Acute Myocardial Infarction

- **Irreversible death of heart tissue,** while **Angina** is chest pain due to ischemia but still heart tissue is **alive**
  - *Subendocardial* (partial) infarct, 20-40m → **Non-STEMI**
  - *Transmural* (whole wall thickness) infarct, 3-6h → **STEMI**
  - ***Stable angina*** → relieved by rest/medications
  - ***Unstable angina*** → doesn't respond to rest/medications

Presentation

- **Chest pain** (central chest pain may not be the main symptom):
  - Three quarters of patients present with characteristic **central** or **epigastric** chest pain **radiating** to the arms, shoulders, neck, or jaw
  - The pain is described as **substernal pressure**, **squeezing**, **aching**, **burning**, or even **sharp** pain
  - Radiation to the **left arm** or **neck** is common
  - Chest pain may be associated with **diaphoresis (caused by increased sympathetic activity)**, **nausea**, **vomiting**, **dyspnea**, **fatigue** and/or **palpitations**
  - **SOB**: may be the patient's anginal equivalent or a symptom of heart failure
  - Could be **painless** in **DM (autonomic neuropathy)** → ***Silent MI***

Atypical presentations *are common and tend to be seen in women, older men, people with diabetes and people from ethnic minorities. Atypical symptoms include* ***abdominal discomfort*** *or* ***jaw pain****; elderly patients may present with* ***altered mental state***

Investigation

- **Troponin** (increase within 3-12h, peaks at 24-48h and return to baseline over 5-14 days)
- **CK-MB** → return to baseline after 48-72h, specificity and sensitivity are not as high → useful to detect reinfarction (10%)

Cardiac enzymes aren't raised in ***unstable angina***

Management of acute attack [MONA]

- **Morphine IV**
  - To relieve pain
  - Avoid intramuscular (IM) → *absorption is* ***unreliable*** *and the injection site* ***may bleed*** *if the patient later receives thrombolytic therapy*
- **Oxygen**
  - In signs of *hypoxia*, *pulmonary edema* or *continuing myocardial ischemia*
  - If O2 saturation <94%
- **Nitrates**
  - Such as: ***GTN*** (sublingual/IV) to treat angina
- **Aspirin 300mg**
  - Should be given before arrival to the hospital
  - Clopidogrel should also be given

Management of Pulmonary edema

- MONA but replace the A with F (**frusemide**) → ***MONF***

Heparin *or* ***LMWH*** *(enoxaparin sodium) should also be considered*

Complications of MI

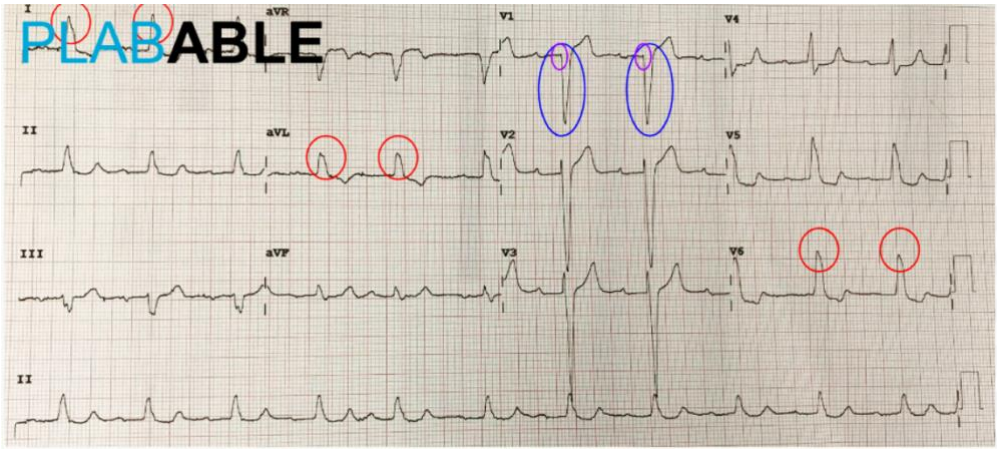
- **Ventricular aneurysm**
  - When the cardiac muscle partially dies during a myocardial infarction, a layer of muscle may survive, and being severely weakened, start to become an aneurysm (a balloon)
  - ***4-6 weeks*** post MI
  - ECG → ***Persistent ST elevation*** and ***left ventricular failure***
  - CXR → ***Cardiomegaly*** with ***an abnormal bulge at the left heart border***
  - ECHO → ***Paradoxical movement of ventricular wall***
  - **Thrombus** may form within the aneurysm increasing the risk of a stroke
- **Dressler's syndrome**
  - 2ry autoimmune form of pericarditis that occur post MI
  - ***1 week-several months*** post MI
  - Features: ***Fever***, ***pleuritic pain***, pericardial and pleural ***effusion***

