MEDICINE 2020

CARDIOLOGY

CARDINAL SYMPTOMS:

- DYSPNOEA
- CHEST PAIN OR DISCOMFORT,
- SYNCOPE,
- PALPITATIONS,
- COUGH
- HEMOPTYSIS
- EXCESS FATIGUE

DYSPONEA

Abnormally uncomfortable awarness of breathing

Acute

- Pulmonary edema
- Asthma
- Pneumothorax
- ARDS
- The test of choice to differentiate

• ARDS from acute LVF- PCWP

LVF↑ ARDS (Normal)

• The test of choice to differentiate COPD from LVF- BNP estimation

Salient features of PE

Commonest symptom → Dyspnoea

Commonest sign → Tachypnoea

Commonest vein of origin → Iliofemoral vein

Clinical & progrostic classification

1. Massive Embolims -> 50% of lumen of MPA obstructed.
   C/F →Hypotension, Cyanosis ± syncope
2. Submassive Embolism → Normotension, Cyanosis ± syncope
   < 50% of lumen of MPA obstructed.
3. Pulmonary Infarction → chest pain + hemoptysis prominent
   Obstruction of distal branches of pulmonary arteries.
4. Paradoxical embolism:- Direct shunt from RA → LA by PFO in 2/3 and O.S ASD in 1/3 of cases.

• Screening test of choice → D. Dimer Assay.
• Diagnostic test of choice → Spiral CT scan of test
• Role of angiography – Indicated only when surgery is planned.
• ECG → Commonest sinus tachycardia least sensitive and specific – S_1 Q_3 T_3 pattern (10% cases).
• Echo → R.V. hypokinesia with sparing of apex → Mc Conwel's sign.

• CXR →

1. Hamptor's Hump
2. Westermark's sign
3. Palla's Sign

- V.P. Scan → Only in pregnancy and CRF

Therapy

Medical ← → Surgical

- Heparin — Only when drug are contraindicated
- Warfarin
- Thrombolsis only in Massive embolism

Chronic

- COPD
- LV dysfunction
- Pleural effusion
- Severe anaemia

The classification of dyspnoea most commonly by NYHA

1. Class I -→ Presence of heart disease without limitation of ordinary physical activity
2. Class II -→ symptoms on ordinary physical activity
3. Class III -→ Symptoms on less than ordinary physical activity
4. Class IV -→ Symptoms at rest

American Thoracic Society Scale of Dyspnoea

Paroxysmal Nocturnal dyspoea is due to interstitial pulmonary edema and sometimes intra alveolar edema and is most commonly of left ventricular origin

Angina Pectoris

Discomfort in the chest and / or adjacent areas associated with myocardial ischemia but without myocardial necrosis

Typical – Substernal, characterized by a burning, heavy, or squeezing feeling precipitated by exertion or emotion and promptly relieved by rest or nitroglycerin

Atypical- Located in left chest, abdomen back, or arm in the absence of mid chest pain, sharp or fleeting, repeated, very prolonged unrelated to exercise and not relieved by rest or nitroglycerin.

Levin's sign- Clenching of the fist in front of sternum while describing the sensation. Levine's sign is a strong indication of an ischemic origin of the pain

Angina is classified by NYHA classification and Canadian cardiovascular society functional classification

Chest Pain

Life threatening causes	Non-life threatening causes
• Aortic Dissection • ACS • Pneumothorax (Tension) • PTE	• Pericarditis • Oesophageal • Herpes Zoster

Palpitation

Unpleasant awareness of the forceful or rapid beating of the heart

Common causeare extrasystoles, atrial fibrillation, atrial flutter, Thyrotoxicosis, anaemia febrile states, tobacco, coffee, tea, alcohol, anxiety state and menopausal syndrome

Syncope

Transient loss of consciousness with full recovery.

Causes:

- Most common cause -→ Vaso-vagal or neuro-cardiogenic syncope
- CVS causes-→
  - LVOT obstruction
  - RVOT obstraction
  - LA obstruction -→ LA myxoma
  - Arrythmias: VT is the most common arrhythmia causing syncope
- Neurological causes:
  - Migraine
  - Seizure
  - V.B.I
- Less caommonly metabolic causes
  - Hypoglycemia, Hypoxia

Test of choice for diagnosing vasovagal syncope-→ till table test. (HUTT)

IMPORTANT SIGNS:

- **EDEMA**
- **CYANOSIS**
- **CLUBBING**
- **PULSE**
- **JVP**

Edema

Soft tissue swelling due to abnormal expansion of interstitial fluid volume. Edema fluid is a plasma transudate that accumulates when movement of fluid from vascular to interstitial space is favoured

Minimal excess of fluid to cause edema > 3 liters

Localized	**Generalized**
– Venous or lymphatic obstruction – Local injury (thermal, immune infectious, mechanical)	– Severe malnutrition – Cirrhosis – Nephrotic syndrome – Heart failure – Renal failure – Drugs

Cyanosis

Bluish discolouration of the skin and/ or mucous membranes

- Reduced Haemoglobin>5 gm/dl
- Methemoglobin>1.5gm/dl
- Sulfhemoglobin>0.5gm/dl
- Arterial saturation (S_pO_2)<85%

Central Cyanosis

1. Decreased atmospheric pressure (high altitude)
2. Impaired pulmonary heart disease
3. Pulmonary AV fistula
4. Haemoglobin abnormalities

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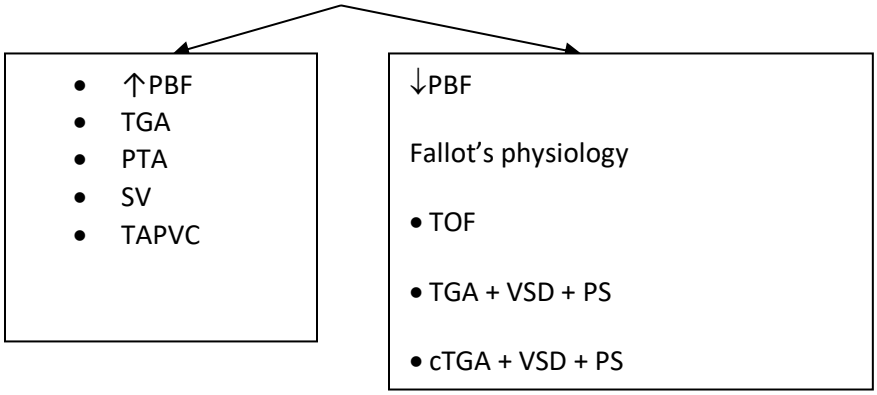
Peripheral cyanosis

1. Reduced cardiac output
2. Cold exposure
3. Redistribution of blood flow from extremities
4. Arterial obstruction

Venous obstruction
Differential cyanosis – PDA with reversal of shunt
Reverse differential cyanosis – TGA with PDA

Approach to congenital cyanotic heart disease (Primary)



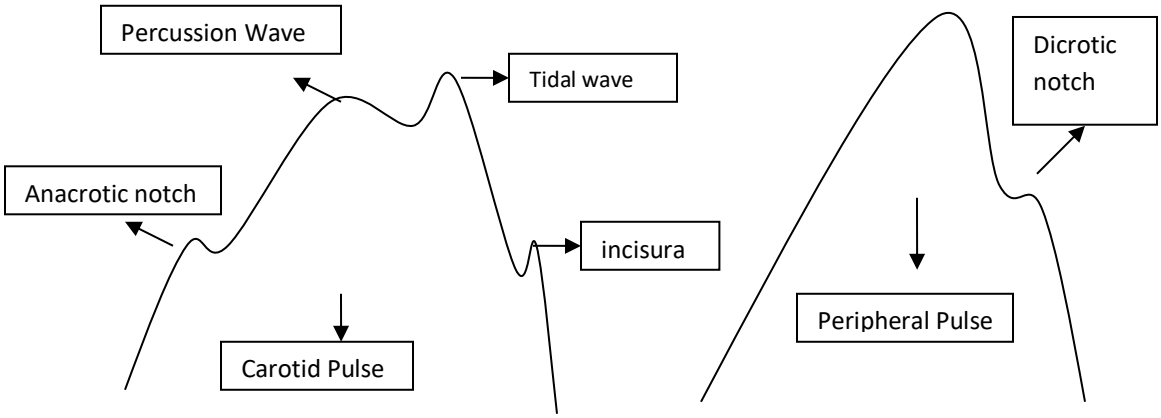
Clubbing

The selective bullous enlargement of the distal segments of the fingers and toes due to proliferation of the connective tissue, particularly on the dorsal surface, is termed clubbing. The growth is mediated by PDGF is normally metabolized in the lungs.

Causes:

- Cyanotic congenital heart disease
- Lung abscess
- Bronchogenic carcinoma
- Bronchiectasis
- Infective Endocarditis
- Idiopathic
- Cirrhosis
- Crohn's disease
- Cystic fibrosis
- Mesothelioma

The mechanism of clubbing is unclear, however it appears to be secondary to a humoral substance which causes dilation of the vwessel of the finger tips



Pulse

Collapsing pulse- aortic regurgitation, severe anaemia, Beri Beri, Thyrotoxicosis, Paget's disease, AP window, PDA

Bisferiens – HOCM, Aortic Regurgitation, Severe AR & AS

Pulsus alterans – LV dysfunction, cardiomyopathy

Pulsus parvus et tardus – Aortic stenosis

Anacrotic pulse – Aortic stenosis

Dicrotic pulse – Fever, cardiac tamponade, severe heart failure, hypovolemic shock →one peak in systole one in diastole

Pulsus paradoxus- Cardiac tamponade, chronic constrictive pericarditis, emphysema, asthma, hypovolemic shock, pulmonary embolus, pregnancy

Reverse pulsus paradoxus – hypertrophic obstructive cardiomyopathy

Radiofemoral delay – coarctation of aorta

Jugular Venos Pulse

Upper limit of normal – 4 cm above the sternal angle

Seen best in right internal jugular vein

Abdomnio Jugular reflux

This is tested by applying firm pressure to the periumbilical region for 10 to 30 seconds with the patient breathing quietly while the jugular veins are observed.

Normally JVP rises 3 cm of H20 and only transiently while abdominal pressure is continued Cause of abnormal response.

- Right ventricular failure
- Left ventricular failure
- Tricuspid regurgitation
- Constrictive pericarditis

A wave: Venous distension due to right atrial systole
X descent: Atrial relaxation and descent of floor of right atrium during RV systole
C Wave: Ventricular contraction (simultaneously with carotid pulse)- bulging of T.V into R.A.
V wave: Right atrial filling when Tricuspid Valve is closed (ventricular systole)
Y descent: decline in right atrial pressure when tricuspid valve reopens
Absent a waves – Atrial fibrillation

Cannon a waves – Av dissociation → CHB, VT, JT

Large a waves – Tricuspid stenosis, RVH, pulmonary hypertension, Ebstein's anomaly (Himalayan P)

Large X descent – Cardiac teamponde, large pericardial effusion, Atrial Septal defect

Decreased or absent X descent – Tricuspid regurgitation, Atrial fibrillation
Large V wave – Tricuspid regurgitation, Atrial fibrillation.

Absent Y- cardiac tamponade – most important
Large Y descent – Constrictive pericarditis, RVMI, Severe rt. Sided failure, AF

Decreased Y descent – Tricuspid stenosis

Steeply rising H wave - Restrictive cardiomyopathy, Constrictive pericarditis, right ventricular infarction.

Equal a and v wave – Atrial Septal defect

- Kussmaul's sign-Chronic Constrictive pericarditis, Tricuspid stenosis, Right sided failure, Right ventricular MI, restrictive cardiomyopathy.

- **Precordial Palpation**
- **Heart Sounds**
- **Murmurs**
-

Precordial Palpation

Cardiac apical impulse is normally localized in the fifth intercostals space, midclavicular line Abnormalities include.

1. Forceful apical trust – LV hypertrophy
2. Lateral and downward displacement – Eccentric dilation of LV
3. Heaving or sustained – LVH, Aortic Stenosis
4. Double – systolic apical impulse – Hypertrophic obstructive candiomyopathy.
5. Dyskinetic (outward bulge) impulse ventricular aneurysm, large dyskinetic area post MK, cardiomyopathy.
6. Sustained lift at lower left sternal border Right ventricular hypertrophy.

Heart Sounds:

Loud S_1- Mitral stenosis, short PR interval hyperkinetic heart, thin chest wall
Soft S_1 -Long PR interval, hear failure, mitral regurgitation thick chest wall, emphysema, calcified MS
Widely split S_1 – Right bundle branch block, Ebstein's anomaly.

S_2: Normally A_2 precedes P_2 and splitting increase with inspiration. Abnormalities include

1. Widened splitting – RBBB, Pulmonic stenosis, ASD, TAPVC
2. Fixed spilling – Atrial septal defect.
3. Paradoxical splitting – Aortic stenosis, Left bundle branch block, CHF
4. Loud A_2 – Systemic hypertension
5. Soft A_2- Aortic stenosis
6. Loud P_2 – Pulmonary arterial hypertension
7. Soft P_2 – Pulmonic stenosis

S_3: Low pitched, heard best with bell of stethoscope at apex, follows S_2
Normally found in children, athletes, pregnancy
Pathological in LV failure, volume overload
Non pathological in MR
S_4 – Low pitched board best with bell at apex, precedes S_1, reflects atrial contraction into a noncomplaint ventricle. It is found in aortic stenosis. Hypertension, HOCM and CAD acute MR

S_4 is absent in patents with atrial fibrillations

Murmurs

Systolic Murmurs

1.Ejection type

- Aortic outflow tract
  - Aortic valve stenosis
  - Hypertrophic obstructive cardiomyopathy
  - Aortic flow manner.

- Pulmonary outflow tract
  - Pulmonic valve stenosis
  - Pulmonic flow murmur

2.Holosystolic type-
- Mitral regurgitation
- Tricuspid regurgitation
- Ventricular septal defect

3.Late Systolic type
- Mitral or tricuspid valve prolapse.

Diastolic Murmurs

Early diastolic
- Aortic valve regurgitation
- Pulmonic valve regurgitation

Mid to late diastolic
- Mitral or tricuspid stenosis
- Flow murmur across mitral or tricuspid valves (ASD)

Pain diastolic
- Any diastolic murmur with ↑ severity

Continuous murmur
- Patent doctors arteriosus
- Coronary A V fistula
- Ruptured sinus of valsalva aneurysm
- Mammary souffle
- Cervical venous hum – physiologycal
- Annomalous left coronary artery from pulmonary artery (AJ CAPA)
- Intercostal AV fistula
- Co-arctation of Aorta
- Severe Fallor's with Aorto-pulmonary collaterals
- MS + ASD (Lutembacher)

Grahm Steel murmur.
It is a murmur of pulmonary hypertensive pulmonary regurgitation and begins with a loud P_2 Congenital PR is not graham steel.

Austin flint murmur
Seen in Aortic regurgitation. It is believed to result from antegrade flow across AV valves that we closing rapidly during filling of the recipient ventricle.

Carrey Coomb's murmur
Mid diastolic aplical murmur in acute rhematic fever.

Opening Snap: High pitched sound following S_2 by 0.06-0.12 sec, heard best at lower left sternal border and apex in mitral stenosis. The more severe the MS, the shorter the S_2- OS interval. Indicates valve mobility.

Election Clicks: High pitched sounds following S1: observed in dilation of aortic or pulmonary artery, congenital AS (loudest at apex) or PS (Upper left sternal gborder). The ejection click of PS decreases with inspiration.

