specific:**

  Drugs (Aspirin + B-blockers) ⟶ Drug Induced Asthma

  Exercise ⟶ Excercise Induced Asthma

  Occupation ⟶ Occupational Asthma

GERD symptoms (Heartburn) → Acid Reflux Asthma

- **Childhood background**

  Extrinsic Asthma → Childhood onset

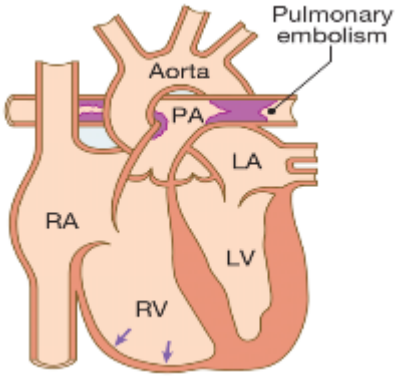
  Intrinsic Asthma → Adult onset

Acute Exacerbation of COPD:

- Smoker (long time)
- Age > 40 years
- Who has:
  - Chronic cough
  - White mucoid sputum
- Now complaining of:
  - Increase severity of cough (syncope\vomiting)
  - Acute Dyspnea
  - Fever (chest infection)
  - Change in color of sputum from white to green or yellow

Pulmonary Embolism:

- **Triad of:**
  - Acute Dyspnea
  - Hemoptysis
  - Chest pain
- History of Unilateral Leg Swelling (DVT)
- Risk Factors:
  - Oral contraceptive
  - Post-Operation



Pneumonia:

- Acute Dyspnea (days)
- Pleuritic chest pain (stabbing\lateral\increase by inspiration)
- Cough
  - Early dry
  - Later on productive (green\yellow sputum)
- Fever

Pneumothorax:

- Acute Dyspnea
- Chest pain

- H\O:
  - Trauma
  - Long term steroid use
  - Smoking (emphysema)

Chronic Dyspnea

Causes:

1. Congestive Heart Failure
2. Interstitial Lung Diseases (pulmonary fibrosis)
3. Anemia

Congestive Heart Failure (Biventricular Failure):

- **Ask about Symptoms of low COP:**
  - Dizziness\Syncope
  - Fatigue
  - Oliguria (polyuria if taking diuretics)
  - Bilateral leg swelling
  - Intermittent claudication
- **Ask about Symptoms of Left Ventricular Failure (pulmonary congestion):**
  - Chronic dyspnea
  - Hemoptysis
  - Recurrent chest infection
  - Orthopnea
  - PND
- **Ask about Symptoms of Right Ventricular Failure (systemic congestion):**
  - Abdominal distension (Ascites\Gases)
  - Jaundice (congested liver)
  - Right hypochondrial pain (congested liver)
  - Dyspepsia (congested stomach & intestine)
    - Belching
    - Borborygmi
    - Pre-mature satiety
  - Bilateral leg swelling (congestion of lower limbs)

Pulmonary Fibrosis:

- Pulmonary Fibrosis is 50% Idiopathic (most common cause), and 50% is other disease

- Triad of:
  - Dyspnea without wheeze
  - Chronic cough without sputum
  - Fine crepitation without ronchi

- **Ask about Occupation:**
  - **Pneumoconiosis:** Coal Dust (coal workers pneumoconiosis)
  - **Asbestosis:** Asbestos Dust
  - **Silicosis:** ⟶ Silica Dust (silicon)
- **Ask about Drugs:**
  - **Amiodarone** (class III antiarrhythmic agent)
  - **Methotrexate** (used to treat certain types of cancer of the breast, skin, head and neck, or lung)
  - **Nitrofurantoin** (an antibiotic used to treat urinary tract infections)
  - **Methysergide** (used prophylactically in migraine and other vascular headaches)
- **Ask about Joint pain:**
  - SLE ⟶ + malar rash
  - RA ⟶ + morning stiffness
  - Back pain ⟶ Ankylosing Spondylitis
  - Crest syndrome

    C ⟶ Calcinosis
    R ⟶ Raynaud's phenomenon (color changes in fingers)
    E ⟶ Esophageal dysmotility ⟶ Dysphagia
    S ⟶ Sclerodactyly (infarction of the fingers and toes)
    T ⟶ Telangiectasia
- **Ask about Sarcoidosis:**
  - Erythema Nodosum (painful rash in lower limbs)
  - Uveitis (H\O red eye)
  - Symptoms of exacerbation at summer time or sun exposure

Anemia:

- **Ask about Symptoms of Anemia:**
  - Fatigue
  - Dizziness
  - Excessive sleep
  - Headache
  - Lack of concentration
- **Ask about Symptoms of the Cause:**
  - Blood Loss
    - Menorrhagia in young females
    - GIT { Hematemesis, Melena

Chest Pain

Is it central or lateral?

- **Central** → Cardiac or GIT (esophageal)
- **Peripheral** → Respiratory or Musculoskeletal

Central Chest Pain:

1. Ischemic heart diseases
2. Aortic Dissection
3. Pericarditis
4. Myocarditis
5. Pulmonary Embolism
6. Aortic Stenosis
7. Hypertrophic Obstructive Cardiomyopathy (HOCM)
8. Esophageal Diseases

Ischemic Heart Disease:

- Central chest pain, **poorly localized**, described as:
  - Heaviness
  - Burning
  - Constricting
- **Radiating to:**
  - Neck
  - Jaw
  - Left arm
  - Forearm
- **Precipitated by:**
  - Exertion
  - Emotional upset
  - Exposure to cold
- **Relieved by:**
  - Sub-lingual Nitroglycerine
  - Rest
  - If not relieved → Acute Coronary Syndrome
- **Risk Factors:** DM, HTN, Hyperlipidemia …etc.

Aortic Dissection:

Sudden, sever, **tearing** chest pain

- **Radiating to:** the back
- **Associated with:**
  - HTN
  - Dysphagia
  - Hoarseness of voice

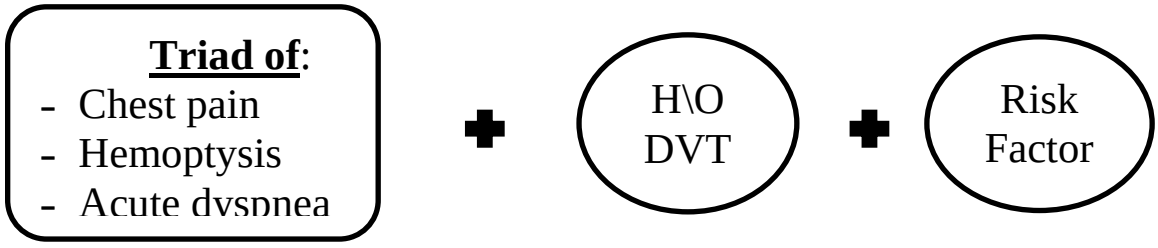
Pericarditis:

- **Stabbing** chest pain, increase by deep inspiration
- **Relieved by:** pending forward & NSAIDs
- **Preceded by:** URTI (viral)
  - With or without H\O TB (Tuberculous pericarditis)
  - With or without Malar rash (SLE)

Myocarditis:

- Acute onset chest pain, **burning** + fever
- **Preceded by:** URTI

Pulmonary Embolism:



Aortic Stenosis:

- Chest pain on exertion (Angina) + Syncope

HOCM:

- Chest pain on exertion + Syncope + H\O sudden death in the family

Esophageal Diseases:

- **GERD:**
  - Heart burn after meal
  - Relieved by antacids
  - Increase with supine position
  - Associated with cough and hoarseness of voice
- **Esophageal Spasm**
  - Chest pain partially relieved by nitroglycerine
  - With acute onset of Dysphagia

Palpitations

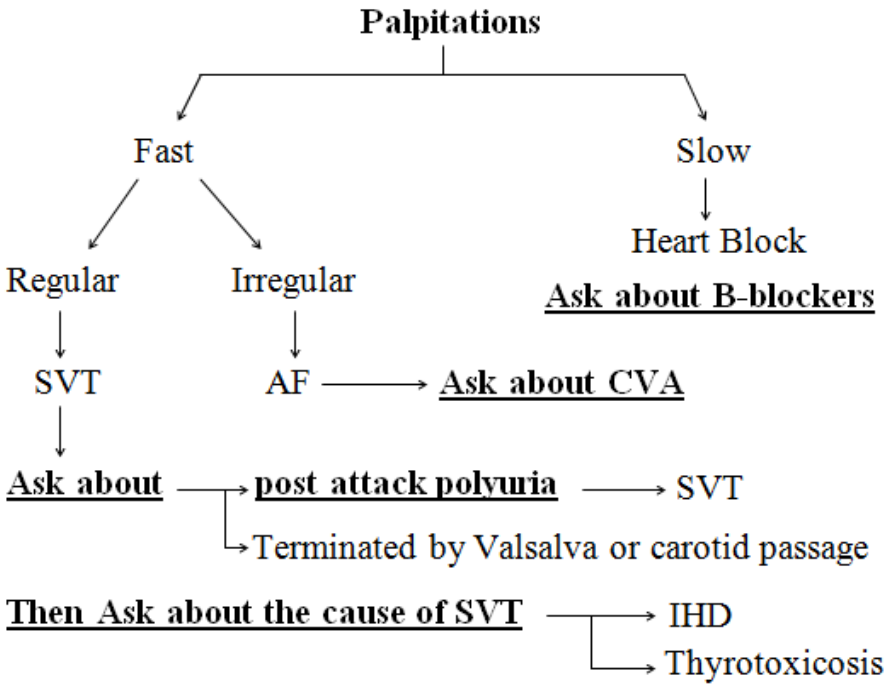
Causes:

1. Cardiac Arrhythmias
2. Hyper-dynamic Circulation (Anemia, Thyrotoxicosis)
3. Drugs

4. Anxiety
5. Coffee & Tea

Cardiac Arrhythmias:

- **Ask is it fast or slow**



Hyper-dynamic circulation:

- Anemia:
  - Headache
  - Fatigue
  - Dizziness
  - Lack of concentration
  - Blood loss (GIT, Menorrhagia)
- Thyrotoxicosis:
  - Wight loss
  - Increase Appetite
  - Insomnia
  - Sweating
  - Heat intolerance

Drugs:

- Diuretics
- Digoxin
- Vasodilators (ACE Inhibitors, Calcium Channel Blockers)
- Salbutamol
- Theophylline

Anxiety:

Fear Exogenous ⟶ Physiological

Endogenous → Pathological

Coffee & tea: Ask about coffee & tea intake

Syncope

Causes:

1. Cardiac Arrhythmias
2. Postural Syncope
3. Vasovagal Syncope
4. Carotid Sinus Hypersensitivity
5. Vertebrobasillar Insufficiency
6. Cough Syncope
7. HOCM
8. Aortic stenosis

Cardiac Arrhythmias: (see palpitation)

Postural Syncope:

Ask if syncope related to posture

If yes → Ask about symptoms of **DM** *(Autonomic neuropathy)*

Ask about symptoms of Addison's disease

- Hyperpigmentation
- Wight loss
- Polyuria
- Fatigue

Vasovagal syncope:

Ask about → Emotional upset
→ Severe painful stimuli

Carotid sinus hypersensitivity:

Ask about → relation of syncope to Shaving
→ Turning Head

Vertebrobasillar Insufficiency:

Ask about → Occipital headache
→ Vertigo
→ Ataxia

+ Risk factors of Atherosclerosis *(DM, HTN, high lipids ….etc)*

Cough syncope:

- Syncope after prolonged attack of cough

Leg swelling

- **Bilateral:**
  1. CCF (congestive cardiac failure)
  2. Liver Cirrhosis

3. Renal Diseases
4. Hypothyroidism
5. Malnutrition
6. Drugs

CCF:

Ask about symptoms of low COP
Ask about symptoms of pulmonary & systemic congestion

Liver cirrhosis:

Ask about jaundice
Then Ask about → Alcohol
→ Hepatitis B\C infection
Female → Primary biliary cirrhosis:-
-Hyperpigmentation
-Itching
Male → Hemochromatosis:-
-Hyperpigmentation
- Infertility
-Polyuria (DM \ DI)
Wilson Disease → Abnormal movement

Renal Disease:

Ask about

Periorbital Edema
(+)

Nephritic Syndrome
Dark urine\Hematuria
Oliguria
HTN (headache\convulsion)
If +ve
↓
Then Ask about
URTI before
→ < 4 days → IGA nephropathy
→ 2-3 weeks → Post-streptococcal glomerulonephritis

Nephrotic Syndrome
Frothy Urine (proteinuria)
If +ve
↓
Ask about:
DM, Rheumatoid Arthritis

Hemoptysis → Wegener's granulomatosis
Malar rash → Lupus nephritis

Hypothyroidism:

Ask about:

- Wight gain
- Decrease appetite
- Increase sleep
- Menorrhagia
- Constipation

Malnutrition

Ask about Chronic Diarrhea

Drugs:

Ask about:

- Amlodipine
- Other vasodilators as ACEI

• Unilateral leg swelling:

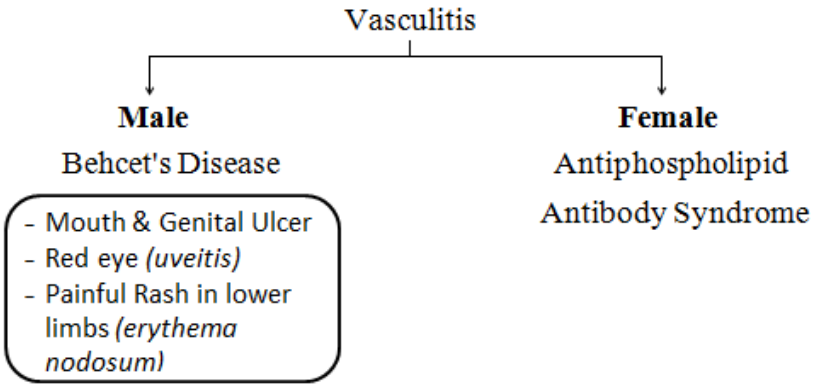
1. DVT
2. Cellulitis
3. Compartment Syndrome
4. Rupture Baker's Cyst
5. Lymphedema

DVT:

Unilateral leg swelling + Pain + Redness + Risk factors

Ask about Factors that making endothelium Rough:

1. Vasculitis



2. Trauma
3. Previous DVT
4. Varicose Veins

Ask about factors leading to Stasis of blood:

1. Prolonged bed rest
2. Long journey *(travel abroad)*
3. Pregnancy
4. Pelvic mass

Ask about factors change the Composition of blood:

1. Protein C & S deficiency

- Family history
- Young > 40 years
- Previous H\O DVT

2. Oral contraceptives *(inhibit protein C & S)*
3. Hyperviscosity Syndrome
   e.g: severe diarrhea & vomiting

Ask about recent Surgery, especially orthopedic

Ask about symptoms of Pulmonary Embolism:

Triad of:

- Chest pain
- Acute dyspnea
- Hemoptysis

+ H\O Anticoagulant Use

Cellulitis:

Ask about:

- H\O fever & Rigor
- H\O DM
- H\O skin wound or diabetic foot
- H\O redness & pain

Compartment Syndrome:

Ask about:

- H\O fracture with Cast
- H\O painful extension of Toes

Rupture Baker's Cyst:

- Patient known case of rheumatoid arthritis
- H\O swelling over the back of knee joint
- If Ruptured $\longrightarrow$ Pain + Redness + Hotness

Lymphoedema:

- Painless swelling
- No Redness
- No Hotness
- Chronic Swelling
- H\O Trauma \ Tumor in lower limbs
- Walking Bare feet *(filariasis)*

Systemic Review

Respiratory System:

Cough → ACEI

Wheeze → Aspirin
→ B-blockers
→ Cardiac Asthma

Hemoptysis → Aspirin
→ Warfarin
→ Pulmonary Congestion

GIT:

Abdominal Pain:

- Gastritis → Aspirin
- Digoxin Toxicity
- Inferior wall MI
- Liver Congestion

Jaundice:

- Liver Congestion
- Amiodarone induced Hepatotoxicity
- Statin induced Hepatotoxicity

Abdominal Distention:

- Gases → Dyspepsia
- Fluid → Ascites

Constipation: Nitroglycerine

CNS:

Embolism from the heart:

- Weakness
- Dyspnea
- Amaurosis Fugax *(transient loss of vision)*

Locomotor:

- Back pain (Sacroiliitis) → Ankylosing Spondylitis
  ↓
  Aortic Regurgitation
- Pain at Big Toe → Gout
  - Side effect of Diuretics
  - Hyperuricemia
- Knee pain → Rheumatoid Fever

Urinary System: → Hematuria Infective Endocarditis
→ Warfarin Overdose

Past Medical History

DM:

- IHD
- Metformin contraindicated in HF
- Peripheral vascular disease

HTN:

- Left ventricular hypertrophy
- IHD
- AF

Asthma:

- B-blockers & Aspirin are C/I

Past-Surgical History

- Anticoagulant Warfarin should be stopped 5 days before surgery
- Aspirin should be stopped 7-10 days before surgery
- Dental procedures are risk for Infective Endocarditis
- Coronary Angiography $\longrightarrow$ Coronary artery disease
- Percutaneous coronary intervention $\longrightarrow$ (stent) in Ischemic Heart Disease
- Coronary artery bypass graft (CABG) $\longrightarrow$ severe Ischemic Heart Disease

Drug History

<u>Anti-failure Drugs</u>

- Diuretics
- ACEI
- B-blockers
- Digoxin
- Spironolactone
- Nitrates

<u>Anti-anginal Drugs</u>

- Statins
- Aspirin
- Clopidogrel
- B-blockers

<u>Anti-coagulants Drugs</u>

- Warfarin
- Heparin

<u>Anti-arrhythmic Drugs</u>

<u>Diuretics</u>

- **Loop Diuretics:**
  - Furosemide
  - Ethacrynic Acid
  - Metolazone
  - bumetanide

Side effects:

- Hypokalemia
- Hyponatremia
- Hypocalcemia
- Hyperglycemia
- Hyperlipidemia
- Hyperuricemia (gout)
- Hypercalciuria
- Hypersensitivity
- Impotence

➢ **ACEI:**

- Lisinopril
- Enalapril

Side effects:

- Dry cough
- Hyperkalemia
- Angioedema
- Teratogenicity

Contraindications:

- Bilateral renal artery stenosis
- Pregnancy

B-blockers:

- Bisoprolol
- Carvidilol
- Metoprolol
- Atenolol

Side effects:

- Asthma
- Bradycardia & Heart block
- Hyperlipidemia

Digoxin:

Mechanism of Action:

Blocks Na^{+} - K^{+} ATPase $\longrightarrow$ Intracellular Ca^{++} $\downarrow$ Contractility (+Ve Inotropic)

Pharmacokinetics:

- Toxic Drug (low therapeutic index)
- High volume of distribution (non-dialyzable)
- Long T1\2 (36-38 hours) $\longrightarrow$ given in loading dose
- Excreted by kidney $\longrightarrow$ C\I in renal disease *(use Digitoxin)*

Toxicity: *(increased by low K^{+}, mg^{++})*

- GIT $\longrightarrow$ Abdominal pain, Diarrhea,Vomiting
- Cardiac $\longrightarrow$ Tachy & Brady-Arrhythmias *(except prolonged Q-T interval and mobitz type2 second degree heart block)*
- CNS $\longrightarrow$ Color vision disturbance (green\yellow), Convulsion, Coma

N.B: It may lead to Gynecomastia

Treatment:

- Stop the Drug
- Do ECG
- Send plasma level for Digoxin
- Treat cardiac arrhythmias
- Give Antidote $\longrightarrow$ Digoxin antibodies (Digibind)

Spironolactone:

K^+ Sparing Diuretic (anti-aldosterone)

Side effects:

- Hyperkalemia
- Gynecomastia (eplerenone will not lead to gynecomastia)

Nitrates:

- Nitroglycerin (parentral)
- ISMN *(Isosorbide mononitrate)(oral)*
- ISDN *(isosorbide dinitrate)(oral)*

Mechanism of Action:

Venodilator (↑c-GMP $\longrightarrow$ ↑Nitric Oxide)

Side effects:

- Headache
- Hypotension
- Palpitations
- Tolerance *(Drug Holiday)*

Statins:

- Simvastatin
- Atorvastatin
- Rosuvastatin

Mechanism of Action:

HMG CoA Reductase Inhibitors $\longrightarrow$ Inhibits Cholesterol Synthesis

Side effects:

- Hepatitis
- Myositis (rare)

Aspirin: *(low dose)*

Mechanism of Action:

Inhibits CoX Enzyme in platelets

$\downarrow$

$\downarrow$ Thromboxane A2 $\longrightarrow$ Anti-platelets effect

Sid effects:

- Bleeding
- Asthma
- Gastritis
- Renal impairment

Clopidogrel: *(Plavix)*

Mechanism of Action:

Inhibits ADP receptors on platelets ⟶ Anti-platelets effect

Side effect:

- Bleeding
- Neutropenia

Anticoagulants:

- **Heparin:**

  Mechanism of Action:

  Inhibits intrinsic pathway by binding to Anti-thrombin III, leading to potentiation of it. ⟶ Prolongation of APTT *(activated partial thromboplastin time)*

  - Given only parenteral (I.V or S\C) but not I.M.

