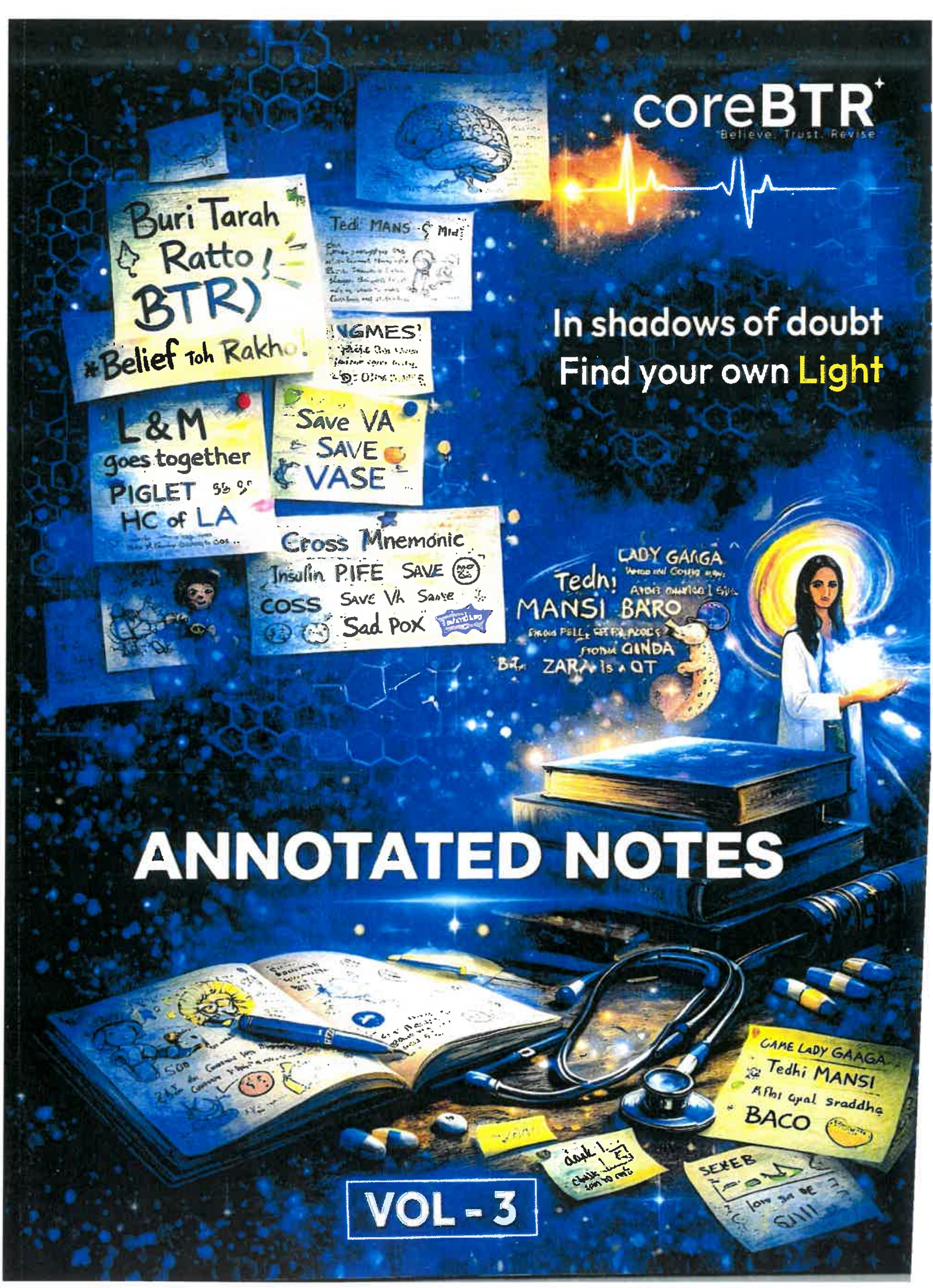

coreBTR
Believe. Trust. Revise
In shadows of doubt
Find your own Light
Buri Tarah
Ratto
BTR)
*Belief Toh Rakho!
L&M
goes together
PIGLET
HC of LA
Save VA
SAVE
VASE
Cross Mnemonic
Insulin PIFE SAVE
COSS Save Vh Save
Sad Pox
LADY GAGA
Tedhi
MANSI BARO
ZARA is a QT
ANNOTATED NOTES
VOL - 3
CAME LADY GAAGA
Tedhi MANSI
BACO
SEXEB

Table of Content

16. Cardiovascular system (CVS) 555
17. Hematology 588
18. Neurology 619
19. Renal system and Electrolytes 653
20. Respiratory system 686
21. Endocrinology 720
22. Gastrointestinal / Hepatobiliary systems (GI/HB) 742
23. Rheumatology 768
24. General Physiology 788
25. General Pharmacology 804
26. General Pathology 829
27. Immunology 845

16

CHAPTER

CARDIOVASCULAR SYSTEM (CVS)

Action Potential

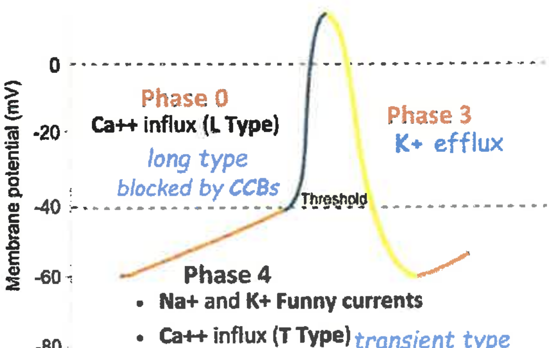
Action potential

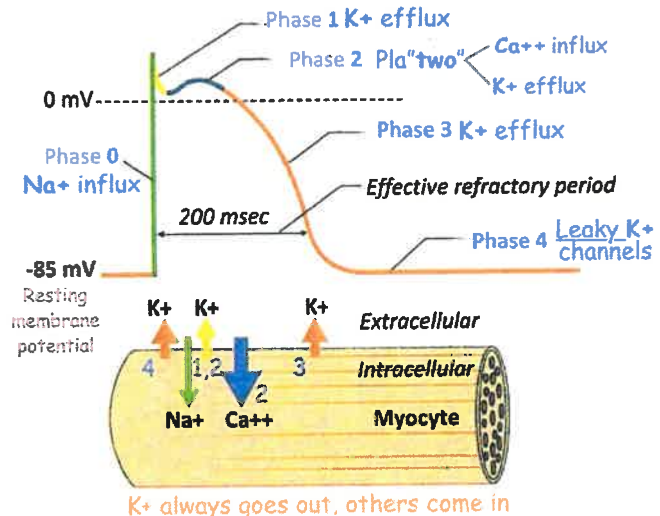
Pacemaker action potential

- Phases present: **4 - 0 - 3**
- *No true resting potential*
- Pacemaker cells generate **rhythm**

Myocyte action potential

- Corresponds to **QT interval**
- Phases present: **0 - 1 - 2 - 3 - 4**
- **Phase 4** is **resting potential**





Funny Currents

- Funny current activates near **–60 mV** and initiates **Phase 4 spontaneous depolarization** responsible for cardiac automaticity.
- It is a mixed **inward cation current** carried mainly by: **Na⁺** and **K+** through HCN (*Hyperpolarization-activated Cyclic Nucleotide–gated*) channels
- Unlike most channels, it opens on **hyperpolarization**, not depolarization *(hence called funny)*

Drugs Acting on Pacemaker Action Potential

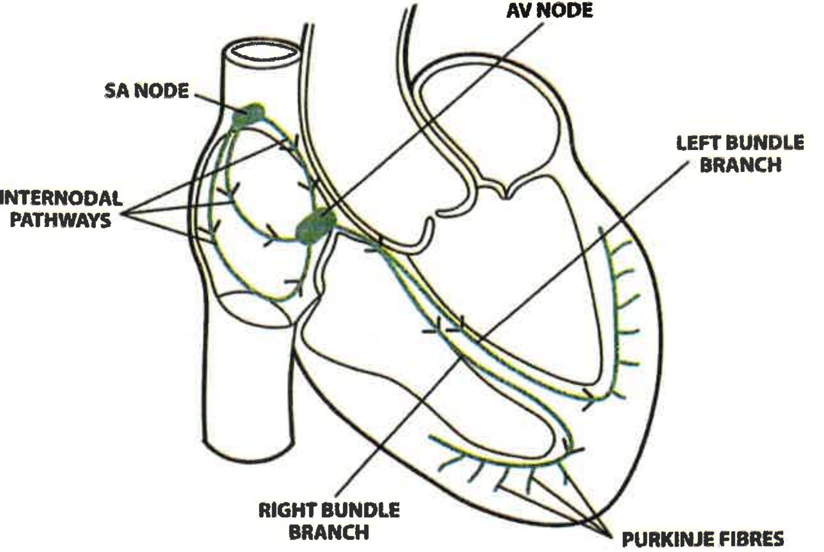
A B C D drugs for rate control

- **Acetylcholine** : Blocks funny current and hence reduces firing of SA and AV node
    - *On the contrary, **Catecholamines increase funny currents** and cause increased firing of SA and AV nodes*
- **Beta blockers** : Blocks **funny current** and **L type** calcium channels
- **Calcium Channel Blockers** (Non DHP type Verapamil and Diltiazem) : Block **L-type** calcium channels
- **Digoxin** : Reduces SA nodal firing via **vagal stimulation** *(also has positive ionotropic effect)*