Indicates valve mobility – cephaled doming.

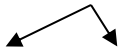
ECG

Bundle Branch Block

Complete >3 small squares
Incomplete <3 small squares
RBBB – Normal variant
- ASD
- Ischemic

LBB: Hypertensive heart Disease
- Aortic stenosis
- Cardio myopathy

Left Hemiblock



LAHB ↓ Left axis
LPHB ↓ Right axis deviation

- Bifascicular and Trifascicular block
- Differential Diagnosis of St Segment Elevation
    1) BRUGADA syndrome
    2) Early repolarisation
    3) Hypercalcemia
    4) Hyperkalemia
    5) MI
    6) Pericarditis
    7) Prinzmetal's angina
    8) L.V. Aneurysm

ECG IN PTE

Sinus tachycardia most common along with T wave inversion in VI – V3
- Others are AF, AF flutter
- S1 Q3 T3 – Suggestive of acute cor pulmonale
- Acute onset RBBB

Paper speed – 25 mm/s
Normal standardization is 1.0 mv per 10 mm
Heart rate = 1500 divided by number of small boxes between each QRS
Mean axis = -30° to + 100°
Intervals
PR (0.12 to 0.20) sec)
Short – Preexcitation syndrome
- Nodal rhythm (inverted p in a VF)

Long: First degree AV block
QRS (0.08 to 0.11 sec)
Widened
- Ventricular premature beats
- Bundle branch blocks
- Toxic levels of some drugs (e.g. quindine)
- Severe hyperkalemia

QT (≤ 0.43s; < 50% of RR interval)

Prolonged

- Congenital
- Hypokalemia
- Hypocalcemia
- Drugs (Quindine, Procainamide, Tricyclics)

Shortened

- Digoxin
- Hypercalcemia
- Digitalis toxicity
- Hyperthermia

P waves

Absent – AF, Junctional rhythm, Sinoatrial block, Severe Hyperkalemia

P pulmonale – pulmonary HT, right atrial enlargement.

P mitrale – Left atrial enlargement (MS)

Inverted P waves – Dextrocardia, ectopic atrial activity, nodal rhythm

Right ventricular hypertrophy

R > S in V_1 and R in V_1 > 5 mm

Deep S in Lead 1 & V_6 ; RAD

Left ventricular hypertrophy

S in V_1 plus R in V_5 or V_6 > 35 mm or

R in aV_L > 11 mm

Infarction

Pathological Q waves (>0.44s and >25% of total QRS height) & ST segment elevation

V_{1-3}	Anteroseptal
V_{4-6}	Apical
I, aVI, V_{5-6}	Lateral
I, aVL V_{2-6}	Anterolateral
II, III a Vf	Inferior
V_{1-2} (Tall R waves)	True posterior

Tall R waves in lead VI-RBBB, posterior wall MI, RVH, Duchene's muscular dystrophy

Hypokalemia

- ST segment depression
- U waves
- QT prolongation
- Reduced amplitude of T waves

Hyperkalemia

- Tall – tented T waves → Earliest change
- Decreased or absent p waves → last change
- Short QT interval
- Widening of QRS complex
- Sine- wave pattern

Hypocalcemia

- QT prolongation
- Flat or inverted T waves

Hypercalcemia

- Short or absent ST segment
- Decreased QT interval

Hypomagnesemia

- Prolonged PR interval

- Prolonged QT interval
- AF
- Torsades de pointes
- T waves flattening

Hypermagnesemia

- Short PR interval
- Prolonged QRS interval
- Peaked T waves

ECG in CNS disorders

1. CVA
2. F.A
3. Muscular dystrophies
4. Mitochondrial myopathies
5. Myasthenia gravis

Long QT interval

Acquired Congenital

Congenital LQT syndromes

Romano ward syndrome	Jervell large Nielsen
- AD	AR
- Na channel	K^+ channel
	Sensineural deafness

Therapy of congenital LQT →

- Beta Blockers
- Left cardiac sympathetic denervation
- Pacing

Acquired Causes:

- Hypokalemia
- Hypocalcemia
- Hypomagnesemia
- Drugs
- SA tt
- Liquid Protein diet

Long QT syndrome (LQTS)

QT interval corrected for heart rate using Bazett's formula is 400 to 460 ms in men and 400 to 480 ms in women.

LQT1, LQT2, and LQT3, appear to account for more than 99% patients with clinically relevant LQTs.

LQT1 represents the most common genotypic abnormality (KCNQ1, 11p chromosome).

They fail to shorten or actually prolong their QT interval with exercise. The T wave in patients with LQT1 tends to be broad and comprises of majority or prolonged QT interval. The common triggers are exercise followed by emotional stress. More than 80% of male patients have their first cardiac event by age 20 years. Patients respond to beta blocker therapy

LQT2 is the second most common genotypic abnormality (KCNH2, 7q chromosome). The T wave tends to be notched and bifid. The precipitant is auditory stimulation and postpartum period.

Despite the occurrence during sleep, patients typically respond to beta blocker therapy well.

LQT3 is due to a mutation in the gene that encodes the cardiac sodium channel located on chromosome 3p (SCN5A). Prolongation of the action potential duration occurs because of failure to inactivate this channel.

They have late onset peaked narrow based T waves or asymmetric peaked T waves

LQT3 is the poorest of all the LQTs in prognosis.

Most events in LQT3 patients occur during sleep, suggesting that they are at highest risk during slow heart rates.
Beta blockers are not recommended exercise is not restricted in LQT3.

Long QT syndrome
Primary prevention with prophylactic ICD implantation should be considered in male patients with LQT3 and in all patients with marked QT prolongation (>500 ms), particularly when coupled with history of syncope and an immediate family history of SCD.

Therapy → Intravenous magnesium for acquired causes

Therapy
1. Beta Blockers
2. LCSD : Left sided cervicothoracic sympathetic ganglionectomy
3. Overdrive pacing

Drugs with CVS side effects:
1. Angina exacerbation
   a. Alpha blockers
   b. Beta – blocker withdrawal
   c. Nitrate withdrawal
   d. Excessive thyroxine
   e. Ergotamine
   f. Methysergide
   g. Sumatriptan
   h. Vasopressin
2. Acute chest pain (non ischemic)
   a. Bleomycin
3. Arehythmias
   a. Adriamycin
   b. Antiarrhythmic drugs
   c. Astemizole
   d. Cisapride
   e. Daunorubocin
   f. Digitals
   g. Erythromycin
   h. Lithium
   i. Pentamidine
   j. Phenothiazines
   k. Thyroxine
   l. Terfenadir
   m. Tricycle.
4. AV Block
a. Beta blockers
b. Digoxin
c. Clonidine
d. Verapamil
e. Methyldopa
5. Cardiomyopathy
a. Daunorubocin
b. Doxorubicin
c. Lithium

d. Phenothiazines
6. Fluid retention/congestive Heart failure
a. Beta blockers
b. Calcium channel blockers
c. Estrogens
d. Thiazolidenediones
e. Indomethacin
f. Steroids
7. Pericarditis
a. Emetine
b. Hydralazine
c. Methysergide
d. Procainamde
8. Thromboembolism
a. Oral contraceptive

AV conduction disturbances

1. First Degree AV Block

Prolonged AV conduction, characterized by a PR interval >20 seconds. Causes are drugs such as digitalis, beta- blockers, and calcium channel blockers.

2. Second Degree AV block

i. Mobitz type I second – degree AV block (AV Wenckebach block) is characterized by progressive PR interval prolongation prior to block of an atrial impulse. The pause that follows is less than compensatory, and the PR interval of first conducted impulse is shorther than the last conducted atrial impulse prior to the blocked P wave. The block is localized to the AV node and associated with a normal QRS duration. It is seen with inferior wall MI, drug intoxication particularly digitalis, beta – blockers, and occasionally calcium channel antagonists. It can be seen in normal individuals with heightened vagal tone.
Progression to complete heart block is rare except in case with acute inferior wall myocardial infection. Even then heart block is well tolerated because the escape pacemaker usually arises in the proximal his bundle and provides a stable rhythm.

II. Mobitz type II second – degree heart block is characterized by sudden failure of AV conduction without a preceding change in PR intervals. It is generally due to a disease of the His-purkinje system and is most often associated with a prolonged QRS duration. It may occur in the setting of anteroseptal infarction or in the primary or secondary sclerodegenerative or calcific disorders of the fibrous skeleton of the heart. There is a high incidence of progression to complete heart block with an unstable, slow, lower escape pacemaker. Therefore, pacemaker implantation is indicated in symptomatic patients.

3. Third-Degree AV block

It is present when no atrial impulse propagates to the ventricles.
If the QRS complex is of normal duration, occurs at a rate of 40 to 55 beats per minute and increases with atropine or exercise, AV nodal block is probable (e.g. congenital complete AV block)
If the escape rhythm of the QRS is wide and associated with rates 40 beats per minute, block is usually localized in, or distal to the His bundle and mandates a pacemaker, since the escape rhythm in this setting is unreliable.

Class I Indications for Permanent Pacing

1. Acquired AV block in Adults
i. Complete heart block associated with any one of the following
- Symptomatic bradycardia
- CHF

- Asystole > 3 second
- Escape rate < 40 beats per minute
- Post AV junction ablation, myotonic dystrophy.

ii. Second degree AV block with symptomatic bradycardia

iii. Atrial fibrillation, atrial flutter, or rare cases of supraventricular tachycardia with complete heart block, bradycardia or any of the conditions listed under (i)

2. After myocardial infarction:

i. Persistent advanced second- degree AV block or complete heart block with block in His-Purkjinje system
ii. Transient advance AV block with associated bundle branch block.

3. Bisfascicular Trifascicular Block

i. Bifascicular block with intermittent complete heart block associated with symptomatic bradycardia.
ii. Bifascicular or trifascicular block with intermittent type II second – degree AV block without symptoms

4. Sinus node dysfunction with documented symptomatic bradycardia → commonest indication of pacing

5. Hypersensitive carotid sinus

i. Recurrent syncope associated with clear, spontaneous events provoked by carotid sinus stimulation.
ii. Minimal carotid sinus pressure induces asystole of >3 second duration in the absence of any medication that depresses the sinus node or AV conduction.

CARDIOMYOPATHIES

DCM	HCM	RCM
Systolic dysfunction	Diastolic dysfunction	Diastolic dysfunction

- Most common cause – Idiopathic
- Treatment of choice – Cardiac transplantation
- Test of choice – Echo cardiography

DCM Causes

Idiopathic	CAD	Reversible causes

- Hemochromatosis
- Hypothyroidism
- ↓↓ P_{O4}
- HIV

Therapy

Normotension Diurectics
- ACE
- Beta Blockers
- Spironolactone

Hypotension
- Vasopressors
- IABP

Anti coagulants, anti arrhythmic,
ICD – to prevent SCD in low EF (<35%)

Disease that involves the myocardium primarily and are not the result of hypertension or congenital, valvular, coronary, arterial, or pericardial abnormalities. Primary myocardial involvement.

1. Idiopathic (D.R.H)
2. Familial (D.H.)
3. Eosinophilic Endomyocardial disease ®
4. Endomyocardial fibrosis ®

Secondary myocardial involvement

1. Infective (D)
   Viral, Bacterial, fungal, protozoal, metazoal, spirochetal, rickettsial
2. Metabolic (D)
3. Familial storage disease (D.R)
   Glycogen storage disease, hemochromatosis mucopolysaccharidooses.
4. Connective tissue disorders (D)
   SLE, PAN rheumatoid arthritis,
   Progressive systemic sclerosis.
5. Infiltrative & granulomas (R,D)
   Amyloidosis, Sarcoidosis, Malignancy.
6. Neuromuscular (D)
   Muscular dystrophy, myotonic dystrophy Friedreich's ataxia (hypertrophic)
7. Toxin (D)
   Alcohol, Doxubocin, cyclophosphamide, 5-FU, cocaine
8. Others
   Peripartum heart disease (D)

Dilated cardiomyopathy (DCM)

→ Presents with left ventricular failure
→ RV failure is an ominous sign
→ Pulsus alternans is common low pulse pressure

- Gallop rhythm
- Manifestation of MR and TR present
- Arrhythmias and systemic embolism common.

Potentially reversible
↓ Phosphate, ↓ calcium, TFT and iron studies.

DILATED CARDIOMYOPATHY

Symmetrically dilated left ventricle, with poor systolic contractile function; RV is commonly involved later in the course.
Manifestations –CHF, Arrthythmia, raised JVP, rales, diffuse & dyskinetic LV apex, S3, hepatomegaly, murmur of MR& TR is common
ECG – LBBB and ST – T wave abnormalities.
CXR – Cardiomegaly, pulmonary vascular redistribution, pleural effusion
Echocardiogram – LV & RV enlargement with globally impaired contraction

Treatment of heart failure

1. ACE inhibitors, nitrates, Digoxin
2. β blockers in ambulatory patients
3. Anticoagulation with warfarin for LV clot or thromboemblic manifestation.
4. Implanted internal defibrillation for symptomatic or sustained arrhythmias.
5. Cardiac resynchronization therapy
6. Immunosuppressive drugs.
7. Cardiac transplantation.

HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY:

HCM →Hypertrophy which is present without any cause to account far it

- There is selective involvement of ventricular septum compared to LV Posterior wall where septal thickness: LV posterior wall is about 1.5 times. In absolute valves septal thickness 15mm i.e there is ASH. Most common genetic cause is missense mutation of beta MHC in chromosome 14.
- Presents with diastolic dysfunction like **RCM** and unlike **DCM**
- Cardinal symptoms are (1) Shortness of breath in 90% of cases
  (2) Angina in 75% of cases
  (3) Syncope
- Important physical

Finds = Double or triple apical

- impulse
- Brisk carotid upstroke
- Pulsus Bisferians
- Reserved pulsus paradoxus

Auscultation – Fusion murmur of (MR + MS)

Dynamically of LV out flow tract obstruction changes with (increases)

(1) ↓ After load
(2) ↓ Pre load
(3) ↑ Cardiac contractility.

The obstructive component is due to

- ASH + systolic anterior motion of mitral leaflet.

Difference with valvular AS

ECGT – Features of LVH

- Deep T wave inversion S
- Q waves inferior leads

Arrhythmias – Ventricular → ¾ cases
Supra ventricular → ¼ cases.

Echocardiogram – Test of choice

1. L V hypertrophy, often with asymmetric septal hypertrophy (>1.3 times thickness of LV posterior wall)
2. Good LV systolic function with small end-systolic volume
3. Systolic anterior motion (SAM) of mitral valve
4. Mid systolic partial closure of aortic valve
5. Doppler shows LV out flow gradient.

Treatment

1. A void strenuous exercise.
2. Beta blockers, verapamil, disopyramide are useful.

3. Amiadarone to suppress atrial or ventricular arrhythmias
4. Implantable automatic diefibrillator for patient with high-risk ventricular arrhythmias.
5. Dual chamber pacemaker, controlled septal infarction by ethanol injection in septal artery.
6. Surgical myectomy in pts. Unresponsive to medical therapy
7. Digoxin, diuretics & vasodilators are contraindicated.

Features of High risk patients.

- Young age (<30 years) at diagnosis
- Family history of sudden death
- Abnormal blood pressure response to exercise
- Market hypertrophy
- Ventricular tachycardia on holter
- Syncope
- Genetic abnormalities associated with increased prevalence of sudden death.

