ECG BASICS

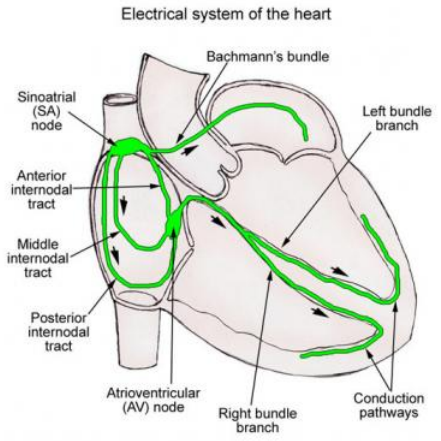
12-lead ECG:

- Praecordial (chest) leads (V1-V6)
- Limb leads (I, II, III, aVR, aVL, aVF) → NORMALLY = **aVR is ALWAYS NEGATIVE**
- **DO NOT ACCEPT ECG W/ ARTEFACTS OF BASELINE SHIFTS**
- **Progression of MI: LAD > RCA > Left circumflex**

Location	Leads	Artery
Right atria	V1, aVR	
Inferior + AV node	II, III and aVF	**RCA → AV node + SA node**
Antero-Septal	V1-V4	**LAD** (MOST common)
Antero-lateral	V4-5, I, aVL	**Left circumflex (LA + LV) = major infarct**
Lateral	I, aVL +/- V5/6	**Left circumflex** → marginal + 1st diagonal branch of LAD
Posterior	• ST depression V1-3 • Dominant R wave V1-2	**Posterior desc artery (branch of RCA)**

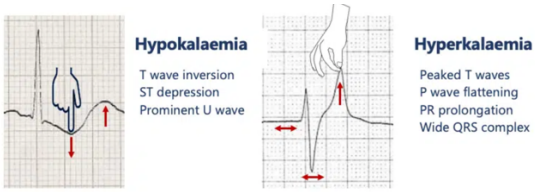
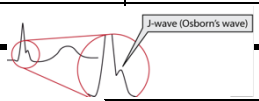
Intrinsic rate:

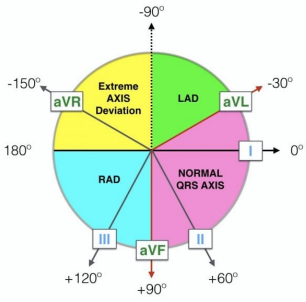
- SA node (60-100bpm) > AV node (40-60bpm) > Purkinje (20-40bpm)
- Slowest conduction = AV node
- R-R variability = SA node misfiring



NB: left ventricular branch divides into the left anterior and left posterior branch

***RV = most anterior = most likely to be stabbed**

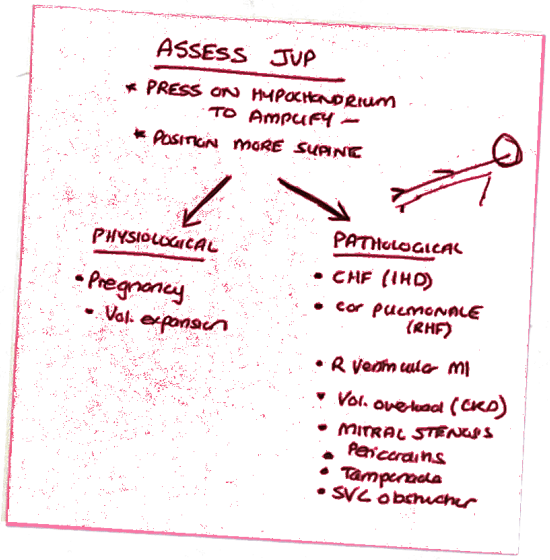
	Duration	Mechanical event	Pathology
P wave	< 0.12s	atrial depol (**atrial arrhytmias**)	• **ATRIAL HYPERTROPHY** → Bifid "M" like = P mitrale → LA enlargement due to MS • **P Pulmonale** → Peaked P wave > 2.5cm • **No P waves = AF / flutter / junctional rhythm**
P-R interval	0.12-0.2 s (3-5 squares)	Conduction (**Heart blocks**)	**Shortened = Accessory pathway** – WPW vs SVT (no P wave) → valsalvre, adenosine (**cardiovert 1st if shock**) **Prolonged = AVN dysfunction/block** → causes include: • Inferior MI, electrolyte disturbance, increase vagal tone/athletes • AV blocking drugs (e.g. digoxin, amiodarone) • Inflammation (IE)→ autoimmune (SLE/SSc) → infiltrative diseases (amyloidosis)
QRS complex	0.08-0.12s	ventricular depolarisation (**Ventricular strain or BBB**)	***Narrow (supraventricular)*** • SVT, atrial flutter, junctional escape ***Broad*** • **ectopic**, LBBB or RBBB, wide complex tachycardias (VT, AF + BBB, torsades) ***Very tall*** • **S wave (V1) + tallest R wave (V5/6) ≥7 BIG squares → LVH** (HTN, AS, AR, MR, HOCM) • **Dominant R wave (V1) + Dominant S wave (V5/6) → RVH** (*pulm HTN, MS, PE*) ***Short*** • **effusion/tamponade, pneumothorax** ***Tall Q wave*** • established/previous full thickness MI ***Normal*** • ***R wave progression*** (normally – most negative V1/dominant S → most positive V6/dominant R • **Dominant R wave in V1 (RVH, posterior MI,** chronic lung disease)
QT interval	< 0.45s **(less than half of R-R)**	Lead II, V5, V6 **[Dictates HR]**	• **Shortened** – Hypercalcemia, digoxin • **Prolonged** (> 440ms) [Torsades de Pointes [polymorphic VT] -**HypoCa, HypoK, HypoMg** o **Other** Causes = TCAs, sotalol, amiodarone, low electrolytes, macrolides, anti-psychotics o Corrected using → anti-bacterials (e.g. quinolones (-acins) and macrolides (-mycins)
ST segment	< 0.15s	**ischemia, electrolyte issue**	• **Elevation** = STEMI (infarction or if in every lead → pericarditis (concave), cardiac tamponade) • **Depression** = NSTEMI, UA (ischeamia, posterior MI)
T wave	0.1 – 0.25s **(usu. 1/3rd height of QRS)**	ventricular repolarisation	**T-wave Inversion** • Normal in III, avR and V1 (Right leads) • ***Pathological*** = **ischemia, electrolyte** PE, RVH, LVH, BBB, digoxin Rx • **Biphasic** o Up → down= ischeamia o Down → Up = HypoK 
U wave	0.08s		Normal or **pathological** (**- hypokalaemia**, hypothermia or with anti-arrhythmic)
J wave			Osborne wave → hypothermia, hyper Ca, SAH



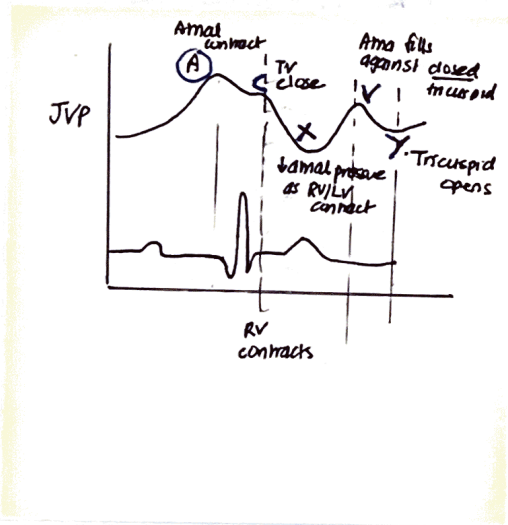
	Lead I	AVF	Normal Variant	Pathology	
RIGHT AXIS deviation			• Children • Tall thin adults • COPD (vertical heart) • Dextrocardia	• **RBBB** • **Left posterior hemiblock** • **Anterolateral MI** (*delayed conduction on left side*)	• **RVH** (in PE, lung disease, **PHTN)** • **Na channel blockade** • **WPW syndrome**
LEFT AXIS deviation			• Pregnancy, • **obesity**, ascites • Abdo distension, tumour	• **LBBB** • **Left anterior hemiblock (most common cause)** • **Inferior MI**	• **LVH**, ?HOCM • VT • RV pacemaker,
EXTREME AXIS deviation			• Emphysema	• **Lead transposition** → can cause both RAD/LAD • **Hyperkalemia** → can cause both RAD/LAD • Pacemaker • VT	

NORMAL ECG FINDINGS IN PAEDS

- HR > 100bpm (correct for age)
- Short PR/QT
- Inferior+lateral Q waves
- RAD, inverted T waves in anterior leads (RV larger than Lv)

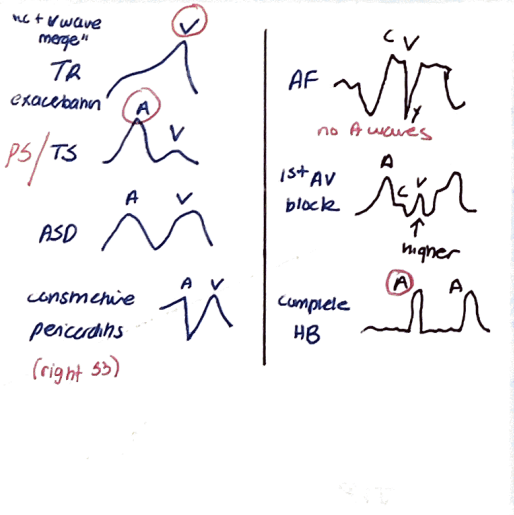


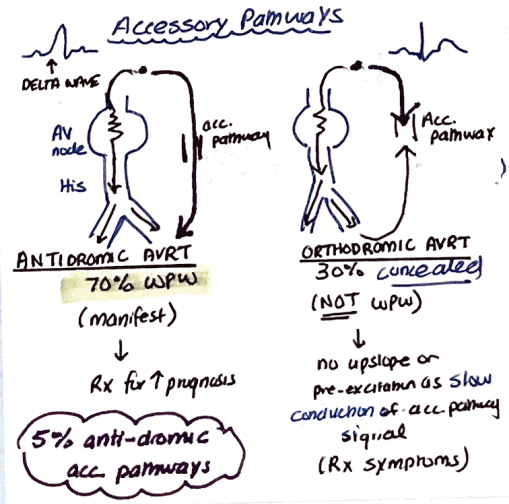
	Unwell	Well
Too slow	Speed them up	
Too fast	Slow them down	
Exciting rhythm	Make them boring	