- Above mentioned drugs **lower heart rate** and **prolong PR interval**
- *Contraindicated in **WPW syndrome***
- **Ivabradine** is another drug that blocks the funny currents and lowers heart rate

- **Effective refractory period** is the period during which: A new propagated action potential cannot be generated, even if a strong stimulus is applied.
    - *Long effective refractory period of cardiac muscle **prevents tetanization***
- **Speed of conduction** in cardiac musculature :
  Bundle of HS/Purkinje fibres > Ventricels > Atria > **AVN**
    - *AV node is slowest due to **least gap junctions** and is responsible for **AV nodal delay***
- **Endocardium** ⇌ **Epicardium** (Depolarisation →, ← Repolarisation)

Both electrical vectors point roughly in the same direction



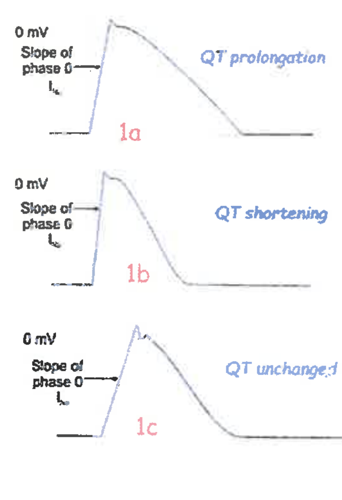
Anti Arrhythmic Drugs (Vaughan-Williams classification)

Class 1 - Blocks Na+ channels ***(has to be avoided in hyper-kalemia)***

1A : K+ channel blocker - *Quinidine, Procainamide, Disopyramide*
- Mnemonic : *Queen proclaimed Diso's pyramid*
- *QT prolongation*

1B : K+ channel opener - *Lignocaine, Phenytoin*
- *Shortens QT interval*
- *DOC for ischemic arrhythmias and digitalis induced arrhythmias*

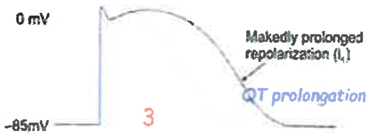
1C : No action on K+ channel - *Propafenone, Flecanaide*
- *Contraindicated in ischemic arrhythmias and digitalis induced arrhythmias*



Class 2 - Beta Blockers
- Slow down phase 4 of **pacemaker action potential** by blocking sympathetic stimulation
- Reduces **myocardial oxygen demand**

Class 3 : Blocks K+ channels ***(has to be avoided in hypo-kalemia)***
- *Amiodarone, Ibutlide, Dofetilide, Sotalol* **"A.I.D.S"**
- QT prolongation
- Side effects of **Amiodarone (40% Iodine)** - has the **longest half life (T½)**

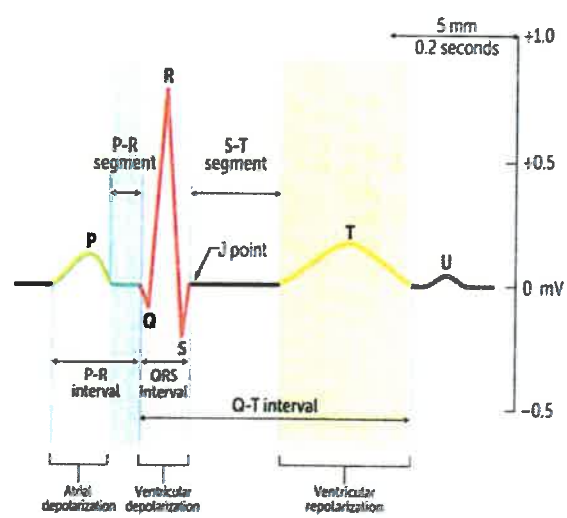
Please	Pigmentation (ceruloderma), Peripheral neuropathy and Photosensitivity
Check	Corneal deposits
LFT	Hepatotoxic
PFT	Pulmonary fibrosis
TFT	Thyrotoxic



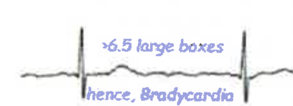
Class 4 : Blocks Ca++ channels
- **Non DHPs :** Slows heart rate and force of contraction (*Verapamil, Diltiazem*)
- **DHPs :** Used as anti hypertensives (eg. Amlodipine)

Class 5 : Adenosine
- IV Adenosine *(fast bolus)* is **DOC for stable PSVT**
  - Adult dose : **6** mg - **12** mg - **12** mg (max dose **30** mg/day)
  - Pediatric dose : **0.1** mg/kg
  - Adenosine is **inhibited** by **Caffeine** and **Theophylline**
- For UNSTABLE SVT : **Synchronized DC cardioversion**

ECG



Heart Rate
- **300/large squares**
- <3 squares : Tachy
- >5 squares : Brady



Normal intervals
- **PR interval** : 0.2 secs
- **QRS** : 0.1 secs
- **QT** : 0.4 secs

2-1-4

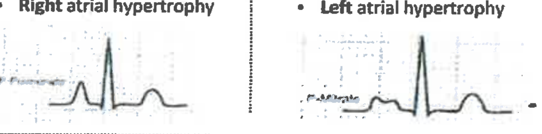
Corrected QT interval

Buzzet's formula :

$$QTc = \frac{QT}{\sqrt{RR \text{ interval}}}$$

Tall P waves
- P pulmonale
- Right atrial hypertrophy

Bifid P wave
- P mitrale
- Left atrial hypertrophy



- **Himalayan P waves seen in Ebstein's anomaly**
- **Pseudo-P-Pulmonale seen with Hypokalemia**

ECG continued

Wave at J point

Osborn wave *seen with* Hypothermia

Epsilon wave *seen with* ARVD

Delta (δ) wave

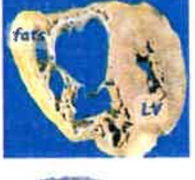
- **WPW syndrome**
- Abnormal pathway : Bundle of Kent
- **Reduced PR** (faster path)
- **Increased QRS** (normal QRS superimposed on abnormal)
- Patient can be *asymptomatic*
- Prevalence *decreases with age*
- TOC : Radiofrequency ablation of abnormal path
- DOC : Flecainide
- In emergency : **IV Procainamide**

Delta wave

Rate lowering drugs** (ABCD drugs) **are avoided

Arrhythmogenic Right Ventricular Dysplasia (ARVD)

- Familial condition
- F/h/o *sudden cardiac death*
- Mutation in **desmosomal proteins**
- **Fibrofatty replacement** of cardiac myocytes
- Naxos syndrome : *ARVD + woolly hair + thick palms/soles*



Uhl's anomaly

Infant + severe right heart failure + **"parchment like RV"**

Causes of Sudden Cardiac Deaths in young

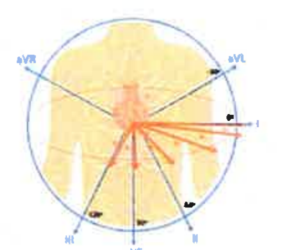
- HOCM
- ARVD
- Long QT Syndrome
- Brugada Syndrome
- Commotio cordis

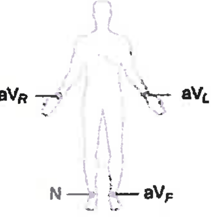
ECG - Axis deviation

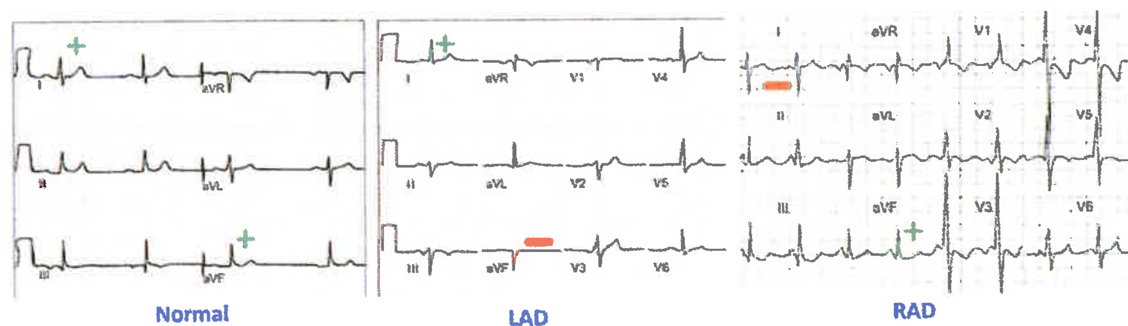
Check lead 1 and aVf

"Check 1 foot"