  Side effects:

  - Bleeding
  - Thrombocytopenia *(heparin-induced thrombocytopenia)*
  - Osteoporosis
  - Alopecia

  Can be given in pregnancy

  Low Molecular Wight Heparin (LMWH) is C\I in renal impairment

  LMWH ⟶ Less risk of Thrombocytopenia
  LMWH ⟶ Less risk of Bleeding

  Antidote: Protamine Sulphate

- **Warfarin:**

  Mechanism of Action:

  Inhibits Vit-K ➔ **inhibition of extrinsic pathway ➔ Prolongation of PT** *(prothrombin time)* or INR *(international normalized ratio)*

  - Oral Anticoagulant
  - Delayed onset of Action (72 hours)
  - Toxic Drug ⟶ Bleeding
  - C\I in Pregnancy

  Side effects:

  - Bleeding
  - Teratogenicity
  - Hepatotoxicity
  - Skin Necrosis

  Antidote: vitamin K, fresh frozen plasma

Antiarrhythmic Drugs: *(Will be discussed in Arrhythmias)*

Family History

Familial Hypercholesterolemia → Autosomal Recessive
HOCM → Autosomal Dominant
Dilated Cardiomyopathy → X-linked Recessive

Social History

H\O:
Smoking \ Alcohol → IHD
Occupation → stress → IHD
Effect of disease on lifestyle

Cardiovascular System Examination

General Examination:

By general examination we need to check for:

1. Signs of Heart Failure
2. Signs of Pulmonary Hypertension
3. Signs of Infective Endocarditis
4. Signs of Over Anticoagulation

** Every cardiac disease will be complicated with one or all of these complications.*

Signs of Heart failure:

- Orthopnea
- Weak Pulse with Tachycardia
- Cold Hands
- Peripheral Cyanosis
- Low BP
- Raised JVP *(volume overload)*
- Jaundice *(liver congestion)*
- Lower Limb Edema

Signs of Pulmonary Hypertension:

- Raised JVP with prominent a-wave
- Bilateral leg Edema

Signs of Infective Endocarditis:

- Fever
- Pallor (Anemia)
- Clubbing
- Splinter Hemorrhage
- Janeway lesion (non-tender)
- Osler's Nodule (tender)
- Purpura
- Conjunctival petechia
- Oral Hygiene
- Lymphadenopathy

Signs of Over Anticoagulation:

- Purpura
- Echymosis

Risk Factors for Ischemia:

- Hypertension (HTN)
- Xanthelasma
- Arcus Lipidimicus
- Tender Xanthomas
- Weak peripheral pulsation
- Scar of Saphenous Graft (CABG)

Important points in CVS examination:

Commonest Cases in Exam are:

- Mitral Stenosis (MS)
- Mitral Regurgitation (MR)
- Aortic Stenosis (AS)
- Aortic Regurgitation (AR)
- Prosthetic Heart Valves

All of these diseases can be complicated by:

- Heart Failure
- Pulmonary HTN (except AS)
- Infective Endocarditis
- Over Anticoagulation

By Examination of Pericardium, you have to know some basics:

- Diseases of the heart are either → Volume Overload / Pressure Overload
- Diseases of **Volume Overload:**
  - MR
  - AR
  - VSD *(ventricular septal defect)*
  - PDA *(patent ductus arteriosus)*
- Diseases of **Pressure Overload:**
  - AS
  - Systemic HTN
  - HOCM
- Diseases of **Volume Overload** have common signs:
  - Forcible Non-Sustained Displaced Apex beat
  - S3 Heart Sound
  - High volume collapsing pulse in early cases
- Diseases of **Pressure Overload** have common signs:
  - Forcible Sustained Non-Displaced Apex beat
  - S4 Heart Sound
  - Usually have Low volume pulse

Murmurs of the Heart:

Systolic Murmurs at Apex → Pan-Systolic Murmur
(Cannot be Ejection Systolic Murmur)

Diastolic Murmurs at Apex → Mid-Diastolic Murmur
(Cannot be Early Diastolic Murmur)

Systolic Murmurs at Aortic area → Ejection Systolic Murmur
(Cannot be Pan-Systolic Murmur)

Diastolic Murmurs at Aortic area → Early Diastolic Murmur
(Cannot be Mid-Diastolic Murmur)

Thrill:

Palpable Murmur

If there is Thrill → the murmur is Systolic *(diastolic murmur not commonly cause Trill)*

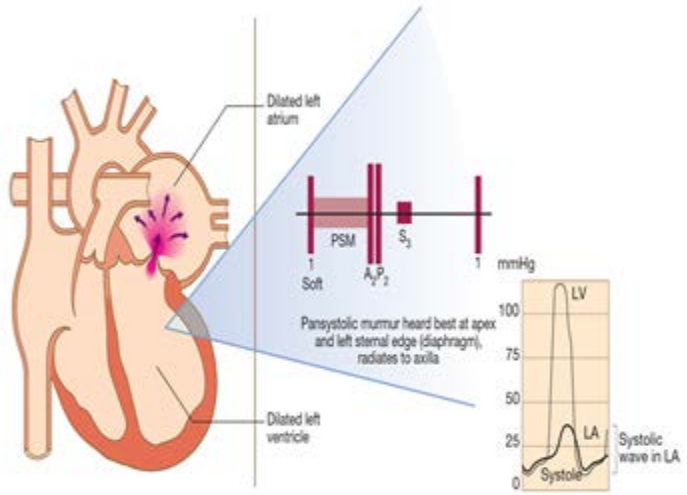
Signs of Mitral Regurgitation

Examination:

1. Pulse:
   High volume pulse *(Collapsing)*
   (Weak pulse in Heart Failure)
2. JVP:

- Normal in early cases
- Raised if there is Pulmonary HTN

3. Pericardium:



➢ **Inspection:**

Pulsation →
- Apex (forcible Apex)
- Left parasternal thrust → Pulmonary HTN
- Right Hypocondrial → Liver Congestion *(Pulmonary HTN)*

Deformity

Pectus Excavatum (in Marfan's Syndrome associated with MR)

➢ **Palpation:**

Apex beat →
- Forcible Non-Sustained Displaced *(early)*
- Weak Apex *(Heart Failure)*

Thrill → may be +ve *(Systolic Murmur)*

Left parasternal heave → +ve if there is pulmonary HTN

Palpable S2 → +ve if there is pulmonary HTN

➢ **Auscultation:**

S1 → Muffled

S2 →
- Muffled
- Can be Loud in pulmonary HTN

S3 may present *(Volume Overload)*

Pan-Systolic Murmur at the Apex, Radiated to the Axilla

** If Murmur not radiate to Axilla, that means it's not MR*

There may be murmur *(pan-systolic murmur)* at **Tricuspid Area** *(that's indicates pulmonary HTN)*

There may be two murmurs at Pulmonary Area:

1. Ejection Systolic Murmur
2. Early Diastolic Murmur *(Graham-Steel Murmur)*

Both indicate pulmonary HTN

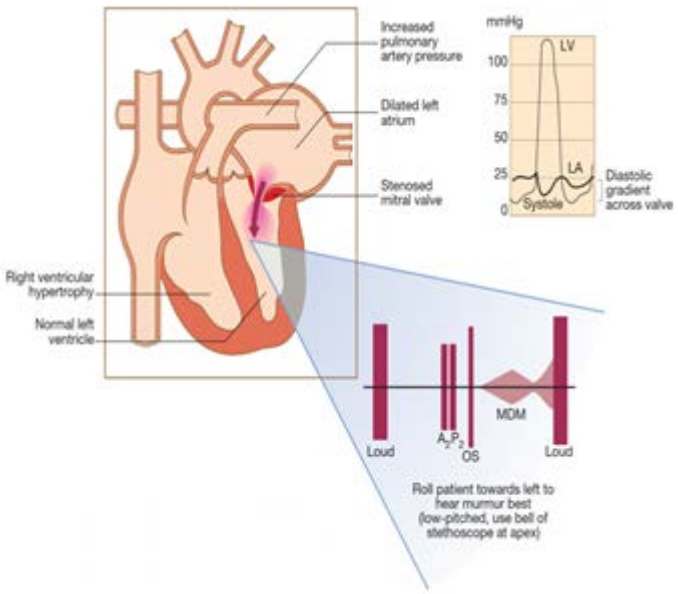
Factors that indicate Severe MR:

1. High volume collapsing pulse
2. Heart Failure signs
3. Pulmonary HTN signs
4. S3
5. Forcible Non-Sustained Displaced Apex

Signs of Mitral stenosis

History:

- Hemoptysis
- Dysphagia
- Syncope (AF)
- CVA *(Cerebrovascular Accident)*



Examination:

1. Pulse:

- **Low** volume
- AF *(Irregular)*

2. JVP:

- Normal early
- Raised in Pulmonary HTN

3. General:

- Malar Flush
- Signs of Heart Failure
- Signs of Infective Endocarditis
- Signs of Over Anticoagulation
- Signs of Pulmonary HTN

4. Pericardium:

➢ **Inspection:**

Pulsation:

At Apex ⟶ Tapping Apex *(palpable S1)*

Left parasternal Thrust ⟶ Parasternal Heave *(Pulm. HTN)*

Right hypochondrial ⟶ Liver congestion *(pulm. HTN)*

Scars ⟶ Left Submammary Scar (mitral valvotomy or valvoplasty)

➢ **Palpation:**

Apex beat ⟶ Tapping (palpable S1)

B.N: Tapping means ⟶ Non-Displaced

⟶ Loud S1 on auscultation

Trill ⟶ Uncommon
Heave ⟶ May be, if there is pulmonary HTN
Palpable S2 ⟶ May be, if there is pulmonary HTN
N.B: *S2 in pulmonary HTN will be widely split*

- **Auscultation:**

S1: Loud *(Muffled in fish mouth stenosis)*
S2: Normal *(Loud in pulmonary HTN)*
Opening snap:
- Heard if the valve still Mobile, because of **calcification** of the valve
- Reflects pressure in the left Atrium

N.B: *the closer the opening snap to S2, the more the severe the MS*
- **Mid-Diastolic rumbling murmur at Apex**:
  - Increase by left lateral position
  - Heard best by Bell of stethoscope
- **Pre-Systolic Accentuation of murmur** ⟶ caused by contraction of left Atrium pre-systole
- Pre-Systolic Accentuation Disappear with Atrial Fibrillation

N.B: *the longer the murmur, the more the severe MS*

Signs of Severity of Mitral Stenosis:

1. Low volume pulse
2. Tapping apex
3. Longer Duration of murmur
4. Closer opening snap to S2
5. Loud S1
6. Pulmonary HTN

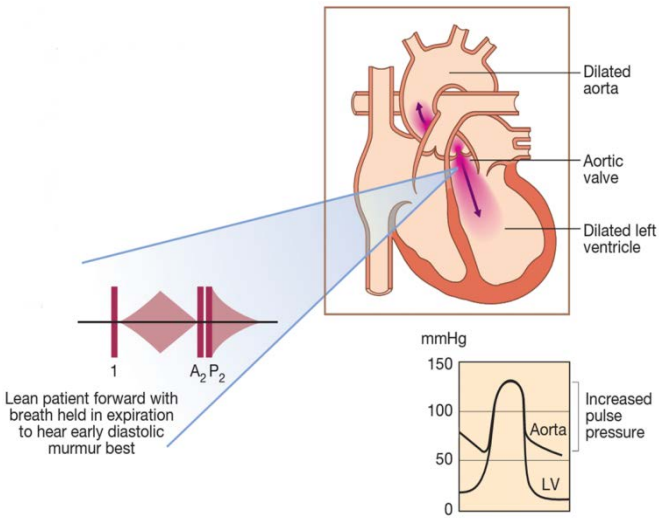
N.B: *MS starts with right ventricle* ⟶ *Left Ventricular Failure*
N.B: *MR starts with left ventricle* ⟶ *Right Ventricular Failure*

Signs of Aortic Regurgitation

Examination:

1. Pulse:
- **High** volume *(Collapsing)*
- Low volume *(in HF)*

2. Peripheral Signs ***(Signs of increase COP)***
- De Musset's Sign *(Head Nodding)*
- Corrigan's Sign *(Dancing Carotids)*
- Quincke's Sign *(Capillary pulsation under nails)*
- Wide pulse pressure
- Muller's Sign *(Pulsation of uvula)*



- Pistol Shot sign *(Systolic murmur at femoral artery by pressure of stethoscope)*
- Durozier Sign *(Systolic & Diastolic murmur over femoral artery by pressure of stethoscope)*
- Hill's Sign *(BP in lower limbs higher than BP in upper limbs by more than 20 mmHg)*

3. JVP:

- Increase ⟶ pulmonary HTN
- Normal ⟶ No pulmonary HTN

4. Pericardium:

➢ **<u>Inspection:</u>**

Pulsation

Apex beat ⟶ Forcible Non-Sustained Displaced Apex

Substernal Thrust ⟶ Parasternal Heave *(Pulm. HTN)*

Right Hypochondrial ⟶ Pulsatile Liver *(Tricuspid Regurgitation)*

↓

Because of pulmonary HTN

➢ **<u>Palpation:</u>**

Apex beat ⟶ Forcible Non-Sustained Displaced

⟶ Weak or Impalpable *(in HF)*

Left parasternal Heave ⟶ pulmonary HTN

Thrill ⟶ Unlikely *(Low intensity murmur)*

Palpable S2 ⟶ Pulmonary HTN

➢ **<u>Auscultation:</u>**

At Aortic area:

S1 ⟶ Normal

S2 ⟶ Muffled *(AR)*

⟶ Loud *(Pulmonary HTN)*

May be **S3** *(volume overload)*

Early Diastolic Murmur, increase by pending forward and holding breath in expiration

B.N: *the longer the murmur, the more severe AR*

<u>At Apex:</u>

May be Mid-Diastolic Murmur *(Austin-flint murmur)* in severe AR

<u>At Tricuspid area:</u>

Pan-Systolic Murmur ⟶ functional tricuspid regurgitation in case of Pulmonary HTN

<u>At Pulmonary area:</u>

Loud S2 ⟶ Pulmonary HTN

Ejection Systolic Murmur ⟶ Pulmonary HTN *(functional)*

Early Diastolic murmur *(Graham Steel Murmur)* in Pulmonary HTN *(functional)*

Signs that indicate sever AR:

1. S3
2. High volume pulse
3. Wide pulse pressure
4. Forcible Non-Sustained Apex
5. Austin flint murmur
6. Longer duration of diastolic murmur
7. Pulmonary HTN

Signs of Aortic Stenosis

Examination:

1. **Pulse:**

- **Low** volume
- Slow rising pulse

2. **JVP:** Not raised
3. Pericardium:

➢ **Inspection:**

Pulsation:

Apex ⟶ Forcible

Sustained Apex

➢ **Palpation:**

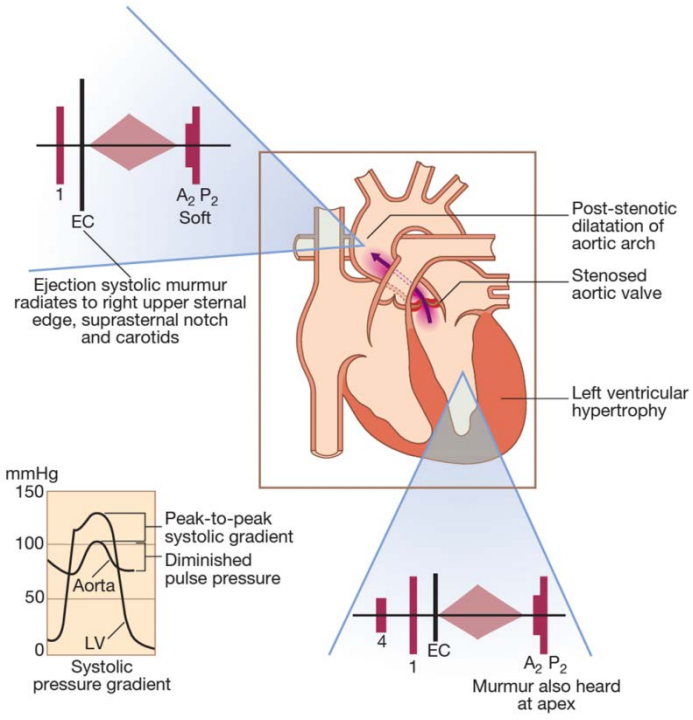
Apex beat ⟶ Forcible

Sustained Non-Displaced Apex *(Pressure overload)*

No Heave

Thrill over Aortic area *(systolic) (↑ Intensity murmur)*

No palpable S2



➢ **Auscultation:**

At Aortic area:

S1 ⟶ Normal

May be **Ejection Click** after S1 *(opening of aortic valve)*

Ejection Systolic Murmur, harsh and radiating to **both Carotids**

It can be heard **all over the pericardium**, but **not Axilla** *(Gallavardin Phenomenon)*

S4 *(in severe AS)*

Signs indicate severity of AS:

1. Low volume pulse
2. Slow rising pulse
3. Narrow pulse pressure
4. Forcible Sustained Non-Displaced Apex
5. S4

D\D of Systolic Murmur:

Pan-Systolic Murmur at Apex

- **MR** ⟶ Radiate to Axilla
- **TR** ⟶ ↑ JVP
- **VSD** ⟶ all over pericardium & maximum at left sternal border opposite to 3^{rd} & 4^{th} intercostal space\ No radiation to Axilla

Ejection Systolic Murmur at Aortic area

- **AS** ⟶ Radiates to both Carotids with low volume pulse
- **Aortic sclerosis** ⟶ Not radiating with normal pulse
- **Hemic Murmur** *(hyperdynamic circulation)* ⟶ Not radiating with high volume collapsing pulse e.g: Anemia & thyrotoxicosis

D\D of Diastolic Murmur at apex:

- **MS**
- **AR** (Austin Flint Murmur)
- **Rheumatic Fever** *(Carey Coombs Murmur)*
- **Atrial Myxoma** ⟶ Tumour plop sound looks like murmur

Heart Failure

Definition:

Inability of the heart to pump blood adequate to tissue needs

Epidemiology: affect 2% of adult population, Affect 10% of those above 75 years old.

Pathophysiology:

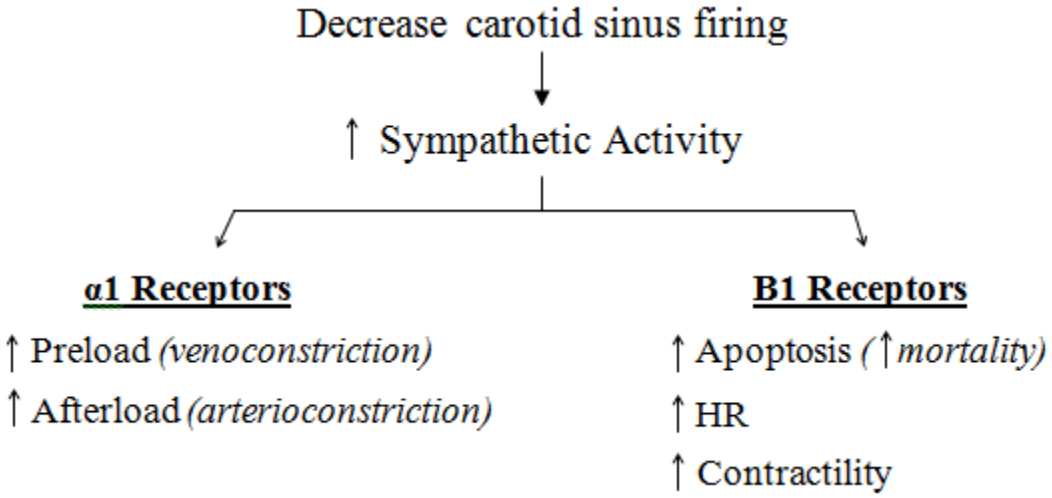
Factors that control cardiac performance are:

1. Preload (volume overload) ⟶ Venous
2. Afterload (pressure overload) ⟶ Arterial
3. Contractility
4. Heart Rate

Change in one of these factors ⟶ Heart Failure

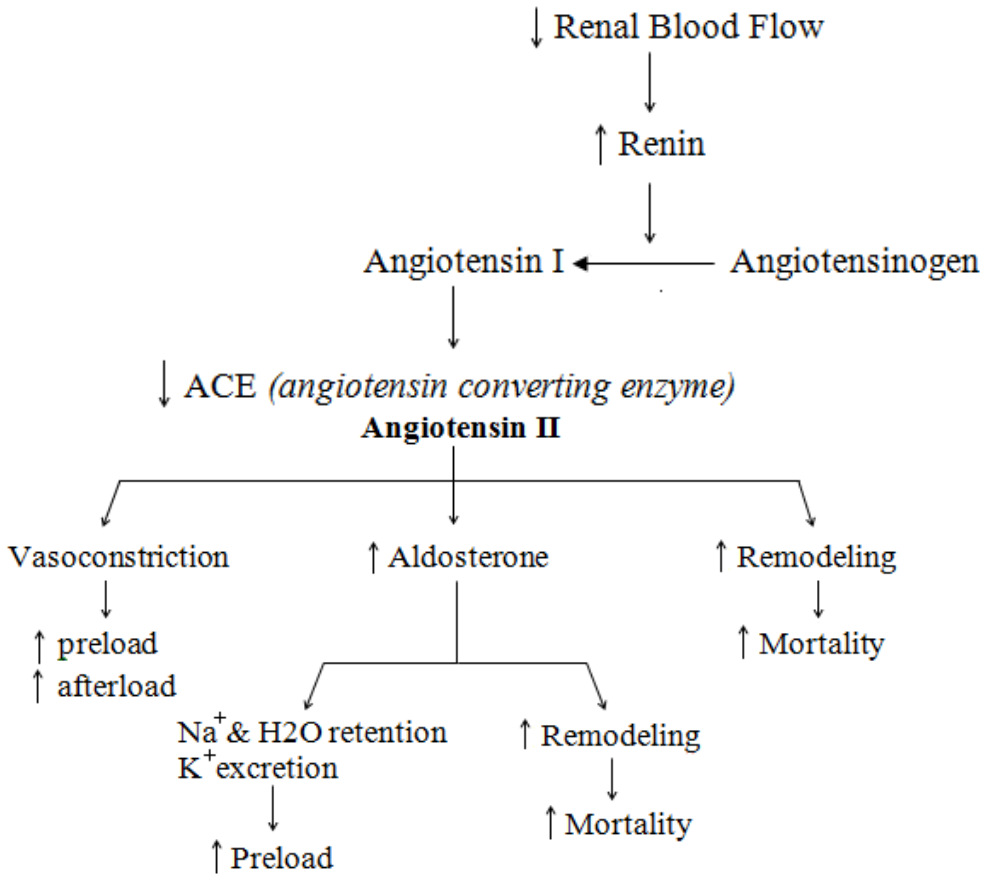
Changes in heart failure are divided as following:

1. **Fast (neural) mechanism:**



N.B: *B-blockers improve survival in Heart Failure because it decrease Apoptosis*

2. **Slow (hormonal) mechanism:**



- Increase in beta natriuretic peptide in response to hypervolemia ➔ diuresis, VD
- Increase ADH ➔ fluid retention

NOTE: ventricular remodeling: it's the change of ventricular muscle size and Configuration (Hypertrophy or dilatation) caused by (catecholamine & Ag II) BUT this hormones by time will lead to apoptosis of cardiac muscle which lead to aggravation of symptoms and increase mortality rate.

So: If given to pateint with heart failure drugs as (ACEI or b-blockers) will decrease mortality rate and improve prognosis.

N.B: Goals of Treatment in Heart Failure:

1. Decrease Preload → Diuretics, Venodilators
2. Decrease Afterload → Arteriodilators
3. Increase Contractility → +ve Inotropic
4. Decrease Mortality → ↓ Remodelling, ↓ Apoptosis

Classification of Heart failure:

1. **Left sided heart failure \ Right sided heart failure**
2. **Systolic heart failure \ Diastolic heart failure**

Diastolic heart failure

- Failure to relax ⟶ ↓ End diastolic volume ↓ COP.
- Normal Ejection Fraction on Echocardiography

Causes include:

1. Aortic stenosis
2. HOCM
3. Hypertension
4. Hypothyroidism
5. Restrictive Cardiomyopathy
6. Cardiac Tamponade

* Digoxin is contraindicated

Treatment ⟶ B-blockers
⟶ Verapamil or Diltiazem

3. **High output \ Low output Heart Failure**

High output heart failure:

- COP is high
- It is caused by increase in tissue demand
- Causes ⟶ Anemia, beri beri
⟶ Thyrotoxicosis, pagets disease
- Digoxin is contraindicated

4. **Acute \ Chronic Heart Failure**

- Acute heart failure (emergency)
- No time for compensation ⟶ Cardiogenic Shock
- Causes include:
    1. Acute MI
    2. Acute MR
    3. Acute AR
    4. Aortic Dissection
    5. Massive Pulmonary Embolism

Causes of Heart failure:

1. **Causes of Left Ventricular Failure**

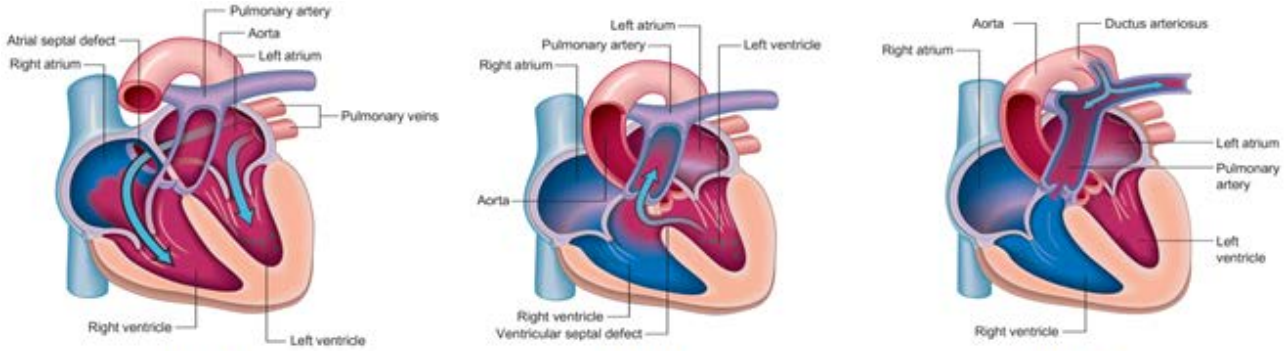
- Most common cause is Ischemic Heart Diseases
- Myocardial Diseases:
    1. MI
    2. HOCM
    3. Myocarditis
    4. Dilated Cardiomyopathy
    5. Alcohol
    6. HOCM
- Endocardial Disease:
    1. Infective Endocarditis
    2. Valvular Heart Diseases (MR, AS, AR)

N.B: *Mitral Stenosis causes Right ventricular failure before left ventricular failure*

- pericardial diseases
- Cardiac Arrhythmia
- Drugs ⟶ Doxorubicin
- Systemic hypertension
- Congenital heart diseases (VSD, PDA, Coarctation of aorta)

2. Causes of Right Ventricular Failure:

- Most common cause is left ventricular failure
- Myocardial Disease:
  1. MI
  2. Myocarditis
  3. Restrictive Cardiomyopathy
  4. Dilated cardiomyopathy
- Pericarditis & Cardiac Tamponade
- Tricuspid Valve Endocarditis
- Tricuspid & Pulmonary Valve Disease
- Pulmonary HTN
- Pulmonary Embolism
- Congenital Heart Diseases:
  1. ASD
  2. VSD
  3. PDA



ASD VSD PDA

NOTE: left side heart failure can cause right side heart failure, but right side failure cannot cause left side failure.