RESTRICTIVE CARDIOMYOPATHY

Increased myocardial stiffness impairs ventricular relaxation. Diastolic pressures are elevated

Manifestation-

Right sided heart failure often predominates. Raised JVP, Hepatomegaly, peripheral edema, murmur of tricuspid regurgitation, Kussmaul's sign

ECG – Low limb lead voltage, Sinus Tachycardia, ST-T wave abnormalities.

CXR – Mild LV enlargement

Echocardiogram-

1. Bilateral atrial enlargement
2. Increased ventricular thickness ("speckled pattern") esp. in amyloidosis
3. Systolic function is usually normal.

Treatment

1. Salt restriction
2. Diuretics
3. Digoxin in cases with impaired systolic function. There is increased sensitivity to digitalis in amyloidosis.
4. Anticoagulation, particularly in patients with eosinophilic endomyocarditis.
5. Deferrioxamine & phlebotomy in pts of hemochromatosis.
6. Surgical excision of fibrotic endocardium and replacement of the involved AV valves in Endomyocardial fibrosis is useful.

ARVD (Arrvthmogenic Right Ventricular Dysplasia)

Right ventricular myocardial cell replacement with fibrous and adipose tissue

Substrate for malignant ventricular arrhythmia. Epsilon wave in ECG is characteristic feature.

Therapy – Anti arrhythmic

Catheter ablation

ICD implantation

- **MRI of heart – Test of choice**

MYOCARDITIS

Causes -

1. Infections – Coxasackie virus B. HIV, Adenovirus, Parvovirus, influenza virus
   Diphtheria, staphylococcus
   Chaga's disease (T. cruzi)
   Lyme carditis.
2. Drugs

3. Radiation

Manifestation
Fever, fatigue, palpitations, viral myocarditis, viral myocarditis, may be preceded by upper respiratory tract infection
Tachycardia, soft S1: S3 common.
Death is generally due to congestive heart failure arrhythmias, and heart blocks.
ECG – Transient ST – T wave abnormalities arrhythmias
CXR – Cardiomegaly
Cardiac enzymes may be raised
Echocardiogram-
Depressed LV function, regional wall motion abnormalities in some
Pericardial effusion may be present.

Treatment
1. Rest
2. Diuretics, digitalis, salt restriction.
3. Immunosuppressive, (steroids and azathioprine) may be considered if RV biopsy shows active inflammation.
4. Antitoxin incase of diphtheria.
5. Heart block may require pacemaker implantation.

PERICARDITIS
1. Idiopathic
2. Infections.
    a. Viral (Coxsackie A & B, Echovirus, HIV)
    b. Pyogenic (Pheumococcus, Staphylococcus, Legionella)
    c. Tuberculosis- Commonest cause in India.
    d. Fungal (Histoplasmosis, Candida)
    e. Syphilis, parasitic
3. Acute myocardial infarction
4. Metastatic neoplasm
5. Radiation therapy (up to 20 years earlier)
6. Chronic renal failure
7. Connective tissue disorder (RA, SLE)
8. Drugs reaction (Procainamide, Hydralazine)
9. Autoimmune following heart surgery & MI (Dressler's syndrome)

Manifestation
Painless pericarditis – Uremia, TB, malignancy
What is characteristic of pericardial pain?
Ans-Radiates to Trapezius ridge and resolves on leaning forward position
Treatment modality – Colchicine in resistant cases.
Complications of pericarditis
1. Effusion.
2. Tamponade
3. Construction

Intense chest pain which is sharp, pleuritic and positional (relived by leaning forward) Fever & palpitations are common.

Rapid or irregular pulse
Coarse pericardial friction rub (loudest with pt. sitting forward)

ECG: Diffuse ST elevation (concave upward) usually present in all leads except a VR and VI
- PR segment depression or elevation is the hall mark.

After several days ST returns to baseline, then T wave inversion develops
CXR: Increase size of cardiac silhouette if large (250ml) pericardial effusion is present
Water – bottle configuration.
Echocardiogram-Pericdardial effusion which commonly accompanies acute pericarditis

Treatment
1. Aspirin, indomethacin
2. Steroids
3. Antibiotics in cases with pyogenic pericarditis
4. Pericardiectomy for prolonged pain or recurrent episodes – for very limited cases.

CARDIAC TAMPONADE
Life threatening emergency resulting from accumulation of pericardial fluid under pressure
Causes:
- Neoplasm
- Idiopathic
- Uremia
- Cardiac trauma
- Myocardial perforation during catheter or pacemaker placement.

Pathophysiology: Rapid accumulation of pericardial fluid exerts a pressure on cardiac chambers and impairs cardiac filling hence compromise cardiac output
Rate of rise of pressure is more important than amount of fluid collected
Manifestation
BECK's TRIAD → Hypotension + Muffled sounds + JUP engorged
EWART'S SIGN → Bronchial sound, left scapular region

Manifestation:
Tachycardia, hypotension, pulsus paradoxus raised JVP with preserved X descent, but loss of Ydescent

ECG- Low limb lead voltage, large effusions may cause electrical alternans (alternating size of QRS complex due to swinging of heart)

CXR – Enlarged cardiac size
Pericardial Fat pad sign
CT & MRI- Both invaluable and better man Echo in imaging the pericardium and also measure its thickness. Analysis of pericardial Fluid.
(1) WBC, hematocrit and protein
(2) Staining and culture far TB
(3) Maligancy
(4) ADA
(5) Cholesterol

Echocardiogram – RV collapse during diastole is the hallmark finding.
IVC plethora
CT MRI – Analysis of P.fluid.

Treatment – immediate pericardiocentesis and IV volume expansion. Diuretics are C/I.

CONSTRICTIVE PERICARDITIS

Rigid pericardium leads to impaired cardiac filling, elevation of systemic and pulmonary venous pressures. And decreased cardiac output.

Causes

- Tuberculosis
- Viral
- Previous cardiac surgery
- Uremia
- Neoplastic pericarditis.

Manifestations:

Gradual onset of dyspnoea, fatigue, pedal edema, abdominal distension.
Symptoms of LV failure uncommon
Raised JVP, Kussmaul's sign, hepatomegaly ascites, peripheral edema
"Pericardial knock" following S_2 sometimes present.

Venous wave form = 'W' or 'M' shaped pattern because both x and y descent are present

- **Kussmaul's sign** present not seen in tramponade
- Also seen in Restriction, Tricuspid stenosis and RV, myocardial infarct
- Pulsus paradoxus in 1/3 of cases
- Square root sign in ventricular pressure waveform

DIFFERENCE BETWEEN CONSTRICTION & RESTRICTION

Feature	Constriction	Restriction
Paradoxical pulse	1/3 cases	Absent
Pericardial Knock	Present	Absent
Pulmonary HTN	Absent	Present
Square root sign	Present	Present
Septal bounce	Present	Absent

Pericaditis in HIV – Commonest cardiac manifestation

- Present in 20% of cases
- Constriction is rare
- Lymphomas and Kaposi's sarcoma are most common neoplasm
- Mycobacterium species most common causes of infective pericarditis

ECG – Low limb lead voltage, atrial fibrillation
CXR – Rim of pericardial calcification in up to 50% of patients

Echocardiogram- Thickened pericardium, normal ventricular contraction, abrupt halt in ventricular filling in early diastole.

CT or MRI are more precise than echocardiogram in demonstrating thickened pericardium
Right ventricular endomyocardial biopsy may be required to differentiate if from restrictive cardiomyopathy

Cardiac Catheterization:

1. Equalisation of diastolic pressure in all chambers of heart

2. Ventricular pressure tracing show "dip and plateau" appearance.

Treatment:
Surgical stripping of the pericardium.
Anti tuberculous therapy
Cholesterol or Gold paint pericarditis seen in Hypothyroidism.
Low pressure Tamponade seen in Uraemia, Hypovolemia.
Pulsus para doxus in tamponade not seen with cases with ASD, AR.

ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION:

- 30 days mortality rate is around 15-20% with more than half the deaths occurring before the stricken individual reaches hospital.
- First year mortality in survivors of acute MI is 4%.
- Maximum number of cases occurs in morning within few hours of awakening.
- Chest pain is the most common presenting complaint.
- Painless SDTEMI is seen in diabetic patients and elderly.
    - $1/4^{th}$ patients with anterior infarction have manifestations of sympathetic nervous system hyperactivity (tachycardia / hypertension).
    - One half of patients with inferior infraction show evidence of parasympathetic hyperactivity (bradycardia / hypotension).
    - Signs of ventricular dysfunction include dyskinetic apical impulse, S_3 and S_4, decreased intensity of S_1 and paradoxical splitting of the S_2.
    - Pericardial rub may be heard.
    - Acute stage < 7 days
    - Healing stage – 7 to 28 days
    - Healed - ≥ 29 days.

ECG – Most patients with STEMI develops. Q-wave – MI. some of them may not develop Q waves (non-Q waves MI).

Serum Cardiac biomarkers –

- CK (MB) – Rises with 4 to 8 hours and comes back to normal by 48 to 72 hours. A ratio of CKMB mass: CK activity ≥ 2.5 is more specific.
- Cardiac specific Troponin T (cTnT) and cardiac specific Troponin 1 (cTnl) are the preferred biochemical markers for MI. levels of cTnT & cTnl start rising with 6 hours after acute MI and remain elevated for 7 to 10 days.
- Myoglobin is the first serum cardiac biomarkers to rise, but is nonspecific and comes back to normal within 24 hours of onset of infarction.
- Total quantity of protein released correlates with the size of the infarct. The peak protein concentration does not correlate well with infarct size.
- Reperfusion leads to earlier & higher peaking of cardiac enzymes.

Cardiac imaging:

- 2D Echocardiography may reveal wall motion abnormality and LV dysfunction. It may also detect RV infarction, ventricular aneurysms, pericardial effusion and LV thrombus.
- Myocardial perfusion imaging with $T1^{201}$ and Tc^{99m} – sestambi reveals a defect (cold spot) in most patients. It neither can nor however distinguish chronic scar from acute infarcts.

Ref:http://circ.ahajournals.org/

Definition of myocardial infarction

Criteria for acute myocardial infarction

The term myocardial infarction should be used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischaemia. Under these conditions any one of the following criteria meets the diagnosis for myocardial infarction:

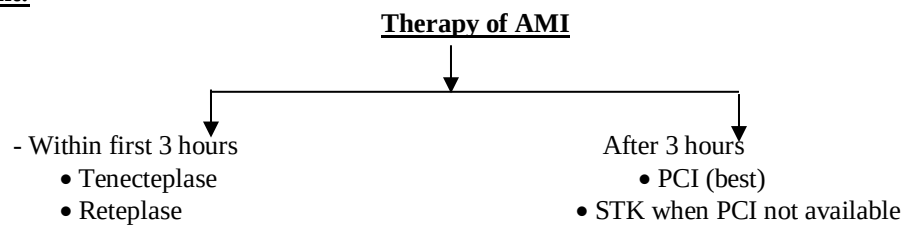
- Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit (URL) together with evidence of myocardial ischaemia with at least one of the following:
  - Symptoms of ischaemia;
  - ECG changes indicative of new ischaemia [new ST-T changes or new left bundle branch block (LBBB)];
  - Development of pathological Q waves in the ECG;
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Sudden, unexpected cardiac death, involving cardiac arrest, often with symptoms suggestive of myocardial ischaemia, and accompanied by presumably new ST elevation, or new LBBB, and/or evidence of fresh thrombus by coronary angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood.
- For percutaneous coronary interventions (PCI) in patients with normal baseline troponin values, elevations of cardiac biomarkers above the 99th percentile URL are indicative of peri-procedural myocardial necrosis. By convention, increases of biomarkers greater than 3 × 99th percentile URL have been designated as defining PCI-related myocardial infarction. A subtype related to a documented stent thrombosis is recognized.
- For coronary artery bypass grafting (CABG) in patients with normal baseline troponin values, elevations of cardiac biomarkers above the 99th percentile URL are indicative of peri-procedural myocardial necrosis. By convention, increases of biomarkers greater than 5 × 99th percentile URL plus either new pathological Q waves or new LBBB, or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium have been designated as defining CABG-related myocardial infarction.
- Pathological findings of an acute myocardial infarction.

Criteria for prior myocardial infarction

Any one of the following criteria meets the diagnosis for prior myocardial infarction:

- Development of new pathological Q waves with or without symptoms.
- Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischaemic cause.
- Pathological findings of a healed or healing myocardial infarction.

Treatment:



1. Nitrates – Sublingual or IV. NTG can be used. They are avoided in patients with hypotension (SBP < 90 mm hg) and evidence of RV infarction (infarction of ECG, raised JVP, clear lungs and hypotension).
2. Morphine
3. Beta blockers – Metoprolol 5mg IOV every 2-5 minutes for a total of three doses provided the patient has a heart rate > 60 bpm, systolic pressure > 100mm Hg, PR interval < 0.24 Sec and rales < 10cm above diaphragm.
4. ACE inhibitors
5. Aspirin
6. Clopidogrel
7. Primary Percutaneous Coronary Intervention (PCI) is more effective than fibrinolysis
8. Fibrinolysis-
    - Door to needle time ≤ 30 minutes.
    - tPA, TNK, rPA and STK can be used.
    - tpA – 15mg i.v. bolus, 50mg i.v. over 30 minutes, followed by 35 mg over the most 60 minutes.
    - rPA -10 MU bolus over 2-3 minutes followed by second 10 MU bolus 30 minutes later.
    - STK – 1.5 million units in i.v. infusion over 1 hour.
9. Bolus Fibrinolytics – TNK, RTP, concept of rescue PCI, Primary PCI and Facilitated PCI.

Reperfusion is assessed by Timi Flow

Grade	Flow pattern
0	No flow
1	Some penetration of contrast
2	Full penetration delayed
3	Normal flow

Reperfusion arrythmias – accelerated idioventricular rhythm (AIVR) is characteristic but it is not specific.

Contraindications to fibrinolysis

Absolute

- H/O cerbrovascular hemorrhage anytime in life
- Known structural cerebral vascular lesion (e.g., arteriovenous malformation)
- Known malignant intracranial neoplasm
- H/O non hemorrhagic stroke within 3 months except within 3 hrs
- Significant closed head or facial trauma within 3 mo
- Suspected aortic dissection
- Active internal bleeding (excluding menses)

Relative

- Marked hypertension
-

SBP>180mmHg
DBP>110mmHg → STK can be given

- Current use of anticoagulants (INR ≥ 2)
- Recent (< 2 weeks) invasiye or surfical procedure, prolonged (> 10 min) CPR
- Known bleeding diathesis
- Pregnancy
- Hemorrhagic ophthalmic condition.
- Active peptic ulcer disease
- H/o. Severe hypertension that is currently controlled
    - Patient should not receive STK if that agent has been used in preceding 5 days to 2 years.
    - Minor hypotension with STK can occur in 4-10 % of patients.
    - Hemorrhagic stroke is the most serious complication and occurs in 0.5 to 0.9% of patients. Treated with these agents. The risk is higher in elderly and higher with tpA ir rPA than with STK.
    - Rescue PCI should be considered in patients with failure of reperfusion (persistent chest pain and ST- segment elevation >90 minutes)

10. Heparin – More beneficial when used with fibrin-specific agents i.e. tpA and rPA
Patients with anterior infarction, severe LV dysfunction. CHF, a history of embolism, echo evidence of mural thrombus, atrial fibrillation should be given full therapeutic does of heparin followed by at least 3 months of warfarin therapy.
11. HMG CoA reductase inhibitors (statins) can be given to all patients for plaque stabilization.