1. **Both positive** : Normal
2. 1 + and aVf - : Left axis deviation
3. 1 - and aVf + : Right axis deviation
4. **Both negative** : Extreme axis deviation







Normal

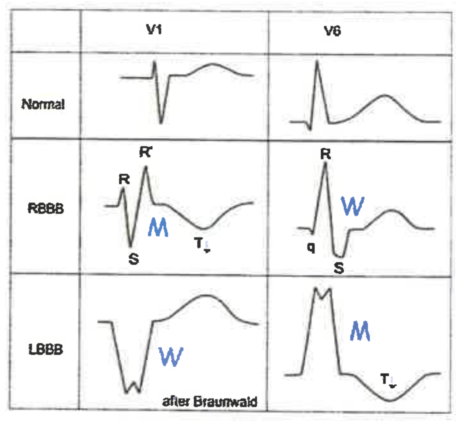
LAD

- MCC : **Left anterior fascicular block**
- Also seen in **LVH** and **inferior MI**

RAD

- MCC : **Right ventricular hypertrophy**
- Common in **lung disease** (COPD, pulmonary htn)

ECG - Bundle Blocks



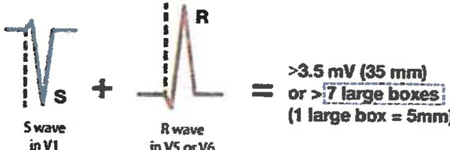
Check V1 and V6

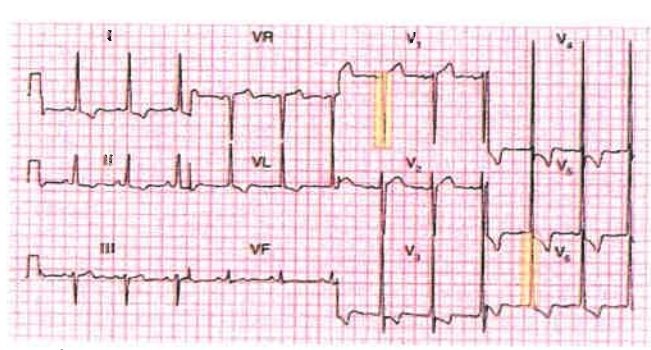
M and W : MarroW (Right bundle branch block)
Marrow is right

W and M : *WilliaM* (Left bundle branch block)

ECG - LVH/RVH

Sokolow-Lyon criteria for LVH



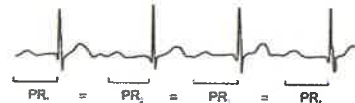


S wave in V1 : 7 boxes + R wave in V6 : 7 boxes = *14 large boxes*

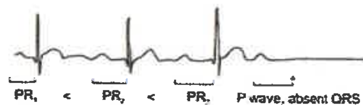
Right Ventricular Hypertrophy (RVH)

R wave in V1 ≥ 7 mm

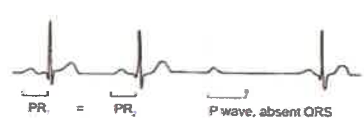
ECG - Heart Blocks



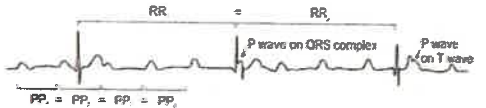
1st degree
Constant and prolonged PR (> 0.2 s)



2nd degree/ Mobitz type 1
Gradually increasing PR - skips beat
Wankebach phenomenon
Normal breakup



2nd degree/ Mobitz type 2
PR constant but suddenly skips a beat
Bad breakup (ghosted)



3rd degree
P and Q not related
Complete AV dissociation
aka Complete heart block

Eg. Stokes Adams Sx (Syncope + Complete Heart block)

Pacemaker needed for

- 2nd degree type 2
- 3rd degree
- *Sick sinus syndrome*
  (bradycardia + irregular rhythm in elderly)

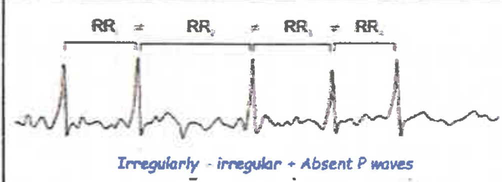
Sick Sinus Syndrome

ACLS Bradycardia Protocol

Unstable/ Symptomatic with HR < 50/min

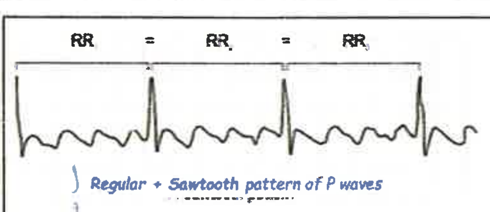
- Treat the **underlying cause** *(Beta-blocker toxicity, MI, Hyperkalemia, Hypoxia)*
- *First-Line Drug: **Atropine*** 1 mg IV bolus
  (if ineffective)
  ↓
- **Dopamine infusion / Epinephrine infusion / Transcutaneous pacing**

Supra - Ventricular Tachyarrythmias



Atrial fibrillation

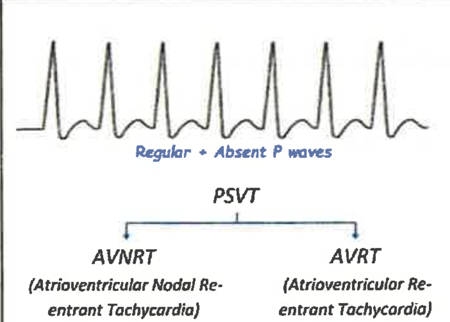
- Unstable patient : Synch. Cardioversion (200 J)
- Stable patient : Rate control
  - Esmolol (DOC) / Verapamil (in asthmatics)
- Acute heart failure + A-fib : Digoxin
- Coagulation in A-fib : Based on CHA²DS²VASc



Atrial flutter

(Regular rhythm, sawtooth pattern)

- Unstable patient : Cardioversion (25-50 J)
- Stable patient : Esmolol / Ibutilide



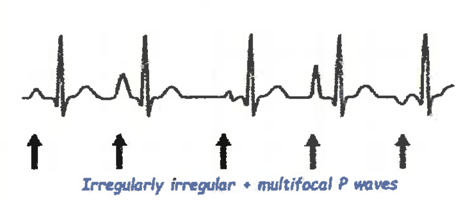
PSVT

- AVNRT (Atrioventricular Nodal Re-entrant Tachycardia) — More common > 60%
- AVRT (Atrioventricular Re-entrant Tachycardia) — eg. WPW syndrome

PSVT

(Paroxysmal Supraventricular Tachycardia)

- Unstable patient : Cardioversion
- Stable patient
  - 1st line : Carotid sinus massage (vagal maneuvers)
  - 2nd line : IV Adenosine
  - Esmolol > Verapamil *(for rate control)*
- FROG sign : visible pulsations in the neck during tachycardia seen in AVNRT



MAT

(Multifocal Atrial Tachycardia)

- DOC for rate control : Verapamil
- Associated with hypoxia - COPD exacerbation *(most common cause)* and Pneumonia
- ***Cardioversion has no role***

Risk of stroke in AF

CHA²DS²VASc score

- CHF
- Hypertension
- Age > 75 (2)
- Diabetes
- Stroke/TIA/TE (2)
- Vascular diseases
- Age > 65
- Sex : *Female*

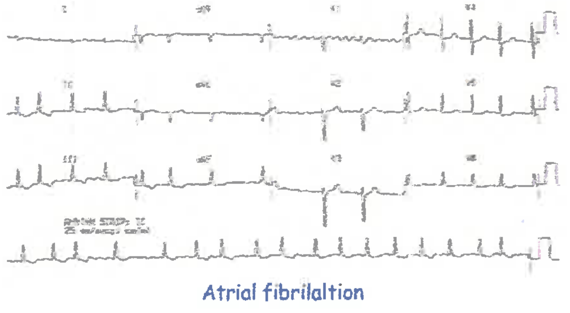
Max score : 9
- 0 : no anticoag.
- 1 : Aspirin
- ≥ 2 : DOACs

Warfarin is the the anticoagulant of choice in
- **Mod-severe MS**
- **Prosthetic valve**