Corepulmonale: Any chronic lung parenchyma or chest wall diseases lead to isolated right ventricular hypertrophy with or without failure.

<u>Clinical features of Heart failure:</u>

1. <u>Symptoms of Left ventricular failure:</u>

- Symptoms of Low COP
  - Fatigue
  - Oliguria
  - Bilateral leg swelling
  - Intermittent Claudication
  - Dizziness (pre-syncope)
  - Syncope

- Symptoms of Pulmonary Congestion
  - Dyspnea
  - Orthopnea
  - Paroxysmal Nocturnal Dyspnea (PND)
  - Hemoptysis
  - Recurrent Chest Infection

2. **<u>Symptoms of Right Ventricular failure:</u>**
   - Symptoms of Low COP *(as left ventricular failure)*
   - Symptoms of Pulmonary Congestion
     - Jaundice
     - Abdominal distension (ascites & gases)
     - Dyspepsia (Borborygmi, belching, premature satiety)
     - Bilateral leg Swelling
     - Right hypochondrial pain

<u>Signs of Heart failure:</u>

- Left ventricular failure & Right ventricular failure have signs in common:
  - Cold Hands
  - Peripheral Cyanosis
  - Decrease pulse volume + Rapid pulse
  - ↑ JVP → systemic congestion (RVF)
    → Volume overload (LVF)
- Bilateral pedal edema
- Pericardium → Weak or impalpable apex
  → S3 gallop
  → Murmur of MR
- Fine crepitation → LVF
- Hepatomegaly & Ascites → RVF

NB. Pulsus alternans: alternating strong & weak beats or pulse (**In advanced stage**) in LVF

<u>Manifestations of advanced HF:</u>

1. Bradycardia without effect of digitalis or b blockers.
2. Hypotension.
3. Pulses alternans.
4. Cheyne stock breathing (periodic breathing occur in advanced severe H.F)
5. Hyponatremia (delutional hyponatremia due to increase ADH secretion).

Precipitating Factors of Heart failure:

1. Abstinence from Drugs
2. Increase Na^+ in diet (common)
3. Chest infection
4. Steroids & NSAIDs
5. Thyrotoxicosis
6. Psychological Stress
7. High dose B-blockers
8. High BP (common)
9. Cardiac Arrhythmias (common)
10. Anemia < 10 g\dl
11. Alcohol (common)

Severity of Heart failure:

NYHA Classification

- Class I HF ⟶ Grad I Dyspnea
- Class II HF ⟶ Grad II Dyspnea
- Class III HF ⟶ Grad III Dyspnea
- Class IV HF ⟶ Grad IV Dyspnea

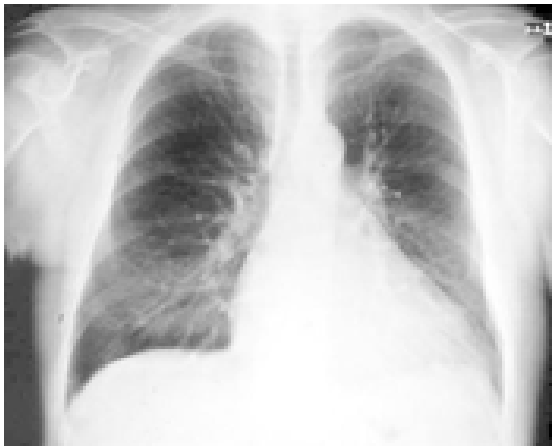
(Class III & IV ⟶ Moderate to Severe HF)

Investigation:

➢ **Routine Investigation:**

- **CBC** ⟶ Anemia (precipitate HF)
  ⟶ Leukopenia (Captopril side effect)
- **RFT**
  - Hypokalemia:
    - Complication of HF
    - Diuretics
    - ↑ Digoxin Toxicity
  - Hyperkalemia:
    - Digoxin
    - Spironolactone
    - Renal Impairment
    - ACEI
  - Hyponatremia:
    - Diuretics
    - Severe HF (bad prognosis)
  - Hypernatremia:
    - Over-Diuresis
      Increased Urea & Creatinine ⟶ ↓ COP ⟶ ↓ Renal perfusion ⟶ Renal impairment
- **LFT**
  - Increased liver enzymes ⟶ Liver congestion
- **INR** ⟶ Warfarin use
  (target INR 2-3 in HF)

➢ **Chest X-Ray:**



- Cardiomegaly
- Bat's Wing Sign
- Kerley-B line
- Pleural Effusion

➢ **ECG:**
- Low Voltage
- Tachycardia

➢ **Echocardiography:**
- Decreased Ejection Fraction
  If < 60% ⟶ Heart Failure
  If < 30% ⟶ moderate to Severe Heart Failure
- Chamber enlargement
- Pulmonary pressure
- Cause of HF

- **Grade I** (EF= 60%) but there is structure heart disease.
- **Grade II** (40-59%).
- **Grade III** (21-39%).
- **Grade IV** (= 20%)

➢ B-Natriuretic Peptide (BNP):
- High BNP ⟶ HF
- Normal BNP ⟶ No HF

The higher the BNP, the higher the Mortality
BNP differentiate between Cardiac & Pulmonary dyspnea

<u>Treatment of Heart failure:</u>

➢ **Non pharmacological treatment:**
- Low salt diet
- Stop smoking
- Low grade exercise
- Vaccination (pneumococcal & H.influenza).
- Supine position
- Education of patient

➢ **Pharmacological treatment:**

1. <u>Diuretics:</u>
- Loop diuretics, Furosemide, Ethacrynic Acid
- To all pt. of HF
- Treat symptoms only
- No effect on mortality
- Decrease preload

- It's better to combine diuretics with ACEIs (to avoid hypokalemia).
- Diuretics are the most effective treatment for symptoms of CHF.
- **Side effects:**

1. Hyponatremia
2. Hypovolemia
3. Hypokalemia
4. Hypomagnesemia
5. Hypocalcemia
6. Hupercalciuria ➔ Stone
7. Hyperglycemia
8. Hyperlipidemia
9. Hyperuricemia
10. Hypersensitivity
11. Ototoxicity &nephrotoxicity

2. <u>Vasodilators:</u>

a. ACEI (arterio and venodilators)

- Lisinopril, Enalapril, Ramipril *(best one)*
- decrease Afterload
- decrease Remodeling ⟶ improve survival
- to all patients
- **S\E** ⟶ Hyperkalemia
  ⟶ Dry cough
  ⟶ Angioedema
- **C\I** ⟶ Pregnancy
  ⟶ Renal artery stenosis

N.B: *Captopril causes loss of taste and neutropenia*

b. Angiotensin receptor blockers

- Losartan, Valsartan, Telmisartan
- Used in case of S\E from ACEI
- Contraindications of both ACEI&ARBS:
  1. Pregnancy &lactation
  2. Hyperkalemia serum K > 5.5.
  3. Bilateral renal artery stenosis or unilateral stenosis with nephrectomy to other kidney.
  4. Advanced renal failure (e.g. creatinine > 3 mg/dL)

c. Nitrates (venodilator) & Hydralazine (arteriodilator)

- Used in case of C\I of ACEI or ARBs
- Improve survival
- Hydralazine S\E ⟶ SLE

3. B-blockers:

- Cardio-selective B-blockers
  - Bisoprolol, Carvidilol (α1\B1 blocker)
  - Metoprolol, Acebutolol, bucindolol

- To all patients (used with causion in class IV)
- Improve survival
- Not used in acute pulmonary edema
- Not used in heart rate < 70\min
- Start with low dose

4. Digoxin (intropics):

- **Used in:**
  1. Class III\IV heart failure
  2. Heart failure & AF
  3. Repeated admission to hospital
- No effect on mortality
- Only symptomatic patients
- Mechanism $\longrightarrow$ Inhibits $Na^+ - K^+ -$ ATPase
- Digoxin also acts on CNS ↑ vagus activity $\longrightarrow$ ↓ conduction through the AVN (delay).
- Pharmacokinetic $\longrightarrow$ half time 38 days
  $\longrightarrow$ Given in loading dose in emergency
- Low therapeutic index *(toxic)*
- Wide volume distribution *(dialysis has no role in treatment of toxicity)*
- C\I in Renal Disease *(use Digitoxin)*
- On ECG: sagging depression of ST segment *(inverted tick sign)*
- **<u>Toxicity:</u>**
  - Early $\longrightarrow$ Abdominal pain, Diarrhea & Vomiting
  - Then $\longrightarrow$ Cardiac arrhythmias (all arrhythmias except prolonged Q-T interval)
  - Eye $\longrightarrow$ Defect in green-yellow colors
  - Convulsion & Coma
  - **Precipitating factors increasing toxicity:**
    (Old age, renal failure, Hypokalemia, Hypomagnesaemia, Hypercalcemia, Thyroid disorders, Drugs: quinidine).
  - **Treatment of toxicity:**
    1. Stop drug
    2. Do ECG
    3. Send Digoxin blood level
    4. Treat arrhythmia
    5. Give Antidote $\longrightarrow$ Digoxin antibodies (Digibind)
- **Contraindications:**

1. Digitals toxicity.
2. Ventricular tachycardia (VT).

3. Partial heart block.
4. Peptic ulcer

- **To avoid toxicity:**
  - Decrease the dose &Drug holiday.
  - Routine estimation of serum level of digitalis (N=0.5-2ng/ml).

5. Spironolactone:

- Potassium Sparing Diuretic *(aldosterone antagonist)*
- Improve survival
- Class III\IV heart failure
- S\E ⟶ Hyperkalemia & Gynecomastia

N.B: *Eplerenone (aldosterone antagonist) not associated with gynecomastia*

6. Anticoagulation:

- Warfarin *(very low ejection fraction)* ⟶↓ Risk of Thrombosis
- INR ⟶ (2-3) ⟶ (target)

7. Cardiac Resynchronization Therapy (CRT):

- **Indications:**
  1. NYHA Class IV
  2. Ejection fraction <30%
  3. Wide QRS in ECG (>140ms)
- CRT increase COP ⟶ Improve symptoms
- Improve survival

Treatment that improve survival in HF

1. ACE inhibitors and (Ag II blockers).
2. Spironolactone especially in (NYHA IV).
3. Beta-blockers.
4. Hydralazine/long-acting nitrates.
5. ICD (Implantable cardiac Defibrillator).
6. Cardiac Resynchronization.

➢ **CARDIAC TRANSPLANTATION**

- End stage cardiac disease, High NYHA functional class
- Failure of maximal medical or other surgical intervention.
- Poor 6 month prognosis & absence of contraindications

  Complications: rejection, infection, malignancy, silent MI

<u>Complication of Heart failure:</u>

1. Hypokalemia &hyperkalemia
2. Hyponatremia
3. Renal Impairment, liver cirrhosis
4. Metabolic Acidosis
5. Bed Rest ⟶ DVT, thromboembolism.

6. Sudden Death
7. Depression
8. Cardiac arrhythmias

Prognosis of Heart failure:

- Bad prognosis (80% die within 6 years)
- Sudden death occurs in up to 50% of patients with heart failure
- cause of death cardiac arrhythmia

Poor prognostic criteria of heart failure:

1. Male + old age + Severe symptom
2. ↑ BNP concentration + Impaired renal function (BUN)
3. Coronary artery disease.
4. ↓ Ejection fraction & ↓ Hypotension.
5. Hypokalemia & hyponatremia

Ischemic Heart Diseases (IHD)

There are two coronary arteries – left & right – originate from the root of ascending aorta.

1. **Left coronary artery:** Passes forward & to the left in the left atrioventricular groove for a short distance & then divides into:
   a. **Anterior descending artery:** passes downward in anterior interventricular groove to the apex & then turns backward to meet posterior *descending artery*.
   b. **Circumflex artery:** runs posteriorly in the left atrioventricular groove to meet the *right coronary*.
2. **Right coronary artery:** runs in right atrioventricular groove to the posterior surface of the heart to meet the *circumflex artery*. posteriorly, it gives the **posterior descending artery** which runs in the posterior interventricular groove to meet the *anterior descending artery*.

Balanced circulation:

- The left coronary artery supplies:
  LA, LV, anterior wall of interventricular septum.
- The right coronary artery supplies:
  RA, RV, posterior wall of interventricular septum.

Left coronary predominance:

The left coronary supplies also: posterior wall of right ventricle.

Right coronary predominance:

The right coronary supplies also: posterior wall of left ventricle
IHD is most common cause of Death in the world

Etiology:

1. Coronary Atherosclerosis (the most common cause – 90%)
2. Vasculitis (polyarteritis Nodosa, Kawasaki Disease, SLE)
3. Anemia
4. Aortic Stenosis
5. HOCM
6. Coronary Vasospasm
7. Cocaine Abuse
8. Small Vessel Disease *(syndrome-x):*
   - Angina with normal angiography
   - DM
   - HTN

- Obesity
- Increase lipids
- Hyperuricemia
- Hyperfibrinogenemia

Pathophysiology of IHD:

- Decrease in myocardial oxygen Supply *(Coronary Atheroma)*
- Increase in myocardial oxygen Demand
    - Increase HR
    - Increase force of Contraction

e.g: Anemia, Thyrotoxicosis, Ventricular hypertrophy

Risk Factors of IHD:

Risk Factors for Atheroma:

Modifiable risk factors:

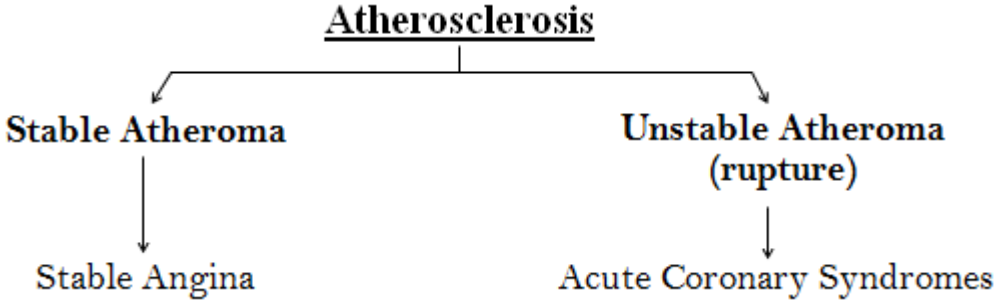
- MD
- HTN
- Hypercholesterolemia
- Obesity
- Sedentary lifestyle
- Hyperfibrinogenemia
- Hyperhomocysteinemia
- Type A personality
- Poor oral hygiene
- Stress
- ↑ CRP

Non Modifiable risk factors:

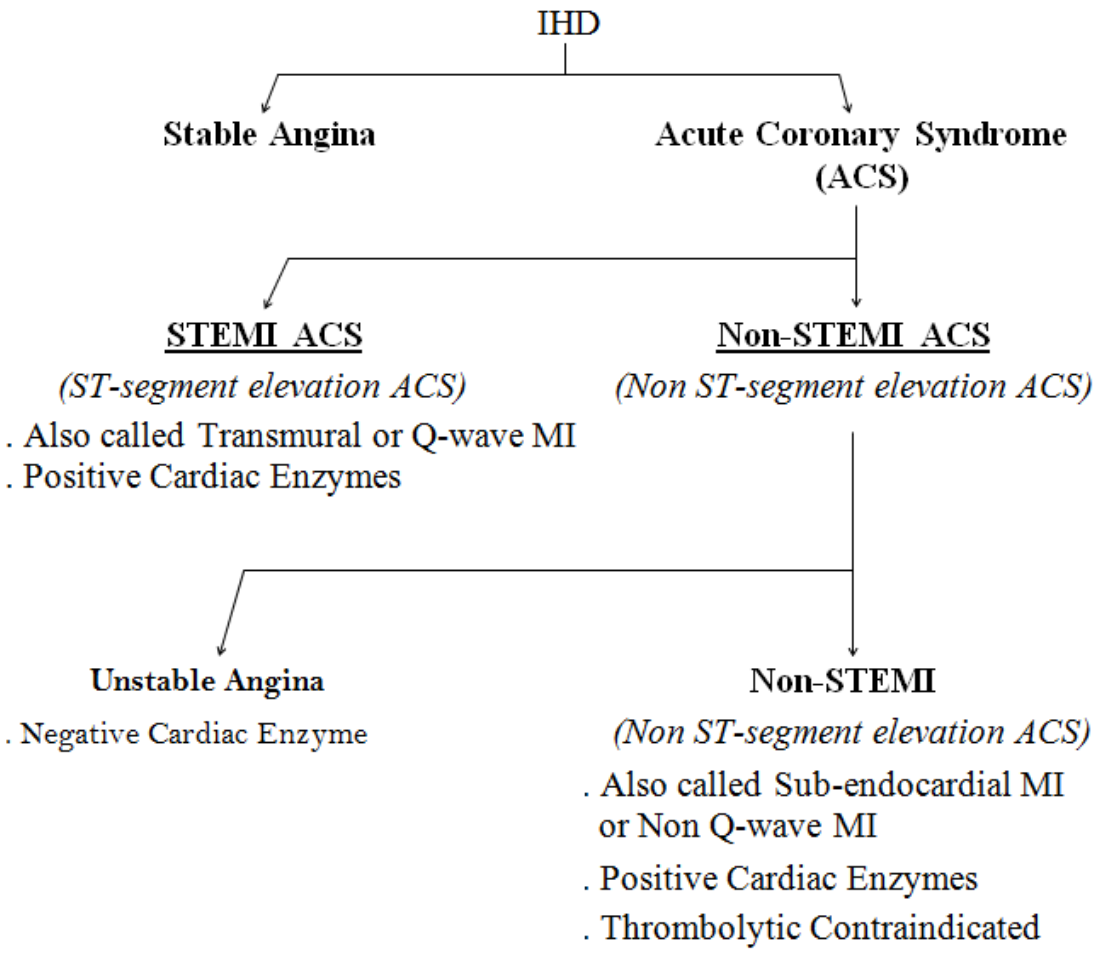
- Age > 40 years
- Sex: M > F, but after menopause M=F
- Family History (first degree relative with MI < 55 years)

Moderate alcohol consumption ↓ risk of IHD, while excessive intake ↑ risk

Pathology:



Classification of IHD:



Stable Angina

It's a clinical syndrome characterized by:

- Chest pain of Ischemic nature
  - Central
  - Poorly localized
  - Burning\Heaviness or Constricting
  - May radiate to: Jaw, Neck, Shoulder, Arm, Forearm & Hand
  - Aggravated by: Exercise, Emotion & Cold exposure
  - Relieved by: Rest & sublingual Nitroglycerine
  - In a risky patient: DM, HTN
- Cardiac Enzymes will be Negative
- It's Not Emergency *(ischemia but no necrosis)*

Some other Types of Stable Angina:

1. **Vasospastic Angina (Variant, Atypical, Prinzmetal)**

- Coronary vasospasm
- Not related to exertion
- Associated with S-T segment elevation in ECG
- B-blockers are contraindicated

- Aspirin will increase the symptoms
- Drug of choice in treatment is Calcium Channel Blockers
  - Diltiazem
  - Verapamil
  - Amlodipine

  Second Choice is Nitrates
  - Short duration of pain
  - Good prognosis. May be associated with Raynaud's Phenomenon

2. **Second Wind Angina:**

- Angina at the beginning of exercise
- Disappear later on with continuing exercise

3. **Decubitus Angina:**

- Angina in supine position
- Indicate severe coronary artery disease

4. **Angina of Lewis(nocturnal angina)**

- Associated with Aortitis
- As syphilis

chest pain in (stable angina)

	Common (classic)	Less common	never
site	**Retrosternal** Often the patient places his clenched hand over the upper Sternum.	**Any site of chest**: ✓ Scapular. ✓ Infraclavicular. ✓ Epigastrium.	Left infra mammary Patient never points with his finger.
character	✓ Compressing. ✓ Constricting.	✓ Heaviness. ✓ Squeezing. ✓ Burning. ✓ Discomfort.	Stitching. Pricking. Stabbing.
Radiation	✓ Left shoulder & inner side of the left arm up to little finger. ✓ Neck, jaw or teeth.	✓ Right shoulder. ✓ Back. ✓ Epigastrium.	Below epigastrium
Duration	✓ Less than 10 or 15 min	✓ More than 15 min	Never >30 min.

Clinical Picture:

Symptoms: *(see before)*

Signs: Usually no signs, but look for:

- **G\E:**
  - Obesity
  - HTN
  - DM

- Signs of Hyperlipidemia eg: Xanthelasms
- Pallor ⟶ Anemia
- Peripheral pulsation
- (Weak ⟶ Atheroma)
- Carotid Bruit *(carotid Atheroma)*

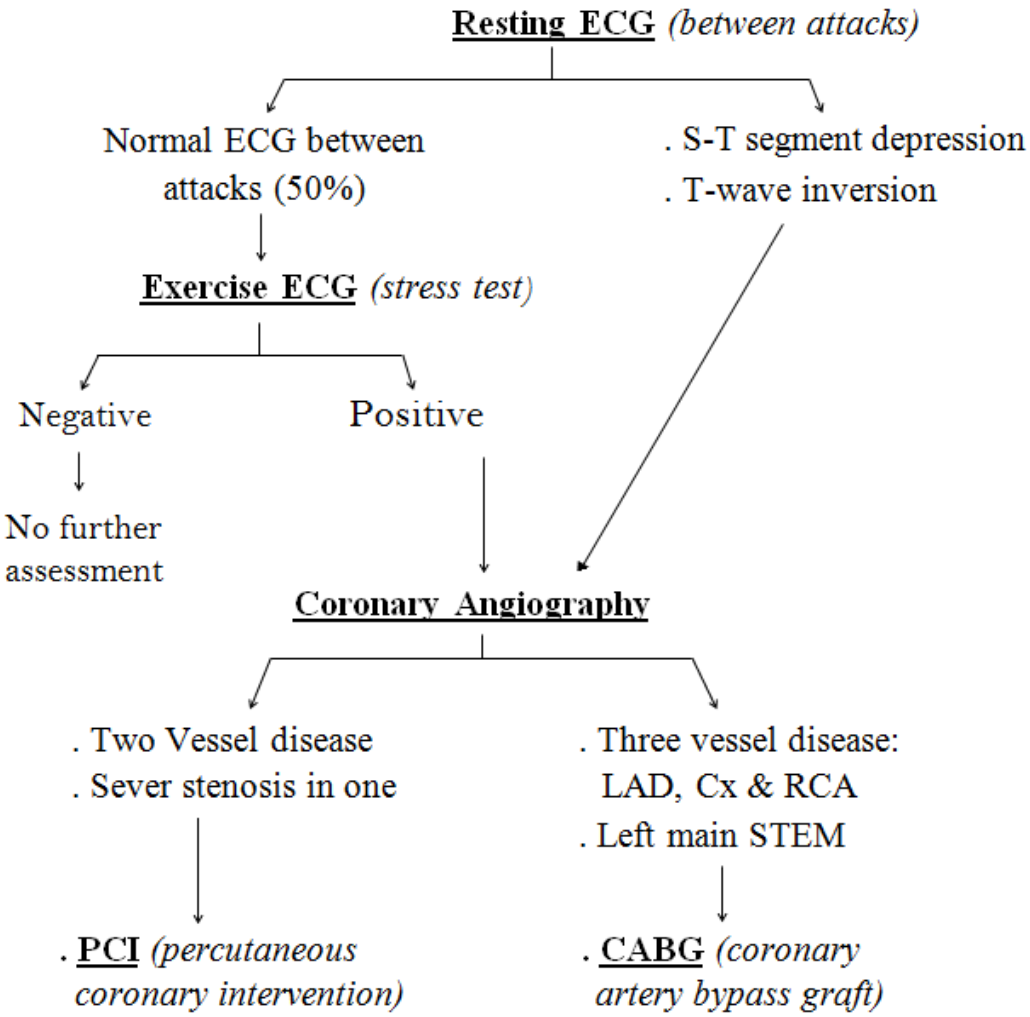
➢ **L\E:**

- Sea gull murmur ⟶ Systolic murmur at Apex (papillary muscle dysfunction (ischemia))
- S4 ⟶ HTN

Investigations:

1. **Blood Investigations:**
   - CBC ⟶ Anemia
     ⟶ ↓ Platelets (Aspirin & Plavix)
     ⟶ Leukopenia
   - Lipid profile:
     - High LDL
     - High TG
     - High VLDL
     - High total Cholesterol
     - Low HDL
   - Blood glucose + HbA1c ⟶ DM
   - LFT ⟶ Statins ⟶ Hepatitis
   - RFT ⟶ Aspirin ⟶ Renal Impairment
2. **Special Investigations:**

   Resting ECG (between attacks)



Exercise ECG:

Indications:

1. Suspected IHD
2. Exercise induced arrhythmias
3. To assess prognosis following myocardial infarction.
4. To assess outcome after coronary revascularization (PCI or CABG).