Complications & their management
1. Ventricular dysfunction.
After STEMI ventricular remodeling occurs, can be prevented by ACE inhibitors and nitrates.
Killip classification.
Class I – No signs of pulmonary/venous congestion.
Class II – Moderate heart failure, rales at lung bases, S3 gallop, venous or hepatic congestion.
Class III- Severe heart failure, pulmonary edema
Class IV- Shock with S B P < 90 mm Hg, mental confusion, oliguria, peripheral cyanosis.
This classification has prognostic value with highest mortality in class IV patients.

2. Cardiogenic shock -
This results when there is infarction of ≥ 40% of left ventricle. The patient has

i. SBP<90 mm Hg

ii. Pulmonary capillary wedge pressure>18mm Hg
The management of cardiogenic shock is best carried out by invasive means (Primary PCI). Use of intra aortic balloon pump and i.v. vasopressors may be beneficial.

3. Right ventricular infarction

- Clinically significant RV infarction is rare and occurs in patients with interior infarction.
- ST elevation in V4 R is common in first 24 hours.
- Signs of severe RV failure jugular vinous distension. Kussmaul's sign, hepatomegaly, with hypotension may be present.
- 2-D echocardiography is useful for diagnosis and assessment of severity.
- Therapy consists of volume expansion to maintain adequate RV preload.

4. Pericarditis:

- Common in first week of transmural MI
- Radiation of pain to trapezius muscle is helpful in distinguishing it from ischemia.
- Pericardial rub may be present.

- Therapy is aspirin 650mg qid and withholding anticoagulants. Steroid is C/I.

5. Thromboembolism
   - 10% cases of STEMI have clinical evidence of thromboembolism
   - Many cases are clinically silent.
   - It may present as hemi paresis, marked hypertension (in case of renal artery involvement)
   - 2-D echocardiography may reveal mural thrombus.
   - Systemic anticoagulation for 3 to 6 months should be given in cases with clear evidence of thromboembolism.

6. Left ventricular aneurysm
   - True aneurysm is dyskinesis or local expansile paradoxical wall motion.
   - Most common site is apex
   - Double, diffuse or displaced apical impulse can be appreciated clinically.
   - Complications are CHF, arterial embolism and ventricular arrhythmias.
   - Pseudoaneurysm is a myocardial rupture contained by local area of pericardium along with organizing thrombus and hematoma.
   - Pseudoaneurysm should be surgically repaired if recognized.

7. Sinus bradycardia
   - Treatment is indicated if it causes hemodynamic compromise
   - I.V. atropine is given
   - Persistent bradycardia (< 40 / min) despite atropine is treated with electrical pacing.

8. AV Block
   - Prognosis is poorer in patients with anterior infarction and AV block as compared to inferior infarction and AV block.
   - Inferior infarction – due to increased vagal tone
   - Anterior infraction-ischemic malfunction of conduction system
   - Electrical pacing is required (in RV infarction dual-chamber AV sequential pacing may be required)

9. Ventricular tachycardia & fibrillation.
   - Prophylactic antiarrhythmic therapy is not recommended.
   - In hemodynamically unstable patient D.C. shock is given.
   - In stable patients i.v. amiodarone (150 mg bolus over 10 minutes, followed by infusion of 1.0 mg/min for 6 hours and then 0.5 mg/min) or procainamide can used.
   - Long term survival is good in patients with primary VF i.e. VF that is a primary response to acute; ischemia and not due to CHF, shock, bundle branch or ventricular aneurysm.

Patients with secondary VF have a poor long term survival and should be considered for electrophysiologic study and implantation of cardioverter defibrillator.

Risk factors after AMI

a) Persistent ischemia
b) Depressed LV ejection
c) Rales over lung bases
d) > 75 years age
e) Diabetes mellitus

UNSTABLE ANGINA AND NON-ST – ELEVATION MYOCARDIAL INFARCTION

Acute coronary syndrome has 3 types of presentations

- Unstable angina
- Non-ST-elevation myocardial infarction

– ST-elevation myocardial infarction

Unstable angina is defined as angina pectoris or equivalent ischemic discomfort with at least one of the 3 features.
1. It occurs at rest usually lasting > 10 minimum
2. It is severe and of new onset (< 6 weeks)
3. It occurs with crescendo pattern

NSTEMI = UNSTABLE ANGINE + ELEVATED CARDIAC BIOMARKERS

Pathophysiology - Four path physiologic processes have been identified.
1. Plaque rupture or erosion with superimposed thrombus (most common cause)
2. Dynamic obstruction (Prinzmetal's variant angina)
3. Progressive mechanical obstruction.
4. Secondary unstable angine (e.g.anemia)

White thrombi (Platelet rich) are more common in these patients. Red thrombi are more often seen in patients with acute STEMI.

Clinical presentation
- Chest pain, substernal is the hallmark.
- Diaphoresis, pale cool skin, sinus tachycardia, $3^{rd}/4^{th}$ heart sounds and sometime hypotension may occur.
- Hypotension may occur.

Electrocardiogram
- ST-segment depression, transient ST- segment elevation and/or T-wave inversion occur in 30 to 50% of patients.
- ST segment depression is an important predictor of adverse outcome.

Cardiac biomarkers
- Elevated CK-MB and troponins are predictors of increased risk for death or recurrent MI.
- Patients with elevated cardiac biomarkers are those with NSTEMI.
- C-reactive protein, B-type natriuretic peptide, and CD-40 ligand also correlate independently with adverse outcome.

Prognosis- TIMI risk score for UA/NSTEMI
- Age ≥ 65 years
- ≥ 3 CAD risk factors
- Prior stenosis > 50%
- ST deviation
- ≥ 2 anginal events ≤ 24 hours
- Aspirin use in last 7 days
- Elevated cardiac biomarkers
- 0-3 Mild risk
- 4-5 Moderate risk
- 6-7-High risk

Treatment

1. Nitrates – Initially given sublingually or by buccal spray .i.v. NTG is recommended is there is no relief.
   C/I - Hypotension
   - Use of sildenafil in last 24 hours.

2. Beta blockers – IV beta blocker followed by oral beta blockers targeted to a heart rate of 50-60 beats / min.
3. Morphine sulfate 1 to 5 mg iv. If there is no pain relief with NTG and Beta blockers.
4. Aspirin 162-325 mg nonenteric formulation stat followed by 75-160 mg/day
5. Clopidogrel 300 mg staff followed by 75 mg/day
6. Heparin
    - UFH-Bolus 60 -70 u/Kg i.v. followed by 12-15 u/kg sc every 12 hours
    - Enoxaparin – 1 mg/KG sc every 12 hours.
    - Dalteparin -120 u/kg sc every 12 hours.
7. GP IIb / IIIa inhibitors
    - Abciximab – it is useful in patients undergoing PCI
    - Tirofiban, Eptifibatide — useful in patients treated medically & those undergoing PCI
8. Calcium channel blockers are used.
    - Patients whose symptoms are not controlled with nitrates and beta blockers.
    - Patients unable to tolerate Beta blockers
    - Variant angina
9. Invasive strategy

In high risk patients coronary arteriography should be carried out within 48 hours of admission followed by revascularization (PCI or CABG) depending on the coronary anatomy Indications.

- Recurrent angina
- Elevated TnT or TnI
- New ST-segment depression
- CHF
- EF < 0.40
- Decreased BP
- Sustained VT
- PCI< 6months, pror CABG

10. Lipid management as in chronic stable angina
11. Lifestyle management.
12. ACE inhibitors

Thrombolysis is C/I.

PAINZMETAL'S VARIANT ANGINA

- This syndrome is due to a focal spasm of epicardial coronary artery, leading to serve myocardial ischemia.
- Younger patients with history of cigarette smoking.
- The clinical diagnosis is made with the detection of transient ST- segment elevation with rest pain.
- Coronary angiography is gold standard for diagnosis. Focal spasm commonly accompanied by stenosis within 1 cm of spasm is the hallmark (most commonly in right coronary artery)
- Ergonovine, acetylcholine and hyperventilation have been used to provoke and demonstrate focal spasm

o Treatment- Nitrates and Calcium Channels Blockers are the mainstay of treatment.
    - Prazosin can be useful
    - Aspirin and Beta blockers are avoided.
o Long term survival is excellent

STABLE ANGINA PECTORIS.

- It is due to transient myocardial ischemia
- Typical chest pain lasting 2-5 minutes. Levine's sign may be positive.

- Pain can radiate to left shoulder and to both arms, especially to the ulnar surfaces of the forearm and hand. Angina is rarely localized below the umbilicus or above the mandible.
- Typically caused by exertion or emotion.
- Relieved by rest and sublingual nitroglycerine.
- The severity can be expressed by the Canadian cardiac society functional classification.
- On examination, during an anginal episode presence of third/fourth heart sound, dyskinetic cardiac apex, mitral regurgitation and even pulmonary edema may be appreciated.

o Lab examination-Hematocrit, blood glucose, creatinine, lipid profile, chest x-ray should be done in all cases.

o ECG-It is normal in half of angina. Typical ST segment and T-wave changes that accompany the episodes of angina pectoris and disappear there after are more specific.

o Treadmill Testing
- Most widely used test for diagnosis of IHD
- Recording of ECG before, during, and after exercise.
- Standard Bruce Protocol is followed
- Ischemic ST response is destined as that depression of ST segment >0.1 mv below baseline and lasting longer than 0.08 sec.
- 85% of maximal heart rate for age & sex should be achieved, otherwise exercise test is considered nondiagnostic.
- When interpreting ECG stress test, the probability that CAD exists in the patient i.e. pretest probability should be considered.
- False positive test is common in
    - Men < 40 years
    - Premenopausal women
    - Patients on cardio active drugs e.g digoxin;
    - Resting ECG abnormalities
    - Ventricular hypertrophy
    - Abnormal serum potassium levels.
- False negative test result can occur in obstructive disease of left circumflex coronary artery.
- Contraindications
    - Acute systemic infection
    - Acute MI ≤ in 48 hours
    - Uncontrolled cardiac arrhythmia
    - Severe symptomatic aortic stenosis
    - Acute myocarditis, pericarditis
    - Uncontrolled heart failure
    - Acute infective endocarditis
    - Acute aortic dissection
    - Acute pulmonary embolism or pulmonary infarction

– Exercise test can be safely performed in patients as early at 6 days after uncomplicated myocardial infarction.

o Cardiac imaging.

– When resting ECG is abnormal (e.g. WPW, >1mm ST segment depression, LBBB, paced ventricular rhythm) stress myocardial perfusion imaging after i.v. administration of thallium 201 or technetium 99 m sestamibi using exercise or pharmacologic stress in useful.

– IV dipyridamole or adenosine can be used in place of exercise.

– Stress (exercise or dobutamine) echocardiography can also be used.

– Advantages of stress echocardiography
1. Higher specificity
2. Versatility
3. Greater convenience

4. Lower cost

Advantages of stress perfusion imaging

1. Higher sensitivity
2. Higher technical success rate
3. Better accuracy when multiple resting L.V. regional wall motion abnormalities present

- Coronary angiography is indicated in
  - Severely symptomatic patient who are candidates for revascularization
  - Diagnostic difficulty on noninvasive tests
  - Survivors of cardiac arrest
  - Ischemia with left ventricular dysfunction
  - High risk of future coronary event

Prognosis

Critical stenosis (> 70%) of coronary arteries and 5 year mortality rate
One artery – 2%
Two arteries – 8%
Three arteries – 11%

50% stenosis of left main coronary artery has a mortality rate of about 15% per year

- Other poor prognostic factors are
  - advanced age (> 75 years)
  - Diabetes
  - Morbid obesity
  - Accompanying peripheral vascular and or cerebrovascular disease
  - Previous myocardial infraction
  - High levels of plasma CRP
  - Evidence of LV dysfunction

Treatment

1. Identification & treatment of aggravating conditions like aortic stenosis, hypertrophic cardiomyopoathy, hypertension and obesity
2. Lifestyle modification
3. Aspirin
4. β Blockers
5. Calcium antagonists
   - If β blockers are contraindicated
   - With β blockers to control symptoms
6. Sublingual nitroglycerin
7. Lipid lowering (LDL < 100 mg / dl)
8. CABG is indicated in
   - Significant left main disease
   - TVD/DVD with reduced LV function
   - DVD with LAD DISEASE
   - Diabetes
   - Prior CABG PCI with recurrent restenosis
   - Abnormal stress test
9. PCI is indicated when
   - One to three vessel diseases with normal LV function
   - Lesion suitable for PCI
   - No diabetes

Hyperlipoproteinemias

Type I : Increased chylomicrons
Raised levels of Triglycerides
Patients can be asymptomatic
At Tg > 1000 mg / dl: - Eruptive xanthomas, small orange – red papules can appear on trunk and extremities
- Lipemia retinalis can occur
- Pancreatitis is the major risk

Type II
II a – Isolated hypercholesterolemia
Raised LDL
Tendon Xanthoma, tuberous xanthomas and xanthelesma are common
IIb- Raised VLDL & IDL
Elevation of triglycerides & cholesterol
Patients are usually asymptomatic until vascular disease develops
Familial combined hyperlipidemia is the commonest lipid disorder

Type III
Raised VLDL, LDL
LDL is normal
Patient has elevated total cholesterol and triglycerides. They are usually asymptomatic until vascular disease develops. Patients may have palmar or **tubero eruptive xanthomas**.

Type IV: Increased levels of VLDL, raised levels of triglycerides
Patients are generally asymptomatic, increased risk of vascular disease
Type V: Raised VLDL and chylomicrons, symptoms and manifestations are similar to type I

LDL Colesterol Goals and Cut points for Therapeutic Lifestyle Changes (TLC) and Drug therapy in Different Risk Categories

Risk Category	LDL Goal (mg/dl)	LDL level of which to initiate TLC (mg /dl)	LDL level at which to consider Drug therapy (mg/dl)
CHD or CHD risk Equivalents	< 100	≥ 100	≥ 130 (drug optional between 100 and 129)
2 + Risk Factors	< 130	≥130	≥ 160 (drug optional between 130 and 159)
0-2 Risk factors	< 160	≥ 160	≥ 190 (drug optional between 160 and 189)

Clinical Identification of the metabolic syndrome – Any three Risk Factors

Risk Factor	**Defining level**
Abdominal obesity (waist circumference) Men Women	 > 40 inches > 35 inches
Triglycerides	> 150 mg/dl
HDL Cholesterol Men Women	 < 40 mg/dl < 50 mg/dl
Blood pressure	> 130/85 mmHg
Fasting glucose	> 110 mg/dl

Type 2 diabetes
Overall the risk of type 2 diabetes in patients with the metabolic syndrome is increased 3 to 5 fold.
In the FOS,s 8 years follow up of middle aged men and women, the population attributable risk for developing type 2 diabetes was 62% in men and 47% in women.

Nonalcoholic fatty liver disease
Fatty liver is relatively common
Both triglyceride accumulation and inflammation coexist.

Hyperuricemia
It reflects defects in insulin action on the renal tubular reabsorption of uric acid wheas the increase in asymmetric dimethylarginine an endogenous inhibitor of nitric oxide synthase relates to endothelial dysfunction.

Polycystic ovary syndrome
It is highly associated with the metabolic syndrome with a prevalence between 40 & 50%

Obstructive sleep apnea
OSA is with obesity hypertension increased circulating cytochines IGT and insulin resistance.