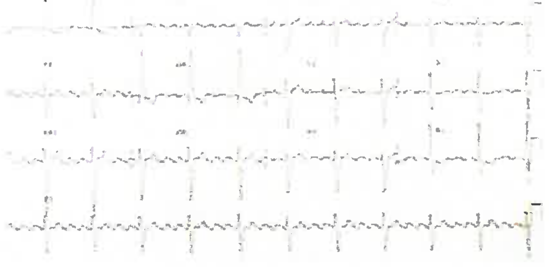
Monitoring of Warfarin therapy is done with INR

Target INR in AF with severe mod-severe MS or mechanical valves is 2 - 3

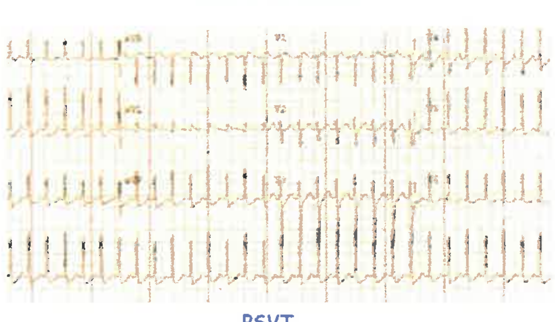
With DOACs (Direct Oral Anticoagulants) routine coagulation monitoring is NOT required



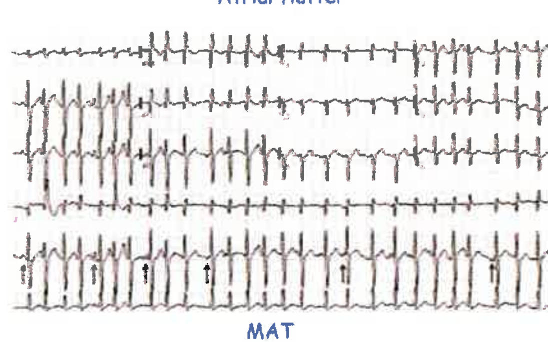
Atrial fibrilaltion



Atrial flutter



PSVT



MAT

Ventricular Tachyarrythmias

No discernible rhythm pattern

V-Fib
(Ventricular Fibrillation)

Wide complex regular tachycardia

Monomorphic Vent - Tachycardia

For V-fib/V-tach

- Unstable patient : Defibrillation
- Stable patient : IV Amiodarone
- *Lignocaine was historically used*

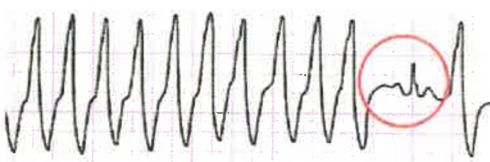
Polymorphic Vent - Tachycardia
aka Long QT syndrome
aka Torades de pointes

- DOC : IV $MgSO_4$
- Associated syndromes :
  - **RomaNO Ward Sx : NO SNHL**
  - **Jerwell-Lange-Nelson Sx : SNHI (+)**
- **Drugs** as described further *"ABCDE"* can predispose to long QT syndrome
- Metabolic abnormality that can predispose are :
  - Hypokalemia
  - Hypomagnesemia, Hypocalcemia
  - Hypothermia

Special beats in Ventricular arrythmias

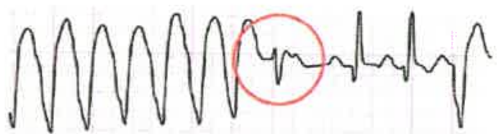
1. Capture beats

- SA node briefly conducts through AV node
- Produces one normal-looking QRS complex amid wide VT complexes



2. Fusion beats

SAN signal and Ventricular signal fuse to form a partially wide QRS



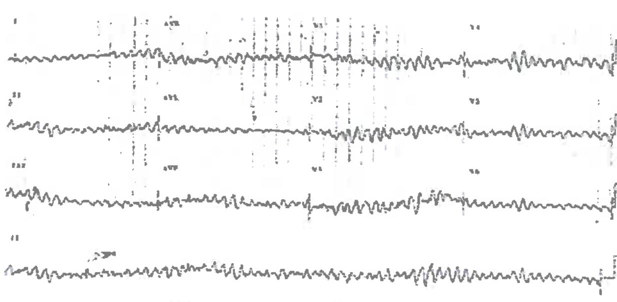
Drugs that can cause long QT syndrome :

1. Anti - **A**rrhythmics : Class 1A, Class 3
2. Anti - **B**iotics/ ATT/ Anti-fungals : Macrolides, Fqn/ Bedaquiline, Pretomanid/ Azoles
3. Anti - **C**-ychotics : Haloperidol, Ziprasidone, Quetiapine, Risperidone
4. Anti - **D**epresents : TCA, SSRI
5. Anti - **E**metics : 5-HT3 blockers - "setrons" (*except Palonosetron*), Domperidone

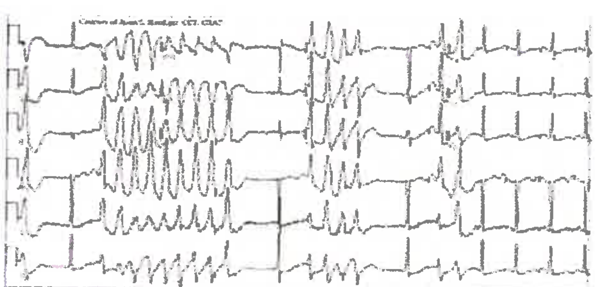
- Other drugs : **Chloroquine & HCQ, Methadone, Protease inhibitors** ("-navir" drugs)
- **CAT drugs** *(withdrawn due to QT prolongation)* : **C**isapride, **A**stemizole, **T**erfenadine

Other uses of $MgSO_4$

1. **Eclampsia** *(seizure prophylaxis)*
2. **Fetal Neuroprotection** (<32 wks POG)
3. **Tocolytic agent**
4. **Acute exacerbation of asthma**
5. **Cellulitis** dressing
6. **Aluminium phosphide** (ALP) **poisoning**
7. **Oral** $MgSO_4$ used as **Laxative**



Ventricular fibrilalltion



Torsades de pointes

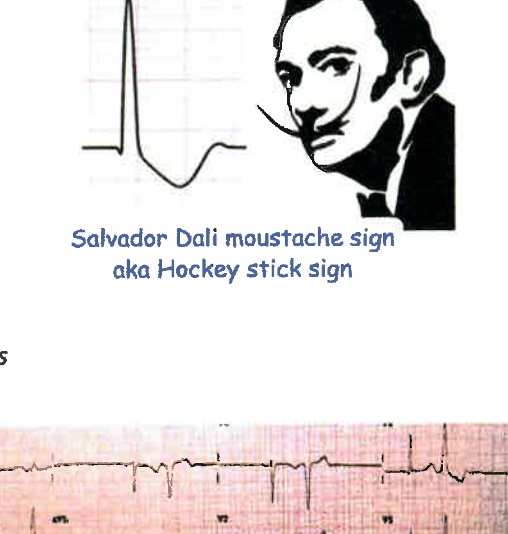
Important ECGs

Digitalis

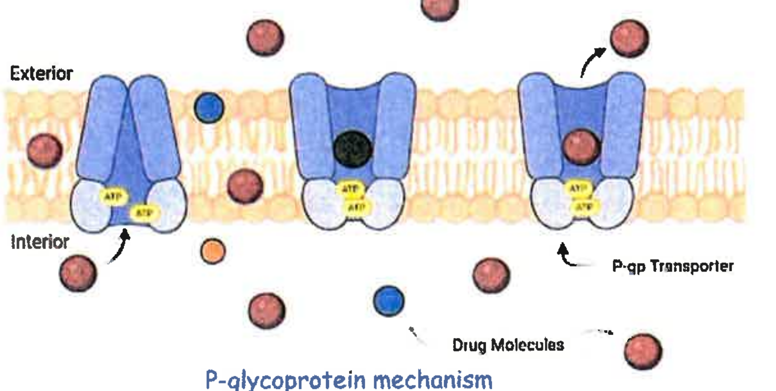
- Digitalis is derived from the plant **foxglove** (*Digitalis purpurea*).
  *Active drug used clinically is - **Digoxin***
- Acts by : **Inhibiting Na^+/K^+-ATPase**
  - Increases **Intracellular Ca^{2+}** - increased cardiac **contractility** *(ionotropic effect)*
  - Increased Vagal tone - **slows AV conduction** *(rate control effect)*
- Side effects : **Hyperkalemia**, **Cholinergic effects**, **Yellow vision** (*xanthopsia*)
- Most Common Arrhythmia : **Ventricular Bigeminy**
- Most Specific Arrhythmia : **NPAT** (*Non-Paroxysmal Atrial Tachycardia*) with AV Block
- ECG Changes (*Digitalis Effect in therapeutic levels*)
  a. Shortened **QT interval** *(faster repolarization)*
  b. Flattened **T wave**
  c. Scooped **ST depression** : "*Hockey stick sign*" or "*Salvador Dali moustache sign* "
  d. **PR interval** prolonged *(AV nodal slowing)*
- Factors Precipitating Toxicity :
  a. **Electrolytes** : ↓ K+, ↓ Mg, ↑ Ca *(enhance digoxin binding to Na^+/K^+ pump)*
  b. **Renal Failure** : Digoxin is renally excreted
  c. **Drugs** : **Verapamil, Amiodarone, Clarithromycin, Cyclosporine, Quinidine**
  (increase serum digoxin level due to P-glycoprotein inhibition)
- Management : **DIGIBIND** *(monoclonal antibody fragments)*, **Lidocaine** - *for arrythmias*