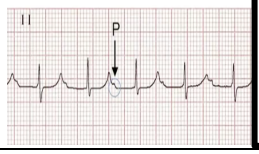
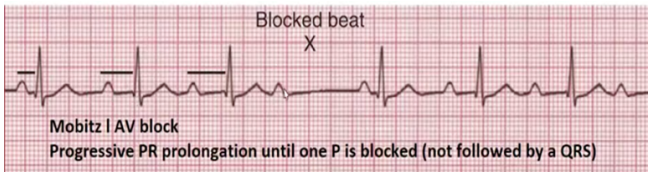
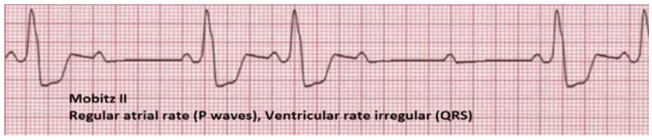
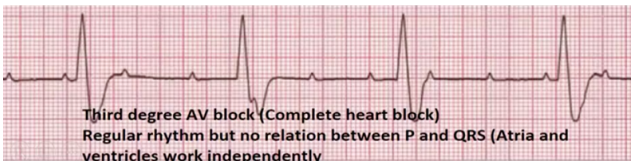
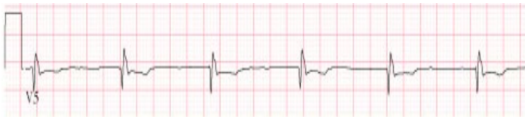
Differentiate carotid vs. JVP

JVP	Carotid
2 peaks	1 peak
impalpable	palpable
obliterated by pressure @ base of neck	cannot/hard to obliterate
moves with resp. to ↑VR	little movement with resp.





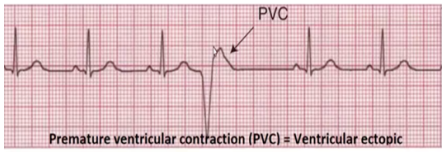
AV Blocks or Bradyarrhythmia

	Key Sign	Temp. Pacing	ECG
Sinus brady (**SA node**)	• **Every p wave followed by QRS complex**	**No**	
1st degree AV block (**within atrium**)	• **Fixed prolonged PR interval** (>200 ms) • ***Cause***: STEMI (inferior), AV nodal disease, myocarditis pro-**cholinergic** (M2) activity e.g. BB, CaB • **NO treatment needed** • Ddx: brugada	**NO**	
2nd degree AV block (Mobitz I Wenckebach) **AV node**	• **Progressive PR interval elongation until** dropped QRS complex (beat) • **Regularly irregular** rhythm ***Cause***: • High vagal tone (esp. children, drugs) • Reversible ischaemia ***Rx: none usually*** ➢ If symptomatic ➔ atropine or pacing	**NO**	
2nd degree AV block (Mobitz II) **Bundle of His or Purkinje**	• **PR interval is fixed (+/- prolonged) with dropped beats** • **Regular** atrial rates • **Irregular** ventricular rate (QRS) ***Cause***: • Infarction • Myocardial fibrosis	**YES** 1. 500mcg **atropine** IV (repeat up to 6x to 3g) 2. **Transcutaneous pacing** – needs ECG leads to monitor and defib pads to pace *(Increase from 70bpm and 70mA)* 3. PPM when available	 Indication for pacemaker (can develop into complete HB) **Atropine A/E = ↓ PSNS = mydriasis, urinary retention, dry eyes, constipation**
3rd degree AV block (*complete HB = 100% SPECIFIC FOR VT*) Anywhere **after AV node**	• **Regular bradycardic rhythm (≈ 30-40bpm)** • **AV dissociation** = P waves and QRS complexes **are completely unrelated (i.e.** atria and ventricles work independently) Ventricular activity maintained by **escape rhythm:** • **narrow QRS**= AV node or bundle of His • **broad QRS** = Distal Purkinje tissues ***Cause***: • AV nodal blocking drugs (BB, CaB, digoxin) • Inferior MI • Degenerative disease		 **CANNON A WAVES = Raised JVP** Cause = stokes-Adams attack after fall (abrupt transient LOC due to sudden loss in CO)
Accelerated Junctional rhythm *"likely electrolyte issue"*	Increased automaticity within AV node or common bundle of His Purkinje system. • No p waves • Narrow QRS complex • rate < 130 bpm.	**No**	 **Rx: Vagal manoevre and adenosine**

Atropine	*Doesn't work in CHB*
⬇ Adrenaline	*Treats hypotension*
⬇ Isoprenaline	*Causes hypotension*
⬇ Transcutaneous pacing	*It Hurts!*
⬇ Transvenous pacing	*Needs time to organise*

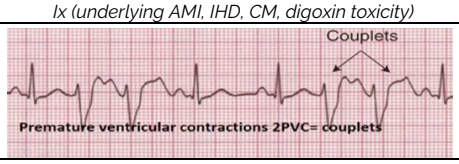
Ectopic Beats & Pacemakers

Frequent Ventricular ectopic (Everyone has it):
- *Common* = Premature ventricular contraction/beats (PVC)
- **Abnormal broad QRS at abnormal time** followed by pause
  - **If ectopic are present with underlying IHD (e.g. cardiomyopathy)→ VT risk**
  - **If NOT → give Mg or K (avoid tea/coffee)**
- *CAUSES*: ***Caffeine, electrolyte imbalance, RV outflow tract***

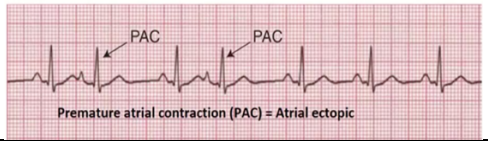


- ***Benign*** = *reassurance*
- ***Referral if red flags*** – *chest pain, syncope, FHx of sudden death* ➔ *further Ix (underlying AMI, IHD, CM, digoxin toxicity)*

Ventricular bigeminy (regular Ventricular ectopic):
- *Abnormal premature ventricular complexes after every normal complex*
- *NB:*
  - ≥ 3 couplets = VT *while*
  - < 3 couplets = non-morphic VT (dizziness & syncope)

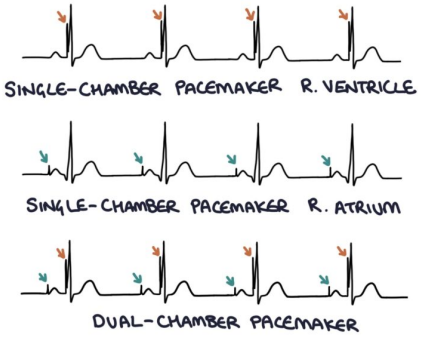


Atrial ectopic: [*can progress to AF*]
- Premature atrial contraction (PVA) – irregular rhythm
- **Narrow QRS +/- Early P-waves**
- Generated by atrium independently from P-waves



Pacemakers (battery powered - lasts 5 years)
- Pulse generator w/ pacing leads exciting relevant heart chamber
- Implanted in left anterior chest wall or axilla
- Modern pacemakers are MRI compatible
- Remove pacemakers prior to cremation

	Location	Indication
Single-Chamber	RA or RV	tachy-brady syndrome
Dual-chamber	RA **and** RV	2nd degree HB (Mobitz type 2) complete HB (3RD deg)
Triple-chamber (biventricular)	RA, RV **and** LV	**SEVERE Heart failure** for cardiac resynchronisation therapy (CRT) - to sync contractions
ICD		HOCMs Shockable arrythmia (VT, VF)



Supraventricular Arrhythmias

Atrial fibrillation (commonest arrythmia – 20% when >80)

1. **Trigger zone** = ectopic beats in left pulmonary vein
2. **Irregularly irregular** +/- tachycardia (rapid ventricular rate)
3. **absent P waves** (NOT SINUS rhythm)

Complications:

4. HF (due to poor ventricular filling during diastole)
5. Ischaemic stroke risk (emboli formation in **atrial appendage**)

Causes (Mrs SMITH)	General Rx
PIRATES • Pulmonary (OSA, PE, COPD) • **IHD** • RHD • Anaemia / alcohol • **Thyrotoxicosis** / toxins • Electrolytes • **Sepsis** (infection)/ sick sinus syndrome • Other: **HTN**, **mitral valve** (MS, MR)	***1) Rate control*** *(safer to proceed with)* **1. BB** (metoprolol 25mg PO or 2.5mg IV - ***fast onset*** — *negative inotrope* **2. Digoxin** (500mg IV or PO) – ***neutral inotrope*** — *vagal mediated but takes 4-6 hrs* **3. Amiodarone** (300mg over 1st hr then 900mg over 24 hrs - ***neutral inotrope*** — *Prolongs QT and multiple side effects* ***2) Rhythm control*** *(if < 48 hrs and haem unstable and no signs of left atrial dilatation)* **1. Flecainide (50-100mg PO)** — only if structurally normal heart without CAD **2. Amiodarone** ***(same as above)*** — *prolongs QT, slow 24 h*, **Less efficacy than flec** **3. Synchronised DC Cardioversion** — *Needs* **sedation**, ***immediate safe, + pacing***