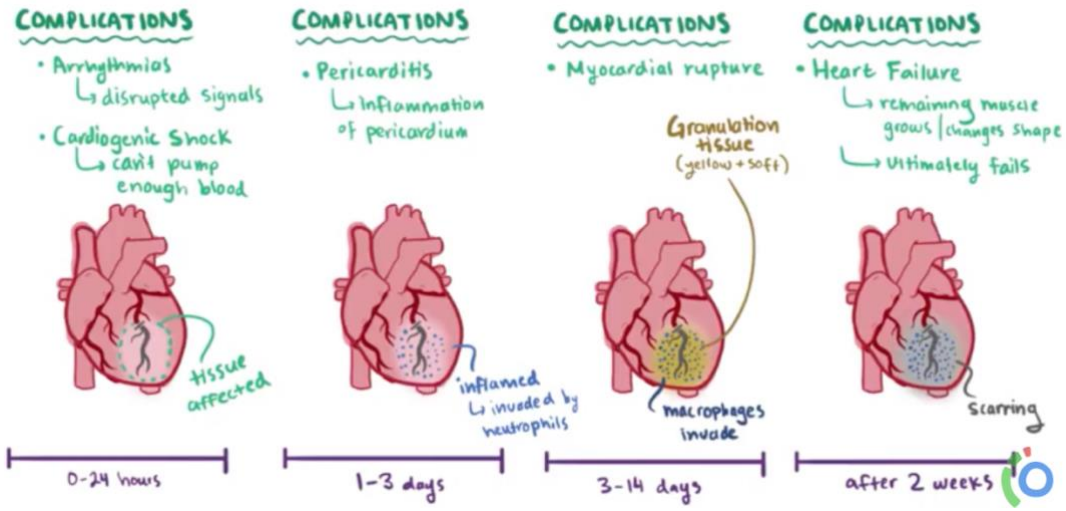
- **Left bundle branch block (LBBB)**
  - Activation of the **left** ventricle of the heart is delayed, which causes the **left** ventricle to contract later than the right ventricle

    [WiLLia**M]**
  - ORS in **V1** → looks like **W**
  - QRS in **V6** → looks like **M**

RBBB, QRS complex: [**M**aRRo**W**]
- **V1** → looks like **M**
- **V6** → looks like **W**

Wide QRS + **positive** V1 → **RBBB**
Wide QRS + **negative** V1 → **LBBB**





Management of CHF [ABSD]

- 1st line → ***ACE-inhibitor*** and ***beta-blocker*** (e.g. Carvedilol)
  - It's better to start one drug at a time
  - DM or signs of fluid overload → start with *ACE-inhibitor*
  - Ejection fraction ≤40% → start with *ACE-inhibitor*
  - Angina → start with *beta-blocker*
  - When starting a beta-blocker, start low and go slow
- 2nd line → ***Spironolactone*** (especially when K^+ <3.2 as it's a K-sparing diuretic)
- ***Digoxin*** → Heart failure + Atrial Fibrillation