Bariatric surgery is an option for patients with the metabolic syndrome who have a body mass index (BMI) of >40 kg/m2 or > 35 kg/m2 with comorbidities. Gastric bypass result in a dramatic weight reduction and improvement in the features of metabolic syndrome. At present, however, a survival benefit has yet to be realized.

RHEUMATIC FEVER:
Group A streptococci (Lancefield classification – 3% develop RF
Peak incidence 5-15 years of age more common in socially & economically backward population
Serotype 1,3,5,6,18 are most commonly associated

2002–2003 World Health Organization Criteria for the Diagnosis of Rheumatic Fever and Rheumatic Heart Disease (Based on the 1992 Revised Jones Criteria:

MAJOR(Mn:CASES)	*MINOR(Mn:FLEAP)*
• **Carditis** • **Arthritis(Poly)** • **Sydenham Chorea** • **Erythema marginatum** • **Subcutaneous nodules**	• **Clinical: Fever, Arthralgia(poly)** • **Laboratory: ↑ESRor *Leucocytes*** **(previously it was CRP instead of *Leucocytes* (WBC)** • **Electrocardiogram: Prolonged P-R interval**

Essential criteria: Supporting evidence of a preceding streptococcal infection within the last 45 days

- **Elevated or rising anti-streptolysin O or other streptococcal antibody, *or***
- **A positive throat culture, *or***
- **Rapid antigen test for group A streptococcus, *or***
- **Recent scarlet fever[e] *(It was not there in previous criteria in 1992)***

Diagnostic Categories	Criteria
1.Primary episode of rheumatic fever[a]	**Two major or one major and two minor criteria PLUS Essential criteria**
2. Recurrent attack in a patient without established rheumatic heart disease	**DO i.e. (Two major or one major and two minor criteria PLUS Essential criteria)**
3.Recurrent attack in a patient with established rheumatic heart disease[b]	**Two minor manifestations plus Essential criteria[c]**
4. • Rheumatic chorea • Insidious onset rheumatic carditis[b]	**Other major criteria or Essential criteria NOT required**
5. Chronic valve lesions of rheumatic heart disease (patients presenting for the first time with pure mitral stenosis or mixed mitral valve disease and/or aortic valve disease)[d]	**Do not require any other criteria to be diagnosed as having rheumatic heart disease**

[a]Patients may present with polyarthritis (or with only polyarthralgia or monoarthritis) and with several (3 or more) other minor manifestations, together with evidence of recent group A streptococcal infection. Some of these cases may later turn out to be rheumatic fever. It is prudent to consider them as cases of "probable rheumatic fever" (once other diagnoses are excluded) and advise regular secondary prophylaxis. Such patients require close follow up and regular examination of the heart. This cautious approach is particularly suitable for patients in vulnerable age groups in high incidence settings.
[b]Infective endocarditis should be excluded.
[c]Some patients with recurrent attacks may not fulfil these criteria.
[d]Congenital heart disease should be excluded.

Jones criteria for RF :
Major criteria

1. Carditis – seen in 50 – 60%
2. Migratory polyarthritis → most common, and least specific (70%)
3. Sydenham's chorea → 20% involves, caudate lobe
4. Subcutaneous modules
5. Erythema marginatium 3 – 5%

- Migratory polyarthritis is the most common presentation (75% of patients)

- Mitral valve involvement is the most common (Mitral regurgitation is the earliest lesion), followed by aortic valve
- Rhyeumatic pericardities does not lead to constrictive pericarditis
- Sydenham's chorea, occurs several months after initial streptococcal infection
- 80% of the patients with acute rheumatic fever have raised antistreptolysin O titer at presentation and 95% if two other tests are used.

Treatment and Prevention of RHD

Primary prevention

1) Benzathine Penicillin > 27 kg – 1.2 MU deep I m once
2) Oral penicillin V – 500 mg BD X 10 days
3) Allergic – Erythromycin – 40 mg /kg x 12 days

Secondary

Benzathine penicillin 1.2 MU deep IM every 3 weeks

Sulfadiazine 1 gm → For

Pencicillin V 250 mg BD → for

Allergic – Erythromycin

Duration of prevention

1) No carditis – 5 years or age 20
2) Carditis, no residual valve disease → 10 years or upto 40 years
3) Carditis + residual valve disease → preferably life long

Therapy of manifestations

1) Salicylates – 100 – 120 mg/ kg X 12weeks
2) Steroids - in severe carditis and CCF

Major Criteria

1) Cardities = Pancarditis manifestations are
    a. Valvulitis murmur 9carrey combs)
    b. S_3
    c. Cardiomegaly
    d. Pericardial Rub
        - Most specific
        - Mitral valve most common followed by aortic
        - Tricuspid – Rarely pulmonary
        - Sole diagnosis by Echo not recommended
        - No constriction seen
2) Arthritis
    a. Migratory polyarthritis
    b. Painful
    c. Do not involve small joints, rarely hip joint
    d. Marginal response to salicylates
    e. Lasts 2 – 3 weeks
3) Chorea : Late manifestation
    Diagnosis of RF can be made solely from S.C.
4) Subcut Nodules: - firm, Painless
    Extensor surface
    Overlying skin normal
5) Erythema Marginatum - evanescent
        - Macular and non pruritic
        - Not on face

- Pale centre serpiginous

Pathology

- Aschoff's nodules – Hall mark of RF
- Fibrinoid degeneration of collagen
- Not seen in extracardiac sites
- Found in 30 – 40 % cases

DETECTION

- Direct isolation of GAS by 3 throat swabs
- Serological tests – ASO, Anti DNASeB

Anti Hyaluronidase

4) VALVULAR HEART DISEASE

MITRAL STENOSIS

Most common etiology is rheumatic heart disease

More common in females

Critical MS is valve area < 0.6 cm^2 / m^2 BSA

Manifestations: Principal symptoms are dyspnoea and pulmonary edema precipitated by fever, excitement, anaemia pregnancy etc.

- Palpable S1; opening snap (OS /follows A2 by 0.05 to 0.12 sec)
  - A2 – OS interval is inversely proportional to severity of obstruction
  - Diastolic rumbling murmur with presystolic accentuation
  - Duration of the murmur correlates with severity of obstruction

ECG – Atrial fibrillation, LA enlargement when sinus rhythm is present, RAD and RVH when pulmonary HT is present.;

CXR – LA and RV enlargement and kerley B lines

Echocardiogram – Inadequate separation, calcification and thickening of valves. Valve area can b calculated

ECHO Grading in MS

- Valve thickening
- Valve mobility
- Valve calcification
- Sub valvular apparatus

Max score = 3 x 4 = 12

Score <8 = BMV

>8 = MVR

Lutembacher syndrome: Mitral stenosis with ASD

Treatment

1. Prophylaxis for rheumatic fever and infective endocarditis
2. medical therapy for heart failure
3. Digitalis, beta blockers, or verapamil to slow ventricular rate in AF
4. In uncomplicated MS, percutenous balloon mitral valvuloplasty is the procedure of choice (C/I in grade II MR, Calcification, LA Clot)
5. Mitral valve replacement

MITRAL REGURGITATION

RHD, mitral value prolapse, IHD with papillary muscle dysfunction, hypertrophic cardiomyopathy, infective endocarditis, congenital

Manifestations

Fatigue, weakness and exertional dyspnoea
Soft S1, loud holesystolic murmur
S3 are common findings
Hyperdynamic apex

Echocardiogram: Enlarged LA: Doppler is helpful in diagnosing and assessing severity of MR.

Treatment:

1. Diuretics and digoxin
2. After load reduction (ACE inhibitors, hydralazine on iv nitroprusside)
3. Prophylaxis for endocarditis
4. Surgical treatment, either mitral valve repair or replacement in the presence of symptoms or evidence of LV dysfunction
    a. LVEF < 60% or
    b. End – systolic LV diameter > 50 mm/m^2

MITRAL VALVE PROLAPSE

Idiopathic, Marfan syndrome, redundant mitral value tissue with myxomatous degeneration and elongated chordae tendinea

Manifestation

- More common in females
- Most patients are asymptomatic
- Atypical chest pain and SV and ventricular arrhythmias are common
- Most important complication is severe MR resulting in LV failure
- Sudden death is very rate complication
- Mid or late systolic click followed by late systolic murmur exaggerated by valsalva and standing.

Echocardiagram – displacement of mitral leaflect (anterior or posterior or both) more than 2mm behind the mitral annulus in left atrium.

Treatment

1. Asymptomatic pts should be reassured
2. Prophylaxis for IE in pts with significant MR
3. Valve repair on replacement for pts with severe MR

AORTIC STENOSIS

1. Types
a. Congenital
b. RHD, senile
2. Level of Stenosis
   a. sub valvular
   b. Valvular
   c. Supra valvular

Congenital, RHD, Idiopathic calcification AS in elderly –more common in males

Manifestations

- Dyspnoea, angina, and syncope are cardinal symptoms
- Heaving apex, soft A2, S4 common, crescendo – decrescendo systolic murmur

Cardinal feature – Soft A_2

Pulsus parvus et tardus

S_3 uncommon: paradoxical splitting

Associated mitral valve disease – almost always points towards RHD

Gallavardin's dissociation

ECG & CSR – LV hypertrophy – post stenotic dilatation and calcification of valves
Echocardiogram – Thickening of LV wall, and thickening of aortic value cusps.
Critical AS, valve area < 0.5 cm^2 /M^2 BSA – Eccentric value closure in bicuspid valves

Treatment

1. Symptoms are the only guide towards operation owing to enhanced mortality in asymptomatic patients.
2. Avoid strenuous activity
3. Treat heart failure but avoid after load reduction
4. Surgical replacement of valve is indicated in
    - Symptomatic patients with angina, syncope on heart failure
    - Valve area < 0.5 cm^2/ m^2
    - Peak systolic pressure gradient > 50 mm Hg
5. HMG CoA reductase inhibitors are useful in retarding the progression of calcific aortic stenosis
6. Balloon aortic valve plasty is useful in
    a. Congenital aortic stenosis
    b. Patients of aortic stenosis unfit for surgery
    c. As a bridge between medical treatment and surgery

Prognosis: In patients not Treated surgically the average time to death after onset of various symptoms is
Angina – 5 Years
Syncope – 3 years
Dyspoea – 2 Years
CHF – 1.5 – 2 years

AORTIC REGURGITATION

More common in males, rheumatic in 70%, syphilis, infective endocarditis are other causes

Manifestations

Extertional dyspnoea and awareness of heart bear angina pectoris and signs of LV failure collapsing pulse, Quincke's sign, and de Musset's signs pistol shots over femoral artery are common
Decrescendo, diastolic, blowing murmur along left sternal border. Austin flint's murmur is heard in moderate to severe AR
ECG & CXR – signs of LV enlargement

Echocardiogram

LV & LA enlargement Doppler studies are careful in detection and quantification of AR

Treatment

1. Standard therapy for LV failure
2. Surgical therapy is indicated in
    - Symptomatic patient
    - Asymptomatic pts with LV dysfunction
        1. Surgical therapy is indicated in
    - Symptomatic patient
    - Asymptomatic pts with LV dysfunction
        1. LVEF < 50%

2. LV end systolic volume > 55 ml / m^2

TRICUSPID STENOSIS

Usually rheumatic more common in females almost always associated with MS
Manifestations
Hepatomegaly, ascites, edema, jaundice raised JVP Diastolic rumbling murmur along left sternal border increased by inspiration with loud presystolic component
TS:

Giant 'a' wave
Slow 'y' descent
Pre systolic liver pulsation
Functional TS- ASD
Treatment valve replacement

TRICUSPID REGURGITATIION
Usually functional and associated with RV dilation and pulmonary hypertension

Manifestations
Severe RV failure, edema hepatomegaly prominent V waves and rapid y descent systolic murmur along sternal edge is increased by inspiration (Carvallo's sign)
Intensive diuretic therapy. In severe cases, tricuspid annuloplasty or valve replacement

TR – Cardinal feature are
1) Prominent C –V wave and deep y descent
2) Systolic pulsation of liver
3) Increase of murmur on inspiration

Condition where these features are not present – Ebstein's anomaly
1) Carcinoid
2) I.V. drug abuse
3) Endorcardial cushion
4) Ebstein anomaly
5) Combined TR + TS is seen with primary valve involvement by RF

CONGENITAL HEART DISEASE

ATRIAL SEPTAL DEFECT
Usually asymptomatic until third or fourth decade. More common in females
Ostium secundum is the most common type
Manifestations: Exertional dyspnoea, fatigue, and palpitations
Parasternal RV lift, wide fixed splitting of S2 diastolic murmur in tricuspid area, prominent but equal 'a' & 'v'wave in JVP.

ECG – Incomplete RBBB with RAD in ostium secundum type
LAD – OSTIUM PRIMUM DETECT, 50% had increased PR interval
CXR – increased pulmonary vascular markings prominence of RV and main pulmonary artery
Echocardiogram – RA and RV enlargement Transatrial flow can be seen by Doppler and contract echo.

Treatment
1. ASD with pulomanry-to-systemic flow ratio (PF:SF)>2.0 should be surgically repaired.
2. ASD closure device
3. medical management for atrial fibrillation and CHF

COARCTATION OF AORTA
Aortic constriction just distal to the origin of left subclavian artery
More common in males
Commonly associated with bicuspid aortic valve and aneurysms of circle of willis

Manifestations
Headache, fatigue, claudicating of the lower extremities

Hypertension in upper limbs, radio femoral delay, pustule collaterals in the intercostals spaces, systolic murmur over the mid – upper back

ECG –LV hypertrophy

CXR – Notching of ribs

"Figure 3" appearance of distal aortic arch

Treatment

1. Surgical correction
2. Coarctoplasty
3. Antibiotic Prophylaxis against endocarditic is required even after correction

INFECTIVE ENDOCARDITIS

- Acute IE = STAPH AUREUS
- Neonatal IE = Normal Tricuspid valves
- Common congenital heart Diseases associated with IE= →VSD, PDA and bicuspid aortic valve
- RHD = Mitral valve followed by aortic valve
- IE in HIV – Salmonella sp and streptococcus pneumonia
- Prosthetic valve Endocarditis – Coagulase (1) negative staph in early (2)

Streptococcus in late

Acute – hectic febrile illness associated with damage to cardiac structures, seeding of extracardiac sites, and progression to death within weeks if left untreated.

Subacute – indolent illness associated with slow or no destruction of cardiac structures and rate metastatic infection.

Etiology

Acute	-	Staphylococcus aureus
Subacute	-	Streptococcus viridans
Early PVE	-	Coagulase negative staphylococcus
IV drug user	-	Staphylococcus aureus
Late onset PVE	-	Streptococcus Viridans
Fungal causes	-	Candida, Histoplasma, Aspergillus

Duke Criteria

Pathological criteria – demonstration of micro – organism by culture and histology

Clinical criteria

Major criteria:-

1) Blood culture – Typical micro organisms from 2 separate blood cultures

Single positive culture far C. Burnetii

Endocardial involvement

1) Oscillating intra cardiac mass
2) Abscess
3) Vavular Regurgitation

Minor

1. Predisposition
2. Fever ≥38.0°C - most common sign and symptom
3. Vascular phenomenon – arterial embolic, ICH myotic aneurysm
4. Immunologic phenomenon –GN, Osler node, Roth spots R factors
5. Microbiologic evidence
6. Echocardiogram

Clinical diagnosis: 2 major criteria

or 1 major + 3 minor criteria
or 5 minor criteria

Roth's spots – collegen vascular disease + Anemia
Osler's nodes – Tendia sub cut nodules
Janeway lesions – non tender, hemorrhage, macular

Trans thoracic Echocardiography has a sensitivity of about 65% & can detect vegetation as small as 2mm
Trans – esophageal echocardiography has a sensitivity of about 90% & can detect vegetations as small as 1mm

HACEK group (Haemophillus, Actionobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kengae) are important cause of culture – negative endocarditis.