Types of cardioversion

1. **Pharmacological** – flecainide OR amiodarone (if structural HD)
   1) **Flecainide** = *more efficacious but needs healthy heart*
2. **Synchronised DC cardioversion** = needs sedation + works bests if drugs given
   1) TTE + warfarin for 4/52 (check INR)
   2) TTE prior to cardioversion to check for clots in LA appendage)
   3) Single shock ➔ 50J (flutter) or 100J for AF
   4) **Immediate** (AF < 48 hrs or unstable)
   5) **Delayed** (AF > 48hrs + stable) + need 3 wks anti-coagulation + rate control prior to prevent embolic stroke

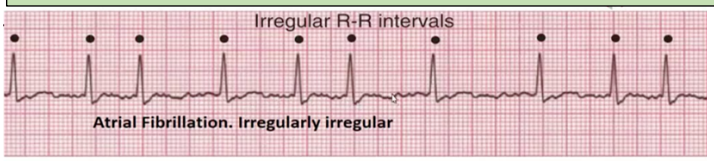
Letter	Risk factor	Score
H	Hypertension	1
A	Abnormal renal and liver function (1 points each)	1 or 2
S	Stroke	1
B	Bleeding	1
L	Labile INRs	1
E	Elderly (e.g., age >65 years)	1
D	Drugs or alcohol (1 point each)	1 or 2

CARDIOVERSION CI:

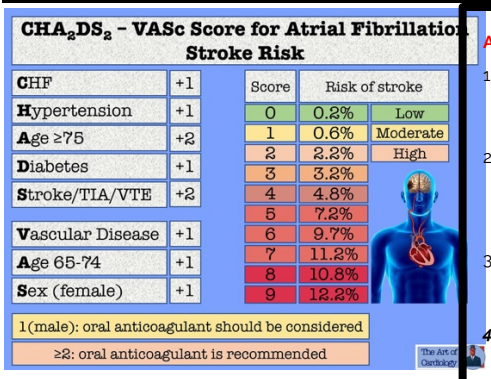
- Old > 70yo
- obese

Reconsider DOAC if HAS-BLED Score > 3

AF + fast V. rate ➔ unstable haemodynamics ➔ urgent cardioversion



Types	*Definition*	*Rx*
AF + ventricular changes	AF with normal Vent fn	**flecainide (I)**
	AF with LV dysfunction (LVEF <40%)	**amiodarone (III)**
	AF w/ controlled ventricular response (+/- on meds)	**pacemaker**
Lone AF	**esp. young children**	**ablation**
Paroxysmal AF	episode < 7 days & self-terminating BUT reoccur	**Flecainide** (if not atrial flutter)
Persistent AF	> 7 days not self-terminating	Cardiovert or Digoxin
Permanent AF	cardioversion fails	rate control



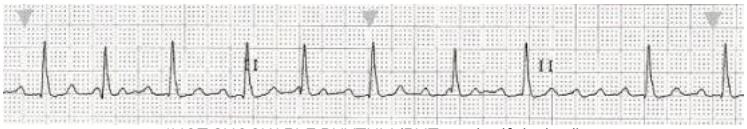
Anti-coagulation w/ DOAC

1. no monitoring required, minimal drug interactions, reduced bleed risk
2. **Reduces risk of stroke by 2/3rd (risk of stroke sig. outweighs the risk of bleeding.)**
3. no need to give aspirin (no evidence for preventing strokes)
4. ***Consider Lifestyle advice*** *= + manage DM, OSA, HTN, HC*

Atrial flutter (SAWTOOTH appearance)

- **Trigger zone in RA** = constant **automaticity firing of large (macro) re-entry circuit (foci),** within **RA around tricuspid annulus**

Character	Causes	Comp.
• Self-perpetuating loop • **May be regular /irregular (variable block)** • Reveal w/ adenosine • NOT normal P waves • Regular 2: 1(V. rate = 150) • Regular 3: 1(V. rate = 100) • Regular 4: 1(V. rate = 75)	• **HTN** • **CM** (atrial enlarged) • **IHD** • **Cong HD** • **Lung disease** • **Infection** • ↑**SNS** (pain, thyrotoxicosis)	Stroke/TIA



*NOT SHOCKABLE RHYTHM (BUT can be if desired)

TREATMENT:

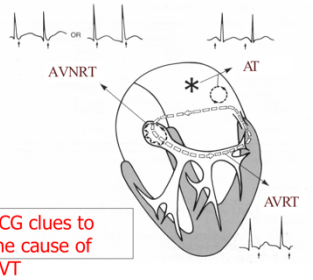
- + treat underlying cause
- **Rate/rhythm control** = BB
- **Anti-coagulate** (based on CHADS-VASC socre)
- **RF Ablation** of reentry rhythm usu at **Cavo-tricuspid isthmus)**

Supraventricular tachycardia (SVT): (3 types)

- **No P-wave**
- **Regular Narrow complex tachycardia** (150-220bpm)

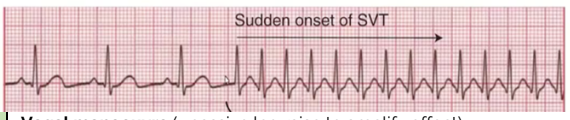
Multiple mechanisms (main 3 types)

1. **AVNRT** (fast + slow conduction) = **most common – re-entry point is back through AV node**
   RP shortest = narrow complex tachy
2. **AV re-entrant tachycardia** (orthodromic **WPW**)
   RP longer due to longer distance
3. **Atrial tachycardia**
   Prolonged RP → causes AV block → paroxysmal tachy



RP interval length = AVNRT < AVRT < AT

**RP interval* = retrograde P wave (bottom → top)



1st line

Vagal manoeuvre (+ passive leg raise to amplify effect)
- carotid sinus massage – *beware in elderly*
- valsalvre - cough, poo

2nd line (to return sinus rhythm)

	Pros	Cons
Adenosine 6 or 12 mg 20mL flush	• Short $t_{1/2}$ • ↓Ca influx • ↑K efflux	• Impending doom + • facial flush • MI • Bronchospasm (asthma) • No prophylaxis
Verapamil 2.5-5mg IV	• Avoids A/E of adenosine • Ongoing prophylaxis	Negative ionotrope

3rd line or if drug CI

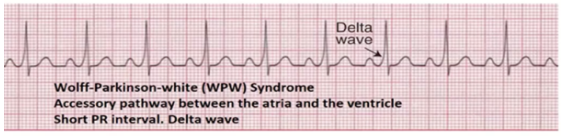
Synchronic cardioversion
- Caution with AF in WPW
- No ongoing prophylaxis
- **Avoid AVN blockade** → causes retrograde conduction → creating VT → need DC cardioversion
- **Selective RF ablation** near AV node (always successful!)

3) Wolff Parkinson-white (WPW) syndrome

- **Accessory pathway** (**bundle of kent**) between atrium and ventricle
- **Usu. accessory pathway** is located on *posterior left ventricular* free wall → resting ECG should show RBBB → early activation

Key features

- Young unheralded syncope
- Short PR interval + wide QRS complex
- Delta wave (slurred upstroke on QRS complex)
- **Type A WPW** = left-sided accessory pathway (dominant R wave V1)
- **Type B WPW** = right-sided accessory pathway (dominant S wave V1)



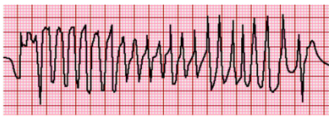
- **WPW + Afib/SVT = 600bpm = irritation + ischeamia = SUDDEN DEATH**
- ***CI*: AV-node blocking drugs** (e.g. BB, CCB, adenosine) – promotes pathway
- If haem stable → slow conduction temporarily → procainamide (before ablation)
- ***Rx*: RF surgical ablation fo accessory pathway (esp. if driver, pilot)**

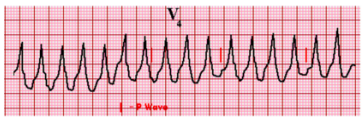
Wide-complex Tachycardia (LIFE THREATENING)

Ventricular tachycardia (4 types)

- **Trigger zone:** Abnormal automaticity of ischaemic tissue or by re-entry within **scarred cardiac tissue** (usu. **Right ventricle**)

Susceptible Heart	Precipitating event
Conduction (ECG) - Channelopathy - Pre-excitation - Long QT	Mechanical - Wire - Tamponade - Trauma
Myocardium (ECHO) - IHD - Cardiomyopathy - Myocarditis	Serological - Electrolytes - Drugs/Toxins - Catecholamines - Hypoxia - Endocrinopathy
Idiopathic VT (EP)	





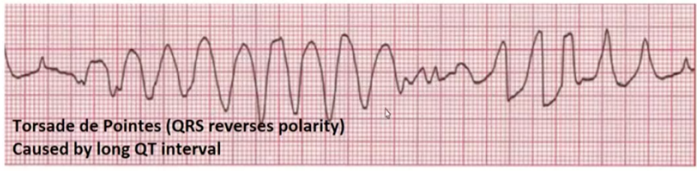
Monomorphic (conscious) VT	• Late sustained VT → permanent arrythmic re-entry circuits **Dead**: ALS + DC cardioversion **Almost Dead**: Synced DC cardioversion +/- sedation **Unwell**: Sedation → synced cardioversion **Well**: Drugs – **amiodarone, lignocaine, BB** ➢ **Avoid verapamil if signs of HF**
Polymorphic VT = torsades	• Look for QT prolongation • **Rx: defibrillation & temp. pacing wires (if bradycardia)** • **Remove causes (i.e. meds)** • **NO amiodraone = prolongs QT**
Sustained VT	***(> 30s) -*** heamodynamic compromised → get ready to shock
NSVT	***(< 30s or > 3 beats)*** → check electrolytes & troponin, put on BB