Contraindications:

1. Recent MI (< 5 days), or Unstable angina
2. Aortic Stenosis
3. Myocarditis\Pericarditis
4. Aortic Dissection
5. Pulmonary Embolism
6. Complete Heart Block
7. Uncontrolled HTN
8. Osteoarthritis
9. Obesity
10. The test should be **stopped immediately**, If the patient develops one of the following:
    1. Chest discomfort
    2. Severe Dyspnea
    3. Dizziness

4. Lowering of Systolic BP> 10mmHg
5. ventricular arrhythmias
6. ST segment depression > 2 mm

Sensitivity: 75% (false positive especially in patients with (left ventricular hypertrophy (strain), digoxin therapy, LBBB, WPW).

Alternative to exercise ECG:

- Dobutamine Echo (stress Echo)
- Thallium scan

Coronary angiography:

Injection of contrast: Fluoroscope showing coronary arteries

N.B: *RFT is important for contrast nephropathy*

Treatment:

- **Lifestyle modification:**
  - Stop smoking
  - Reduce weight
  - Exercise (30 minutes 4d/week.)
  - No stress
  - Control DM (FBS <120, HA1c <8), HTN (<135/85mmHg) & high lipids (LDL<100, HDL >35).
- **Medical:**
  1. **Antiplatelets:**
     - Low dose Aspirin 75mg
     - Clopidogrel 75mg
     - Improve survival
  2. **Statins:**
     - Simvastatin
     - Atorvastatin
  3. **B-blockers:**
     - Decrease HR, Force of contraction & Decrease Demand
     - Atenolol, Bisoprolol, Carvedilol
  4. **Nitrates:**
     - Oral $\longrightarrow$ ISMN, ISDN
     - Sublingual or transdermal patch $\longrightarrow$ Nitroglycerine *(Angised)*
  5. **Calcium Channel Blockers:**
     - Used if there is contraindication to B-blockers
     - Amlodipine
     - Deltiazem
     - Also used in Vasospastic Angina

6. **Potassium Channel Blocker:**
   - Nicorandil
   - Used if not controlled by other Drugs
7. **Ivabradine:**
   - Inhibits peacemaker *(SA node)*
   - Decrease HR *(alternative to B-blockers)*
8. **Ranolazine:**
   - Decrease Na^+ ⟶ current Decrease HR
     ⟶ Decrease Contractility

➢ **PCI (Percutaneous Coronary Intervention)**

Indications:

1. Poor response to medical treatment
2. Significant stenosis (>70%) in one or two coronary vessels
3. Post-CABG angina

Procedures:

1. **Stent Insertion:**
   - Best type is Drug-eluting stent
   - Clopidogrel for 12 month to reduce risk of stent thrombosis
   - **Complication**: Stent Thrombosis ⟶ MI
     Cholesterol embolism
2. Coronary Balloon Dilation

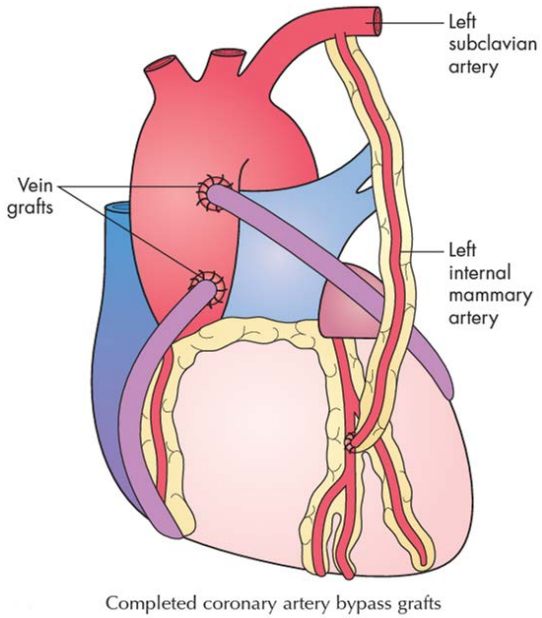
➢ **Coronary artery Bypass Graft (CABG)**

Indications:

1. Three Vessel Diseases (LMS, LAD, RCC)
2. Left main Stem Disease
3. Post-MI (failed PCI)
4. Angina unresponsive to drugs & PCI

Procedure:

- Graft from small saphenous vein (closes at 10 years) *(Aspirin prevent closure)*
- Graft from internal mammary artery *(Lives longer, but causes chest wall numbness)*



Completed coronary artery bypass grafts

<u>Complications:</u>

1. Post-CABG Angina
2. Dressler Syndrome *(2-3 weeks after)*
3. Intraoperative mortality is low

4. Continue Aspirin for life

Syndrome X:

- Typical angina on effort + ischemic changes on stress ECG testing + angiographically normal coronary artery, etiology unknown.
- The carries a good prognosis and respond to treatment with anti-anginal therapy.

Acute Coronary Syndrome (ACS)

Definition:

Unstable Angina with evolving MI

Classification:

1. STEMI ACS (ST-segment elevation ACS)
2. Non-STEMI ACS (non ST-segment elevation ACS)

   Divided into:

   1. Unstable Angina (-Ve Cardiac Enzymes)
   2. Non-STEMI (+Ve Cardiac Enzymes)

	Stable angina	Unstable angina	NSTEMI	STEMI
Mechanism	Stenosis of> 75% of the supplying vessel	Stenosis of> 90% of the supplying vessel	Near total Occlusion of the supplying vessel➡ Subendocardial infarction	Total Occlusion for >30min of the supplying vessel➡ Transmural infarction
C/P	See before	See before	See before	See before
ECG	'Resting is normal Exercise is abnormal	ST segment depression & /or New T wave inversion No Q-wave		ST segment elevation Q-wave
Cardiac enzymes.	No elevation	No elevation after 12 hours enzymes	Elevation of cardiac enzyme	
Treatment	Rx Risk factors Aspirin Nitrate B-blocker/CCB	Rx Risk factors Aspirin + Clapidogrel Unfractionated or LMW heparin Nitrates blockers or CCB Do not administer Thrombolytic therapy		Rx Risk factors Aspirin + Clapidogrel Unfractionated or LMW heparin Nitrates blockers or CCB Thrombolytic therapy

Diagnosis of ACS:

Diagnosis of MI by at least (2) of the following:

1. Chest pain persisting> 30 min.
2. Typical ECG finding (ischemic changes in 2 successive leads or more)
3. Elevated cardiac enzyme.

1. **Patient Symptoms:**

   Ischemic chest pain not related to exercise, not relieved by sublingual Nitroglycerine or rest & may be associated with:

   - Angor animi(sensation of coming death)
   - Sweating
   - Vomiting

   <u>Unstable Angina defined as:</u>

   Angina of increasing severity, duration & frequency *(Crescendo Angina)*

   MI can be without chest pain (silent MI) in:

   - Elderly
   - Diabetics
   - Autonomic neuropathy
   - Heart transplantation

2. **ECG:**

   Role of ECG in Acute Coronary Syndrome:

   1. Classification into STEMI\non-STEMI (new LBBB indicates STEMI)
   2. Determination of the age of MI:

      Minutes (Hyper acute)

      Hyper acute T-wave

      Hours (<12 hours) (Acute)

      ST-segment elevation

      > 12 hours (Passed MI)

      Pathological Q-wave

      Days (Recent MI)

      Pathological Q-wave + T-wave inversion

      Weeks & Months or years (Old MI)

      Only pathological Q-wave

   3. Site of MI:

      - Circumflex artery

        Lateral wall MI $\longrightarrow$ lead I, aVL, V5, V6

      - Left main stem

        Anterior wall MI $\longrightarrow$ V1, V2, V3, V4, V5, V6+I, aVL

      - LAD

        Septal wall MI $\longrightarrow$ V2, V3, V4

      - Right coronary artery

        Inferior wall MI $\longrightarrow$ lead II, III, aVF

- Right coronary artery (posterior descending artery)
  Posterior wall MI ⟶ - Tall R-wave in V1
  - ST-segment depression in V1, V2
  - ST-segment elevation in V7, V8, V9 *(posterior leads)*

NOTE: *In Inferior (MI) right sided chest leads should be-put-to look for right Ventricular infarction*

4. Complications of MI:
   a. Heart Block ⟶ inferior wall MI *(right coronary artery AV-nodal artery)*
   b. Ventricular Arrhythmias **(VF its most common cause of death in first hours in patient with MI)**
   c. Ventricular Aneurysms *(persistent ST-segment elevation)*
   d. Pericarditis *(ST-segment elevations)*
   e. Re-infarction *(ST-segment elevations)*
5. Follow up of treatment:
   ST-segment should decrease by >50% of the original ECG after thrombolytics in 1 hour.

3. Cardiac Enzymes:

Increase after Acute MI

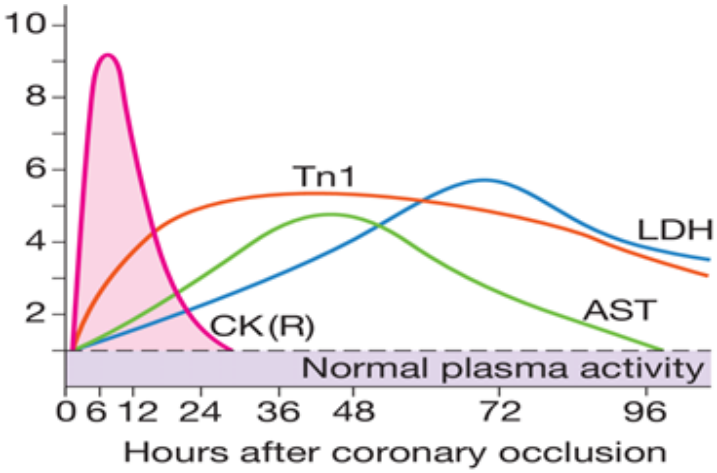
a. Creatinine phosphokinase (CK-MB):
   - Selective to Heart
   - Increases after 4-6 hours
   - Peak at 24 hours
   - Decreases at 48 hours (back to normal)
   - Can be increased in other non-cardiac diseases as:
     - Myositis
     - Pulmonary Embolism
     - Renal Failure
     - Myocarditis
   - Used in diagnosis of Re-infarction

b. Troponin T & I:
   - More sensitive than CK-MB
   - Increases after 4-6 hours
   - Peak at 24 hours
   - Decreases after 2 weeks
   - Not used to diagnose Re-infarction

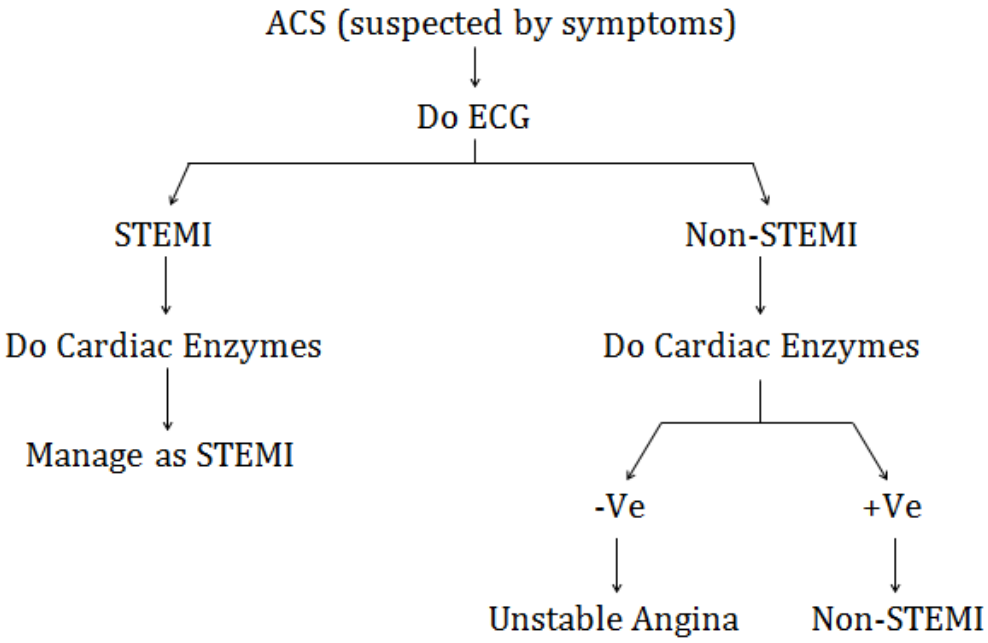
- Highly specific to heart
- Used for Risk Stratification, if +Ve ⟶ high risk MI
- Can be high in:
  - Renal Failure
  - Pulmonary Embolism
  - Myocarditis

c. Myoglobin:
   - Earliest cardiac enzyme to rise

d. LDH, AST:
   - Less specific to heart
   - Can be high in:
     - Liver Disease (AST)
     - Hemolytic Anemia (LDH)



Management of Acute Coronary Syndrome:



a. Pre hospital management:
   1. O2 by mask *(O2 Saturation >94%)*
   2. S\L Nitroglycerine
   3. Crushed Aspirin 300mg
   4. I.V Metoclopramide *(vomiting)*
   5. I.V Morphine 2.5 *(pain\anxiety)*
   6. Send by Ambulance

(Remember Minutes Means Muscles)

Note: *Avoid IM injections because poor skeletal muscle perfusion delays onset of action & if thrombolytic therapy will be given.*

b. Hospital Management:

STEMI

Do ECG & Attach to monitor

↓

O2 by Mask

↓

. I.V Access *(send investigations)*
. Cardiac enzymes
. Complete blood count (leukocytosis)
. Coagulation profile
. RFT
. LFT

↓

Brief clinical assessment

*** History** ⟶ Contraindications of thrombolytics

*** Examination:**

- Pulse ⟶ Arrhythmias\shock\bradycardia (inferior wall mi)
- B.P ⟶ Both arms *(aortic dissection) (C\I of thrombolytics)*
- JVP ⟶ Tamponade\Right Ventricular Infarction
- Murmurs ⟶ MR\VSD (pansystolic murmer)
- Signs of Heart Failure

NB: Chest X-Rays:

- CXR may give clues to an alternative diagnosis aortic dissection or pneumothorax.
- CXR may show complications of (MI) such as heart failure.
- Do not wait CXR for thrombolysis

Echocardiography:

- Ventricular wall motion abnormalities.
- Complications: MR, myocardial aneurysm
- **Abnormalities:** of wall motion are usually presents, But acute STEMI cannot be distinguished from an old myocardial scar or from acute severe ischemia.

- Useful when the ECG is not diagnostic of STEMI and it can HELP in management decisions, such as IF patient should receive reperfusion therapy (e.g. fibrinolysis or percutaneous coronary intervention (PCI).

Start Anti-Ischemic therapy

1. Nitrate
2. B-blockers
3. ACEI
4. Statins
5. Aspirin
6. Plavix (Clopidogrel)
7. LMWH (heparin)

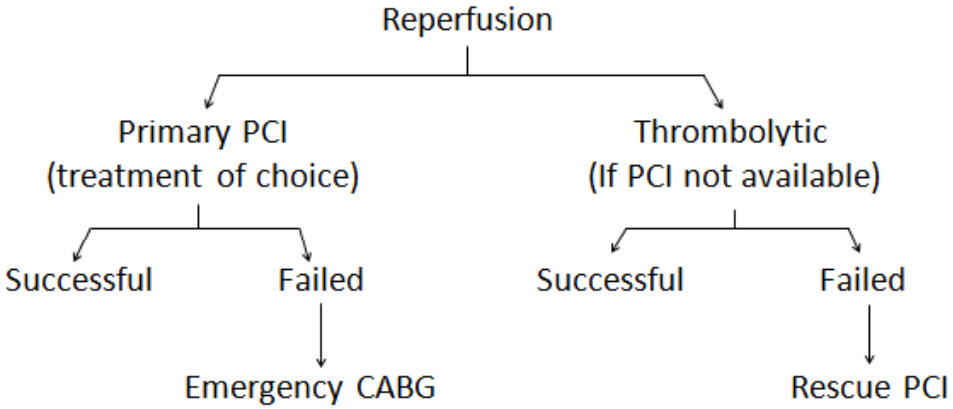
Note: *Do not use nitrates in patient with recent use of phosphodiesterase inhibitors for erectile dysfunction (sildenafil or tadalafil).*

Subcutaneous Heparin: ↓ risk of reinfarction after successful thrombolysis and reduce the risk of thromboembolic complications.

Beta- blockers: (↓ myocardial O2 consumption relive pain), limit infarct size, and decrease arrhythmias so it reduces mortality.

ACEI: reduce Mortality in patients following acute MI and should be prescribed within 24hours of hospitalization for person with (STEMI)

➢ **Reperfusion Treatment:**



➢ **Primary PCI:**

- Treatment of choice
- Patient should be given either:
  - Abciximab
  - Tirofiban
  - Eptifibatide

} (Glycoprotein IIb\IIIa inhibitors)

Thrombolytics (fibrinolytics):

Clinical benefit:

1. Coronary thrombolysis restores coronary patency.

2. Preserves left ventricular function
3. Improves survival by decreasing hospital mortality of myocardial infarction (25%-50%)

Note: *Successful thrombolysis leads to reperfusion with relief of pain, resolution of acute ST elevation.*

- The earlier that thrombolytic therapy is given after the onset of chest pain, the greater the benefit (thrombolytic therapy is beneficial up to 6 hours but may be given for up to 12hours).
- Two types:
    1. Streptokinase
    2. Tissue plasminogen activator (r-TPA)

    Mechanism of action:

    Dissolve thrombus by activation of plasminogen $\longrightarrow$ plasmin

    Streptokinase:
    - Antigenic (leading to hypersensitivity)
    - Higher risk of bleeding

    r-TPA: (Alteplase \ Tenecteplase \ Reteplase)
    - Non-antigenic
    - Lower risk of bleeding
    - Expensive

(Both given IV infusion), Side effects of both: Bleeding & Reperfusion, Arrhythmias.

Note: *(t-PA) have better survival rate than Streptokinase (but with higher risk of intracerebral bleeding.*

Criteria for Thrombolysis use in Acute MI

Indications:

1. Chest pain consistent with Acute MI.
2. ST-elevation > l mm in limb leads or >2mm in precordial leads, in at 2 contiguous leads or new LBBB.
3. < 6 hours from onset of symptoms up to 12 hours if pain persists)
4. Patient is younger than 75 years (greater risk of hemorrhage)

Contraindications:

- **Absolute:**
    1. H\O brain hemorrhage
    2. H\O Ischemic stroke < 3 months
    3. A-V malformation
    4. Brain tumor
    5. Recent head trauma < 2 weeks

6. Major internal bleeding
7. Aortic Dissection (do CXR if suspected)
8. Pericarditis
9. Myocarditis
10. Previous allergy from streptokinase

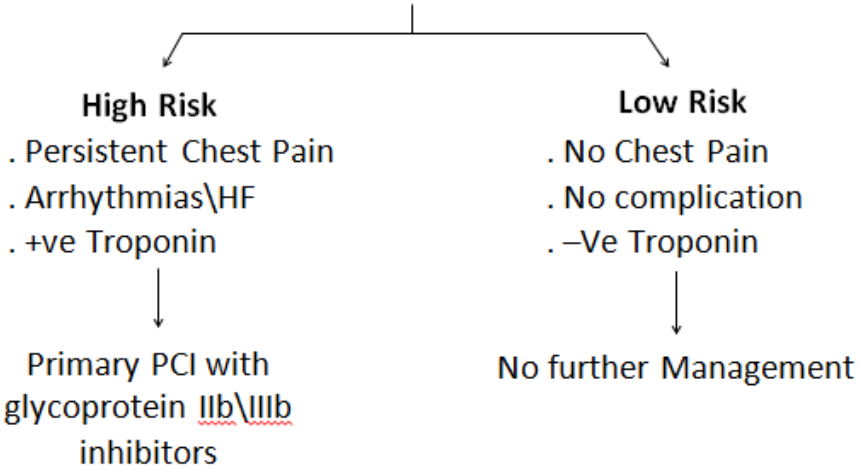
- **Relative:**
  1. Warfarin use
  2. Pregnancy
  3. Prolonged resuscitation (>10 min)
  4. Bleeding tendency
  5. Uncontrolled HTN (>185\110)
  6. Renal \ Liver diseases
  7. Arterial puncture in non-compressible site
  8. Active proliferative diabetic retinopathy

N.B: *Heparin (LMW) given after r-TPA, but not after streptokinase*

Non-STEMI

Anti-Ischemic treatment:

Nitrate \ B-blockers \ Statins \ Aspirin \ Clopidogrel \ LMW heparin



Further management of MI:

1. Echocardiography (Assess Ventricular EF)
2. Exercise ECG (after 5 days)
3. Angiography
4. Secondary prevention (control risk factors)
5. Full anti-Ischemic treatment
6. Hospital stay 7 days

Cause of death:

Early: arrhythmia especially ventricular type.

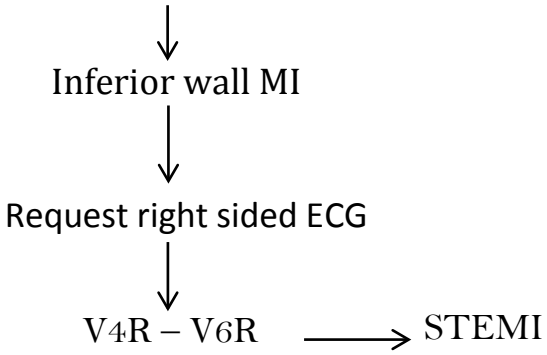
Late: heart failure.

Return to normal life:

- Mobilization in 2 days in absence of HF.
- The usual duration of hospitalization for uncomplicated STEMI is about 5 days.
- during the first 1-2 weeks pt. should be encouraged to activity by walking inside house and-out doors in good weather, Normal sexual activity may be resumed.
- Most patients will be able to return to work within 2-4 weeks.
- Car driving after 4 weeks.