Lab Investigations
Serological tests useful in Bartonella, Chlamydia and C. Burnetti
ESR – elevated
Urinalysis – Proteinuria and hematuria

Treatment
1. Effective antibiotic therapy
2. Surgery is required in
3. Fistula in pericardial sac
4. Moderate to sever CHF due to valve dysfunction
5. Unstable prosthetic valve
6. Relapse PVE
7. S. aureus PVE with intracardiac complications
8. Fungal or brucella endocarditis
9. Persistent bacteremia despite optimal antimicrobial therapy

VIRIDANS STREPTOCOCCI NUE
- Aqueous penicillin BG – 12 – 18 million – 4 -6 weeks
- CEFTRIAXONE- 2 gm once – 4 weeks
- Penicillin + Gentamicin – weeks

ENTEROCOCCI – Penicillin G + Gentamicin 4-6 weeks
Vancomycin + Gentamicin – 4 – 6 weeks

STAPHYLOCOCCUS
Methicillin sensitive- Nafcillin or oxacillin – 4 – 6 weeks
Methicillin resistant – vancomycin
Prosthetic valves – Addition of Rifampicin is most
HACEK – CEFTRIAXONE – 2 gm once daily x 4 weeks

Cardiac lesions for which antibiotic prohylaxis is advised
High Risk
1. Prosthetic heart valves
2. Prior bacterial endocarditis
3. Complex Cyanotic congenital heart disease
4. Surgically constructed systemic – pulmonary shunts
5. Repaired shunts within 6 months
6. Valvulopathy after cardiac transplantation
7.

Procedures for which endocarditis prophylaxis is advised in patient at high or moderate risk for endocarditis

1. Dental procedures: extraction, periodontal procedures, implant, placement, root canal instrumentation, intra ligamentary injections (anesthetic)
2. Respiratory procedures; bronchoscopy with a rigid bronchoscope operations involving the muscosa
3. Gastrointestinal procedures; sclerotherapy of oesophageal varices, stricture dilation, ERCP, biliary tract surgery, surgery involving mucosa
4. Genitourinary procedures: urethral dilation, prostate or urethral surgery cystoscopy

Antibiotic regiment for prophylaxis of endocarditis in adults at
Moderate of the high risk

- Congenital cardiac malformation, ventricular septal defect, bicuspid Aortic valve
- acquired aortic and mitral valve dysfunction
- Hypertrophic cardiomyopathy (Asymmetric septal hypertrophy)
- MVP with valvular regurgitation and / or thickened leaflets

Low risk

1. O.S.A.SD
2. CABG
3. Pacemeaker
4. ICD
5. MVP without MR
6. Repaired shunts

Procedures for which endocarditis prophylaxis is advised in patient at high or moderate risk for endocarditis

1. Dental procedures: Extraction, periodontal procedures, implant, placement root canal instrumentation intra ligamentary injections (anesthetic)
2. respiratory procedure: Bronchoscopy with a rigid bronchoscope operations involving the mucosa
3. Gastrointestinal procedures: sclerotherapy of oesophageal varices, stricture dilation, ERCP biliary tract surgery, surgery involving mucosa
4. Genitourinary procedures: urethral dilation, prostate or urethral surgery cystoscopy

Antibiotic Regiment for prophylaxis of Endocarditis in Adults at

Note: for patients at high risk administer a half –dose 6 house after the initial dose

II. Genitourinary and Gastrointestinal tract procedure

A. High – risk patients

1. Ampicillin 2.0 gm IV or 1M plus gentamicin 1.5mg/kg IV or 1M within

30 minutes of procedure, repeat ampicillian, 1.0gm iv or im or amoxicillin 1.0 gm PO 6th later

B. High risk penicillin a- allergic patients

1. Vancomycin 1.0 gm IV or 1M over 1-2 h plus gentamicin 1.5 mg/kg IV or 1M with ub 30 minutes before procedure, no second dose recommended.

C. Moderate risk patients

1. Amoxicillin 2.0 gm PO 1h before procedure or ampicillin 2.0 gm IV or 1M 30 minutes before procedure

D. Moderate risk, penicillin – allergic patients

1. Vancomycin 1.0 gm IV inferred over 1-2 hours and completed within 30 minutes of procedures.

DISEASES OF AORTA:

Aortic Aneurysm:

Abnormal widening of abdominal or thoracic aorta
Most common site is infra renal abdominal aorta

Most common cause of ascending aorta aneurysm is Marfan's syndrome (Fibrillin effect)
Most common cause of aortic aneurysms is atherosclerosis.

Manifestations:

Thoracic aneurysms may result in deep, diffuse chest pain, dysphagia, hoarsensess, dry cough
Abdominal aneurysms may result in abdominal pain or thromboemboli to the lower limbs.
On examination, abdominal aneurysms can be palpated as a pulsatile mass between xiphoid process and umbilicus.

Investigation
CXR: Enlarged aortic silhouette (thoracic aneurysm), rim of calcification (abdominal aneurysms)
USG: best screening test
CT scan – best to monitor the size
MRI – test of choice

Treatment:

1. Strict control of hypertension.
2. Surgical resection of the aneurysm is indicated for
    a) Thoracic aneurysm > 6 cm in size.
    b) Abdominal aneurysm > 5.5 cm in size
    c) Persistent pain despite BP control.
    d) Evidence of rapid expansion
    e) In patients with Marfan's syndrome thoracic aortic aneurysm > 5 cm

Aortic Dissection:

It is a potentially life threatening condition which disruption of aortic intima allows dissection of blood into vessel wall.

DeBakey classification –

Type I – Dissection involves ascending to descending aorta.
Type II- Dissection is limited to ascending aorta.
Type III – Dissection involves descending aorta only.

Stanford's Classification –

Type A – Dissections that involve ascending aorta independent of the site of tear and distal extension
Type –B Dissections involve transverse and / or descending aorta without involving ascending aorta.
The peak incidence of aortic dissection is in the sixth and seventy decades of life with men affected twice as often as women.

Proximal aortic dissection is more common. Ascending aortic dissection is associated with hypertension, cystic medial necrosis, the Marfan syndrome.
Descending dissections are commonly associated with atherosclerosis or hypertension.
Incidence is increased in patients with coarctation of aorta, bicuspid aortic valve, and rarely in third trimester of pregnancy in otherwise normal women.

Manifestations:
Sudden onset of severe anterior or posterior chest pain, with "ripping" quality. Additional symptoms relate to the obstruction of aortic branches (stroke, MI), dyspnoea (acute aortic regulation), or symptoms of low cardiac output due to cardiac tamponade (dissection into pericardial sac).
Sinus Tachycardia is common. Hypotension, pulsus paradoxus, and pericardial rub in cases of cardiac tamponade. Asymmetry of carotid or brachial pulses, aortic regurgitation, and neurological abnormalities associated with interruption of carotid artery flow are common findings.
CSR – widening of the aortic silhouette is the most common abnormally " calcium sign" can be seen. The remarkably high accuracy of MRI has made it the current "gold standard" for diagnosing the presence or absence of aortic dissection.
Trans – esophageal echocardiography should be considered first in suspected aortic dissection other important investigations are angiography and CT Scan.

Treatment
1. Surgical
    a. Treatment of choice for acute proximal dissection
    b. Treatment for acute distal dissection complicated by
        i. Prognosis with vital organ compromise
        ii. Rupture or impending rupture
        iii. Retrograde extension into the ascending aorta
        iv. Marfan's syndrome

2. Medical treatment involves use of IV nitroprusside and IV beta blockers (keeping systolic BP between 110 & 120 mm Hg and heart rate around 60 bpm)

Indications
- Treatment of choice for uncomplicated distal dissection
- Treatment for stable, isolated arch dissection
- Treatment of choice for stable chronic dissection (. 2 weeks)

Takayasu's Disease
- Idiopathic large- vessel vasculitis in young individuals
- Women are affected 10 times more often than men
- It is also known as pulseless disease
- Anorexia, weight loss, fever and night sweats occur, cerebral ischemia, hypertension, loss of pulses in arms is seen
- Glucocorticoid & immunosuppressive therapy may be beneficial, but mortality is high

Non Specific Aorto Arteries
- Incidence mare in females than males
- Involvement of large sized vessels alike giant cell arteritis
- Involvement of ascending aorta and its branches
- Involves origin of arteries, Tends to spare inferior mesenteric artery
- Pan arteritis – Granulomatous with giant cell formation.

Classification
Type I – Ascending aorta

Type II - Descending aorta
Type III – both
Type IV – Pulmonary artery
Type V – Coronary Arteries

- Manifestations defined on which artery is compromised
- Ocular manifestation = "wreath like anastomose in retinal vessels"
- Criteria for diagnosis – ACR criteria
- Diagnosis - Hypertension with multiple bruits
    a. MR angiography
    b. Aorto Graphy with DSA
- High ESR and leucocytosis in active
- Therapy = Medical – Imuno suppressants

Surgical – Angio plasty and
Stent placement

Cardiac Tumours

Myxoma

- Most common type of primary cardiac tumour in adults
- Seen in third to sixth decade of life
- Most common in females
- More are sporadic but some are familial (autosomal dominant)
- Most are solitary and located in left atrium septum near fossa ovalis
- The most common clinical presentation mimics that of mitral valve disease
- "Tumour plop" is a characteristics low - pitched sound which may be audible during early or mid diastole and is thought to result from the tumor abruptly stopping as it strikes the ventricular wall.
- Peripheral or pulmonary embolisation is seen
- Constitutional signs & symptoms include fever, weight loss, cachexia, malaise arthralgia, rash, clubbing, Raynaud's phenomenon anaemia, polycythemia elevated ESR, thrombocytopenia, or thrombocytosis.
- 2 – D echocardiography CT AND MRI are useful investigations

Treatment is surgical e4xcision using cardiopulmonary bypass

Others tumours

- Sarcomas are most common primary malignant tumours of the heart. They are generally right sided and carry a poor prognosis. Angiosarcomas
- Tumours metastatic to heart are many times more common than primary tumours
- In absolute number most common primary originating sites of cardiac metastases are carcinoma of the heart and lung.
- The relative incidence of cardiac metastasis is highest in malignant melanoma
- Cardiac metastases rarely result in clinical manifestation and very rarely are the cause of death

Treatment of cardiac metastases is palliative

- Rhabdomyomas are commonest cardiac tumor in children

HYPERTENSION

- Most common form is essential hypertension 92 -94%
- Most common cause of secondary hypertension is renal parenchymal disease
- Environmental factors – salt intake, obesity, occupation, alcohol intake, family size & crowding.
- 60% of hypertensive are salt sensitive. Primary aldosteronism, bilateral renal artery stenosis, renal parenchymal disease and low rennin essential hypertension are all salt sensitive
- Low rennin hypertension is more common in elderly & diabetics. These patients are salt sensitive and diuretics responsive.
- Normal rennin hypertension (non modulators) is more common in males and post menopausal females. They are salt sensitive.

- High rennin hypertension is characterized by high plasma rennin activity and responsiveness to amgiotensin II antagonists.
- Low calcium intake has been associated with an increase in blood pressure in epidemiologic studies
- Insulin resistance is responsible in some patients with hypertension

Syndrome X is characterized by presence of any 3 out of the following

1. Abdominal obesity - > 40 in. in men
   - > 35 in. in women
2. Hypertriglyceridemia (> 150 mg/dl)
3. HDL cholesterol -< 40 mg / dl in men
   - < 50 mg / dl in women
4. Blood pressure ≥130 /85 mm Hg
5. Fasting glucose ≥ 110 mg/dl

Risk Factors

Smoking	**TOD**
Dyslipidemia	Heart LVI + HF Angina
Diabetes mellitus	Stroke
Age> 60 years	Nephropathy
Sex	PAD
Family History	Retinopathy

JNC – VII

- Concept of pre hypertension – 120-139 systolic
  80-89 mm Diastolic
- Normal < 120 mmHg systolic
  < 80 mm Hg diastolic
- Importance of Therapeutic life style modification in prehypertension
- Stage I – 140 – 159 mmHg systolic
  90-99 mmHg diastolic
- Stage II - > 160 mmHg systolic
  > 100 mmHg diastolic

DASH – Dietary Advice to stop Hypertension

- Glucocorticoid remediable hypertension (11-B hydroxylose give defect)
- Elevated aldosterone activity with normal aldosterone levels.
- Syndrome of apparent mineralocorticoid excess (defect in renal 11 B-hydroxy steroid dehydrogenase)

- Secondary hypertension
  1. Renal causes
     - Renal parenchymal disease
     - Reno vascular steno sis
  2. Endocrine
     - Oral contraceptives
     - Cushing's disease
     - Primary hyperaldosteronism
     - Pheochromocytoma
     - Hyperthyroidism & hypothyroidism

- Acromegaly
- Hyperparathyroidism

3. Neurologic
   - Psychogenic
   - Familial dysautonomia (Riley – Day)
   - Polyneuritis
   - Acute spinal cord section – GB syndrome
4. Others
   - Coarctation of aorta
   - Polycythemia vera
   - Drugs e.g. cyclosporine, glucocorticoids
   - Toxemia of pregnancy
   - Acute intermittent porphyria

- Drug of choice for Cyclosporine induced HTN - CCB

Classification of blood pressure for adults > 18 years old
Joint National committee VI

Category	Systolic pressure in mm Hg	Diastolic pressure in mm Hg
Optimal	< 120	< 80
Normal	< 130	< 85
High normal	130-139	85-89
Hypertension		
Stage I (mild)	140-159	90-99
Stage II (moderate)	160-179	100-109
Stage III (Severe)	≥ 180	≥ 110
Isolated systolic Hypertension	≥ 140	< 90

- Diagnosis of hypertension is based on the overage of ≥ 2 readings taken at each of two or more visits after initial screening.

Diagnosis of Secondary hypertension

It should be suspected in patients with

- Abrupt onset of severe hypertension
- Hypertension of any severity in a patient under the age of 35 or over the age of 55.

Pheochromocytoma-

- History of headache, palpitations, anxiety attacks, unusual sweating, hyperglycemia, and weight loss is a strong indication for investigation to rule out pheochromocytoma.
- Best screening test is 24 hours urine catecholamine and their metabolites.

Cushing's syndrome-

- 24 hours urine test for cortisol
- Dexamethasone suppression tests

Reno vascular hypertension-

- Clinical indicators
  - Abdominal bruit.
  - Deterioration of renal function after giving ACE inhibitors
  - Older patients with diffuse atherosclerotic disease.
- Best screening test – captopril enhanced radionuclide renal scan.
- Most sensitive & specific – The spiral CT scan.
- Gold standard – renal angiography with renal vein renin determinations.

Primary aldosteronism –

- Hypokalemia is common in these patients
- The aldosterone concentration or excretion rate is high and plasma rennin activity is low in primary aldosteronism.

Therapy:

EPLERENONE – used in bilateral adrenal Hyperplasia

- Mineralocorticoid Receptor antagonist
- Co-arctation of Aorta – Commonest cause of HTN is mechanical obstruction.
- Commonest cause in NSAA is renal.