Digoxin
Na^+/Ca^{2+} exchanger
Na^+/K^+-ATPase
Ca^{2+}
Na^+
K^+
SR → ↑↑Ca^{2+} → ↑TnC Ca^{2+} binding → ↑ cardiac contraction

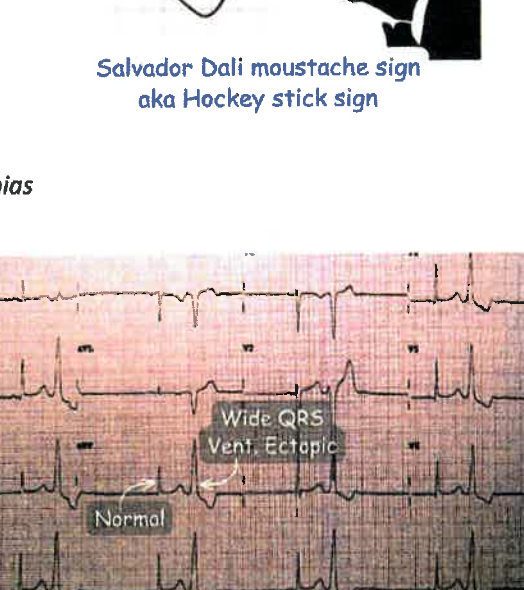
Digoxin MOA



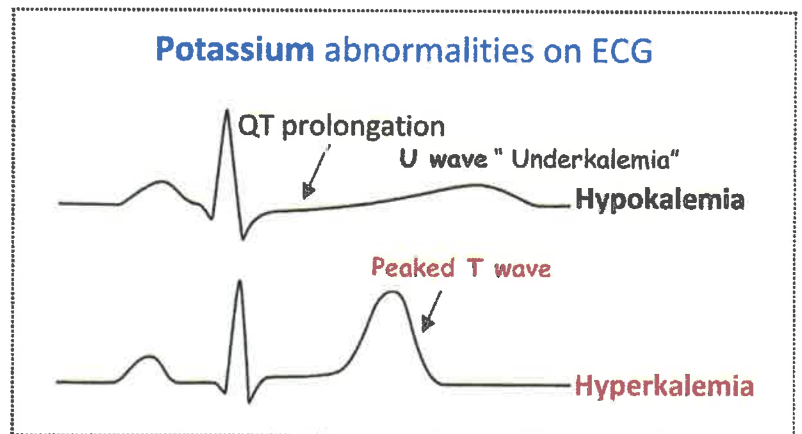
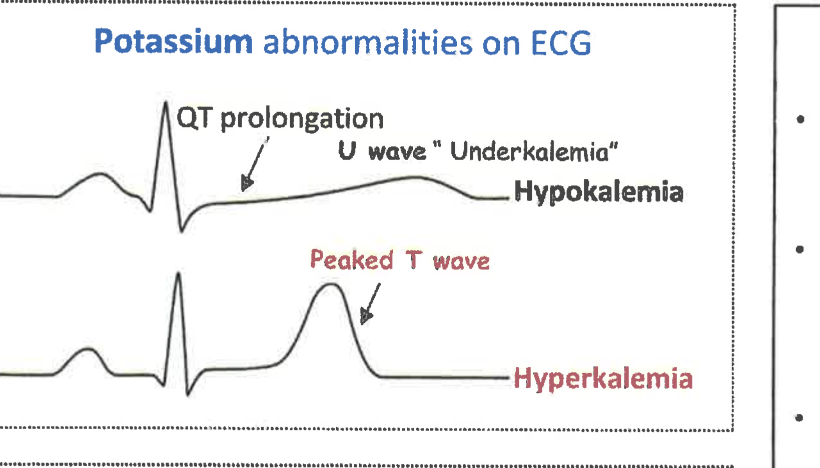
Salvador Dali moustache sign
aka Hockey stick sign



P-glycoprotein mechanism



Ventricular Bigeminy

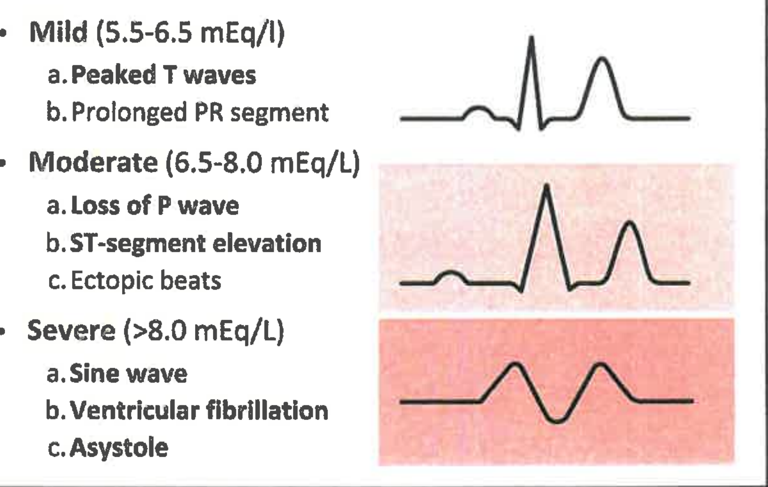
Calcium abnormalities on ECG

QT prolongation

Hypocalemia

QT shortening

Hypercalcemia



ECG changes in hyperkalemia

- Mild (5.5-6.5 mEq/l)
  - a. Peaked T waves
  - b. Prolonged PR segment
- Moderate (6.5-8.0 mEq/L)
  - a. Loss of P wave
  - b. ST-segment elevation
  - c. Ectopic beats
- Severe (>8.0 mEq/L)
  - a. Sine wave
  - b. Ventricular fibrillation
  - c. Asystole

Treatment of Hyperkalemia

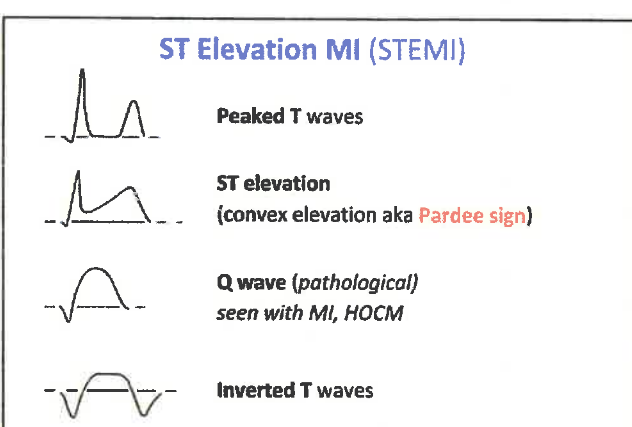
1. **Calcium gluconate** (*Not Calcium Carbonate*) - *Stabilizes cardiac cells, does NOT lower serum K+*
2. **Insulin** + **Dextrose** drip } *Intracellular K+ shift*
3. **Salbutamol** inhalation }

ST Elevation

Causes of ST elevation

1. **Hyperkalemia**
2. **STEMI**
3. **Acute pericarditis** *(concave up, global)*
4. **Prinzmetal angina** *(transient)*
5. **Takotsubo cardiomyopathy** *(mimics STEMI entirely)*
6. Vent aneurysm
7. **Brugada Syndrome** *(in V1 - V3)*

ST Elevation MI (STEMI)

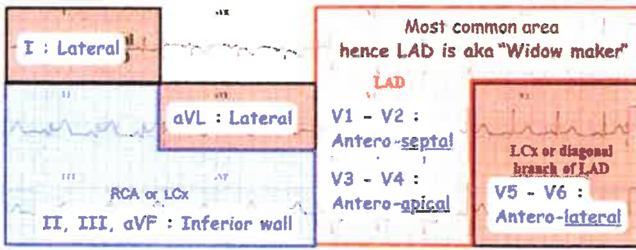


Peaked T waves

ST elevation
(convex elevation aka Pardee sign)