Management of Right Ventricular Infarction:

- Right Ventricular Infarction will lead to ⟶ shock + high JVP + Kussmaul sign (D\D cardiac tamponade)
- Echo will differentiate them
- Nitrates is contraindication
- ACEI is contraindication
- Diuretics is contraindication
- Give Colloids (IVF)
- PCI or thrombolysis
- ECG in right ventricular infarction



- high mortality

Complications of MI:

a. Mechanical Complications:

1. VSD *(2-7 days)* ⟶ New pan-systolic murmur, Shock
2. Acute Mitral Regurgitation (2-10 days)
   - Rupture of papillary muscles
   - New systolic murmur
   - Shock
   - Treat by surgery
3. Early pericarditis (2-3 days)
4. Re-infarction

5. Ventricular wall rupture
6. Left ventricular Aneurysm
   - Late complications (4-6 weeks)
7. Dressler syndrome (2-3 weeks)
   - Pericarditis
8. Cardiogenic Shock
9. Neurogenic shock(severe pain)
10. Left and right ventricular failure.

b. Arrhythmias:

Highest risk in first 2 hours

1. Ventricular arrhythmias
   - Ectopic
   - Tachycardia
   - Fibrillations
   - Can be fatal *(sudden death)*
2. Heart block Inferior wall MI
3. AF\SVT\Flutter
4. Reperfusion arrhythmias

c. Other Complications of MI:

1. Fever
2. Post MI angina
3. Frozen shoulder *(4-6 weeks)*

Bad prognostic signs-in STEMI: Hypotension, Marked LVF, Extensive ECG changes, LBBB or A-V block.

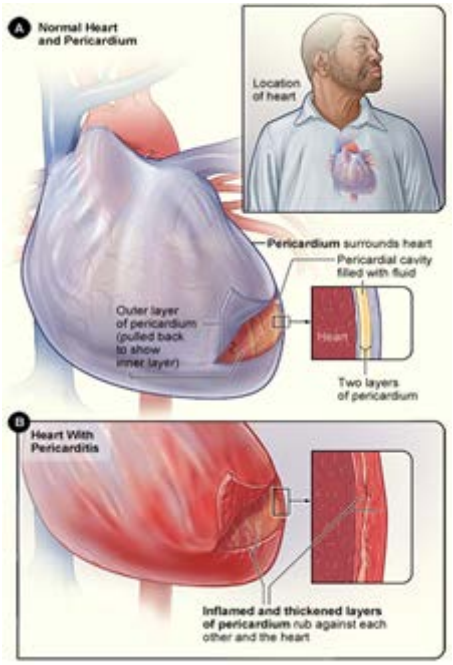
- Worse in anterior MI than inferior infarcts, High enzyme

<u>MI in young people:</u>

1. Cocaine use Coronary $\longrightarrow$ Vasospasm

   Treatment:
   - I.V Diazepam
   - Nitroglycerine
   - No Thrombolytics
   - No B-blockers
2. Vasculitis
   - SLE
   - Kawasaki Disease
   - Polyarteritis Nodosa
3. Familial Hyperlipidemia

Acute pericarditis



Acute inflammation of pericardium

Causes:

- Idiopathic
- Viral infection *(most common)*
  - Coxsackie-virus, EBV
- Bacterial
  - Mycoplasma, Staph, Strepto
- Fungi
- Autoimmune (SLE\RA)
- Metabolic (Uremia)
- Drugs (procainamide, hydralazine)
- Dressler syndrome
- Radiations

Clinical features:

- Chest pain *(pleuritic):* Stabbing, increased by inspiration & reliever by pending forward & NSAIDs
- Fever
- Symptoms of cause as:
  - Fever & URTI $\longrightarrow$ Viral infection
  - Malar rash $\longrightarrow$ SLE
  - MI $\longrightarrow$ Dressler syndrome
- Pericardial friction rub
- Normal S1 & S2
- Normal JVP
- Normal B.P

Investigations:

a. **ECG:**

Diffuse S-T segment elevation (concave upward) present in all leads except (AVR& V1) & P-R segment depression in all leads

	st segment shape	evoluton of st egment
Pericarditis	Concave upwards all leads except AVR &V1	ST remains elevated for days after ST returns to baseline, T waves invert.
Acute MI	Convex upward ST elevation over infracted region.	T waves invert in hours while ST still elevated, with pathological Q wave later on.

b. Echo:

Small amount of pericardial effusion

Treatment: it is self limiting in 3-10 days

1. NSAIDs (Aspirin – Indomethacin)
2. Steroids (Prednisolone) if no response to NSAIDs
3. Colchicine in resistant cases

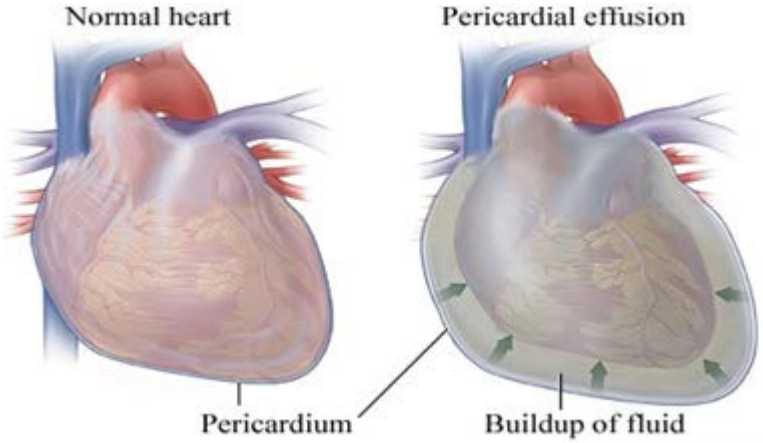
N.B: *Steroids may increase risk of recurrence*

Complication

1. Pericardial Tamponade: due to large effusions.
2. Chronically Inflammations: pericardial thickening & reduced pericardial compliance ⟶ Pericardial constriction.

Pericardial Effusion:

Accumulation of fluid in pericardial space without hemodynamic instability (no drop in BP)



Causes:

1. Can be complication of acute pericarditis, so all causes of acute pericarditis ⟶ pericardial effusion
2. Transudate ⟶ HF, Hypoalbuminemia
3. Exudate ⟶ Trauma, Infection
4. Fibrinous *(serosanguinous)* ⟶ Malignancy
5. Hemorrhagic ⟶ Warfarin overdose

Clinical features:

1. Dyspnea
2. High JVP *(prominent X & Y descent)*
3. Muffled heart sounds

4. Ewart's sign ⟶ Bronchial breathing at the left base of the lung *(large effusion compressing the lower lobe of lung)*

Investigations:

a. CXR:

- Enlarged heart

b. ECG:

- Low voltage ECG
- Electrical alternans

c. Echocardiography:

- increase fluid in pericardial space

Treatment:

Pericardiocentesis technique

Therapeutic & diagnostic *(send fluid for culture\biochemistry)*

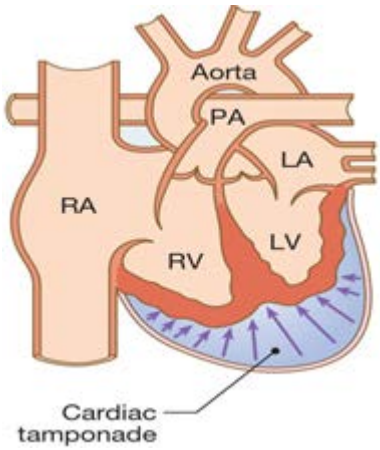
Cardiac Tamponade

Rapid accumulation of fluid in pericardial space ⟶ increase intra-pericardial pressure ⟶ decrease COP

Pathophysiology

Normally, the pericardial space contains about 50 mL of fluid.

Intrapericardial pressure = Intrathoracic pressure, When additional fluid enters the pericardial space, Intrapericardial pressure rises.



If fluid accumulates rapidly:

Tamponade occurs with fluid accumulation of200-250ml.

If the fluid accumulates gradually

The pericardium may contain liters with only small rise in pressure. (Time to compensation)

In diastole ↑ Intrapericardial pressure ⟶ elevation of diastolic pressure in all Cardiac chambers (equilibration of pressures) lead to ↑ ventricular diastolic Pressures impair ventricular filling ⟶ ↑ cardiac output & (JVP).

Causes:

- As pericarditis & pericardial effusion

  +

- Aortic Dissection, MI

- Post-cardiac procedure
- Warfarin overdose

Clinical features:

1. Dyspnea
2. Low pulse volume
3. High JVP (+Ve Kussmaul Sign)
4. **Pulsus paradoxicus** (exaggeration of normal dropping of systolic blood pressure during inspiration (>10mmgH) this due to decrease in LV volume during inspiration 2ry to decreasing pulmonary veins return).
5. Muffled S1& S2

Beck's Triad:

- Low BP
- High JVP
- Muffled heart sounds

Investigations:

- **CXR** ⟶ Flask shaped heart
- **ECG** ⟶ electrical alternans *(Alternating size of QRS complex due to alternation of heart)* + low voltage ECG
- **Echocardiography:**
  - Rim of fluid around heart (> 2cm)
  - Diastolic collapse of right atrium & right ventricle

Treatment:

- Pericardiocentesis

Constrictive pericarditis:

Rigid calcified pericardium impair cardiac filling & elevation of Systemic and pulmonary venous pressure and decreased cardiac output.

Causes: Idiopathic, TB, ureamia.

Clinical features: (symptoms of RVF)

1. High JVP *(Kussmual sign +Ve),* prominent x and y descent,
2. Pericardial knock (early, relatively high-pitched diastolic sound known as (Pericardial knock), this sound occurs early in diastole as a result of the rapid cessation of ventricular filing as the pericardium stretched to its limits.)
3. weak S1 & S2
4. Hepatomegaly

5. Ascites
6. Bilateral leg edema
7. Pulsus paradoxicus: - is present in 1/3 of patients.

Investigations:

- CXR $\longrightarrow$ Calcified pericardium (no increased heart shadow)
- Echo $\longrightarrow$ Diastolic collapse of right ventricle & atrium
- Cardiac MRI $\longrightarrow$ Sensitive
- Cardiac catheterization *(to differentiate from restrictive cardiomyopathy)*

Treatment:

Pericardiotomy *(surgical)*

Cardiomyopathy

Definition:

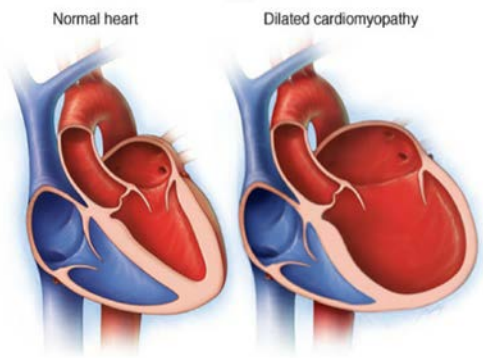
Primary disease of cardiac muscle & not secondary to ischemic, congenital or valvular heart diseases.

Types:

1. **Dilated Cardiomyopathy:**

Dilatation of both right & left ventricle ⟶ congested heart failure

Causes:



1. Idiopathic
2. Familial X-linked, autosomal dominant
3. Alcohol,Doxorubicin, cyclophosphamide
4. Hemochromatosis
5. HTN
6. Autoimmune(SLE)
7. Viral infection
8. Peripartal or postpartal cardiomyopathy
9. Thyrotoxicosis,acromegalym,DM,myxedema
10. Muscular dystrophies (Duchene)
11. Metabolic: Beriberi (Thiamin deficiency), glycogen storage diseases.

Clinical features:

- Symptoms of Heart Failure *(biventricular)*
- Features of the Cause
- **Signs of Heart failure:**
  - Muffled heart sounds
  - S3
  - Murmur (MR)
  - High JVP
  - Weak apex
  - Weak pulse
  - AF

Investigations:

- Blood ⟶ Plasma BNP High
  ⟶ Serum Na^+ is low in severe cases *(Poor prognosis)*
- ECG ⟶ Low Voltage
  ⟶ T-wave inversion
- CXR ⟶ Cardiomegaly
  ⟶ Pulmonary edema

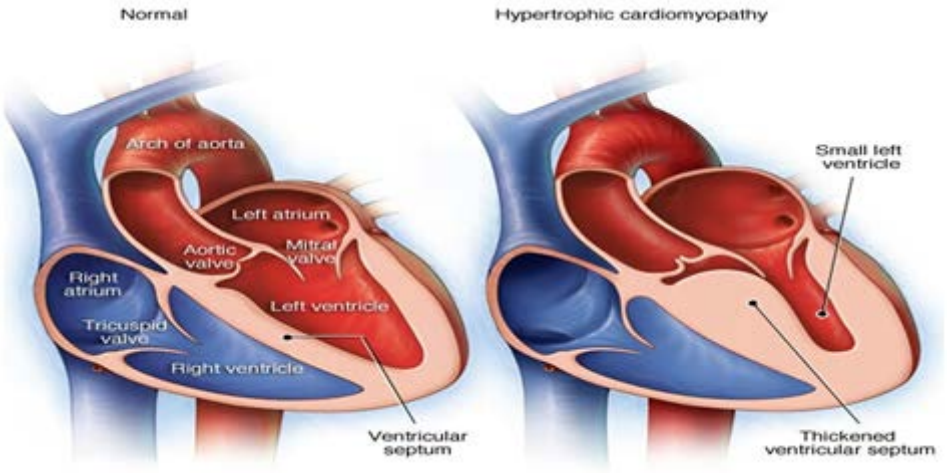
- Echo
  - Diagnostic
  - Low EF
  - Global Hypokinesia
  - Intra-cardiac Thrombus
  - MR (dilated mitral valve ring)

Treatment:

- Treatment of heart failure (seen before)
- Control of the cause
- Cardiac transplantation remains the standard treatment in refractory cases

Prognosis:

- Postpartum Cardiomyopathy can be reversible
- Overall mortality about 40% in 2 years



2. **Hypertrophic Obstructive Cardiomyopathy (HOCM):**

Pathology:

- Hypertrophy of septum (IVS) called ASH *(Asymmetrical Septal Hypertrophy)*
- Left ventricular outlet obstruction
- Pressure gradient between left ventricle & aorta
- Hypertrophy of ventricular wall
- SAM (systolic anterior motion of mitral valve leaflet)
- diastolic heart failure
- Usually affects the left ventricle.

Epidemiology:

- Sex: more common in males than in females.
- Age: The most common presentation is in the third decade of life.
- The most common form of cardiomyopathy.

- The most common cause of sudden death in young athletes (because of ventricular Arrhythmias).
- Autosomal dominant disorder in 60% of cases.

Complications:

1. Sudden death *(ventricular fibrillation)*
2. Diastolic heart failure
3. Left ventricular outlet obstruction → COP → syncope
4. Angina (increase myocardial oxygen demand).

Causes:

1. Familial → Autosomal Dominant
   → Mutations in myosin & tropomyosin
2. Sporadic in 50% of cases

Clinical features:

Symptoms:

1. Angina
2. Exertional syncope
3. Dyspnea
4. Palpitation
5. Sudden death

Signs:

1. Jerky pulse
2. Jerky apex beat (results from a forceful left atrial contraction.)
3. Ejection systolic murmur at aortic area → sternal notch (↑ murmur with Standing & ValSava maneuver)
4. S4

Investigations:

ECG:

- Q waves in V1, V 2 & V3
- LVH
- Ischemia (T-wave inversion)

Echo:

- ASH *(asymmetric septal hypertrophy)*
- SAM *(atrial septal motion)*
- Diastolic dysfunction
- Left ventricular gradient

Exercise ECG

Drop of BP by >10mmHg *(systolic)* with exercise

- Myocardial ischemia

- Exercise induced Arrhythmia

Cardiac catheterization:

- Coronary anatomy
- Hemodynamic measurement

Treatment:

- General measures → Family screening
  → Patient education
- If no symptoms → No treatment needed
- If there is symptoms:
  - B-blockers or Verapamil for diastolic failure
  - Amiodarone for ventricular arrhythmias
  - Implantable Cardioverter Defibrillator for those at high risk of sudden death as:
    1. History of syncope
    2. Family H\O sudden death
    3. H\O ventricular tachycardia
    4. Left ventricular wall thickness >3cm
    5. Drop in BP during exercise
    6. If there is genetic mutation

Surgical Management:

- Alcohol Myomectomy *(ablation)*
- Surgical Myomectomy

Association with HOCM:

- Friedrich's Ataxia
- Fabry's Disease
- WPW syndrome
- Myotonia dystrophica

3. Restrictive Cardiomyopathy

Pathology:

- Diastolic dysfunction because of deposition of certain substance in myocardium
- It affects mainly right ventricle → right ventricular failure

Causes:

1. Amyloidosis
2. Hemochromatosis
3. Loffler's syndrome
4. Sarcoidosis
5. Scleroderma

6. Endomyocardial fibroelastosis
7. Idiopathic

Clinical features:

1. Dyspnea
2. High JVP (prominent X & Y descent)
3. Ascites
4. Liver congestion edema

Differential Diagnosis:

- Constrictive Pericarditis

Investigations:

- Echo $\longrightarrow$ Diastolic dysfunction of right ventricle
- Cardiac catheterization is the investigation of choice to differentiate it from constrictive pericarditis (square root sign)

Treatment:

1. Treat the cause
2. Treat of heart failure

Acute Myocarditis

Acute inflammation of Myocardium

Causes:

1. Idiopathic (50%)
2. Viral (Coxsackie or adenoviral infection.)
3. Bacterial (clostridia, T.B mycoplasma)
4. Protozoa (Chagas disease)
5. Drugs (Cyclophosphamide, Herceptin, Phenytoin, Adriamycin)
6. Toxins

Clinical features:

1. Fatigue
2. Dyspnea
3. Chest pain
4. Fever
5. Palpitations
6. Muffled heart sounds
7. Gallop rhythm

Investigations:

- **ECG:**
  - T-wave Inversion
  - Sort ST-segment
  - Heart block
  - Arrhythmias
- **Troponin T or I** $\longrightarrow$ +ve ESR is markedly raised, Elevated antiviral titers
- **Echocardiogram:** Ventricular function, pericardial-effusion may be present

Treatment:

- Supportive + Treat of the cause

Complication:

- Heart Failure

Rheumatic fever

It is post-infectious autoimmune disease

Epidemiology

- **Incidence:** More in underdeveloped countries, especially people living in overcrowded areas.
- **Age:** typically (5-15) year old, can occur in young adult (20-30)years old.
- **Gender:** Males = Females.

Pathophysiology:

- After infection with *(group A & B hemolytic streptococcus)*
- Acute follicular tonsillitis *(not skin infection)*
- 3 weeks later, patient will develop cross reactivity & will form antibodies *(autoantibodies)* which causes the clinical features:
  1. **Antigenic similarity theory:** Group A (b) hemolytic streptococcus (which has M protein that look similar to some proteins in the heart (mimicry theory), so some antibodies synthesized against bacterial antigens (M-proteins) may cross react with myofibrils of heart.
  2. **Immune complex theory:** the antigens of streptococcus binding to antibody inside blood make (Ag- Ab complex), that attack human tissue most accepted theory.

Pathology

- Sites of immune reaction (inflammation) commonly are:-
- Joints most common 75% $\longrightarrow$ arthritis
- All layers of heart 60% $\longrightarrow$ Pancarditis start from endocarditis.
- Skin lesions.
- Brain (basal ganglia), other serious sacs (pleura, peritoneum)…etc.

Note: *the inflammation takes two forms:*

1. Exudative (no fibrosis): involve serious sacs and heal completely without fibrosis.
2. Proliferative (fibrosis): involve endocardium (valve & mural endocardium) and myocardium heal with fibrosis in form of lesion called (**Aschoffs nodule**).

Aschoffs nodule its microscopic finding, the **pathognomonic lesion** of rheumatic carditis, consists of Central fibrinoid degeneration surrounded with macrophages, T- lymphocyte, plasma cells, and multinucleated giant cells (Aschoffs giant cell), all these are surrounded by fibroblast.

Clinical features:

(Jones Criteria)

- **Major Criteria:**

1. Fleeting arthritis (large joints) (75%)
   (Adults > children)
   (Non-erosive migratory polyarthritis) mainly affecting joints of extremities (knee, ankle, wrist, elbow).
   It **dramatically improve on salicylates (aspirin)**, if not improve the diagnosis is in **doubt**.
2. Carditis (70%) The incidence is decreased with increasing age (children > adults)

- Myocarditis (conduction defect *(heartblock)*)
- Pericarditis (indicates severity)
- Endocarditis
  - Mural endocarditis
  - Most commonly in mitral valve (90%)
  - Then aortic valve (30%)
  - Then tricuspid valve (10%), pulmonary (1%)

Carey-Coombs murmur (mid-diastolic murmur at apex)
Regurgitation commoner than stenosis

3. Chorea (Sydenham's chorea) $\longrightarrow$ 10%)
   - Can be late manifestation after 3-6 months of rheumatic fever
   - Occur due toinflammation in basal ganglia.
   - **(Female > male), and the chronic rheumatic fever also commonin female**.
   - It is characterized by purposeless, involuntary movements of the face& extremities, nervousness, explosive speech, emotional liability. Symptoms are absent during sleep and resolve spontaneouslyin 1-2 weeks
4. Subcutaneous nodules $\longrightarrow$ 25%
   - Painless
   - Over extensor surface of joints
   - It's number & size correlated with severity of carditis
   - Composed of $\longrightarrow$ Central degeneration, surrounded by inflammatory cells + Aschoff's cells + fibroblasts
5. Erythema marginatum $\longrightarrow$ 10%
   - Fading center with marginal erythema

- Over trunk & extremities, It appears for hours and disappear and recurs again

Note: *Erythema nodosum: tender dusky red raised nodules, on front of the shins, they are not common and non-specific in rheumatic fever, not considered in criteria to diagnosis*

➢ **Minor Criteria:**

1. Arthralgia
2. GIT manifestation
3. Fever
4. Prolonged P-R interval
5. Increase ESR & CRP
6. Increase WBCs
7. Previous rheumatic fever

Diagnosis of rheumatic fever requires:
2 major criteria **Or** 1 major criteria **+** 2 minor criteria
+
- Evidence of recent streptococcal infection:
- High Antistreptolysin Titer *(ASO)*
- Throat swab *(culture)*
- Streptozyme Test
- Scarlet fever

Exception

A. If the patient has Rheumatic heart the Diagnosis of recurrent attack needs (2 minor criteria +evidence of GABHS).
B. If Rheumatic chorea present (Other major criteria or evidence of GABHS not required).
C. Insidious onset rheumatic carditis *(Other major criteria or evidence of GABHS not required)*.

<u>Investigation:</u>

- **Echocardiography**:
  - Carditis especially (valvitis)
  - MR
  - AR
- **CBC**: Leukocytosis, ESR\CRP
- **ECG**
- **Throat swab**
- **Manifestations of active rheumatic fever:**

Pallor, fever, tachycardia, ↑ E.S.R, ↑ TLC, ↑ CRP, Muffling of heart sounds

Treatment:

1. **Prophylaxis**

- Improve socio-economic status
- Tonsillectomy

2. **Acute attack**

- Antibiotics ⟶ Penicillin-G (erythromycin or cephalosporin if allergic)
- Anti-inflammatory:
  - High dose Aspirin (8g\day)
  - Aspirin should be continued until the ESR has fallen and then gradually reduced
- Steroids Indicated in cases with significant **carditis, or severe arthritis**, and those who do not respond an adequate dose of aspirin.
- It's not improve prognosis
- Chorea (haloperidol, benzodiazipines)

Complications:

➢ **Acute**

- Heart failure

➢ **Long term**

- 60% ⟶ Rheumatic heart disease *(10-15 years later)*
  - MS
  - MR
  - AS
  - AR
- Infective endocarditis
- Recurrence of R.F ⟶ pregnancy\oral contraceptive

Antibiotic prophylaxis to:

- Prevent recurrence
- Decrease progression to rheumatic heart disease

Benzathine penicillin I.M every month:

Duration:

If no carditis ⟶ 5 years (21 years of age)

If there is crditis but no valvular diseases ⟶ 10 years

If Carditis + Valvular disease ⟶ up to 40 years & may be lifelong

N.B:

- *There may be genetic susceptibility*

- *Long term prophylaxis antibiotic give protection against recurrent* rheumatic fever but does not given protection against infective endocarditis
- Fallow up by echocardiography for future valve lesions.