HTN crisis – Situations requiring rapid reduction of BP.
Urgencies – can be treated relatively slowly.
Emergencies – Immediate therapy within 1 hours
Therapy of HTN
RCT have proved that 10-14 mm Hg of systolic and 6 mmHg in diastolic blood pressure reduction confers 2/5 less stroke 1/6 less CAD
Isolated systolic Hypertension – common in elderly systolic BP > 160mmHg
Drugs of Choice – Diuretics and CCB

Algorithm for therapy of HTN
Not at Goal
<140/90 and
< 130/80 in diabetic and in kidney disease
↓
Initial Drug choice
↓

Without compelling indications	With compelling indications
1st choice – (diuretics)	1) DM
2nd choice – others	2) HF
	3) Priormi
	4) Stroke

Resistant HTN – Failure to reduce diastolic BP 90 mm after use of 3 drugs in full Therapeutic doses one of which includes a diuretic
HTN WITH Diabetes – Goal < 130/80 First choice

- ACE / ARB second choice Diuretic
- Consider change if serum creatinine > 1.5 mg/d1
- Change to DHP and between blockers

Treatment

1. In patients with Stage III hypertension drug therapy should be immediately started.
2. In patients with State I & stage Ii hypertension immediate drug therapy is warranted if.
    - Three or more cardiovascular risk factors are prese
    - Patient is diabetic
    - There is an evidence of target organ damage (e.g renal disease, cardiac disease, retinopathy etc0
3. In patients with low/medium risk and stage I/Stage II hypertension initially lifestyle measure are instituted. If BP is not under control on several occasions then drug therapy is started
4. Surgery may be required in some cases of secondary hypertension.

Class of drug	Indications	Contraindications
1. Diuretics	▪ Heart failure ▪ Elderly ▪ Systolic hypertension	▪ Gout
2.Beta blockers	▪ Angina ▪ Post – MI ▪ Tachyarrhythmias	▪ Asthma & COPD ▪ 2° / 3°
3.ACE inhibitor	▪ Heart failure ▪ LV dysfunction ▪ Post MI ▪ Diabetic nephropathy	▪ Pregnancy ▪ Hyperkalemia ▪ B/L renal artery stenosis
4.Calcium antagonists	▪ Angina ▪ Elderly patients ▪ Systolic hypertension	▪ 2° / 3° AV block
5. Angiotensin II antagonists	▪ ACE inhibitor induced cough	▪ Pregnancy ▪ Hyperkalemia ▪ B/L. renal artery stenosis
6 Alpha Blockers	▪ Prostatism	
7. Minoxidil	▪ Therapy of HTN with renal failure	

Reasons for poor therapeutic response
1) Excessive sodium
2) OCP, steroids
3) Secondary hypertension
4) Non diuretics anti hypertensive.

Malignant Hypertension

- Marked BP elevation (usually) diastolic BP>130mm Hg)
- Papilledema, retinal hemorrhages and exudates.
- Hypertensive encephalopathy – severe headache, vomiting, visual disturbances, transient paralysis, convulsions, stupor, and coma
- Cardiac decompensation
- Acute renal failure
- Characteristic vascular lesion is fibrinoid necrosis of the walls of small arteries and arterioles
- Average age – 40 years, men are more commonly affected.

Treatment

- Reduction of diastolic pressure by one- third but not a level<95mmHG
- Correction of medical complication

• Acute rental failure – fenoldopam
Pheochromocytoma-phentolamine
Others – Nitroprusside, nitroglycerine Esmolol
• Furosemide should be used in all the patients.

- Digoxin may be used if there is an evidence of cardiac decompensation Overall the best drug is fenoldopam.

Overall the best drug is fenoldopam
DES – drug Eluting stents with Rapamcin and sirolimus coated stents used in PCI so as to prevent in stent thrombosis.

Acute Arterial Occlusion
Cause: Embolic arising from heart and aorta
- Cardiac Sources – AF, AMI, ventricular aneurysm cardiomyopathy, IE, prosthesis
- Hematological causes

C/F- 5P's
Therapy – (1) Heparin
(2) Thrombolytics
(3) Surfical
(4) Correction of underlying disorder.

ATHEROEMBOLISM
Embolic in small vessels hence pulsation is intact presents with livdo reticularis, cyanosis and gangrene (blue toe syndrome) biopsy reveals cholesterol crystals.

Framingham Criteria for Congestive Heart Failure
Major criteria
1. Paroxysmal nocturnal dyspnoea
2. Neck vein distension
3. Rales
4. Radiographic cardiomegaly
5. Acute pulmonary edema
6. S_3 gallop
7. Central venous pressure >16 cm, H_2O
8. Hepatojugular reflux
9. Weight loss ≥ 4.5 Kg in 5 days in response to treatment of congestive heart failure.

Minor Criteria
1. Bilateral ankle edema
2. Nocturnal Cough
3. Dyspnoea on ordinary erection
4. Hepatomegaly
5. Pleural effusion
6. Decrease in vital capacity by one third from maximal value recorded
7. Tachycardia (rate ≥ 120 bpm)

Diagnosis of CHF – 2 major criteria or 1 major + 2 minor criteria

Never therapeutics in CCF
1. Drugs
    1. Vasodilators – nitrate

Nebivolol: selective beta1 blocker with vasodilatory properties
Aliskiren: Direct rennin inhibitor
    2. Ca2 + sensitizer with inotropic action – LEVOSIMENDAN
    3. Neutral endopeptidase inhibitor – omapatrialat (doubtful role)

Istaroxime: inhibitor of Na-K ATPase and activator of Ca-ATPase
4. Ventricular Resynchronisation

5. LABP – Intra Aortic Balloon Pump

Management of arrhythmias in HR

a. Amiodarone drug of choice
b. ICD- best option

Poor prognostic markers in HF:
1) Severely depressed ejection fraction (< 15%)
2) Reduced maximal 02 uptake
3) BNP > 500 pg/ml
4) Frequent VPC

CARDIAC ARRHYTHMIAS

Atrial premature complexes:

APCs can be found on 24-h holter monitoring in over 60% of normal adults. They are usually asymptomatic and benign.

They are recognized on ECG as early p waves with morphology that differs from the sinus p waves. Treatment is useful only if they cause palpitations on trigger paroxysmal supraventricular tachycardias. Alcohol, tobacco, or adrenergic stimulants should be identified and eliminated. In their absence, mild sedation or beta blocker may be tried.

AV Junctional complexes:

The site of origin of these complexes is thought to be in the bundle of his, since the normal AV node in vivo possesses no automaticity.
They are most often associated with cardiac disease or digitalis intoxication.
They can be recognized on ECG as normal appearing QRS complexes that are not preceded by a P wave. If symptomatic they should be treated as APCs.

Ventricular premature complexes (VPCs)

Of adult males ≥ 60% will exhibit VPCs during a 24 hours holter monitoring
In patients with previous MI, if frequent (> 10 per hour) or complex VPCs are present, they are associated with increased mortality.
R-on-T VPC have been associated with increased risk of sudden death.
VPCs are recognized by wide (Usually > 0.14 sec), bizarre QRS complexes that are not preceded by P waves. When interval between VPCs is variable then ventricular parasystole is said to be present. VPCs produce a fully compensatory pause i.e., the interval between conducted sinus beats that bracket the VPCs equals 2 basic RR intervals.

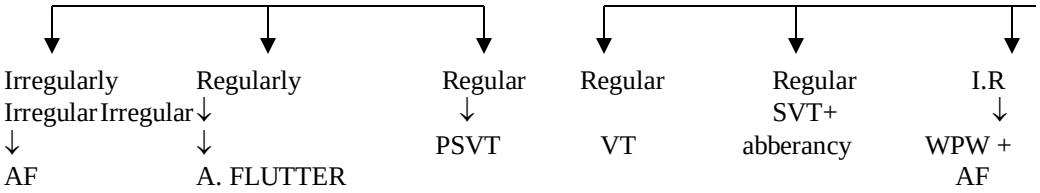
Treatment:

In the absence of cardiac disease, isolated asymptomatic VPCs regardless of configuration and frequency, need no treatment.
Beta blockers are useful in the management of symptomatic VPCs that occur primarily in the daytime or under stressful situations and in specific settings such as thyrotoxicosis and mitral valve prolapse.
In cast trial post myocardial infraction patients who received class IC drugs had increased overall mortality and sudden death rate as compared to patients who received placebo
Implantable cardiac defibrillator (ICD) placement improves prognosis in patients with inducible VT and LV dysfunction.

Approach to Tachyarrythmias

LEAD II

Narrow QRS	Wide QRS
(<12 secs)	(>12 secs)
RHYTM	RHYTM



Therapy of choice for hemodynamically unstable tachyarrthmics → D.C. Cardioversion.

Atrial Fibrillation:

Atrial fibrillation is characterized by disorganized atrial activity without discrete P waves on surface ECG Atrial activity ranges from 350 to 600 beats per minute. The ventricular response is irregularly irregular. The main factor determining the rate of ventricular response is the functional refractory period of the AV node. If in the presence of AF, the ventricular rhythm becomes regular and slow, complete heart block is suggested, and if the ventricular rhythm is regular and rapid, a tachycardia arising in AV Junction or ventricle should be suspected. Digitalis intoxication is a common cause of both phenomena

Patients with AF have loss of a waves in JVP and variable pulse pressures in the carotid arterial pulse. The first heart sound varies in intensity.

Cause of AF

- Rheumatic heart disease
- Non - rheumatic mitral valve disease
- Hypertensive cardiovascular disease
- Atrial septal defect
- Chronic lung disease
- During stress or vasovagal response
- Acute alcoholic intoxication.
- Thyrotoxicosis
- Lone AF

Factors associated with high risk of stroke in AF:

1. Age > 65
2. Hypertension
3. RHD
4. Prior stroke or TIA
5. Diabetes mellitus
6. Congestive heart failure
7. Left atrial dimension>5cm.

Treatment:

1. In acute AF, precipitating cause such as thyrotoxicosis, pericarditis, and alcohol intoxication should be sought and corrected.
2. If the patient's clinical status is severely compromised, electrical cardio version is the treatment of choice.
3. In the absence of cardiovascular compromise, slowing of ventricular rate is the initial therapeutic goal. Beta blockers or calcium channel blockers should be used.
4. Conversion to sinus rhythm is attempted only after control of ventricular rate. All patients with AF>48 hours should be anticoagulated to an INR of at least 1.8 for the prior 3 consecutive weeks. It should be continued for 4 weeks after cardioversion. Quinidine-like drugs (Class IA) or flecainide like agents (Class IC) are used for cardioversion. Pill in pocket for AF is flecainide or propafenone.

DC cardioversion is highly effective method. At least 200 joules of synchronized shock is used. For chemical cardioversion ibutilide, procainamide or amiodarone can be used.

5. If sinus rhythm is restored, quinidine or related agents, class IC agents, sotalol or amiodarone may be used to prevent recurrence.
6. In patients in whom cardio version is unsuccessful or AF has recurred it is better to let the patient remain in AF and control the ventricle rate with Beta blockers, calcium channel blockers or digoxin. Recommendations for long term anticoagulation.

Age (Years)	Risk Factors	Recommendfations
> 65	Absent Present	Aspoirin warfarin
65 – 75	Absent Present	Aspirin or Warfarin Warfarin
> 75	All patients	Warfarin

7. Ablation therapy for cure of AF is successful in a small subset of patients and can cause pulmonary vein stenosis, pulmonary hypertension and stroke.
8. MAZE Procedure is the surgical therapy for AF.

Atrial Flutter:

This arrhythmia occurs most often in patients with organic heart disease. Flutter, may due to precipitating factors such as pericarditis, acute respiratory failure, or following open-heart surgery.

Most commonly if atrial flutter lasts more than a week it will convert to atrial fibrillation. Atrial flutter is characterized by an atrial rate between 250 and 350 bpm.
Classically, flutter waves are seen as regular saw tooth like atrial activity, most prominent in inferior leads.
Activation mapping has revealed that atrial flutter is a form of atrial reentry localized to right atrium.

Treatment

The most effective treatment of atrial flutter is direct – current cardioversion (25 to 50 joules). Higher energies 9100 – 200 joules) are often used because they are less likely to cause AF.
Anticoagulation, as used in AF should also be given in patients with atrial flutter.
Atrial pacing can also be used for converting atrial flutter into sinus rhythm particularly in cases following open heart surgery and recurrent flutter in the setting of acute M1.
Drugs used for conversion of flutter to sinus rhythm are ibutilide, amiodarone, class IA or IC drugs.
Radiofrequency ablation is highly effective in typical form of atrial flutter which is due to reentry around tricuspid valve.

Paraxysmal supraventricular tachycardia

- Reentry is responsible for most of the cases of PSVT.
- AV model reentrant tachycardia is the most common cause of PSVT. It is more common in women. It is initiated by APC. Regular narrow QRS complex tachycardia at rates of 120 to 250 bpm is seen.
- PSVT due to AV reentry incorporates a concealed AV bypass tract as a part of tachycardia circuit. Bypass tract conducts retrograde from ventricles to atria. AV reentrant tachycardia can be initiated by either APCs or VPCs.
- Reentry in the region of the sinus node or within the atria is invariably initiated by APCs. These arrthythmias are less common than AV reentry and are more often associated with underlying cardiac disease. During sinus node reentry P wave morphology is normal, but PR interval is prolonged.

Treatment:

1. In patients without hypotension, vagal maneuvers, particularly carotid sinus massage, can terminate the arrhythmia in 80% of the cases.
2. If hypotension is present raising the BP by I.V. phenylepherine 0.1 mg may terminate arrhythmia alone or in combination with carotid sinus pressure.
3. if these maneuvers are unsuccessful, verapamil (2.5 to 10 mg i.v.) or adenosine (6 to 12 mg iv) is the agent of choice.
4. In symptomatic patients who require chronic therapy radiofrequency catheter ablation of the AV node or AV bypass tract should be considered. Catheter ablation is less successful in patients with sinus node reentry.

WPW syndrome:

1. Short PR interval (< 0.12 sec)
2. Slurred upstroke of the QRS complex, (Delta wave)
3. Wide QRS complex
    - AV bypass tracts are composed of strands of atrial like muscles which may occur almost anywhere around the AV rings.
    - During normal sinus rhythm AV bypass tracts conduct in an ante grade pattern in patients with WPW syndrome
    - During PSVT in WPW the impulse is usually conducted ante grade over the normal AV system and retrograde through the bypass tract (in 95% of cases).
    - Atrial flutter and atrial fibrillation also occur commonly in patients with WPW syndrome. Since bypass tract is fast conducting the ventricular responses during atrial flutter or fibrillation may be usually rapid and may cause ventricular fibrillation.

Treatments:

1. Acute management of episodes of PSVT in patients with WPW syndrome is similar to that of PSVT in patients with concealed bypass tracts.
2. In patients with WPW syndrome and AF Dc cardioversion should be carried out if there is a life threatening, rapid ventricular, and response. In non life threatening situations, lidocaine (3 to 5 mg / kg) or procainamide (15 mg/kg) administered i.v. over 15 to 20 minutes will usually show the ventricular response. Ibutilide can also be used.
3. IV Verapamil and digitalis should be avoided in patients with WPW AND AF because they can increase ventricular response (by shortening the refractory period of accessory pathway). Beta blockers are of no use in controlling ventricular response during AF when conduction proceeds over the bypass tract.
4. Catheter ablation of bypass tracts is possible in>90% of the patie3nts and is the treatment of choice in patients with symptomatic arrhythmias. Surgical ablation may require in an occasional patient in whom catheter ablation fails.