Q wave *(pathological)*
seen with MI, HOCM

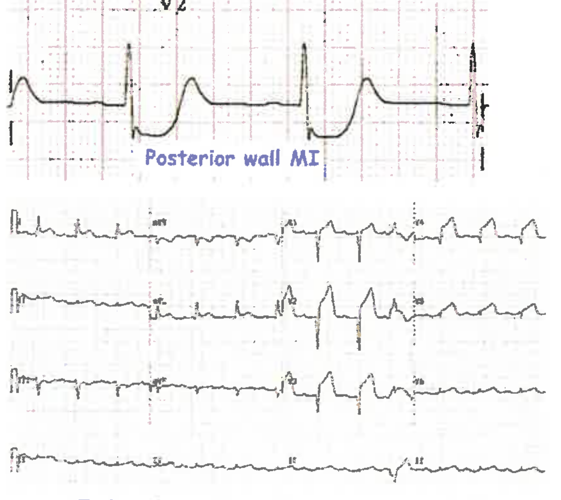
Inverted T waves



Posterior Wall MI *(PDA territory)* - ECG changes

- *ST depression* ***in V1–V3 (reciprocal changes to ST elevation)***
- ***Tall, broad R waves in V1–V3 (reciprocal changes to path. Q waves)***
- ***Upright T waves*** (In NSTEMI there would be inversion of T waves)

Confirmed with **posterior leads V7–V9** : ST elevation ≥0.5 mm



ST elevation in V1 to V4 - Anterior wall MI
*Presents with **cardiogenic shock** due to pump failure*

Acute Pericarditis

Concave up ST elevation

PR segment depression

Acute Chest Pain + Pericardial rub *(Pathognomonic)*

- **Chest pain** is
  - ***Sharp, pleuritic, worse on inspiration***
  - ***Relieved by sitting forward***
- **Viral infections** are the mcc (coxsackie, mumps)
- Post-MI pericarditis - **Dressler syndrome**
- Rx : **NSAIDs** (Ibuprofen/Aspirin)
- Colchicine (*reduces recurrence*)

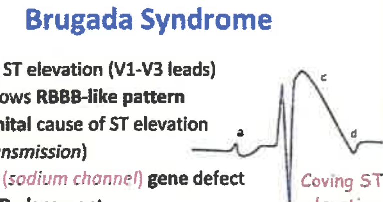
Takotsubo Cardiomyopathy

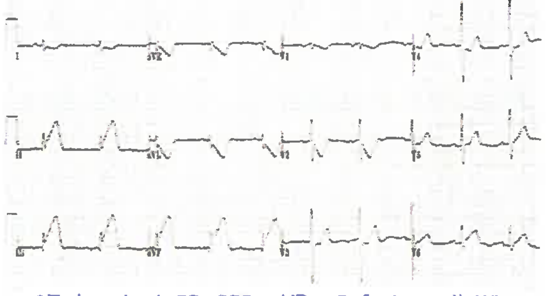
- **Transient** left ventricular systolic dysfunction triggered by **emotional or physical** stress
- aka broken heart syndrome/ stress cardiomyopathy
- **Mid ventricular** narrowing of left ventricle (*like octopus jar*)
- *Hypokinesis of mid ventricle* (apex : normal)
- ***Even cardiac biomarkers are elevated.***
- Differentiated from STEMI only on angiography

Takotsubo = Japanese octopus trapping pot

Brugada Syndrome

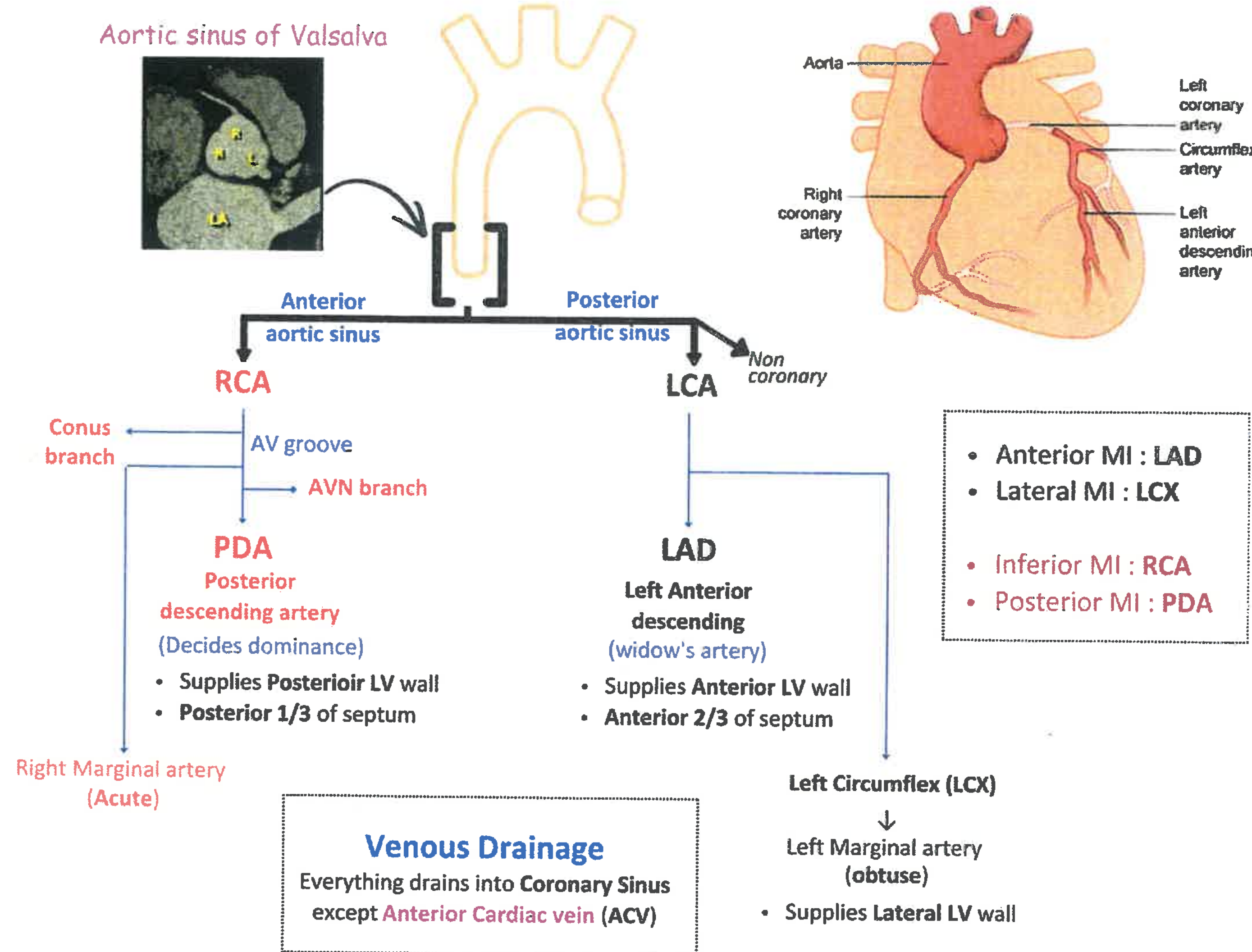
- **Coving** ST elevation (V1-V3 leads)
- ECG shows **RBBB-like pattern**
- **Congenital** cause of ST elevation (*AD transmission*)
- SCN5A (*sodium channel*) **gene defect**
- Mx : **ICD** placement