Mitral stenosis

Failure of mitral valve to open during left ventricular diastole

Causes:

1. Rheumatic heart disease (the most common cause)
2. Congenital
3. mucupolysaccaride
4. carcinoid syndrome
5. methysergide

M.S occur more in women

Normal mitral valve area [4-6 cm^2] it will be reduced:

- Less than 1 cm2 in severe cases
- 1 -1.5 cm2 in moderate cases
- 1.5 – 2 cm2 in mild cases

Pathophysiology:

M.S →left atrial dilatation & left atrial pressure→ ↑ pulmonary congestion → pulmonary hypertension → Right ventricular failure → Left ventricular failure

Symptoms:

- Exertional dyspnea – Hemoptysis
- Thromboembolic because of atrial fibrilation → (CVA, mesentri ischemia)
- Dysphagia
- Hoarseness of voice "Ortner's syndrome"
- Infective endocarditis

Signs: see before

Complications of M.S:

- Left atrial enlargement
- Systemic embolism (CVA, mesentric ischemia)
- Pulmonary HTN
- Functional T.R
- Rt ventricular failure
- Infective endocarditis

Investigations:

1. ECG
2. Echo
3. X- ray
4. Catheterization

ECG

- P. mitral
- P. pulmonal
- A.F
- Rt ventricular hypertrophy
- Rt axis deviation
- R.B.B.B

X- ray

- Congested upper lope veins
- Double cardiac contour (silhouette sign) due to left atrial enlargement
- Mitralization of left heart border
- Horizental left main bronchus "splaying" due to large Lt atrium
- Kerly B lines
- Mitral stone (pulmonary hemosidorosis) due to blood congestion

Echo

a. Diagnostic
b. severity → mitral valve score:-

- Valve area
- Calcification
- Mobility
- Regurge

If <8 → valvotomy – valvoplasty
If >8 → valve replacement

Cardiac catheterization

Diagnosis – treatment → Ballon valvotomy.

<u>Management:</u>

A symptomatic pt in sinus rhythm:

- I.V peniciilin every 3 months to delay progress

Mild symptoms:

- Treat H.F with diuretic
- Treat A.F [control rate/ rhythm – anti coagulants]
- Follow up by Echo until indication of surgery

Moderate to severe ± early pul. HTN [early palpable S2]:

- **Surgical treatment**

Indications of surgery:

1. Pt è severe symptoms & hemoptysis
2. Pt è early pul. HTN
3. Reccurent thromboembolic inspite of adequate anticoagulant
4. Asymptomatic patient with mitral orifice < 1.7cm2.

Types of surgery:

1. Closed commissurotomy [valvotomy] by cathterizaton
   C/I in: thrombus in Lt atrium
   Mitral valve score > 8
   Regurgitation
2. Open commissurotomy
3. Valve replacement

N.B:

- Lutembachter's S/S → M.S + ASD
- Pregnant women became symptomatic in 2nd trimester
- Ortner's S/S → hoarseness of voice or dysphagia is due to left atrial enlargement in M.S
- **Lt atrial myxoma:**
  1. Similar to M.S
  2. Most common 1ry heart tumor
  3. Benign tumor
  4. May be familial
  5. More in teenager females
  6. Tumor plop → clinically heard at apex.
  7. May lead to thromboembolism
  8. More common in left atrium

Mitral regurgitation

Definition: failure of mitral valve to close during left ventricular systole

Pathophysiology:

- During systole: closure of mitral valve and opening of aortic valve but in case of MR failure of closure of mitral valve leading to back flow in left atrium and causing volume overload which lead to dilatation of left ventricle
- In the beginning “in frank –starling limit" compensated:
  - ↑ Volume and ↑ EDV $\longrightarrow$ ↑ C.O.P
  - High volume pulse
  - Forceful non-sustained apex beat
- Later "out of frank –starling limit" non-compensated:
  - Dilatation of left heart $\longrightarrow$ ↓ F.O.C and ↓ C.O.P
  - Low volume pulse
  - Dilatation of left atrium leads to atrial fibrillation
  - Prolonged pulmonary congestion lead to pulmonary HTN
- So MR has many stages:

1. Before frank-starling
2. After frank-starling(HF)
3. Stage of pulmonary HTN, right ventricular hypertrophy, functional TR

Clinical picture: "Symptoms of complication"

- Symptoms of left ventricular failure "LVF"
- Hemoptysis and recurrent chest infection due to “pulmonary congestion"
- Dysphagia due to left atrium enlargement
- Syncope and palpitation due to atrial fibrillation
- CVA symptoms due to thromboembolic complication of AF

Sign:

- **General examination:**
- **Pulse:** high volume → in compensated
  Low volume → in non-compensated "HF"
  Irregular → in atrial fibrillation
- **JVP:** May be high in pulmonary hypertension with prominant wave "V" wave.
- **Precordial examination:**
- **Inspection:** all pulsation possible finding according to stage
- **Palpation:**
  - Apex beat: may be displaced forceful non-sustained in compensated or weak impalpable in non-compensated
  - May be thrill
  - May be parasternal heave
  - May be palpable S2 at pulmonary area
- **Auscultation:**

  In apex area
  - Muffled S1 and S2
  - Maybe S3" severe MR"
  - Pansystolic murmur soft in character, radiating to axilla, increase during expiration

  In pulmonary area
  - Maybe normal or loud S2 if there is pulmonary hypertension
  - Maybe ejection systolic murmur " pulmonary flow murmur, functional murmur" or early diastolic murmur "Graham steel murmur"

  In tricuspid area

- Maybe pansystolic murmur of functional TR due to pulmonary HTN

Factors indicating severity of MR in examination:

1. High volume pulse
2. Wide pulse pressure
3. Raised JVP
4. Displaced forceful non sustained apex beat
5. S3
6. Signs of pulmonary HTN

Causes of MR:

1. RHD
2. Ischemic heart disease
   - Acute MR due to rupture of papillary muscles after acute myocardial infarction
   - Chronic papillary dysfunction due to chronic repeated ischemia associated with special murmur "sea-gull murmur"
3. Connective tissue diseases
4. Mitral valve prolapse " most common cause"

Investigation:

1. ECG:
   - P- mitral and p-pulmonale
   - Af
   - Left ventricular hypertrophy, RVH, RT axis deviation, RBBB
2. X-RAY
   - Mitralaization of left border
   - Prominent pulmonary artery
   - Splaying " widening" of carina
   - Cardiomegaly
   - Pulmonary oedema
   - Maybe late there is calcification of mitral valve
3. Echo
   - Diagnostic
   - Detect severity of MR " LV ESD > 45 mm and EF% < 60%"
   - Pulmonary pressure
   - Thrombus
   - Size of left atrium
   - Valve prolapse
4. Coronary catherization

- To severity
- If MR Associated with CAD

TREATMENT:

MEDICAL:

1. Diuretics to HF
2. ACE-I to HF
3. Warfarin
4. Treatment of AF if present
5. Follow up by ECHO every 6 months

Surgical:

Valvoplasty "valve repair" or valve replacement

Indication for surgery:

1. Sever symptoms
2. LV ESD > 45 mm in Asymptomatic patient
3. EF% < 50% in Asymptomatic patient

Mitral valve prolapse

Definition: degenerative disease of mitral valve leads to prolapse of mitral valve leaflet into left atrium in the middle of systole

Maybe associated with MR in severe MVP

Causes:

1. Idiopathic "most common cause"
2. CT disease
3. Marfan syndrome

Clinical picture

1. Young age
2. Syncope
3. Palpitation
4. Atypical chest pain
5. Any type of arrtyhmia
6. Sudden death may occur if associated with MR
7. Type A personality

MVP syndrome:

Panic attacks

Signs

- Late systolic murmur in apex beat if associated with MR
- Mid diastolic click

Investigation:

- **ECG:** non specific, ST segment depression, T wave inversion, all types of arrhythmia
- **ECHO:** diagnostic, with or without regurgitation and detect severity

Treatment:

- MVP without MR ⟶ follows up by echo 6 months to 1 year
- MVP with arrhythmia ⟶ propanalol
- MVP with MR ⟶ surgery

MVP maybe lead to CVA *(associated with AV malformation in brain, and thromboembolim)*

Aortic Stenosis

Definition Failure of aortic valve to open during left ventricular systole ⟶ *(pressure over load)* and increase in after load.

Pathophysiology:

So aortic Stenosis ⟶ decrease in C.O.P ⟶ VC of Peripheral arterioles ⟶ lead to increase in after load ⟶ left ventricular hype atrophy heart failure.

2 Things maintain function of heart in A.S Asymptomatic:

1. V.C in peripheral vessels
2. contaction of left atrium.S4

N.B:

- *So vasodilator drug is contraindication*
- *Atrial fibrillation leads to decompansated A.S.*

Course:

1. **Asymptomatic:**
   - May take up to 30 years "duration"
   - No treatment only follow up (echo every 6month).
   - Low risk of sudden death
2. **Symptomatic:**
   - Increase risk of sudden death.
   - Urgent surgery

CAUSES

a. **According to age:**

More than 65 year	Less than 65 year
1. calcific (acquired), may be associated with calcified A.V node 2. bicusped aortic valve(congenital)	1. bicusped aortic valve 2. calcific.

b. Drugs:

Methysergide (prophylaxis) in migraine

c. Williams syndrome "supra valvular aortic stenosis + hypercalcemia mucopolysaccradosis

Clinical feature:

Symptoms:

1. Syncope
2. Angina

} Exertional

3. Dyspnea
4. Excersice intolerance
5. heart failure

Signs: see before

Investigation

1. **ECG**
   - Left ventricular hyperatrophy
   - 1st/2nd/3rd heart block.
   - P-mitral (duration of P.wave).
2. **Chest X-ray**
   - Calcification of aortic valve
   - Left ventricular enlargement
3. **Echocardiography**
   1. Diagnosis of aortic Stenosis
   2. Severity
   3. Valve area
   - Normal aortic valve 3-4 cm.
   - If less than 0.5cm it`s severe (AS).
   - valve area>1.5 $\longrightarrow$ Mild.
   - 1-1.5 $\longrightarrow$ moderate.
   - Less than 0.5 $\longrightarrow$ Severe.
   4. Pressure gradient cross aortic valve.
   - If pressure > 50 mmHg this mean sever (AS)
   5. Diastolic dysfunction.
   6. Ejection fraction.
   - < 50% significant heart failure (systolic dysfunction).
   7. Follow up in asymptomatic patient every 6-12 month.
   - (If patient become symptomatic surgery is must).
4. **Cardiac catherization**

Assessment for coronary artery before surgery may be association with disease.

Treatment:

a. **Symptomatic (Survival rate):**
   1. Angina 5 year
   2. Syncope 3 year
   3. Heart failure 2 year

b. **Asymptomatic:**
   1. B-blocker $\longrightarrow$ for B.p
   2. Simvastatin $\longrightarrow$ delay progression of (AS).
   3. Follow up echo every 6-12 month.

Indication of surgery

1. Symptomatic
2. Asymptomatic and Echo finding:
   1. Valve area <0.5.
   2. Pressure gradient >50mmHg.
   3. Ejection fraction<50%.

Remember:

- **(Hayde syndrome)**
  Symptom of (AS) + Angiodysplasia in colon (P.R bleeding)
- **(William's syndrome)**
  Hypercalcimia, syncope because arrhythmia (AS)
- **(D/D of Aortic Stenosis + Syncope)**
  1. Atrial fibrillation.
  2. Heart block.
  3. Arrythemia.
  4. Symptome of A.S.
  5. William`s syndrome

Aortic regurgitation (AR)

Definition: Failure of aortic valve to close during left ventricular diastole

Causes:

Chronic

- Idiopathic
- Ankylosing spondylitis
- Hypertension
- Syphilitic aortitis
- Marfan's syndrome
- Congenital bicuspid valve.
- Acquired (RF SLE).

Acute

- Infective endocarditis.
- Aortic Dissection
- Complication of valvotomy.

Epidemiology: More common in males.

Pathophysiology (Hemodynamics)

During diastole there is regurge of blood from aorta to LV leading to increased LV diastolic volume with LV dilatation and low diastolic BP.

In systole there is increase in CO with elevation in systolic pressure (but within limit because later will develop LVF).

Clinical picture:

- Asymptomatic in mild cases.
- Symptomatic Those of **LVF (dyspnea, orthopnea, PND, fatigue).**

Palpitation (occurs even with mild AR).

Angina

Signs: see before

Investigation

1. **ECG:** show left ventricular hypertrophy (usually).
2. **Chest-x-ray:**
   - LV dilatation
   - Lung congestion
3. **Echocardiography:** initial investigation of choice because confirm diagnosis and assess the severity of regurge

Management

Medical treatment:

- Asymptomatic pt. follows up by echo every 6-12 months.
- Treat arrhythmias and Treat HF.
- ACE inhibitors (to prevent progression of LV dilatation) & digitalis and diuretics to decrease pulmonary congestion.

Surgery treatment Indications:

- Symptomatic even with ordinary activity despite medical treatment.
- Asymptomatic patient with LV dysfunction (LVEF< 55% or end systolic LV diameter by echo >55mm) Operation recommended, before development of CH F.
- AR due to annular dilatation or dissecting aneurysmal dilatation of the aorta immediate surgery.

Type of Surgery: Repair OR Replacement.

Tricuspid regurgitation

Etiology:

Organic:

1. Rheumatic fever.
2. Infective endocarditis especially in (IV) drug abuser.
3. Congenital.
4. Carcinoid syndrome.
5. Complicating valvotomy.

Functional: Due to dilatation of tricuspid ring secondary to RV dilatation in case of right side heart failure.

Note: *carcinoid syndrome is affecting right sided valve (tricuspid & pulmonary)*

Pathophysiology: (Hemodynamics)

During systole part of blood will regurge from RV to RA leading to RA dilatation & Systemic congestion.

During diastole there is increased blood flow from the RA to RV causing, RV dilatation and RV failure.

Clinical picture

It give the clinical picture of Right sided heart failure.

General examination

- ↑ **JVP** & large (V) wave is seen in jugular veins during systole.
- **Pulsatile liver:** Systolic expansion of the liver frequently is present *(Pulsating liver).*

Precordial examination

- **Inspection and palpation:**
  - Apex: Shifted outwards and diffuse due to severe (RVD) right ventricular dilatation.
  - Lt Parasternal pulsations & epigastric pulsations: due to RV dilatation &hypertrophy.
- **Auscultation:**
  - **1st sound:** muffled on tricuspid area (rare).
  - **3rd sound:** may be present on tricuspid area in case of right side failure.
  - **Murmur:** Pansystolic murmur best heard over the tricuspid area, and it increases by inspiration.

Investigation

1. **ECG:** May show Right ventricular hypertrophy & Evidence of RA dilatation (P-Pulmonale).
2. **Chest X-ray:** may show Cardiomegaly due to right ventricular dilatation & May reveal valve calcification.

3. **Echocardiography:** initial investigation of choice because confirm diagnosis and asses the severity of regurge, also can detect combined lesion in same valve or other valves and determine the cause and complication.

Management

- Intensive diuretic therapy when right sided heart failure signs are present.
- In severe cases (in absence of severe pulmonary hypertension) surgical treatment consists of tricuspid (repair) or valve-replacement but this is very uncommon to be done

Prosthetic heart valve

Two types: metallic, bioprosthesis "tissue graft"

metallic	bioprosthesis
- Increase risk of thrombus "need life long anticoagulant" - lives 25 – 30 years "low risk for reoperation" - indicate in: 1. young age 2. most cases	- Decrease risk of thrombus "no need for anticoagulant" - Lives 10 – 12 years "high risk for reoperation" - Indicate in: 1. Old age 2. pregnant

N.B: *if pt has atrial fibrillation→ metallic because pt already on warfarin*

Examination:

- **Inspection:** sternotomy scar
- **Auscultation:** metallic sound in
  - S1 mitral replacement
  - S2 Aortic replacement
  - If metallic sound disappear maybe regurg or thrombus

Any pt. with prosthetic valve presented with loss of consciousness one of the following is the cause:

1. thromboembolic phenomena
2. septic embolism " infective endocarditis"
3. arrhythmia"syncope"

Complication:

1. Valve dysfunction
2. Valve thrombosis
3. Thromboembolic phenomena "CVA"
4. Infective endocarditis

5. Arrhythmia
6. Traumatic hemolysis "MAHA"
7. Complication of anticoagulant

Medical management:

1. Warfarin with target INR 3 – 4 unit, in pregnant switch to heparin
2. Prophylactic antibiotic to infective endocarditis

Pt with prosthetic valve presented with infective endocarditis, this is indication for surgery

-murmer of regurgitation in prosthetic valve indicate dysfunction,while murmer of stenosis does not indicate dysfunction.

Hypertension

Definition: elevation a systolic blood pressure > 140 mmHg or a diastolic blood pressure >90 mmHg of two or more measurements on two or more occasions.

Epidemiology

- Age: increase with age.
- Gender: male > female
- Race: more in Black.

The factors responsible for blood pressure in our body:

1. The degree of contraction of the resistance blood vessels *(the arterioles)* $\longrightarrow$ vascular resistance.
2. Total body sodium (regulated by Aldosterone & Kidney).

Causes:

1. Essential HTN *(idiopathic)* $\longrightarrow$ 95%
   - Unknown cause but a combination of:
   - **Genetic factors:** +ve family history, Black Male
   - **Environmental factors** (Stress).
   - **Life style & habits:** Salt-intake, Alcohol, Obesity, lack of exercise.
   - **DM2** (50% of patients are HTN)
2. Secondary HTN $\longrightarrow$ 5%

   Secondary HTN caused by:

   a. Renal (most common):
   - Glomerulonephritis
   - Polycystic kidney diseases
   - Renal artery stenosis
   - Chronic pyelonephritis

   b. Endocrine:
   - Cushing syndrome
   - Cons syndrome
   - Hyperthyroidism
   - Hyperparathyroidism
   - Pheochromocytoma
   - Acromegaly

   c. Cardiac:
   - Coarctation of aorta
   - Corticosteroids

   d. drugs
   - NSAID
   - Oral CCP
   - Erythropoietin
   - Cyclosporine

   e. Other causes:
   - Oral contraceptives
   - Pregnancy (eclampsia)
   - Vasculitis.
   - ICP
   - Carcinoid syndrome

Definition& classification of Hypertension		
Category	**Systolic BP (mmHg)**	**Diastolic BP (mmHg)**
Hypertension		
Grade 1 **(mild)**	140-159	90-99
Grade 2 **(moderate)**	160-179	100-109
Grade 3 **(severe)**	≥180	≥10
Isolated systolic hypertension		
Grade 1	140-149	< 90
Grade 2	≥160	< 90

Diagnosis & Assessment:

History:

1. Asymptomatic
2. Headache
3. Epistaxis
4. Fatigue

Examination:

- B.P measurement *(both upper limbs & one lower limb)*
- Normally B.P higher in lower limbs than upper limbs
- If B.P in upper limbs > lower limbs ⟶ Coarctation of Aorta
- If B.P unequal in upper limbs *(>20mmHg difference)* ⟶ Aortic Dissection
- Pulse ⟶ AF, radiofemoral delay *(Coarctation of Aorta)*
- Signs of heart failure
- Carotid bruit
- Pallor ⟶ Anemia
- Cushing or Acromegaly
- Auscultate the heart for S4
- Bruit at renal angle ⟶ Renal artery stenosis
- Kidney masses ⟶ Polycystic kidney diseases
- Target organ damage

Investigation:

Aim of investigation to detect target organ damage and assessment of risk factors.

1. CBC: Anemia
2. Urine routine examination ⟶ albumin\ glucose\ RBCs
3. Blood glucose
4. Lipid profile
5. ECG ⟶ left ventricular hypertrophy, AF

6. Echo $\longrightarrow$ left ventricular hypertrophy & diastolic dysfunction
7. RFT
   - Urea\Electrolytes: Renal cause\ glumerulonephritis
   - $K^+ \longrightarrow K^+$ (HTN + Hypokalemia)
   - **Causes:**
     - Diuretics
     - Cushing syndrome
     - Conn's syndrome
     - Renal artery stenosis
     - Liquorice use
     - Pheochromocytoma
     - Glucocorticoid remediable aldosteronism.
8. Investigations for special cause if needed as:
   - Serum aldosterone: Renin Ratio (conn's syndrome)
   - Thyroid function test
   - Growth hormone study

Complications: ***(Target organ damage)***

Systolic HTN is more dangerous than Diastolic HTN

Hypertension $\longrightarrow$ Target Organ Damage (TOD)

1. **Renal (hypertensive nephropathy):**
   - Proteinuria
   - Hematuria
   - Chronic renal failure
   - Renal artery stenosis
   - Chronic glomerulonephritis
2. **Heart:**
   - AF
   - Left ventricular hypertrophy
   - Diastolic dysfunction
   - Heart failure
   - IHD
   - Aortic dissection
3. **CNS:**
   - Hemorrhagic & Ischemic stroke
   - Lacunar stroke
   - Subarachnoid hemorrhage.
   - Hypertensive Encephalopathy *(Headache confusion coma).*
   - Hypertensive vasculopathy *(retinopathy)*

- Grade I: Arteriolar thickening *(Silver wiring).*
- Grade II: Arteriovenous crossing *(nipping)*
- Grade III: Retinal ischemia *(Flame-shaped Hge & Cotton wool spots).*
- Grade IV: Papilledema

Acute complications:

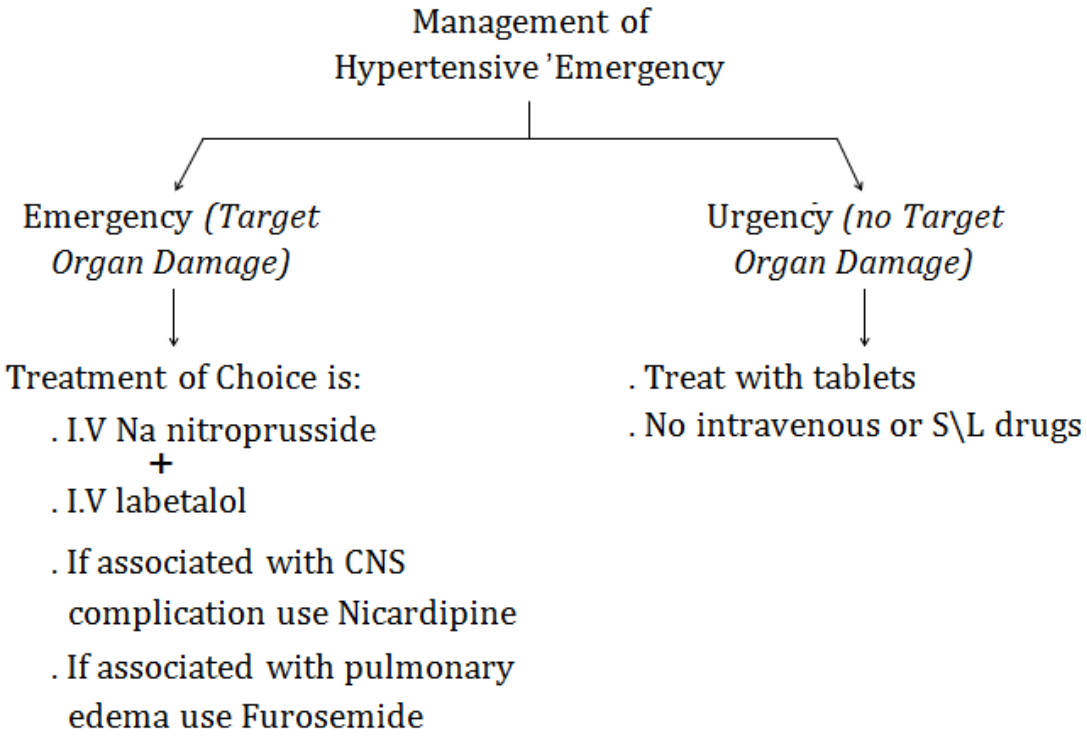
- **hypertensive emergency**
  1. Aortic dissection
  2. Acute pulmonary edema
  3. Malignant HTN
  4. Acute renal failure
- **Malignant hypertension:**
  Increase blood pressure (>220\120) + retinal changes *(papilledema\retinal hemorrhage)*
- **Hypertensive Encephalopathy:**
  Increase blood pressure (>220\120) + Convulsion + decrease in consciousness with localizing signs *(not hemorrhagic)*

Treatment of hypertension:

1. Whom to treat??
   a. All patients with B.P > 160\100 *(sustained)*
   Measured on more than one occasion
   b. B.P 140-159\90-99 + End Organ Damage *(treat with lifestyle + drugs)*
   c. B.P 140-159\90-99 + No End Organ Damage *(treat by lifestyle + reassess every year)*
2. Target blood pressure:
   - If diabetic \ renal diseases \ vascular diseases <130 \ 80
   - If patient has no chronic disease $\longrightarrow$ <140\90
3. Lifestyle modification:
   - Weight reduction (↓ 1kg $\longrightarrow$ ↓ B.P by 1mmHg)
   - Low salt diet (<2 g/day)
   - Stop smoking
   - Exercise
   - Treat risk factors:
     - DM
     - LDL < 100mg *(statins)*
     - Aspirin

4. Pharmacological:
   - Monotherapy:
   - If >55 year or black:
   - Drug of Choice is:
     - Thiazide Diuretics
     - Calcium Channel Blockers
   - If < 55 years ⟶ ACEI (ARBs if ACEI is C/I)
   - Combination therapy:
   - ACEI + Thiazide ⟶ add Calcium Channel Blockers
     Add B-blockers & α-blockers or α-methyldopa
   - Individualize treatment (treat in special situation):

1. HTN + DM ⟶ ACEI
2. HTN + IHD ⟶ B-blockers
3. HTN + Renal impairment ⟶ Calcium Channel Blockers
4. HTN + pregnancy ⟶ α-methyldopa \ labetalol
5. HTN + Raynaod's phenomenon ⟶ Calcium Channel Blockers
6. HTN + CHF ⟶ B-blockers\ACEI
7. HTN + asthma ⟶ avoid B-blockers



N.B: *it is contraindicated to drop B.P rapidly except in:*

- Acute pulmonary edema
- Aortic Dissection
  - Acute renal failure

Infective endocarditis

Definition:

1. Infective Endocarditis: inflammation caused by microbial infection of the endocardium (valves, mural endocardium)
2. Non-infective (sterile) Endocarditis:-results in inflammation without infection by organisms as in collagen diseases e.g. **(SLE)**.