Non paroxysmal junctional tachycardia:

Most common cause is digitalis intoxication. Other causes include inferior wall MI, myocarditis, acute rheumatic fever, valve surgery catecholamine excess.

It is recognized by a QRS complex identical to that of sinus rhymm, Rate is generally between 70 to 150 bpm. When this rhythm is due to digitalis intoxication, it is usually associated with AV block and dissociation.

Treatment:

Discontinuation of digitalis is required. If other serious manifestations of digitalis intoxication, active intervention with lidocaine or beta blocker may be useful, and in some cases digitalis antibodies (Fab fragments) should be considered.
Cardioversion of this rhythm should not be attempted.

Ventricular Tachycardia:
Ventricular tachycardia is defined as 3 or more consecutive VPCs at a rate more than 100 bpm.
Sustained VT is defined as VT that persists for more than 30 second or requires termination because of hemodymanic collapse.
ECG diagnosis of VT is suggested by wide-complex QRS tachycardia at a rate exceeding 100 bpm.
Characteristics of 12 lead ECG that suggest a ventricular origin for the arrhythmia are
1. Wide QRS complex
   > 0.14 second with RBBB configuration
   > > 0.16 second with LBBB configuration
2. AV dissociation (with or without fusion or captured beats)
3. A superior QRS axis in presence of RBBB pattern.
4. Concordance of QRS pattern in all precordial leads.
5. Atypical right or left bundle branch block.

The prognosis of VT depends upon the underlying disease state. If sustained VT develops within 6 weeks following acute MI, the mortality rate at 1 year is 75%.

Treatment:
1. Asymptomatic non sustained VT in a patient without organic heart disease need not be treated.
2. Sustained VT in the absence of heart disease requires therapy. It may respond to beta blockers, verapamil, class IA, IC or III agents (amiodarone).
3. In patient with VT and organic heart disease, if severe hemodynamic compromise is present or there is an evidence of ischemia, CHF or central nervous system hypoperfusion the rhythm should be promptly terminated by dc cardioversion.
4. If the patient with organic heart disease tolerates the VT well, pharmacologic therapy can be given. Procainamide is the most effective agent for acute therapy.
5. In stable patients if the drugs are not able to terminate the tachycardia, overdrive pacing may be need.
6. Surgical techniques following activation mapping have been developed. Catheter ablation can also be performed in selected patients.

Torsades De Pointes:
VT characterized by polymorphic QRS complexes that change in amplitude and cycle length, giving the appearance of oscillations around the baseline. This is associated with QT prolongation.
QT prolongation may result from electrolyte disturbances (particularly hyp0okalemia and hypomagnesemia), antiarrhythmic drugs (quinidine), phenothiazines, tricyclic agents, bradyarrhythmias. It may be due to congenital long QT syndrome.
The electrocardiographic hallmark is polymorphic VT preceded by marked QT prolongation, often in excess of 0.60 second.
Treatment:
1. Correction of metabolic abnormalities and removal of drugs that induced the prolonged QT interval. In the setting of drug induced torsades de points, atrial or ventricular overdrive pacing and the administration of magnesium have also been useful in terminating and preventing the arrhythmia.
2. For patients with congenial prolonged QT interval syndrome, beta blockers have been the mainstay of therapy. Cervicothroacic sympathetomy is not effective as a sole therapy.
3. For recurrent episodes in patients with congenital prolonged QT syndrome despite beta blockers ICD with dual chamber pacing capability and beta blockers have become the treatment of choice.

Accelerated idioventricular rhythm:

It is also called slow VT with a rate that ranges from 60 to 100 bpm. It usually occurs during acute MI often during reperfusion. It may also be seen following cardiac, surgery, in patients with cardiomyopathy, digitalis intoxication, or rheumatic fever.
This rhythm rarely causes hemodynamic compromise or symptoms.
Treatment is rarely necessary. In symptomatic patients atropine is useful.

Indications of ICD Implantations:

a) Cardiac arrest due to VT/VF
b) Sustained VT with structure heart disease
c) Sustained VT on EP study
d) VT not amenable to other treatment.

Newer drugs:

- **Dronedaron**e: It is amiodarone derivative and contains no iodine. It is slightly less effective than amiodarone. Absorption is increased by food. Low prevalence of lung and thyroid toxicity is seen compared to amiodarone. Patients with severe liver dysfunction and heart failure it is C/I.

- **Aliskiren:** It is direct rennin inhibitor. Function is like ACE inhibitor or ARB.
- **Dabigatran:** Direct thrombin inhibitor, it is indicated for non valvular AF.
- **Rivaroxaban/ Apixaban**- oral factor Xa inhibitor, fondaparinaux is Xa inhibitor given by S/C route.

MCQ'S (SET-1)

1. Negative hepato-jugular reflex is seen in
   a. Superior vena caval obstruction
   b. Inferior vena caval obstruction
   c. Constrictive pericarditis
   d. Cardiac tamponade
2. Cardiac tamponade without pulsus paradoxus seen in
   a. Aortic diseection
   b. HIV paricarditis
   c. Uremia
   d. Malignancy
3. A patient demonstrates alternate weak and strong pulse beat with irregular Rhythm. He is having
   a. Pulsus paradoxus
   b. Dicrotic pulse
   c. Pulsus bigeminus
   d. Pulsus alternans
4. ST segment elevation in ECG seen in all except:
   a. Early repolarsation
   b. Constriction
   c. Ventricular aneurysm
   d. Prinzmetal's angina
5. X wave absent in
   a. CHB
   b. Junctional Tachycardia
   c. AF
   d. Atrial flutter
6. Steep H wave is seen in all except
   a. Restrictive cardiomyopathy
   b. Right ventricular MI
   c. Constrictive pericarditis
   d. Tricuspid stenosis
7. Tricuspid regurgitation without peripheral features of it is seen in
   a. Carcinoid syndrome
   b. Ebsterin's anomaly
   c. Rheumatic heart disease
   d. Intra venous drug abuse
8. Forceful and illsustained apex is seen in all except
   a. Pagets disease
   b. AP window
   c. PDA
   d. AR with failure
9. Systolic retraction of apex beat (Broad bent's sign) is seen in
   a. Cardiac tamponade
   b. AR
   c. Constrictive pericarditis
   d. HOCM
10. Triple apex heat is seen in
   a. HOCM

b. DCM
c. RCM
d. Myocarditis

11. A 50-year-old presents with 3 hours history of retrosternal pain and SOB. He had his pain during eating and radiated to back. He was known hypertensive O/E. cold clommy extremities HR-130/m. BP-80/40 mmHg. JVp normal. All peripheral pulses equal. CxR shows left plunar effusion. Most likely
a. Aortic dissection
b. AmI
c. Rupture esophagus
d. PTE

12. Upright tilt table test is used in the evaluation of
a. Syncope
b. Palpitations
c. Dyspnoea
d. Chest pain

13. D/D of early systolic murmur all except
a. Small VSD
b. Very large VSD
c. Acute MR
d. AP window

14. D/D of continuous murmur all except
a. PDA
b. RSOV
c. Co-arctation
d. AR + VSD

15. ECG feature most commonly seen with PTE
a. Sinus tachycardia with ST-T changes in $V_1 – V_3$
b. S_1 Q_3 T_3 pattern
c. Incomplete RBBB
d. Complete RBBB

16. Feature in ECG not associated with hypokalemia
a. ST segment depression
b. QT prolongation
c. PR prolongation
d. Reduced T wave amplitude

17. ST segment elevation is seen in all except
a. BRUGADA syndrome
b. Hypercalcemia
c. Hyperkalemia
d. Hypermagnesemia

18. Mobilz type II heart block is characterized by all except
a. Site of block is distal to bundle of HIS
b. Wide QRS complex
c. Prolonged PR interval
d. Not all impulses in the SA node reach the ventricles

MCQ'S (set-2)

1. The ECG feature of hypokalemia is
a. QT shortening
b. PR prolongation
c. QRS widening
d. U waves

2. Which of the following is the earliest change in ECG in a patient with hyperkalemia
a. Sine wave appearance
b. Tall T waves
c. QT shortening
d. PR prolongation

3. The most common cause of digitalis toxicity is
a. Hypokalemia
b. Acidosis
c. Accidental overdose
d. Dehydration

4. All of the following can be used for the treatment of digitalis toxicity except
a. IV potassium
b. Beat blockers
c. Lignocaine
d. Fab fragments of digitalis antibodies

5. Pulsus paradoxus is a common finding in all of the following conditions except
a. Pericardial tamponade
b. Acute severe asthma
c. Superior vena cava obstruction
d. Hypertrophic cardiomyopathy

6. Bisferiens pulse is present in all of the following cardiac condition, except
a. Aortic stenosis
b. Aortic regurgitation
c. Aortic regurgitation with mild stenosis
d. Hypertrophic cardiomyopathy

7. A pulse with two palpable waves one in systole and one in diastole is known as
a. Pulsus begemium
b. Pulsus alteraus
c. Bisferiens pulse
d. Dicrotic pulse

8. Cannon a waves in JVP are seen in all of the following conditions except
a. Junctional rhythm
b. Tricuspid regurgitation
c. Ventricular tachycardia
d. Complete heart block

9. Single S_2 seen in
a. TOF
b. PAH

c. Pulmonary atersia
d. Corrected TGA
e. Pulmonary stenosis

10. All of the following conditions can cause appearance of rapid y descent in JVP except
a. Right atrial myxoma
b. Constricutive pericarditis
c. Severe right – sided heart failure
d. Severe tricuspid regurgitation

11. Kussmaul's sing is seen all of the following condition except
a. Severe right-sided heart failure
b. Right ventricular infarction
c. Pericardial tamponade
d. Constiuctice pericardiits

12. Which of the following clinical condition will cause soft first heart sound?
a. Tachycardia
b. Mitral stenosis
c. Short PR interval
d. Long PR interval

13. Which of the following conditions will cause paradoxical splitting of second heart sound?
a. Right bundle branch block
b. Severe systemic hypertension
c. Pulmonary stenosis
d. Aterial septal defect

14. Wide & fixed splitting of second heart sound is of considerable diagnostic value in which of the following clinical condition
a. Atrial septal defect
b. Mitral stenosis
c. Aortic stenosis
d. Right ventricular pacemaker

15. A 26 year old man complains of abdominal, swelling of legs and easy fatigue BP – 90/70 mmHg and pulse becomes difficult to feel on inspiration. He has pedal edema Ascites and tench hepatomegaly. Presence of early third heart sound. The probable diagnosis
a. Cr pulmonale
b. TS
c. C pericarditis
d. PS

16. Loud P2 heard in
a. PAH
b. TOF
c. Eissemmenger
d. PS
e. AS

17. In which of the following condition fourth heart sound can not be heard
a. Systemic hypertension
b. Atrial fibrillation
c. Hypertrophic cardiomyopathy
d. Aortic stenosis

18. A person is diagnosed to have congestive heart failure. Which of the following sign will not be present on clinical examination
a. S3 gallop
b. Rales over lung fields
c. Raised JVP
d. Collapsing pulse

19. All of the following drugs can prolong QT interval on ECG except
a. Digoxin
b. Phenothiazines
c. Sotalol
d. Tricyclic antidepressants

20. Osborn wave on ECG is seen in which of the following condition
a. Hypocalcemia
b. Digitalis toxicity
c. Hyperkalemia
d. Hypothermia

21. Which of the following statement regarding the effect of potassium concentration of ECG is true?
a. The earliest ECG sign of hyperkalemia is a reduction in P wave amplitude.
b. Deep symmetrical T wave inversions are characteristic of early hyperkalemia
c. Hyperkalemia predisposes to digitalis induced tachyarrhythmias.
d. Hypokalemia is associated with torsades de points.

22. A 7-year-old male comes with complaints of headache. On examination upper limb B.P. was found to be more than that of the lower limbs. The most likely cause is
a. Co-arctation of aorta
b. HOCM
c. Pheochromocytoma
d. Renal vein thrombosis

23. 'A' wave in the JVP is due to
a. Ventricular systole
b. Atrial systole
c. Filling of the right veins of the heart
d. Transmission of carotid pulsations

24. Pulsus paradoxus is seen in all except
a. Cardiac tamponade
b. Bronchial asthma (acute exacerbation)
c. Hypertropic obstructive cardiomyopathy

d. Pregnancy

25. Kussmaul sign is seen in all except
    a. Constrictive pericarditis
    b. Pericardial tamponade
    c. Acute pulmonary embolism
    d. Right ventricular myocardial infarction

26. Each of the following heart sounds may occur shortly after S2 except
    a. Opening snap
    b. 3rd heart sound
    c. Ejection click
    d. Tumour plop.
    e. Pericardial knock

27. Normal (innocent) murmurs are usually which type of murmurs
    a. Early systolic
    b. Pre systolic
    c. Mid systolic
    d. Holo systolic
    e. Early diastolic

28. Each of the following statements regarding the measurement of cardiac output is true except
    a. In thermodilution method, cardiac output is directly related to the area under the thermodilution curve
    b. The thermodiluation method tends to overestimate cardiac output in low output states
    c. In the presence of tricuspid regurgitation, the Fick technique is preferred over the thermodilution method for measuring cardiac output
    d. A limitation of the Fick method is the necessity of measuring oxygen consumption in a steady state
    e. Cardiac output is inversely related to systemic vascular resistance

29. Means Lerman scratch is heard in
    a. Hypothyroidism
    b. Hyperthyroidism
    c. Beriberi
    d. Homocysteinemia

30. Aortic dissection associated with
    a. HTW
    b. Co-arctation
    c. Pregnancy
    d. Takayaslt's arterths

31. Which of the following association between altered electrolytes and ECG abnormalities is not true?
    a. Hypocalcemia results in prolongation of the QT interval
    b. Hyperkalmia causes QRS widening and diminished P wave amplitude
    c. Hypermagnesemia is associated with polymorphic ventricular tachycardia
    d. Hyperkalemia causes all peaked T waves

32. Least likely ECG in left pneumothorax
    a. Inversion of T wave
    b. Left axis deviation
    c. Small R wave
    d. Electrical altrnans

33. A 73 year old man with a remote history of M1 feels the event of palpitations while driving than awakens having driven his car into a ditch, unaware of what has transpired. The most likely cause is?
    a. Ventricular tachycardia
    b. High degree AV block
    c. Epilepsy
    d. Neuocardiogenic syncope
    e. Hysterical fainting

34. In carcinoid part of the heart most commonly affected
    a. Outflow of RV
    b. Inflow of RV
    c. Inflow of LV
    d. Out flow of LV

35. A 59-year-old man with severe mysomatous mitral regurgitation is asymptomanic with a left ventricular ejection fraction of 45% and end systolic diameter of 2.9 em/m2. The best management
    a. MVR
    b. No treatment
    c. ACE inhibition
    d. Digoxin and Diuretic

Answer key (set-1)

1. B
2. A

3. C
4. B
5. C
6. D
7. B
8. D
9. C
10. A
11. A
12. A
13. D
14. D
15. A
16. C
17. D
18. A

(SET-2)

1. D
2. B
3. A
4. A
5. D
6. A
7. D
8. B
9. A, C
10. A
11. C
12. D
13. B
14. A
15. C
16. A, C
17. B
18. D
19. A
20. D
21. D
22. A
23. B
24. C
25. B
26. C
27. C
28. A
29. B
30. A,B,C,D
31. C
32. B
33. A
34. B
35. A