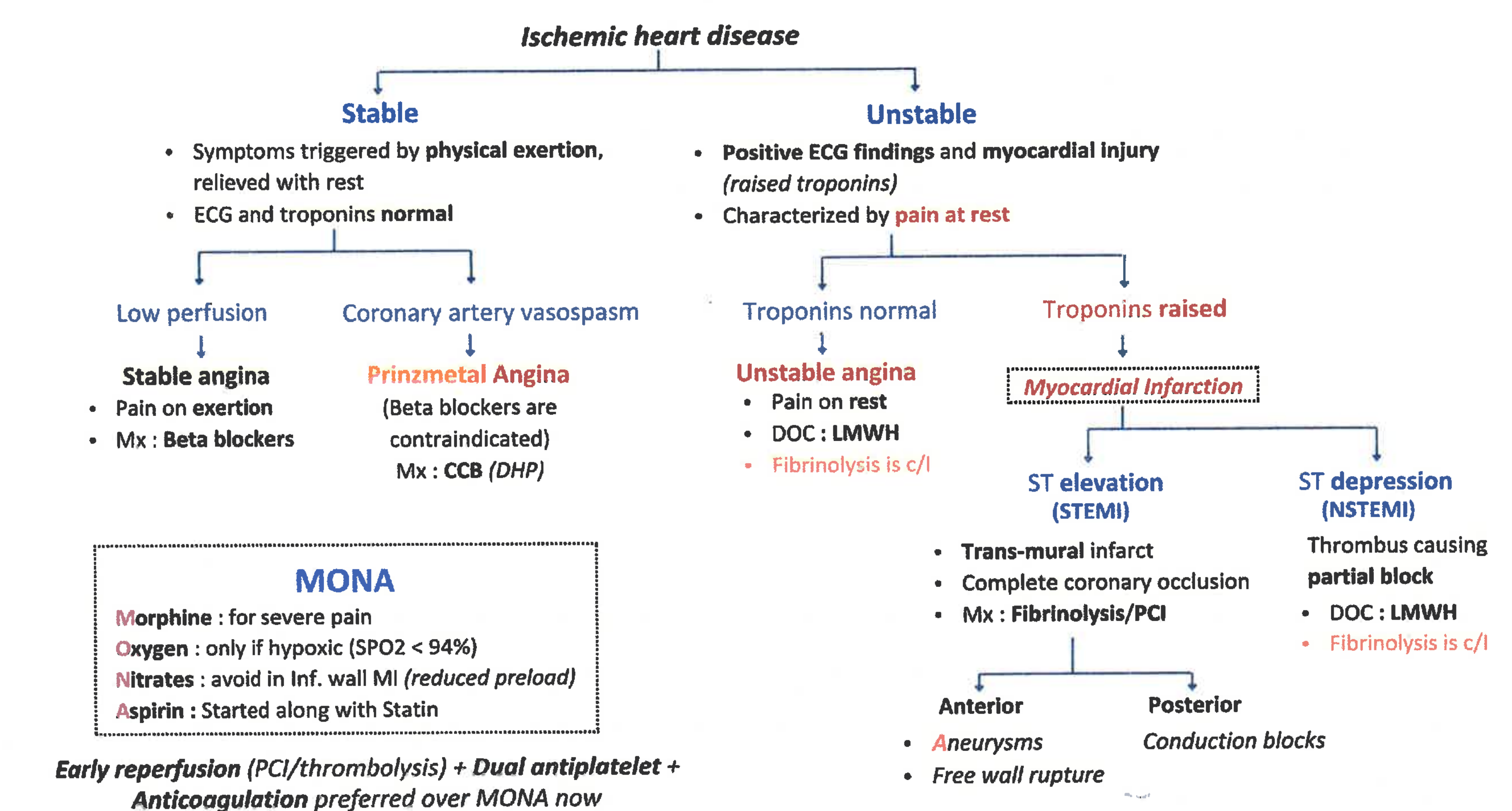


ST elevation in II, III, aVF - Inferior wall MI
*Presents with **bradycardia** to ischemia to SAN and AVN*

Anatomy of Coronary Circulation



Myocardial Infarction



Myocardial infarction continued

	Stable Angina (fixed narrowing)	Unstable Angina (smaller vessels blocked)	NSTEMI (partial block)	STEMI (transmural infarct)	Prinzmetal angina Coronary artery vasospasm
PAIN	On exertion	**Rest/Exertion**	**Rest/Exertion**	**Rest/Exertion**	**Rest/Exertion**
TROPONIN	Negative	Negative	**Positive**	**Positive**	Negative
ECG	Normal	**ST ↓, T inv**	**ST ↓, T inv**	**ST ↑**	*Transient* **ST ↑**
INFARCTION	-	-	***Sub-endocardial***	***Trans-mural***	-
Management	***Beta blockers*** (decreases myocardial oxygen demand)	***LMWH*** *Fibrinolysis is contraindicated*	***LMWH*** *Fibrinolysis is contraindicated*	***PCI/Fibrinolytics***	***DHP - CCBs***
	Statin + Aspirin ± Clopidogrel/Prasugrel				

Timing of PCI in NSTEMI

(for STEMI immediate PCI/fibrinolysis is preferred)

Immediate PCI (<2 hours)
- Hemodynamic instability / cardiogenic shock
- Heart failure

Early PCI (<24 hours)
- Rise/fall of troponin
- Dynamic ST-T changes

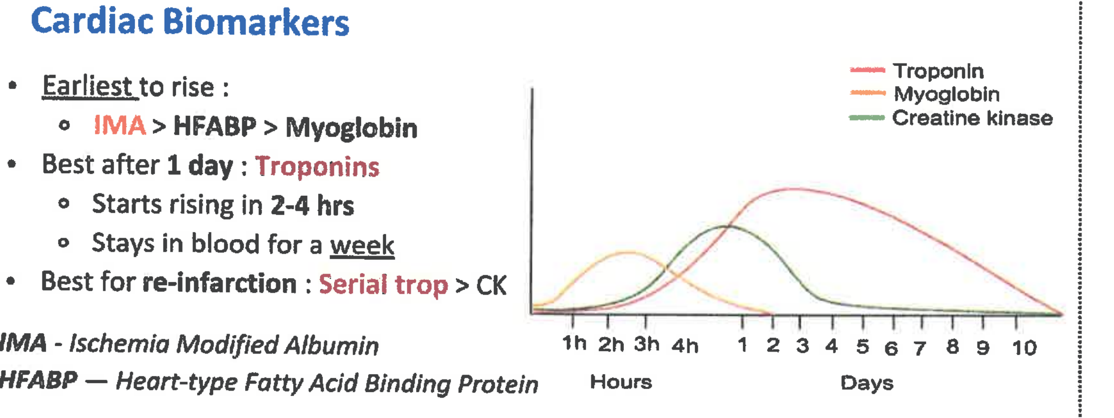
Delayed PCI (24–72 hours)
- Diabetes mellitus *(can have silent MI)*
- Prior CABG *(higher risk)*

Cardiac Biomarkers

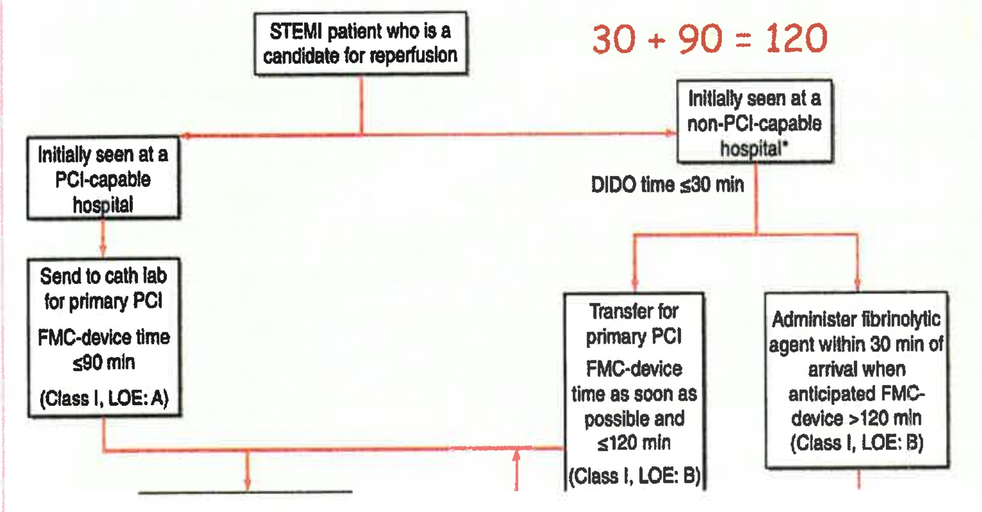
- Earliest to rise :
  - IMA > **HFABP > Myoglobin**
- Best after **1 day** : Troponins
  - Starts rising in **2-4 hrs**
  - Stays in blood for a week
- Best for **re-infarction** : Serial trop > CK

***IMA** - Ischemia Modified Albumin*

***HFABP** — Heart-type Fatty Acid Binding Protein*



Re-perfusion therapy in STEMI



- If hospital **has a cath-lab** then - from **first medical contact** (FMC) to **device placement** (< **90 mins**)
- If hospital **doesn't have Cath-lab**
  - Get out of that hospital (**DIDO**) within **30 mins**
  - **Transfer** to PCI hospital and **device placement** within **120 mins**
  - If not possible to transfer and place device within 120 mins then administer **fibrinolytics** within **30 mins**

Universal Classification of Myocardial Infarction

- **Type 1**: **Spontaneous MI** d/t plaque erosion/rupture, dissection
- **Type 2**: MI due to ischemia secondary **to ↑ demand** or **↓ supply**
- **Type 3**: **Sudden cardiac death** due to MI before biomarkers can be obtained
- **Type 4**: MI associated with **PCI** (4a) / **stent thrombosis** (4b)
- **Type 5**: MI associated with **cardiac surgery** (CABG)