Torsade de Pointes ("twisting of tips")

➢ **Polymorphic VT**
➢ **Usu. self-limiting but can progress to VT**

Causes

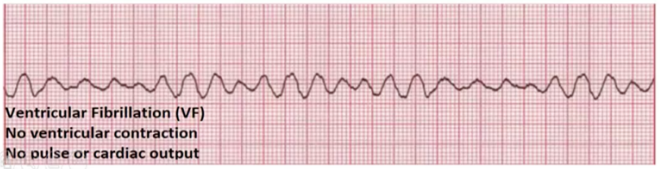
- **Long QT syndrome** (inherited)
- **Medications** (antipsych, citalopram, flecainide, sotalol, amiodarone, macrolide Abx, anti-emetics)
- **Electrolyte** (Low K, Mg, Ca) secondary to XS vomit (alcohol binge)



Acute management	***Long-term***
1. **DC cardioversion** 2. **Correrct/Stop offending drugs** (e.g. anti-arrythmias, anti-microbials, anti-psychotics, anti-emetics, TCA) 3. **IV $MgSO_4$ infusion** 4. Speed up → use bradyarrhythmia Mx	• **Check meds** (e.g. BB (not sotalol)) • **Correct electrolytes** • **ECHO - ?cardiomyopathy** • **PPM or ICD**

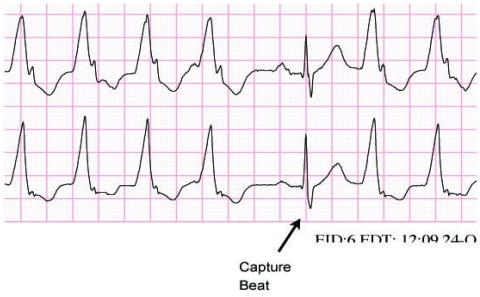
Ventricular fibrillation (VT → VF)

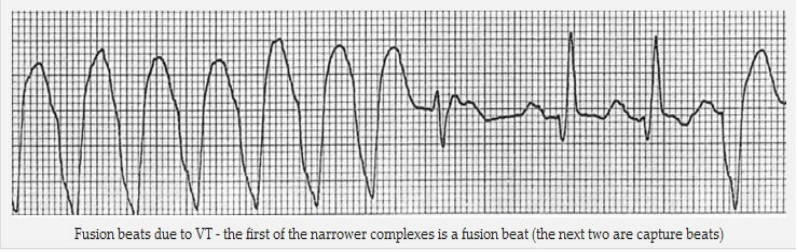
- **Trigger zone:** Multiple re-entrant circuits creating small wavelets that breakdown and depolarise causing high-freq activation of cardiomyofibres ➔ loss of contractility and blood flow ➔ myocardial ischeamia
- **Irregular, shapeless QRST waveforms**
- **S+S: NO** pulse or cardiac output [**NO** ventricular contraction)
- **Ix = rule out cause:**
  - **Troponin**
  - **CTA** = Stenosis, STEMI
  - **ECHO** = heart failure
  - **Cardiac MRI** = Infiltrative, arrhythmogenic cardiodysplasia



AICD + CPR

Beware of idiopathic VF causes: **brugada syndome (Na channel blockage), catecholamine surges → patients need implantable defribillators**





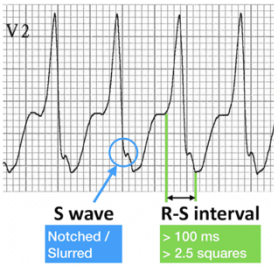
Fusion beat suggests = VT

VT vs SVT with aberrancy

Brugada Criteria (differentiate SVT from VT) – if any Yes = VT

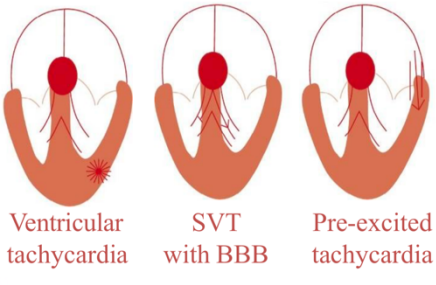
- Broad complex tachycardia
- Extreme RAD
- Absent rS complex (i.e. no RBBB/LBBB)
- AV dissociation or absent P waves
- **Brugada sign:** Onset of R wave to trough/nadir of S wave is > 100ms in leads V1-6
- **Josephson sign**: Notching/slurring near the trough/nadir of S wave

**Broad complex tachycardia (SVT or VT) --> SVT does not give major haemodynamic compromise unlike VT which is life-threatening (hypotensive and unwell – shockable rhythm)*



Capture and Fusion beat

- **Capture beat** = intermittent atrial beat before ventricular beat [normal beats than back to abnormal]
- **Fusion beat** = When a supraventricular/atrial beat is superimposed on an ectopic ventricular beat → hybrid complex Is generally **narrower** than a ventricular beat
- ***indicates*** that there are 2 foci of pacemaker cells firing simultaneously: a supraventricular pacemaker (e.g. the sinus node) and a competing ventricular pacemaker (source of ventricular ectopics).



Wide complex tachycardias

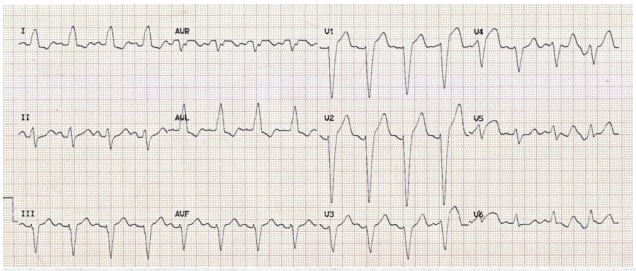
- VT (80% of the time)
- SVT with BBB (20%)
- Pre-excited tachycardia

Rx:

➢ *amiodarone (chemical cardiovert) in consicous pt*
➢ **avoid BB, CaB**

BUNDLE BRANCH BLOCKS: (Wide-QRS complex) – 3 conduction paths

- RBBB
- Anterior fascicle of left bundle branch (i.e. left anterior hemi-block) → marked LAD
- Posterior fascicle of left bundle branch (i.e. right anterior hemi-block) → marked RAD

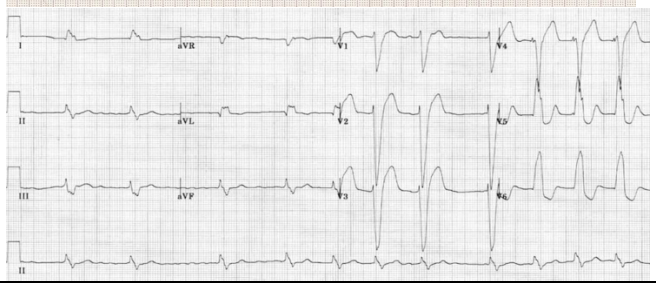


LBBB criteria

- Wide QRS complex (>120ms)
- QRS in V1 = Dominant deep S wave
- QRS in V6 = positive (notched "M" wave)
- No Q waves in lateral leads
- Broad monophasic R wave in lateral leads (I, aVL, V5-6)
- ST segment **discordant** with QRS complex

Cause:

- AS
- IHD
- Systemic HTN
- Anterior MI
- Cardiomyopathy
- Hyperkalemia
- Conduction system fibrosis

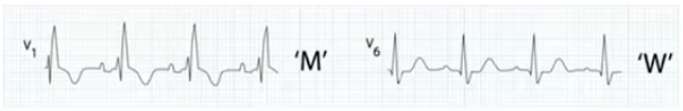


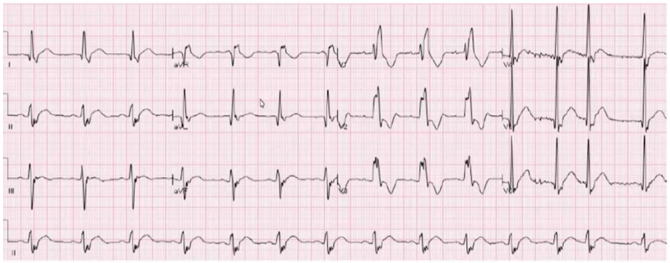
RBBB criteria:

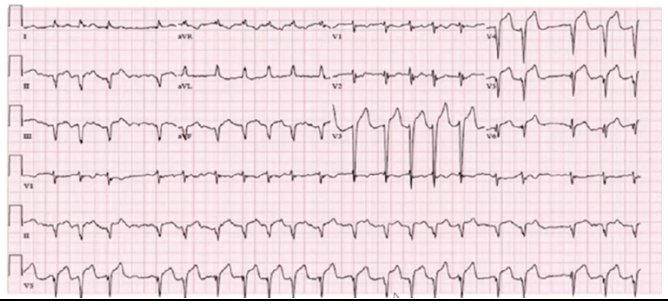
- Wide QRS complex (>120ms)
- QRS in V1-3 = "R" pattern + "M"
- QRS in lateral leads = wide slurred S-wave = "W" pattern
- Up and down P wave (atrial enlargement)
- T wave inversions

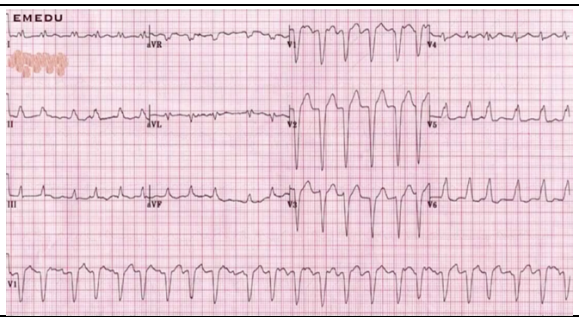
Cause:

- RVH/Cor pulmonale
- PE
- ASD
- IHD
- Cardiomyopathy







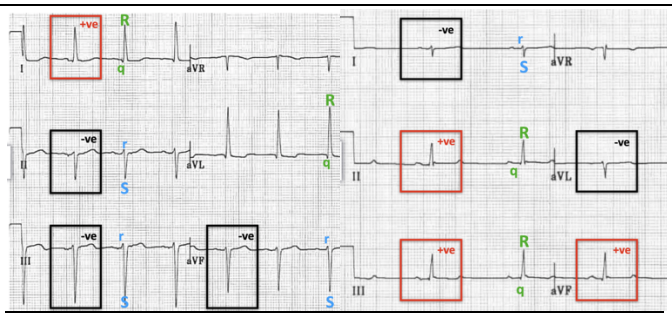


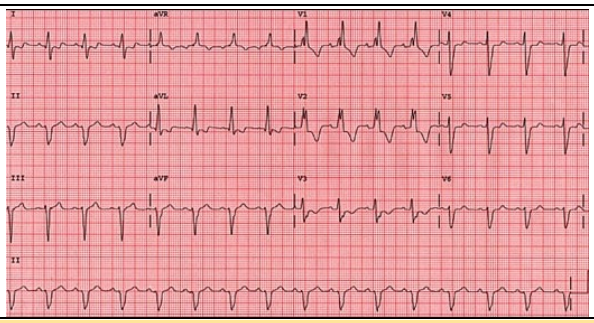
AF with rapid ventricular response rate

- Irregularly irregular tachycardic non-sinus rhythm

AF with rapid ventricular response rate + LBBB

- Irregularly irregular tachycardic non-sinus rhythm
- LBBB → V1 is positive, V6 is negative
  - Deep s wave in V1
  - Wide QRS





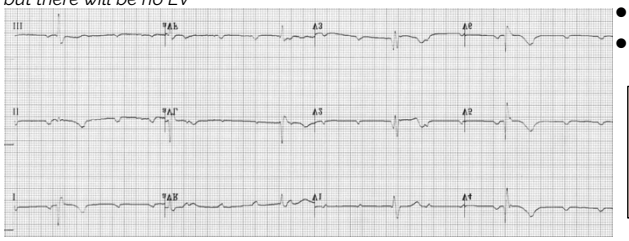
Anterior fascicular block (Q1S3)

- LAD (MOST common cause) – depolarise to inferior lead
- rS (deep S) = II, III and aVF (+ve)
- qR (tall R) = I, aVL**(-ve)**

** In LAFB, QRS voltage in lead aVL may meet voltage criteria for LVH (R wave height > 11 mm), but there will be no LV strain pattern*

Posterior fascicular block (S1Q3)

- RAD – initial conduction to lateral leads
- rS (deep S) = I, aVL **(-ve)**
- qR (tall R) = II, III and aVF (+ve)
- Slightly prolonged QRS (0.1 - 0.12s)



Bifascicular Block = 2 out of 3 fascicles blocked **Either:**

- **RBBB + left anterior fascicular** block (Seen as LAD) OR
- **RBBB + left posterior fascicular** block (seen as RAD) – less common due to *dual blood supply*
- Only one fascicle is left for conduction, and if that fascicle is intermittently blocked: can develop Mobitz 2

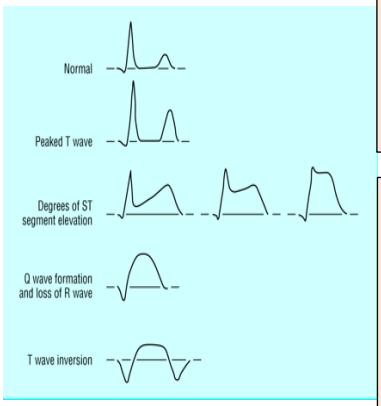
Trifascicular block either presents as

- 3rd degree AV block + RBBB + LAFB or;
- 3rd degree AV block + RBBB + LPFB

Incomplete Trifascicular block

1. May process to complete HB
2. **May be assoc. with syncope**

Evolution of STEMI



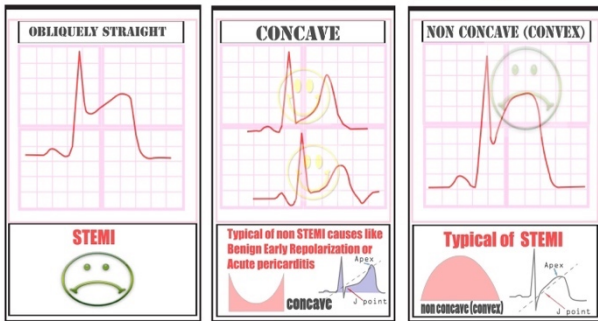
STEMI progression

1. **Normal**
2. **Peak T wave [5-30mins]**
3. **ST elevation [day 1-2**
4. **Pathological Q wave [permanent]** (2-3 days after) if myocardium not perfused
5. **QT prolongation + Reciprocal changes**
6. **T wave inversion [*weeks to mths]***

POST-STEMI progression

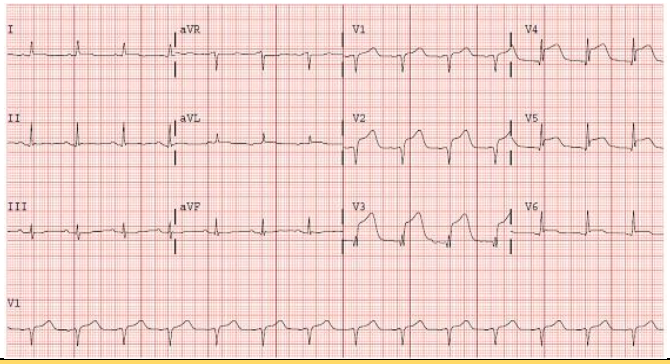
- **0-4hrs= none**
- **4-12 hrs** = coagulative necrosis
- **12-24 hrs** = neutrophil migration (↑ WCC)
- **1-3d** = acute inflame + fibrinous pericarditis
- **3-14d** = macrophages + granulation tissue
  - Free wall rupture = cardiac tamponade
  - Papillary muscle rupture = MR, IV septal
- **2wks-mths** = scar + true ventricular aneurysm + dressler (fever, effusion, arthralgia)

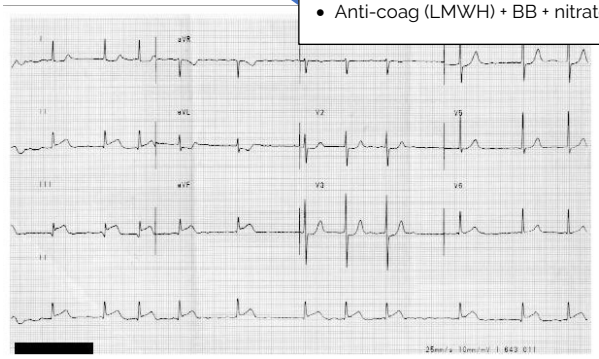
ST segment elevation morphologies



STEMI → MONA BASH ← NSTEM

MONA BASH:
- DR ABCD + defib pads
- 5000units of heparin before PCI (within 12 hrs)
- Anti-thrombolytics (> 1hr)
- Anti-thrombotic (aspirin, clopidogrel)
- Anti-coag (LMWH) + BB + nitrate





Anterior STEMI

- V2-5 suggests anterior pathology LAD artery with pathological Q wave (i.e. full thickness myocardial infarct)

If not chest pain → persistent ST elevation

- Aneurysm to LV, Old infarct?
- Rx: morphine, oxygen (only if sats ,< 92%), nitrates and aspirinn

Acute inferior MI

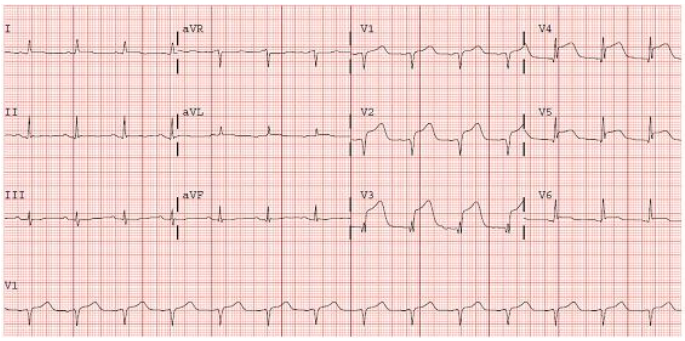
- **> 1mm ST elevation** inferior leads
- **Absence** of Q waves

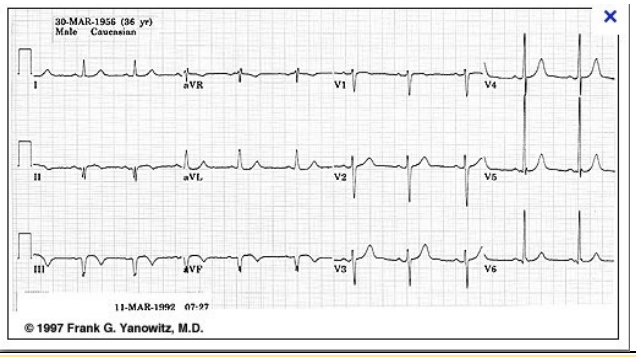
In AF what rhythm disturbance is common with acute inferior MI??

- **Sinus bradycardia**

What medication should be avoided in inferior Mis?