Epidemiology:

Age: More in elderly.

Sex: More in male than female.

Commonest valve affected is **mitral** valve then **aortic** then **tricuspid** then **pulmonary** (left > right, regurge > stenosis) this is due to pressure gradient more in left sided than right side and in regurge more than stenosis.

Pathophysiology:

1. **<u>Abnormal endocardium:</u>**

- Valvular heart diseases
- Congenital heart diseases (e.g. VSD, bicuspid aortic valve, PDA & coarcitation of Aorta)
- Previous infective endocarditis
- Prosthetic heart valves
- Heart transplantation
- IV drug abusers common in right side endocarditis

NB:

- *Large ASD have minimal risk (uncommon) in ASD because low pressure gradient between the 2 atria.*
- *MVP without regurgitation has low risk, but with regurgitation has high risk.*
- *Narrower lesion more risk than large e.g. (small V.S.D more incidence of infection than* Large V.S.D

2. **Bacteremia:**

- High risk procedures as:
  - Dental procedures
  - Colonoscopy
  - Urinary tract procedures

3. **Jet of blood:**

- Blood flow through the heart is increased
- When these factors present $\longrightarrow$ high risk of endocarditis

- Endocarditis more common in left side of the heart
- Right sided endocarditis ⟶ I.V drug abusers
- Vegetation over the valves is the pathological feature of infective endocarditis
- Vegetation composed of:
  - Bacteria
  - Fibrin
  - Platelets
  - Inflammatory cells
- Vegetation ⟶ thromboembolic phenomena
- Valves also may be damaged ⟶ Valvular regurgitation

Classification:

1. **Acute bacterial endocarditis:**
   - Commonly over normal valves
   - Commonest organism staphylococcus aureus
   - High mortality
   - Progress over short time ⟶ HF
   - Usually in drug addicts
2. **Subacute bacterial endocarditis:**
   - Over diseased valve (MR\AR…etc)
   - Caused mainly by streptococcus viridans
   - Run a subacute course

   (1, 2 are called native valve endocarditis)
3. **Prosthetic valve endocarditis**
   a. Early prosthetic valve endocarditis:
      - Before 6 months of replacement
      - Caused by staphylococcus epidermidis

   b. Late prosthetic valve endocarditis:
      - After 6 months of replacement
      - Caused by streptococci
      - Good prognosis

Causes of Infective endocarditis:

- Bacteria:

  Most commonly **gram positive** cocci as:
  - **Staphylococcus aureus** (most common of acute IE & **common in I.V abusers** *(most involved tricuspid valve right side)*

- **Staph. Epidermidis:** most common cause of **(IE)** on **prosthetic valve** in first 6 months of surgery.
- **Strept. Viridans** (the most common organism affecting **native valve**): e.g. in tooth extraction or brushing teeth or chewing.
- **Strept. Foecalis** (enterococci): e.g. GIT or genitourinary procedures (rigid endoscopy).
- **Strep bovis:** e.g. (underlying GI malignancy or colon polyp).

Gram negative bacteria (HACEK) (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella)

- **coxiella burnetii** (Q fever: - H/O of contact with farm animal.Culture -ve endocarditis + hepatosplenomegaly, aortic valve is most involved.
- **Brucella:** H/O contact with goats or cattle & aortic valve is usually involved

- Fungi ⟶ Candida albicans (Culture -ve endocarditis + large vegetation in echo, affecting immune compromisedpatients *(poor prognosis)*)
  - **Others:** Rickettsia, Chlamydia, Klebsiella, Pseudomonas can cause

Clinical features of IE:

a. **Sub-acute infective endocarditis [SIE]**
   Follows an indolent (slow) course, rarely causes metastatic infection, and progresses gradually unless complicated by emboli.

- **Constitutional (general) symptoms: F**ever, **A**norexia, **H**eadache, **M**alaise, Weight loss, Night sweating, myalgia, arthralgia.
  NOTE: (SIE) should be suspected when a patient known to have congenital or valvular heart disease develops a **low grade** persistent **fever**, night sweats or weight loss & develops new **murmur** or heart failure.
- **Systemic emboli:**

1. **CNS embolism:** ischemic Stroke, intracranial hemorrhage due to mycotic aneurysm, Brain abscess, meningitis, encephalitis.
2. **Eye:** Conjunctival-vessel embolism Sub-conjunctival hemorrhage. Sudden blindness: due to embolism of the central retinal artery.
3. **Limb ischemia:** acute limb pain.
4. **Cutaneous embolism: Janeway lesion** (Erythematous, painless flat (patches) lesions on the palms-and soles).
5. **Coronary artery embolism:** chest pain (MI).

6. **Renal-embolism:** (flank pain & hematuria).
7. **Splenic embolism:** left upper quadrant or left shoulder pain.
8. **Mesenteric embolism:** abdominal pain and hematochezia
9. **Pulmonary emboli:** occur in IV drug abuser with tricuspid. Or pulmonic valve endocarditis & presents with sudden dyspnea cough (hemoptysis) & pleuritic chest pain.

- **Immune complex-mediated symptoms:**
  The infected vegetation contains antigens, that trigger immune response production variant of autoantibodies (e.g. Rheumatoid-Factor)
  -Immune complex depositions which may cause. Extra cardiac manifestations but these manifestations are often absent & if present in long standing cases:
  - **Finger clubbing**
  - **Splinter hemorrhage:** longitudinal hemorrhage under the nails due to rupture capillaries.
  - **Osler's nodule:** Small, painful, intracutaneous in the pulps of fingers & toes.
  - **Roth s spots** –by Fundoscopy (area of retinal hemorrhage with pale center).
  - **Glumerulonephritis:** (Red cell cast & proteinuria) cause of death in infective endocarditis.
  - **Splenomegaly** in 30%-40% of patients.
  - **Petechia**

B. Acute infective endocarditis

- It is caused by virulent organism & it can affect even normal valves.
- Presents with fever and rapid damage of heart valves & C/P of CHF with murmurs & (Heart block may occur when-infection extends into conduction system).
- Metastatic infection is common &'It 'can progress to death within weeks.
- The systemic features of **sub-acute** infective endocarditis are **absent**.
- Mortality rate is high

<u>Cardiac features:</u>

- Changing murmur
- Heart failure
- 1st degree heart block (prolonged P-R interval) $\longrightarrow$ Aortic root abscess

Diagnosis of infective endocarditis:

1. **Blood culture:**
   - 3 sets at different times & different sites
   - If +ve 2 cultures, for common organism ⟶ 90% sensitivity for IE *(major criterion)*
   - 10% are culture negative

   Causes of culture –ve infective endocarditis:

   a. Prior antibiotic use
   b. HACEK group
   c. Fungal endocarditis
   d. Atypical organisms
2. **Echocardiography:**
   - Trans-esophageal Echo more sensitive than transthoracic echo
   - Valve regurgitation
   - Vegetation
   - Abscess
3. **ESR & CRP** ⟶ Increased
4. **Urine R\E** ⟶ RBCs cast
5. **ECG** ⟶ Prolonged P-R interval
6. **CBC** ⟶ Anemia
7. **LFT**
8. **RFT**
9. **CXR:** may show abscesses due to septic emboli from right-sided endocarditis

Dukes Criteria for diagnosis of IE:

Major Criteria:

1. **Positive blood culture:**
   - 2 or more 'positive cultures drawn>12 hours apart OR
   - All 3 cultures positive if drawn at-least one hour apart OR
   - A majority positive if 4 or more cultures are drawn.
2. **Endocardium involved:**
   - Positive Echo *(regurgitation\vegetation\abscess)*

Minor Criteria:

- Predisposing condition *(cardiac lesion\I.V drug abuser)*
- Positive culture that doesn't meet major criteria
- Positive Echo that doesn't meet major criteria
- Vascular\Immunological signs
- Fever > 38

Definite endocarditis: (2 major) OR (1 major & 3 minor) OR (5 minor)
Possible endocarditis: (1major & 1 minor) OR (3 minor)

Complication of IE:

1. Heart failure
2. Myocardial abscess
3. Valve regurgitation
4. Aortic root abscess
5. Embolic phenomena
6. Abscess in various organs

Treatment of IE:

1. **General:** Supportive care
2. **Antibiotics:**

- Antibiotic therapy started after sample obtained and before the result (empirical).
- Antibiotics should be bactericidal and combination is needed to prevent the resistance of therapy.
- Period of 4 to 6 weeks of intravenous therapy is required.
- Monitoring treatment: by improvement of clinical picture and Serum CRP is better than the ESR.
- Recurrence of fever may indicate treatment failure, but may also result from hypersensitivity
- Reactions to antibiotics.
- Any source of infection should be removed,for example, a tooth with abscess should be extracted
- Empirical treatment:
  - I.V Benzylpenicillin + I.V Gentamicin (4 weeks)
  - If acute IE add Flucloxacillin (staph)
  - Prosthetic valve endocarditis add Vancomycin + Rifampicin
- If staphylococci $\longrightarrow$ Flucloxacillin + Gentamicin
- Coxiella $\longrightarrow$ Doxycycline
- Fungal $\longrightarrow$ I.V Fluconazole + Flucytosine

Note:

- *Heparin & other anticoagulants are contraindicated in IE for fear of rupture of mycotic aneurysm.*
- *No hepatomegaly in infective endocarditis* ***except in coxiella***

3. **Indications for surgery:**
   a. Heart failure

b. Aortic root abscess\endocardial abscess
c. Prosthetic valve endocarditis
d. Thromboembolic phenomena in spite of antibiotic treatment
e. If no response to medical treatment
f. Large vegetation>5mm $\longrightarrow$ valve obstruction
g. Fungal endocarditis

Antibiotic prophylaxis for infective endocarditis:

Recommended only in 4 conditions:

1. Prosthetic heart valve
2. Previous IE
3. Repaired congenital heart diseases with intra-cardiac device
4. Heart transplantation

Also it is recommended only for certain procedures as Dental procedures

In general

Amoxicillin >2 gram or **clindamycin** 600 mg (if penicillin allergic), given **one hour before** the procedures.

In high risk patients undergoing (genitourinary or gastrointestinal procedures add **Gentamycin**).

(HOW YOU can avoid IE)

A. Correction of the underlying cardiac lesion e.g. closure of VSD.
B. Prevention of infection: **Antibiotics**.
   - **For tooth extraction & upper respiratory tract surgery:** Amoxicillin 3 g orally 1 hour before & 1g orally 6 hours after.
   - **For GIT & genitourinary procedures:** Ampicillin 2 g IV plus Gentamycin: 80 mg IV half an hour before & 6 hours after.
   - **For cardiac surgery:** Cefotaxime 2g IV, 2 hours before & 1 gm. / 6h for 1 day after.

<u>Mortality:</u>

- 30% staphylococci
- 6% Sensitive streptococci

Factors that increase mortality: Age>65 yrs., Aortic valve infection, CHF, CNS involvement, fungal endocarditis, immune suppuration patient.

Arrhythmias

Definition:

Arrhythmia is an abnormality of the cardiac rhythm or rate.

The conducting system of the heart:

Under normal condition, the pacemaker of the heart is **Sinoatrial node** (SAN)

The cardiac impulses arises from **SAN** in a rate (60 – 90 beats/min)

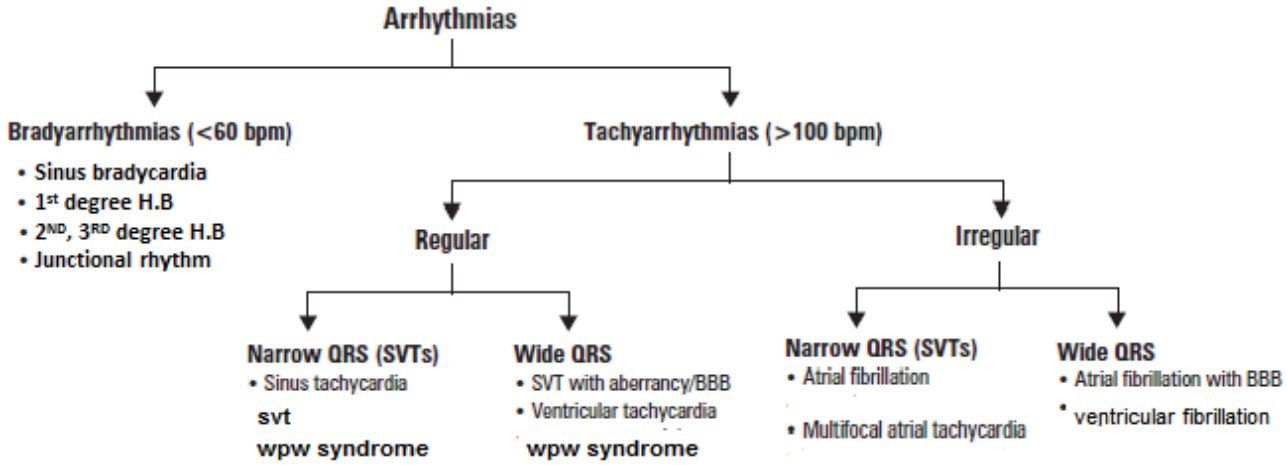
The impulse spreads through the walls of the atria causing them to contract.

Next, the impulse reaches the **AV node**, in which there is a delay of conduction to allow the atria to contract before the ventricles.

Then the impulse reaches **bundle of Hiss** in the interventricular septum, then along the **2 bundle branches** (left & right) & finally **Purkinje fibers** to terminate in the ventricular myocardium causing ventricular contraction.

- Sympathetic stimulation ➔ ↑ the activity of SAN & ↑ the conduction of AVN.
- Parasympathetic stimulation ➔ ↓ the activity of SAN & ↓ the conduction of AVN.
- The ventricles are supplied by sympathetic only (no parasympathetic supply).
- SAN is considered the pacemaker of the heart because its normal rate (60-90b/m) is faster than other cardiac muscle fibers.
- SAN is characterized by its own automaticity (ability to generate impulses) so nerve supply of the heart aims at regulation of heart rate & not initiation of rhythm.
- Normally, the AVN allows passage of impulses from atria to ventricles but not the reverse (no retrograde conduction)

Classification of arrhythmias:



Scheme for arrhythmias

I. Etiology of any arrhythmia:

Tachyarrhythmia	Bradyarrhythmia
1- Myocarditis. 2- Ischemic heart disease (Myocardial infarction). 3- Rheumatic heart disease. 4- Congenital heart disease. 5- Digitalis.	
6- Sympathomimetics. 7- Thyrotoxicosis.	6- Sympatholytics 7- Hypothyroidism.

Exceptions:

- Sinus (tachy or brady) arrhythmias: Physiological & pathological causes.
- Atrial flutter or Atrial fibrillation: begin the etiology by: MS, thyrotoxicosis,HTN,IHD

Mechanisms of Arrhythmias

I. Abnormal Automaticity

Under normal circumstances only cells in the specialized conduction system (SA node, AV node and ventricular conduction system) exhibit natural automaticy. These cells are pacemaking cells. The automaticity of these cells can become abnormally increased or decreased

Ectopic pacemakers.

This mechanism is responsible for the accelerated idioventricular rhythm and ventricular tachycardia that often occurs 24 to 72 h post MI.

II. Alterations in Impulse Conduction

A. Re-Entry Circuits

The presence of self-sustaining re-entry circuit causes rapid repeated depolarizations in a region of myocardium e.g. myocardium that is infarcted/ischemic will consist of non-excitable and partially excitable zones which will promote the formation of re-entry circuits.

B. Conduction Block

Ischemia, fibrosis, trauma, and drugs can cause transient, permanent, unidirectional or bidirectional block

Can lead to bradycardia or tachycardia when impaired conduction leads to re-entry phenomenon

C. Bypas Tracts

Normally the only conducting tract from the atria to the ventricles is the AV node

Congenital/acquired accessory conducting tracts bypass the AV node and facilitate premature ventricular activation before normal AV node conduction (Pre-Excitation Syndromes)

III. **Trigged activity**

Results of secondary depolarization, arising from an incomplete repolarized cell membrane e.g. ventricular arrhythmia as in patients with Ischemic heart disease.

II. Clinical picture:

Symptoms of tachyarrhythmias

1. Asymptomatic.
2. Palpitation: (see approach to palpitation)
3. Manifestations of LCOP (low cardiac output) especially syncope.
4. Precipitation of HF & angina.
5. Features of the cause e.g.: MI, Rheumatic heart disease, digitalis toxicity.

Symptoms of bradyarrhythmias:

The same **but** no precipitation of angina.

Exceptions:

Atrial fibrillation (AF)	+ Thromboembolism
Ventricular tachycardia (VT)	+ Sudden death
Complete heart block	+ Syncope , Sudden death

SVT + Polyuria

Signs:

1. **Radial pulse:** *(test for ventricle)*
   a. **Rate:** ↑ in tachy, ↓ in bradyarrhythmias.
   b. **Rhythm:** all are regular except AF(irregulary irregular) & extrasystoles (regulary irregular)(in AF calculate pulse deficit)
   c. **Response to carotid sinus massage** (in tachy): ↓ HR in any tachyarrhythmia except arrhythmias that originate in the ventricle.

Simply: any arrhythmia contain this word, ventricular, in its name ➔ no effect by carotid massage (no parasympathetic supply)

NB: In bradyarrhythmias: Response to atropine instead.

Respiratory sinus arrhythmia: *(HR is increased during inspiration)*
- Inspiration ➔ ↑ VR ➔ ↑ of SAN ➔ ↑ HR.
- This is a physiological process indicating that the pacemaker is the SAN.

2. **Neck vein:** *(test for atrium)*
   - Rapid A wave in atrial tachyarrhythmias.
   - Loss of A wave in atrial fibrillation.
   - **Cannon A wave** in any arrhythmia containing this word: nodal, either: paroxysmal nodal tachycardia or nodal rhythm.
   - Occasional cannon A wave in: ventricular tachycardia & complete heart block *(Atrio-Ventricular dissociation)*

Cannon A wave:
- It means severe increase of the right atrial pressure.
- It is due to ventricular contraction during atrial contraction.

3. **Auscultation:** *(first heart sound)*
   - Accentuated in any tachycardia.
   - Weak in any bradycardia.

Exceptions:
- Atrial fibrillation
- Ventricular tachycardia } Variable S1
- Complete heart block
- Nodal rhythm → → accentuated S1 inspite of bradycardia
- In AF S4 is lost

III. Investigations:

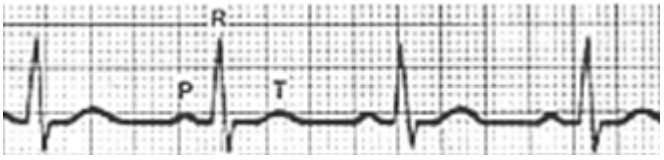
1. ECG:

P wave: *represents atrial contraction.*

- Normal in sinus arrhythmias.
- Abnormal in any other atrial arrhythmias.
- Flutter wave in atrial flutter.
- Fibrillation waves or even absent P wave in atrial fibrillation.

PR interval: *represents the passage of impulse from atria to ventricles.*

- Short in tachycardia.
- Prolonged in bradycardia.
- AV dissociation in: VT, CHB (complete heart block)



QRS complex: *represents the ventricular contraction.*

- Regular except in AF & extrasystole.
- Deformed (bizarre): in VT & CHB.

2. Investigations for the cause:

1. **Echo:** Congenital or valvular heart diseases.
2. Thyroid function tests.

IV. Treatment:

A. Pharmacological

CLASS		DRUGS	MAIN USES
Class I : Na channel blockers *(slows the depolarization)*	Class IA	Qunidine , Procainamide.	Broad spectrum.
	Class IB	Lidocaine , Phenytoin.	Ventricular arrhythmias.
	Class IC	Flacainide , Propafenone.	Broad spectrum.
Class II : β blockers		Propranolol , Atenolol , Esmolol	Tachyarrhythmias. Premature beats.
Class III : K channel blockers		Amiodarone , Bretylium.	**Broad spectrum.**
Class IV : Ca channel blockers		Verapamil , Diltiazem.	Atrial tachyarrhythmias.
Others :		Adenosine(↓automaticity&conductivity) Digitalis (↓automaticity&conductivity)	PSVT Atrial tachyarrhythmias

Side effects of antiarrhythmic drugs:

- **Proarrhythmias:** new arrhythmias induced by the drug.
- **Qunidine:**
  - Allergy & hypotension.

- Cinchonism (headache, vomiting, tinnitus & blurring of vision.
- Digitalis toxicity.

➢ **Lidocaine:** 3m
- Mental confusion.
- Myocardial depression.
- Muscle twitching.

➢ **Amiodarone:** *due to its tendency to accumulate in body tissue it may lead to:*
- CNS: Dizziness, depression, tremors.
- Corneal deposits.
- Thyroid dysfunctions (hyper or hypo thyroidism)
- Pulmonary fibrosis.
- Elevation of hepatic enzymes.
- Constipation.
- Skin pigmentation.

B. Non pharmacological:

1. DC cardioversion.
2. Implantable cardioverter defibrillator (ICD).
3. Radiofrequency catheter ablation.
4. Artificial pacemaker (temporary, permanent)

Sinus tachycardia

Definition:

It is a condition in which the SAN discharges impulses faster than normal (>100 / min)

(Notice that SAN is still the pacemaker of the heart)

Etiology:

1. **Physiological: E**xercise, **E**motions, **E**xcessive coffee.
2. **Pathological: H**ypotension, **H**yperdynamic circulation, **H**yperthermia, **H**eart failure
3. **Pharmacological:** Adrenaline, Atropine.

Clinical Picture:

Symptoms:

- **The same as scheme.**
- Onset & offset: gradual.
- Duration of the disease is usually long as the condition is mostly physiological.