Complications of MI
1. **Cardiac Arrhythmias** (first 48 hours) • **MCC** of death post MI
2. **Peri-infarction Pericarditis** (1-3 days) • Due to **inflammation** over **necrotic myocardium** • *Not auto-immune unlike Dressler's syndrome which is seen after **weeks**.*
3. **Papillary Muscle Rupture** (2-7 days) • Acute **Mitral Regurgitation** *(new onset early systolic murmur)* • **Posteromedial** > Anterolateral papillary muscles (posteromedial has single blood supply - PDA)
4. **Interventricular Septal Rupture** (3-5 days) • Acute **VSD** *(new onset pansystolic murmur)*
5. **Ventricular Pseudoaneurysm** and **Ventricular Free Wall Rupture** (3-14 days) • **Displacement of cardiac apex** • Diagnosed on **echocardiography** • *True ventricular aneurysm* (2 weeks - months)
6. **Post-Cardiac Injury Syndrome** (Dressler Syndrome) - Weeks to months • **Autoimmune pericarditis** after MI

Myocardial Stunning

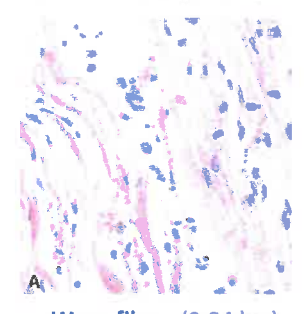
- **Transient LV dysfunction** due to acute ischemia

Myocardial Hibernation

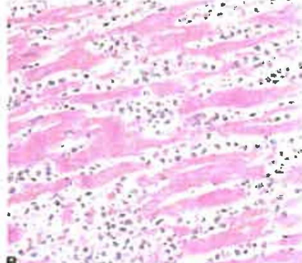
- **Chronic reduced contractility** due to long-standing ischemia

Pathology in Myocardial Infarction

- Earliest change on electron microscopy : Mitochondrial Swelling *(seen within seconds)*
- Earliest change on light microscopy : Waviness of myocardial fibers (*after 30 mins*)
- Earliest change on gross examination : Beyond 4 hours

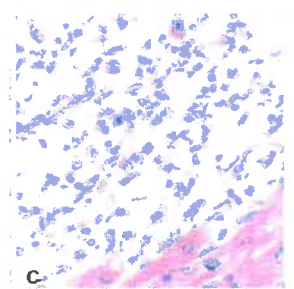


Wavy fibers (0-24 hrs)



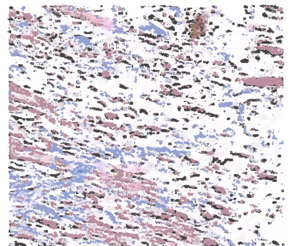
Coagulative necrosis (1-3 days)

- Loss of nuclei : **"ghost nuclei"**
- Marked **eosinophilic** fibers
- **Neutrophil infiltration** *(acute inflammation)*



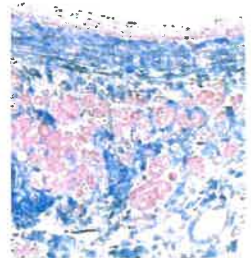
Macrophage infiltration (3-7 days)

- **Macrophages** replace neutrophils
- Myocardial wall **weakest** during this phase *(papillary muscle rupture)*



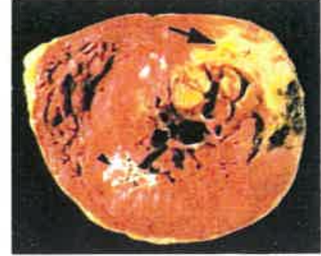
Granulation tissue formation (7-14 days)

Neo-angiogenesis



Scar tissue (>14 days)

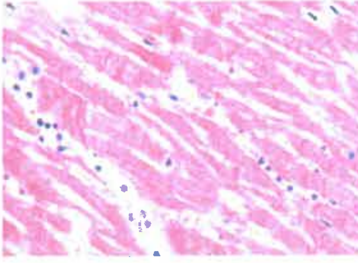
- Increasing **collagen** deposition
- **Decreasing cellularity**
- Progressive **scar formation**



TTC Stain

(Triphenyl Tetrazolium Chloride)

- Normal myocardium : **Stains Red**
- Infarcted myocardium : **White** (no staining)
- *Viable myocardium contains active dehydrogenase enzymes.*



Contraction Bands

hallmark of reperfusion injury

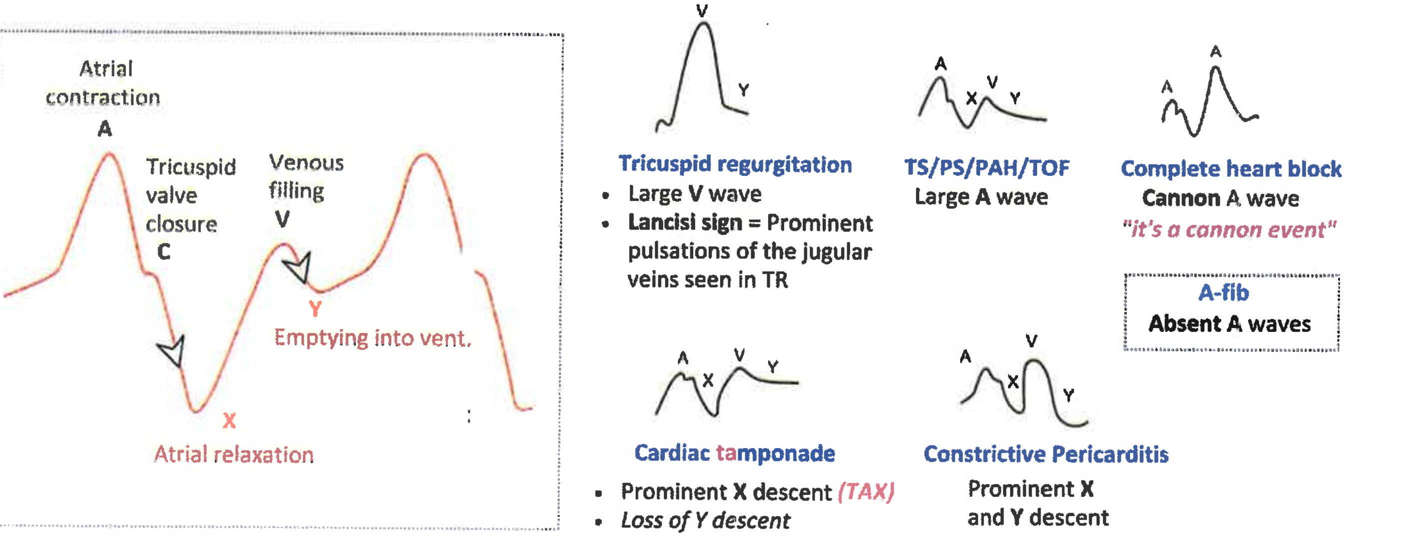
- Intensely **eosinophilic transverse bands**
- Due to **hypercontraction of sarcomeres**
- Caused by **massive calcium influx** into **irreversibly injured myocytes**

LDH Flip

Seen in Myocardial infarction

- LDH-2 > LDH-1 (normal pattern)
- LDH-1 > LDH-2 (in MI)

JVP



Tricuspid regurgitation
- Large **V** wave
- **Lancisi sign** = Prominent pulsations of the jugular veins seen in TR

TS/PS/PAH/TOF
Large **A** wave

Complete heart block
Cannon A wave
"it's a cannon event"

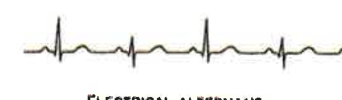
A-fib
Absent A waves

Cardiac tamponade
- Prominent **X** descent *(TAX)*
- *Loss of Y descent*

Constrictive Pericarditis
Prominent **X** and **Y** descent

Cardiac tamponade

- **Beck's triad**
  1. **Hypotension** *(obst. shock)*
  2. **Raised JVP**
  3. **Muffled heart sounds**
- *Pulsus Paradoxus*
- **Ewart's sign** (intra-scapular dullness)
- On Echo : **Diastolic Collapse,** *dancing heart sign*
- **Electrical alternans** on ECG



Constrictive Pericarditis

- **Kussmaul's sign** *(paradoxical **increase in JVP** on inspiration)*
- *Pulsus Paradoxus*
- **Pericardial knock**
- **Low voltage ECG**

*Normally, **systolic BP decreases** slightly (<10 mmHg) during **inspiration** because increased venous return to the right heart reduces left ventricular filling and stroke volume*

Pulsus paradoxus

On inspiration, venous return increases
↓
RV pushes IV septa towards LV, **stroke volume of LV further decreases** and SBP falls by **> 10 mmHg**

1. **Cardiac tamponade**
2. **Const. Pericarditis**
3. **Pulm. Emboli**
4. **Pregnancy**
5. **Asthma**

Reverse pulsus paradoxus
*SBP **increases** with inspiration (seen in **PPV** and **HOCM**)*