- **avoid nitrates - high risk of hypoTN**



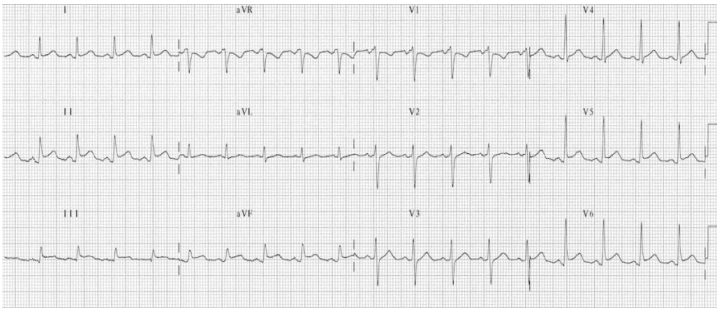


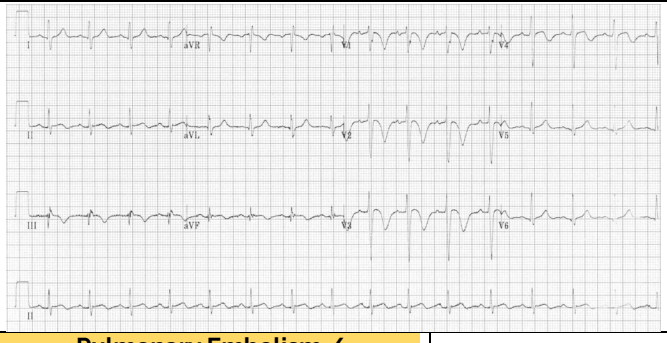
Anterior LV infarction/aneurysm (opp. For posterior LV infraction)

- Dominant **INVERTED** R wave in V1/2 [*Dominant R wave*]
- Horizontal ST elevation in V1-3 [*Horizontal ST depression*]
- **Cause**: usually in apex of heart → after a transmural myocardial infarction
- Echo to confirm

Transmural Inferior infarct (acute occlusion)

- **Pathological Q waves (Criteria)** = Q wave >1/3 of the ensuing R wave in depth and/or >0.04s in duration in at least two leads not aVR
- Has **reciprocal ST depression or TWI**





Pericarditis

- **Stage 1:** Diffuse saddle-shaped ST elevation + **PR depression (specific sign)**
- **Stage 2 (wks 1-3):** flat T wave, normal ST
- **Stage 3 (> 3 wks):** T wave inversion
- **Stage 4 (many wks):** normal ECG
- **Risk of pericardial effusion**

- **Cause** = idiopathic, RHD (GAS), SLE, metabolic, drugs
- **Px**: pleuritic chest pain, relieved on bending forward (friction rub
- **Ix + Rx:** ECG→: NSAIDs or colchicine

Pulmonary Embolism / Acute Right heart strain

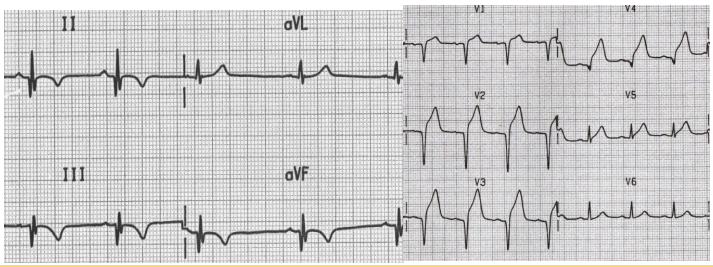
****Acute PHTN = PE until proven otherwise****

1. **Sinus tachycardia (most common)**
2. **Ant. T-wave inversion** (due to R ventricular stress)
3. **RBBB (18%)**
4. **Extreme RAD (16%)**
5. **S1 Q3 T3** = rare

*deep S (lead I), Q wave + T wave inversion (III),

- Chest pain, SOB
- Haemoptysis
- Tachypnoea
- High V/Q mismatch
- RF: post-op, long-flight, immobile, COCP, pregnant

MYOCARDIAL INFARCTION anomalies



Pathological Q waves

- > 40ms (1mm) wide
- > 2mm deep
- >25% of depth of QRS
- Seen in V1-3

Significance = current or prior MI

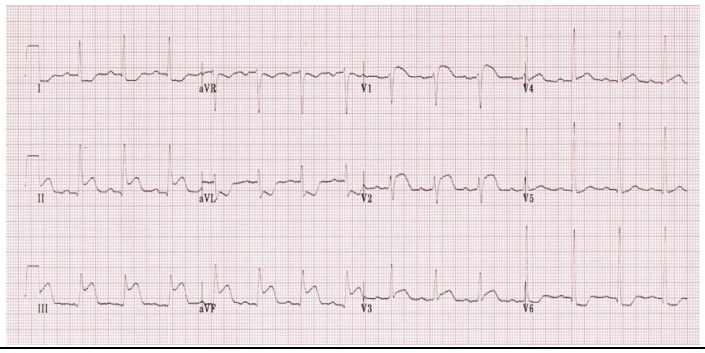
Non-ischaemic ST elevation

Normal
- variant V2-V3
- BERP

Conduction
- Brugada syndrome
- WPW

Plasma
- Hyperkalaemia
- Hypercalcaemia
- Drug toxicity

Myocardium
- Peri/myocarditis
- LV aneurysm
- Takotsubo

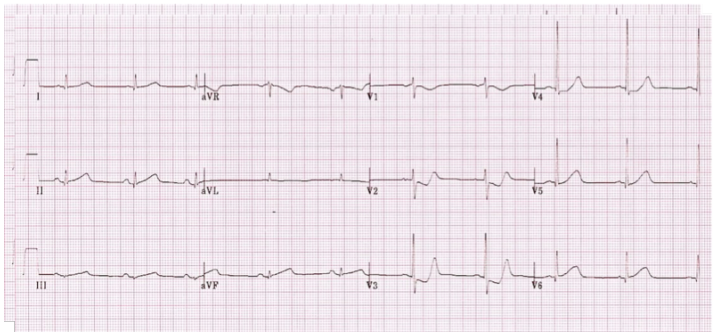


RV infarct

- Kussmaul's sign

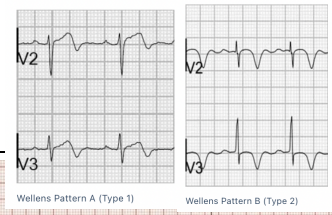
Inferior STEMI. RV infarction is suggested by:

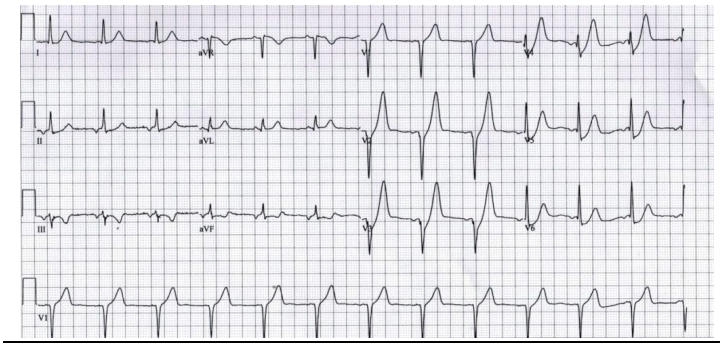
- ST elevation in V1
- ST elevation in lead III > lead I
- **Rx with increased preload to push blood out, hence avoid nitrates**



Posterior STEMI

- tall R waves in V1/2

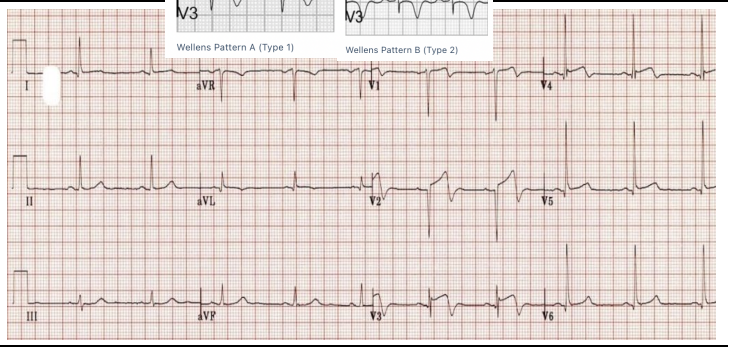




De Winter T wave sign –

Precordial leads (V1-6) with

- upslope ST segment depression > 1mm from J point from **below isoelectric line**
- **Tall prominent symmetrical T waves**
- Reciprocal ST elevation in AVR
- **SIGNIFICANCE=** preceding or post- STEMI



Wellen's syndrome

2 TYPES:

- Wellen type A (25%)– biphasic
- Wellen type B (75%) – Deeply inverted T wave in V2/3

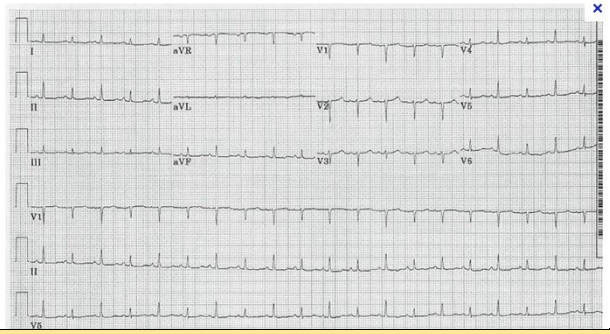
SIGNIFICANCE =Highly specific for critical stenosis of LAD (LV infarct)

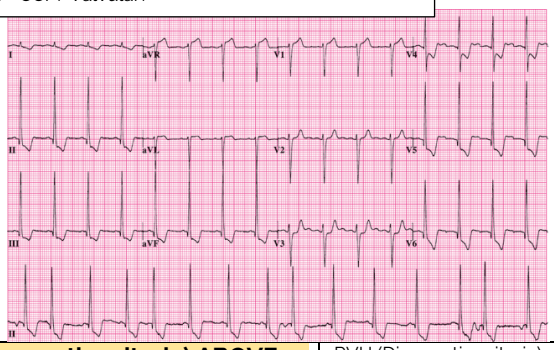
Key notes:

- streptokinase (54% reperfusion) vs PCI (90% reperfusion) --> check if ST elevation drop > 30%

Other rare ECG anomalies

DDx of LVH
- Young = aortic coarctation (Marfan's, Turner's)
- Old = CCF? Valvular?