Signs:

1. **Radial pulse:**
   - **Rate:** > 100 /min but usually less than 160 / min.
   - **Rhythm:** regular.
   - **Response to carotid sinus massage:** ↑ gradual HR
2. **Neck vein:** Normal rapid waves.
3. **Auscultation:** Accentuated S1.

ECG:
- **Rhythm:** regular.
- **Rate:** 100 – 160 / min.
- **P waves:** are normal & each P wave is followed by normal QRS.

Treatment: usually no need
- Treatment of the cause.
- β blockers & sedatives may be needed.

Paroxysmal supraventricular tachycardia

Definition:

It is a paroxysmal condition in which there is an abnormal focus in the atrium - *other than SAN* - which discharges regular impulses more than SAN (150-250/min).

1. AV Nodal Re-entry Tachycardia (AVNRT)

Definition: It is a reentry circuit formed in AVN or just next to it

Notice that the heart neglects the SAN & follows the focus

Etiology:
- Physiological: excessive coffee, smoking.
- Pathological: the same as scheme (thyrotoxicosis,IHD)

Clinical picture: *(in between the attacks the heart is normal)*

Symptoms:
- The same as scheme.
- Sudden onset & offset.
- Duration of the disease: usually long history as the condition is mostly physiological.
- Duration of the attack: Variable, usually few minutes but may lasts for hours.
- **NB:** PSVT that lasts for more than 50 % of the day is considered a permanent PSVT.

Signs: *during the attack*

1. **Radial pulse:**

   Rate: 150 – 250 beats/min. *(uncountable).*

Rhythm: regular.

Response to carotid massage: sudden HR.

2. **Neck vein:** Normal rapid waves.
3. **Auscultation:** Accentuated S1.

ECG:

P wave: In atrial tachycardia: deformed.

QRS: rapid, regular with normal shape.

Treatment: *During the attack*

1. Vagal stimulation: Carotid sinus massage, pressure on eye ball or valsalva maneuver
2. Drugs: ABCD
   Adenosine (DOC), **β** blockers, **C**a channel blockers (verapamil), **D**igitalis. (IV)
3. If there is no response or if the patient is hemodynamically unstable: **DC cardioversion**.

2. <u>Atrioventricular Re-entrant Tachycardia (AVRT)</u>

- **WPW (Wolff-Parkinson-White syndrome)**

Congenitally: It is a reentry circuit between atrium and ventricles with one portion of the circuit is usually the AV node, and the other, an abnormal accessory pathway from the atria to the ventricle known as (bundle of Kent).

Usually silent and asymptomatic because the electricity commonly entering to the ventricle through normal pathway (A-V node pathway) but if the activation of ventricle occur through abnormal way (accessory) the patient usually become symptomatic and called WPW.

Types of WPW:-

1. **Orthodromic tachycardia:** The re-entry circuit passes antegradely through the A-V node & retrograde through the accessory pathway produce inverted P wave& narrow-QRS tachycardia.

2. **Antidromic tachycardia:** the re-entry Circuit passes antegradely through the accessory pathway & no retrograde through the AV node, this produce wide QRS tachycardia with **delta wave** and positive P wave because no retrograde conduction through AV node.

WPW is associated with thyrotoxicosis, mitral valve prolapse, HCM & more common in men.

ECG: short PR Interval and slurred of the QRS complex called a delta wave.

Note: QRS may be narrow or wide according to types.

Management:

- If the patient is unstable DC shock
- Maneuvers ↑ to vagal tone: (Carotid artery-message, Valsalva) usually terminate the attack.
- IF patient is stable **IV Adenosine, procainamide**.
- If frequent and symptomatic give Prophylactic prevention with **flecainide**, propafenone or amiodarone.
- Catheter ablation is the **definitive treatment of choice**.

Note: Calcium channel blocking, or Digoxin are contraindicated because they block the A-V node and allow more transmission through abnormal pathway (accessory).

Atrial Flutter

Definition:

It is a condition in which there is an abnormal focus in the atrium that discharges rapid regular impulses **(250 – 350 /min)**, but due to **physiological block** of AVN,

Not all atrial impulses are conducted to the ventricles, only ½, ⅓, ¼ , .. of the will pass to the ventricles.

(Notice that not all atrial impulses are conducted to the ventricles)

Etiology: *doesn't occur in normal heart*

The same as scheme but begin with: **Mitral stenosis & thyrotoxicosis**

Clinical picture:

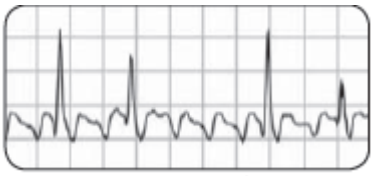
Symptoms:

- The same as scheme.
- Sudden onset & offset.
- Duration of the disease: Short, it is a transient arrhythmia between normal sinus rhythm & atrial fibrillation.

Signs:

1. **Radial pulse:**
   Rate: Variable according the degree of AV conduction, 150, 100, 75beats/m.
   Rhythm: regular.
   Response to carotid massage: ↓ HR in mathematical pattern due to ↑ AV block from 2:1 to 3:1 to 4:1 So, HR from 150 to 100 to 75 beats/min.
2. **Neck vein:** number of A waves is double, triple or quadriple the pulse rate according to the degree of AVN conduction.

3. **Auscultation:** Accentuated S1.


Atrial flutter with variable block

ECG: *(Saw tooth appearance)*

P waves: abnormal, replaced by multiple small flutter (**f**) waves before each QRS

QRS: normal, regular, at a rate of ½, ⅓ or ¼ the atrial rate according to AVN conduction.

Treatment:

1- Drugs: to control the ventricular rate (↓ AVN conduction)

β blockers, **C**a channel blocker (verapamil) or **D**igitalis.

2-DC cardioversion: if the patient is hemodynamically unstable.

Atrial fibrillation

Definition:

It is a condition in which there are rapid **irregular** impulses (400-600/min) arise from the atria by multiple ectopic foci *(so the atria don't contract effectively)* & due to physiological delay at AVN, not all impulses are conducted to the ventricles.

It is the most common sustained cardiac arrythmia (0.5 % of adult population).

(Notice that there are multiple foci ending in ineffective atrial contraction)

Types of AF:

1. **Paroxysmal AF:** i.e. discrete self terminating episodes.
2. **Persistent AF:** i.e. prolonged episodes that can be terminated by electrical or chemical cardioversion.
3. **Permanent or chronic A.F:** i.e. sinus rhythm cannot be restored; Permanent AF is usually preceded by bouts of paroxysmal AF and one or more episodes of persistent AF.

(50 % of paroxysmal AF can occur in normal heart & 20 % of persistent or permanent AF can occur in normal heart)

Etiology:

- Mitral stenosis, thyrotoxicosis, HTN&IHD
- Constrictive pericarditis & Cardiac tamponade
- Lone AF (idiopathic): especially in elderly.
- Other causes: **like scheme.**

Clinical picture:

Symptoms:

- The same as scheme.

- Palpitation: rapid, irregular & may be paroxysmal or sustained.
- Duration of the disease: may be long.

(The patients may accommodate for a new rhythm & palpitation disappears)

Thromboembolism: ineffective atrial contraction predisposes to stasis of blood and may lead to thrombosis & systemic emboli (e.g. hemiplegia)

Signs:

1. **Redial pulse:**

   Rate: usually rapid (100 – 150 /min)

   May be slow as in patients on digitalis.

   Rhythm: marked irregularity *(you can't count 4 successive regular beats)*

   Pulse deficit (apical pulse - radial pulse): > 10/min.

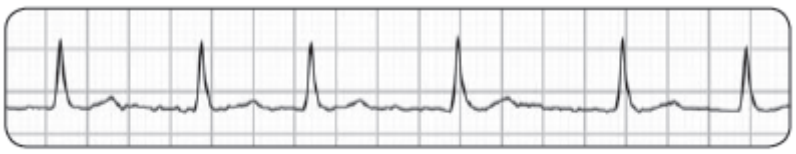
   Response to carotid massage: may ↓ HR due to decreased AV conduction.

   If the radial pulse becomes regular & slow in a case of AF: CHB is suspected & If the radial pulse become regular & rapid in a case of AF: VT is suspected.

2. **Neck vein:** absent A wave.
3. **Auscultation:** Variable intensity of S1.

ECG:

P wave: absent & replaced by fibrillation (F) waves.



Atrial fibrillation (lead II)

QRS: normal in shape but irregular in rhythm.

Treatment:

The acute management of AF involves **3 strategies:**

1. **Control of ventricular rate:** by **β** blocker, **C**a channel blocker, **D**igitalis or amiodarone.
2. **Reversion to normal sinus rhythm:**

Methods: Electrical cardioversion.

Drugs: qunidine, flacinide, propafenone or amiodarone.

Indication:

- Recent onset of AF (<48 hours)
- No history of recent embolism.
- No significant left atrial enlargement or other structural heart disease

Precautions:

Anticoagulant must be given before reversion to decrease the risk of embolization.

Discontinuation of digitalis before electrical cardioversion is a must.

3. **Prevention of thromboembolism:** by warfarin or aspirin.

Anticoagulation should be maintained for at least 1 month following successful cardioversion.

NB: In some cases atrial fibrillation is better treated by anticoagulant therapy &control of ventricular rate without any trial to return to sinus rhythm.

Recurrent AF is treated by long use of propafenone, flecainide or amiodarone.

NB: Perminant AF treatment by anticoagulation for life.

CHADS2 SCORE for AF determines the need for anticoagulation.

Complications of AF:

1. Thromboembolism
2. Angina due to increase HR and decrease COP.
3. It is a precipitating factor of heart failure and pulmonary edema in presence of pre-existing heart disease

Multifocal Atrial Tachycardia (MAT)

Irregular rhythm caused by presence of 3 or more atrial foci (may mimic A.Fib)

atrial rate 100-200 bpm; at least 3 distinct P wave morphologies and PR intervals vary, some P waves may not be conducted

Occurs more commonly in patients with COPD, and hypoxemia; less commonly in patients with hypokalemia, hypomagnesemia, sepsis, and theophylline or digitalis toxicity.

Treatment: treat the underlying cause; calcium channel blockers may be used (e.g. diltiazem, verapamil), β-blockers may be contraindicated because of severe pulmonary disease

no role for electrical cardioversion, antiarrhythmics or ablation

Ventricular tachycardia

Definition:

It is a paroxysmal condition in which there is abnormal focus in the ventricle that discharge impulses more than SAN (150 – 250 / min).

Since the focus is in the ventricle & there is no retrograde conduction in the AVN, So ventricles will follow the ectopic focus & atria will follow the SAN *(AV dissociation)*

(Notice that there is no retrograde conduction in the AVN)

Etiology: *occur in patient with established heart disease*

The most common cause is ischemic heart diseases **(myocardial infarction)**.

Other causes: the same as scheme.

Clinical picture:

Symptoms:

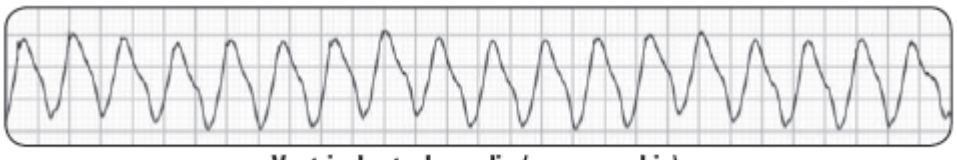
- **The same as scheme.**
- Sudden onset & offset.
- Duration of the disease: short history because it is a serious condition.
- Duration of the attack:
  - Sustained VT: more than 30 seconds (hemodynamically unstable)
  - Non sustained VT: less than 30 seconds.
- **Sudden death:** if converted to ventricular fibrillation.

Signs:

1. **Redial pulse:**
   Rate: 150 – 250 / min (uncountable).
   Rhythm: regular.
   Response to carotid massage: no effect (no parasympathetic supply to ventricles)
2. **Neck vein:**
   Occasional cannon A wave (because occasionally the atria & ventricles may contract together).
3. **Auscultation:** Variable S1, occasionally cannon sounds

ECG:

QRS: rapid, regular & **wide abnormal** (bizarre) shaped.



Ventricular tachycardia (monomorphic)

P waves: Normal rate & shape.

May come before or after the QRS and also may be hidden by the QRS.

No fixed relation between P waves & QRS complexes *(atrio ventricular dissociation)*

NB: Any wide QRS complex tachycardia in any patient with primary heart disease is considered & treated as VT until proved otherwise.

- **Capture beat:** it is when a P-wave that occurs in time at which it becomes conducted ➔ normal QRS Morphology without interruption the tachycardia.
- **Fusion beat:** occurs when activation of ventricules is partly via the-normal His- purkinje system & partly from tachycardia focus.
- **Left axis deviation**

Treatment:

<u>During the attack:</u>

If the patient is hemodynamically unstable:

Immediate cardioversion (start at 100J & repeat if needed & add 100J to each successive shock)

If the patient is hemodynamically stable:

Amiodarone (IV): 150 mg IV over 10min & follow with 1mg/min infusion for 6 hours.

Lidocaine (IV).

Recently,amiodarone has replaced lidocaine as the antiarrhythmic drug of choice in terminating VT.

Adenosine is not effective in VT.

In between the attacks:

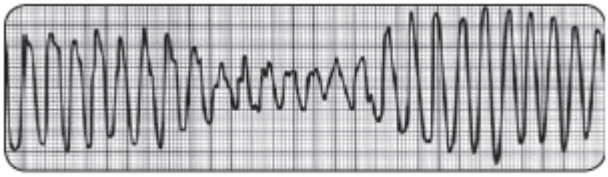
- **A**miodarone.
- **L**idocaine.
- **I**mplantable Cardioverter defibrillator (ICD): in resistant cases.

NB:

- Verapamil is contraindication.
- Digoxin: not effective because work on AVN & can precipitate bigeminy & VT in toxicity.

Torsades de points: (French for twisting of the points)(polymorphic V. tach)

It is a multifocal VT characterized by QRS complexes that change in amplitude & appear to be twisting around the isoelectric line of the ECG & associated with prolonged QT interval.



Torsades de pointes

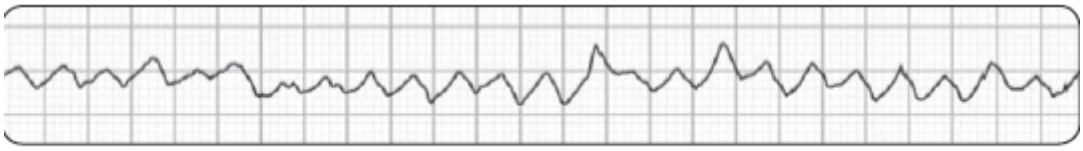
Antiarrhythmic drugs & electrolyte disorders (hypokalemia, hypommagnesemia, hypocalcemia, congenital)

Treatment: Mg & overdrive pacing may be needed

Ventricular Fibrillation (V Fib)

Terminal event, unless advanced cardiac life-support (ACLS) procedures are promptly initiated to maintain ventilation and cardiac output, and electrical defibrillation is carried out

ECG: Bizarre shaped electrical activity, with no P wave, and heart rate may reach to (300 - 600).



Ventricular fibrillation

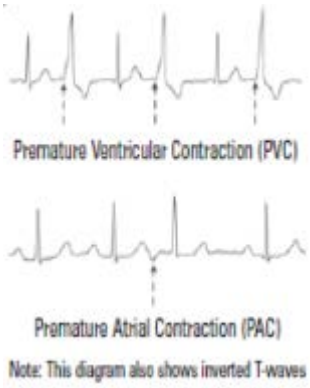
Most frequent cause of sudden death

Treatment: do CPR

Atrial ectopic (PAC)

Atrial premature beats are usually preceded by a P wave of-different shape, The Following QRS complex is normal (narrow). Frequently the ECG demonstrates P waves that are inverted in the inferior leads (leads II, III, AVF) and upright in lead AVR, reflecting the origin of this arrhythmia from the inferior aspect of the atria.

Treatment: No need.



Ventricular Ectopic beats (VEBs)

Ventricular Extra-systole:

A premature cardiac contraction that is out of the normal rhythm and arises in response to an impulse outside the (S A node).

ECG: Ventricular ectopic beat (VEB) are beats which not preceded by a P wave and have a Broad QRS, the QRS could be:

1. Unifocal: identical beats same shape of QRS arising from a single ectopic focus.
2. Multifocal: different morphology QRS from multiple foci.

Definitions

1. **Bigeminy:** IN which every sinus beat is followed by a VEB.
2. **Trigeminy:** in which two sinus beats are followed by a VEB.
3. **Couplets:** Pair of VEB.

Classification of VEB according to severity

<u>Simple ventricular ectopy</u>

- Is present of < 10 PVCs /hour during 24-hour Holter Monitoring. This type is not significant.
- Complex ventricular ectopy (Frequent VEBs)
- Is >10 PVCs / hour during 24-hour monitoring.
- Frequent VEBs are not significant in patient without heart disease but Frequent VEBs are poor prognostic features in patient with (structure heart disease) Acute MI or heart failure.

Note: Frequent VEBs usually signify heart disease so echocardiography to determine the presence of CHD or structural lesion or H.F is important

Treatment:

Ventricular ectopic beats in healthy person (no heart disease):

Is not necessary to treatment unless the patient is highly symptomatic, ß-Blockers can be used.

Ventricular ectopic beats associated with heart disease:

Treatment should always be directed at the underlying condition.

ß-blockers, anti-arrhythmic drugs do not improve and may even worsen prognosis.

Sinus bradycardia

Definition:

It is a condition in which the SAN discharges impulses by a rate less than 60 / min

Etiology:

- **Physiological:** During sleep, Athletes.
- **Pathological:** Obstructive jaundice, Hypothyroidism.
- **Pharmacological:** β blockers, Ca channel blockers, Digitalis.

Clinical picture:

Symptoms: *usually asymptomatic*

- **The same as scheme** *(notice that there is no precipitation of angina)*
- Onset & offset: gradual.
- Duration of the disease is usually long as the condition is mostly physiological.

Signs:

1. **Radial pulse:**

- **Rate:** < 60 /min.
- **Rhythm:** regular.
- **Response to exercise or atropine:** gradual ↑ HR

2. **Auscultation:** Weak S1.

ECG:

- Rhythm: regular.
- Rate: < 60/min.
- P waves: are normal & each P wave is followed by normal QRS.

Treatment: usually no need

- Treatment of the cause.
- Atropine may be needed.
- Artificial pacemaker may be needed in sever chronic cases or when sinus bradycardia is a part of Sick Sinus Syndrome.

Sick sinus syndrome

Combination of symptoms (dizziness, syncope, fatigue, confusion & CHF) due to SA node dysfunction due to degeneration or ischemia its more common in elderly The SA node dysfunction may be:

• Sinus bradycardia or Sinoatrial block sinus arrest.

• Alternating bradycardia with tachycardia (Atrial flutter or AF) (tachy-brady syndrome)

Treatment:

Asymptomatic no treatment required.

Symptomatic patient treated with atropine if still symptomatic (permanent pacemaker)

Nodal (Junctional) rhythm

Definition:

It is a condition in which the heart is controlled by the AVN.

Here, the impulses reach the atria & ventricles in the same time.

Etiology: The same as scheme *(the most common causes are digitalis & MI)*

Clinical picture:

Symptoms:

- **The same as scheme.**
- Sudden onset & offset.
- Duration of the disease: usually short history except if congenital.

Signs:

1. **Radial pulse:**

- Rate: slow (40 – 50 /min).
- Rhythm: regular.

2. **Neck vein:** Cannon A waves.

3. **Auscultation:** accentuated S1 (cannon sounds) This is an exception in bradyarrhythmia.

ECG:

- P wave is inverted, may be before, under or after QRS complex, HR is slow
- P waves: Inverted & come approximately at the same time with QRS so maybe absent
- QRS: Slow, regular with normal shape.

Treatment:

- Treatment of the cause.
- Atropine.
- Artificial pacemaker may be needed in severe cases.

Heart block

Types:

- **Sino atrial block:** failure of impulse to conduct between the SAN & the atria.
- **AV block:** failure of impulse to conduct between the atria & the ventricles.
- **Bundle branch block (BBB):** either in left or right bundles.

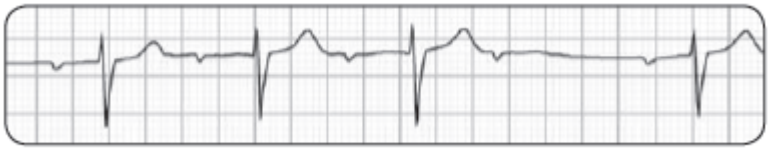
Atrio ventricular (AV) block

First degree heart block: *(Just delayed conduction)*

PR interval is longer than 0.2 second.
All impulses from SAN are conducted to the ventricles.
Etiology: physiologically during sleep or pathologically as in myocarditis, endocarditis, rheumatic fever, IHD
Usually asymptomatic.

Second degree heart block:

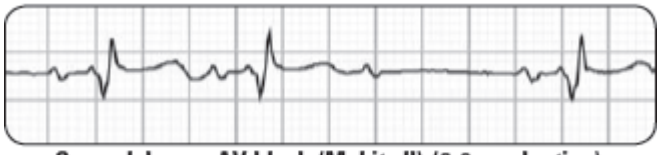
In this condition some impulses from the atria don't reach the ventricles, this causes **"dropped beats".** There are two types:
Type I 2nd degree (Mobitz I, *Wenckebach block*):
Progressive prolongation of PR interval leading finally to the dropout of a QRS complex & then the cycle is repeated. (Notice that there is irregular pulse).
This condition is not too serious and may occur physiologically during



Second degree AV block with Wenckebach phenomenon (Mobitz I)

sleep in athletes.

Type II 2nd degree (Mobitz II):



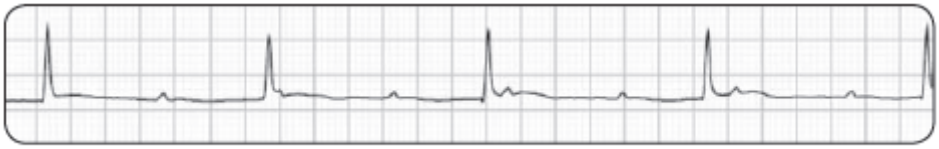
Second degree AV block (Mobitz II) (3:2 conduction)

Intermittently skipped ventricular beat

The AVN transmits one impulse for each 2, 3, 4 or more atrial impulses.

This block may be fixed (e.g. 2:1 all the time) or variable (irregular).

Complete heart block (3rd degree):



Third degree AV block (complete heart block) (lead II)

In this condition all impulses from the atria don't reach the ventricles so, the ventricles will be controlled by idioventricular rhythm.

Notice that the atria are controlled by SAN & the ventricles are controlled by **idioventricular rhythm**.

(Atrioventricular dissociation)

Idioventricular rhythm may originate anywhere from AVN to the bundle branches or purkinje fibers. (The closer the origin to AVN, the faster the rate)

Etiology: The same as scheme plus idiopathic fibrosis of AVN.

Clinical picture:

Symptoms:

- **The same as scheme.**
- Syncope " Adams-Stokes attacks"
- Sudden death.

Signs:

1. **Redial pulse:**
   - **Rate:** 30-40 /min.
   - **Rhythm:** regular.
2. **Neck vein:** normal with occasional cannon A waves.
3. **Auscultation:** Variable S1 with occasional sounds.

ECG:

QRS: slow, regular & **wide abnormal** (bizarre) shaped.

P waves: normal rate & shape.

No fixed relation between P waves & QRS complexes

(Atrioventricular dissociation)

Treatment:

- Treatment of the cause.
- Artificial pacemaker: the treatment of choice.
- Atropine.

In one word:

- ✓ **Sinus bradycardia:** is the same like **sinus tachycardia** but slow.
- ✓ **Nodal rhythm:** is the same like **Paroxysmal nodal tachycardia** but slow.
- ✓ **Complete heart block:** is the same like **ventricular tachycardia** but slow