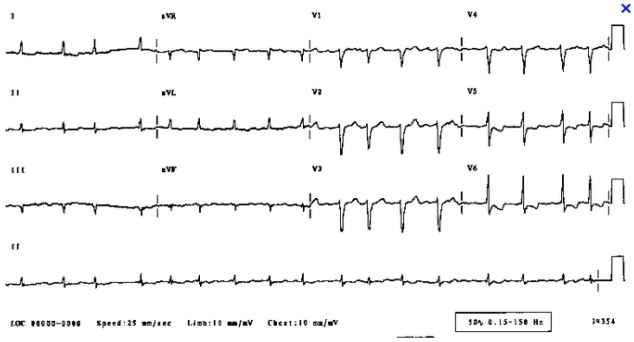
Pericardial effusion/ tamponade

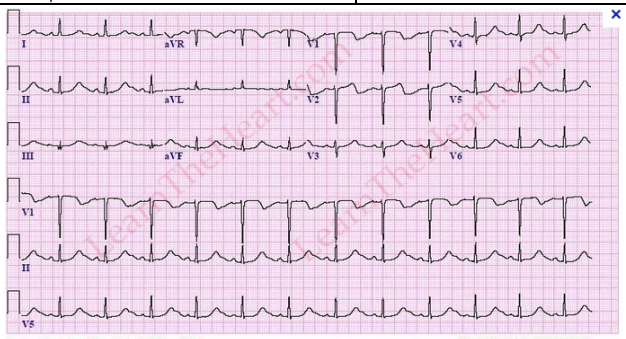
- *Small amplitude* ECG
- ***SPECIFIC SIGNS***
  - Pulsus paradoxus (↓SBP during inspiration due to ↑LV afterload = ↓SV)
    DDx: Acute asthma, COPD, pericarditis, tension pneumothorax
  - Kussmaul's compensatory hyperventilation (JVP falls on inspiration)
- ***NON-SPECIFIC SIGN*** = peripheral oedema, raised JVP

LVH (Diagnostic criteria) ABOVE

- LAD
- Dominant S wave in V1 + tall R wave in V5 or V6] > 7 big squares
- R wave in aVL > 10mm
- T-wave inversion (lateral leads = V5-6)
- Lateral ST depression assoc. with LVH: strain pattern

RVH (Diagnostic criteria)
- RAD
- Dominant R wave in V1
- Dominant S wave in V5/6
- T-wave inversion (right-inferior chest leads = V1-3, II, III, aVF)





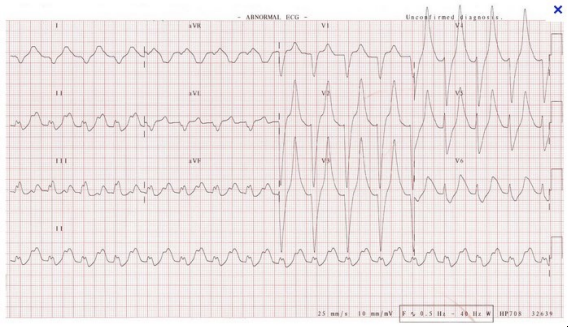
Digoxin Effect [shortened QT]

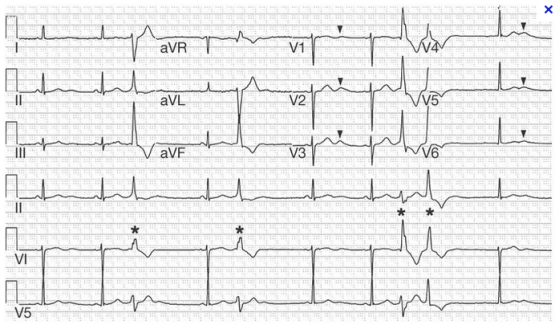
- Prolonged PR
- Short QT + U wave (**reverse tick)**
- Flat T wave
- J point depression

Digoxin blocks Na/K channel = hypokalameia and hyperNa
- Dizzy, headache, GI distress, gynacomastia, xanthopsia (yellow/orange tinge night vision)

Prolonged QT interval > half the R-R interval (DDx: HOCM)

- **Drugs:** amiodarone, TCA's, antibiotics, fluconazole, erythromycin, metoclopramide, quinidine, haloperidol, methadone, ondansetron, SSRI's (citalopram), furosmide
- **Genetic**: cardiac ion channel mutation (Na+, K+), **Romano-Ward syndrome** = most common form of congenital long-QTc syndrome
- **myocardial disease**: MI, RF, 3rd degree HB, cardiomyopathy
- **electrolytes**: low Ca2+, low K+, low Mg2+



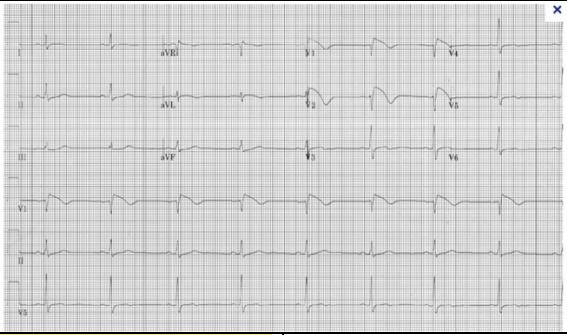


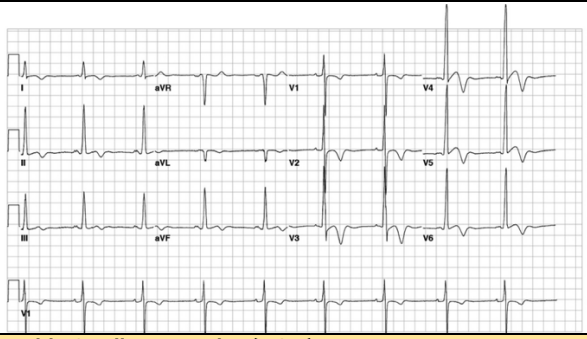
Hyperkalaemia

- **(1) Tall tented T waves → Prolonged PR → Widened QRS + Bradycardia**
- Rx: IV calcium gluconate + insulin with glucose

Hypokalaemia

- **U waves + flattened T waves + prolonged QT interval**
- Frequent VEBs +/- ST depression





Brugada Syndrome

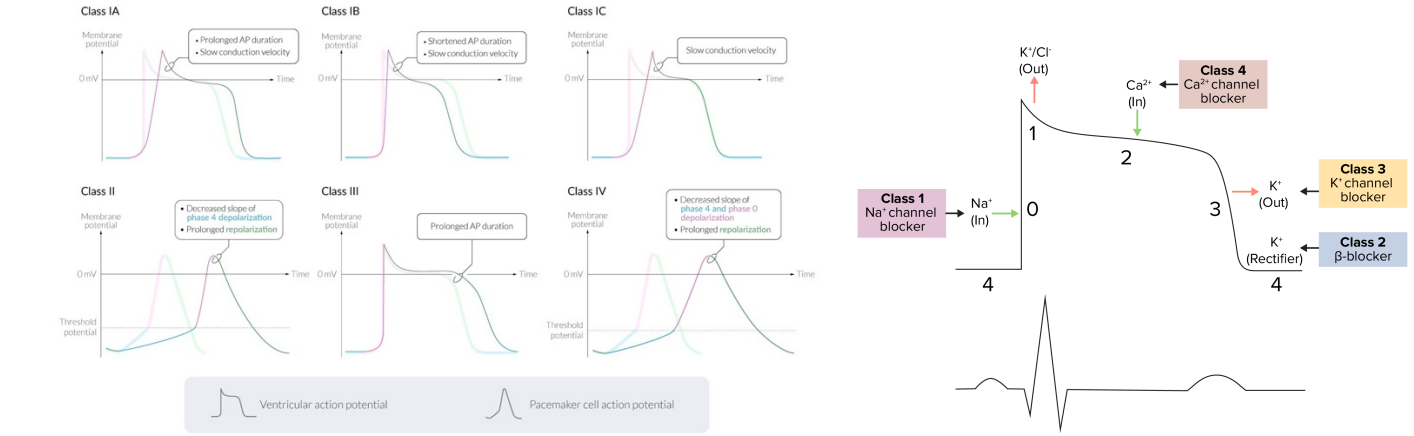
- **RBBB**
- **Cove-ST (V1-V3)**
- **T wave inversion (Brugada sign)**
- **Prolonged Pr**

- **Mostly SE Asians + AD mutation** in Na Channel (SCN5A)
- **Complication** = syncope, VT, VF, **sudden CARDIAC death**
- **Rx: ICD**

Hypertrophic Cardiomyopathy (HCM)

- **LVH + dagger-like Q waves in infero-lateral leads**
- **Px**- angina, collapse → Ejection systolic murmur (increased on standing)
- **DDx**: ARVC – inverted T wave (V1-3) + Fhx of sudden cardiac death
- **ECHO**: Assymetrical left ventricular hypertrophy + IVS thickening
- **Rx: Exercise, BB, anti-hypertensives**

Class	Drugs	MoA	Indications	A/E
1a - NaB	*Quinidinde, procainamide* **(rate control)**	• Moderate Na+ channel blocker – prolong repolariations	SVT, VT	prolonged QT [Torsades] cinchonism = Blurred vision, GI upset, tinnitus, confused
1b – NaB	*Lidocaine, phenytoin* **(rate control)**	• Mild Na blockade • Shorten phase 3 repolarisation	VT or after MI	CNS depression / seizures / tetany AV block
1c - NaB	*Flecainide, propafenone* **(rate control)**	• Marked Na blockade → sig. slows phase 0 upstroke	SVT, VT, AF	Prolonged PR + QRS duration
2 =BB	*Propranolol, metoprolol* **(rhythm control)**	• BB = ↓cAMP = ↓ Ca = ↓ SA/AV nodal activity • Decrease phase 4 depolarisation	SVT, AF	Exacerbate asthma, COPD
3 = K	*Amiodarone, sotalol* **(rate control)**	• Blocks K+ channel → slows AV node → prolong Phase 3 repolarisation	AF, SVT, VT	Prolonged QT, pulm. Fibrosis (amiodarone)
4 = CaB	*Diltiazem* *Verapamil* → **-ve ionotrope**	• CaB = slows phase 4 spontaneous depolarisation	SVT, AF	Headache (nifedipine) AV block (verapamil)
5	**Adenosine**	• Activate Gi protein = ↓cAMP = ↓ Ca = transient AV nodal block • Short-acting → immediate flush	Terminate AVNRT and orthodromic AVRT	Sense of impending doom HypoTN, chest pain, flush
	Digoxin	• Inhibit Na+/K+ ATPase • Increased contractility + ↓ HR	AF, atrial flutter, chronic systolic HF (HFrEF)	Xanthopsia, GI pain/N/V, gynacomastia, hyperK



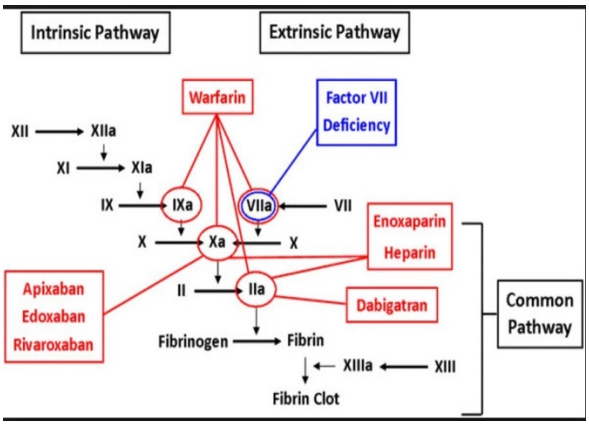
CHA_2DS_2-VASc Score	
CHF (heart failure)	1
Hypertension	1
Age ≥ 75	2
Diabetes	1
Stroke	2
Vascular Disease	1
Age 65-74	1
Sex Category (female)	1

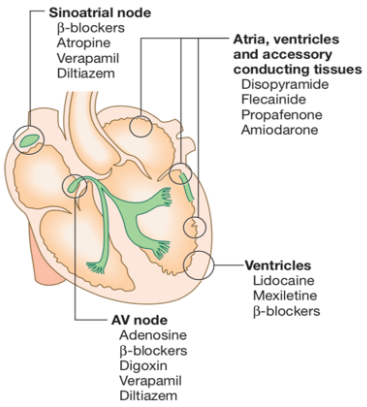
First-Line (for both): DOAC therapy over warfarin

Anticoagulate based on CHA_2DS_2-VASc score

CHEST 2018
≥1 for males or ≥2 for females (i.e. at least 1 non-sex risk factor)

AHA/ACC/HRS 2019
≥2 for males or ≥3 for females (i.e. at least 2 non-sex risk factors)





	Class	MoA	Indication	Benefits	CI
Warfarin PO	Anti-coag – bridging therapy to DOAC	Vit K antag	**ONLY** for AF, atrial flutter, VTE prostatic heart valves (anything protein rich	Cheap, known A/E, ok for AKI **FFP + VIT K over days**	Regular iNR monitor, skin necrosis
DOACS	Anti-coag – continue for > 3/12 (stop when safe - e.g. ongoing leg pain/swell common due to damaged veins for DVT)	Direct Xa inhibitor → apixaban (Eliquis), rivaroxaban (Xarelto)		No HIT, NO obs, few drug-drug interactions	SOME reversible, caution AKI USE, expensive
Heparin (SC)	Anti-coag -	Activate Anti-thrombin III		ok for AKI - **protamine**	Narrow TI, HIT, OP, Regular Obs,
Aspirin	Anti-platelet	COX inhibitor	**More for stroke** *Not for DVT/PE*		
Clopidogrel	Anti-platelet	P2Y12 inhibitor			
Ticagrelor	Anti-platelet	P2Y12 inhibitor			
Abciximab	Anti-platelet	GPIIB/IIIA inhibitor			
Dipyridamole	Anti-platelet	PDE inhibitor		**Used if clopidogrel CI**	
Streptokinase	Fibrinolytics (synthetic)	Convert plasminogen into plasmin for clot breakdown	• **PE, DVT**		•
t-Pa	Fibrinolytics (intrinsic)		**Wait for PCI**		

MANAGEMENT OF ADULT TACHYCARDIA / BRADYCARDIA

Tachycardia

Assessment: (Are they stable/unstable)
1) A-G
2) FiO2
3) IV access - FBC, EUC, CMP, VBG, troponin
4) 12-lead ECG
5) Identify reversible causes (4 H's, and 4 T's)

Bradycardia (complete HB, meds?)

- Cause
  - Vasovagal shock (removal of carotid sheath)
  - HB
- Stable
  - Senior review
  - Dual chamber pacemaker (synchronise the atria and ventricle)
- Unstable (syncope or shock) + detectable pulse
  1) IV 500mcg atropine (max 3mg) every 3-5 minutes
  2) Consider transcutaneous pacing
  3) May need IV adrenaline 6mL (1:1000) infusion or IV isoprenaline infusion
  4) Urgent Dual chamber PPM
- Unstable = NO pulse
  1) PEA algorithm

Transcutaneous pacing

1) Clip hair and dry skin
2) Negative pad on anterior chest
3) Positive pad on back
4) Sedation – iV fentanyl 50-100mcg and IV midazolam 1-5mg
5) Pacing mode → 70mA
6) Increase mA and pacing until captured
7) Set pace 10mA above threshold
8) Ongoing sedation and analgesia

Stable

Narrow complex (QRS < 0.12s)
Any symptomatic tachycardia is NOT sinus tachycardia

Regular

SVT (AVNT, AVNRT, AT)
1) valsalvre + carotid massage
2) IV 6mg adenosine rapid bolus
*consider verapamil if asthma

Atrial flutter
1) control rate - BB

Other
(junctional tachycardia)
(sinus tachycardia)

Irregular

AF
(rate vs rhythm control)
1) BB,
2) digoxin or amiodarone (if known HF)
3) DC cardiovert if hypoTN

Others
(atrial flutter with variable block)
(Multifocal atrial tachycardia)
(Drugs= digoxin toxicity)
(AT with variable block)

Uncertain

Attempt vagal manoevres (i.e. Valsalva with head down tilt)

If reverts = likely SVT
If NOT reverts =consider adenosine and expert advice

Wide complex (QRS > 0.12 s)
Assume VT until proven otherwise

Regular

Monomorphic VT

Pulseless
Treat as shockable rhythm (ALS protocol)

Pulse
- Pulse but compromised → Go to Unstable pathway
- Haem stable → Consider either:
  1) IV amiodarone 300mg in 5% dex 100mL over 20-30min
  2) IV sotalol 1.5mg/kg in 5% dextrose over 15-30mins
  3) iV lignocaine 1-1.5mg/kg

Others
(SVT + BBB)
atrial flutter

Irregular

Polymorphic VT (Torsades - QTc > 450ms)

Unstable torsades → go to unstable Algorithm

Stable Torsades
1) **STOP offending drugs** (e.g. neuroleptic, macrolides, sotalol, amiodarone, flecainide, disopyramide)
2) **check serum K+**

Temporary pacing OR
IV MgSO4 (50% in 4mL) Or
IV isoprenaline OR
IV lignocaine

Bradycardia: 500mcg IV atropine up to 3mg (every 3-5mins)

Others
AF + BBB or
AF + WPW
Aberrant conduction in SVT

Uncertain

Presume VT until proven otherwise

1) Semi-elective cardioversion
2) IV amiodarone 150-300mg in 5% dex over 20-30mins
3) check EUC, troponins

ID and Rx cause
1) FBC, EUC, CMP, VBG, ECG, troponin
2) ECHO, PCI, CXR

Unstable
1) chest pain
2) hypoTN
3) altered LOC

1) IV sedation (RSI) - ketamine and midazolam
2) Maintain airway
3) 3x sync DC shocks - 100-200J
4) Repeat up to 3 times
5) IV 300mg amiodarone in 5% dextrose over 20-30min
6) IV 900mg amiodarone in 5% dextrose over 24 hrs

ROSC

1) Secondary survey (A-G)
2) 12-lead ECG, CXR, NGT, IDC
3) ID and Rx cause – VBG, FBC, EUC, CMP, LFT, CRP, Troponin, ECHO
   a. Urgent PCI, thrombolysis for STEMI or new LBBB
   b. Anti-arrhythmic – 0.6mg/kg/h amiodarone
4) Maintain Temp (fever)
5) Normalise BSL control
6) FiO2 94-98%
7) Normal PaCO2
8) Check any damage due to resuscitation.