Management of STEMI

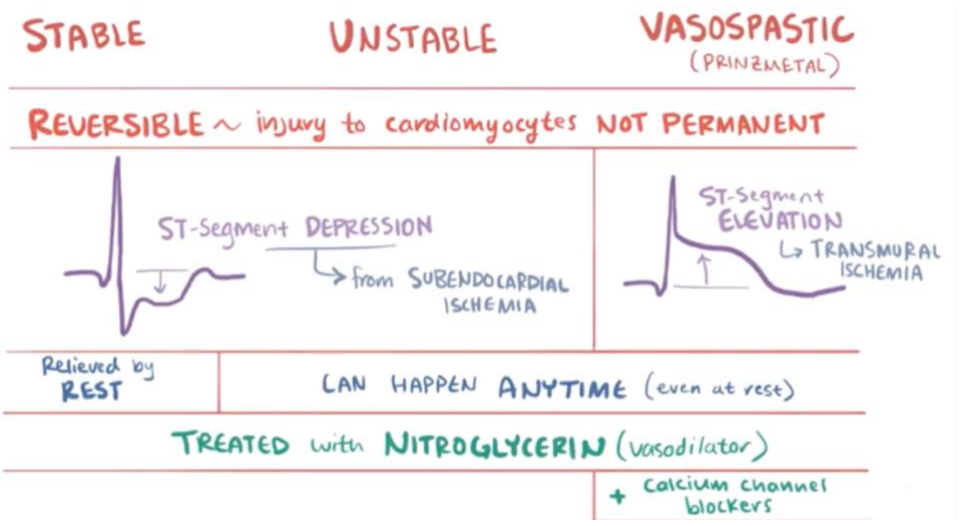
- If patient presents with symptom onset **within 12 hours** and PCI can be done **within 120mins** then withhold fibrinolysis → perform **PCI (percutaneous coronary intervention)**
- If PCI is not available and cannot be delivered **within 120mins** → **thrombolysis** (**Alteplase** or streptokinase)
- Thrombolysis can only be done within 12 hours of symptom onset

Post myocardial infarction management / management of NSTEMI or unstable angina

1. **Dual antiplatelet therapy: Aspirin + Clopidogrel (Plavix)**
   - Aspirin is continued life-long. Clopidogrel can be given for 1 year
2. **Beta blockers**
   - As soon as they're hemodynamically stable
   - In people without heart failure → *For 1 year after an MI*
   - In people with heart failure → *Indefinitely*
3. **ACE inhibitor**
   - As soon as they're hemodynamically stable
   - If intolerant → *ARB*
4. **Statins**
   - Atorvastatin 80 mg, used indefinitely

Once the patient is discharged, he can take a ***CAB*** or ***BAS***
C – Clopidogrel
A – Aspirin
B – Beta-blocker
A – ACE inhibitor
S – Statin

Angina



Vasospastic (Prinzmetal/variant) angina

- May or may not have atherosclerosis
- Ischemia is due to *coronary artery vasospasm*

DD of acute chest pain

Condition	Signs and Symptoms
Ischaemic cardiac pain	Retrosternal "pressure", "tightness", "constricting" Radiates to shoulders, arms, neck or jaw Associated with diaphoresis, sweating, nausea, pallor
Pericarditis	Atypical, retrosternal pain Sometimes pleuritic Positional (relieved on sitting forward)
Gastroesophageal reflux disease	Retrosternal, "burning" Associated with ingestion Pain when supine, after consumption of food, alcohol, NSAIDs Relieved by antacids
Aortic dissection	"Tearing" pain, sudden in onset Radiates to back Diaphoresis, hypotensive, tachycardic Difference in blood pressures and/or pulses Abnormal or absent peripheral pulses
Pulmonary embolism	Acute respiratory distress Diaphoresis, hypotensive, tachycardic, hypoxaemia Pleural rub
Pneumothorax	Pleuritic, sharp, positional, sudden in onset Associated with abrupt breathlessness Diminished breath sounds, hyperresonance to percussion
Pneumonia	Associated with cough, sputum, and fever
Musculoskeletal	Sharp, positional, pleuritic Aggravated by movement such as deep inspiration, coughing, twisting of neck or thoracic cage

- Widened mediastinum on CXR

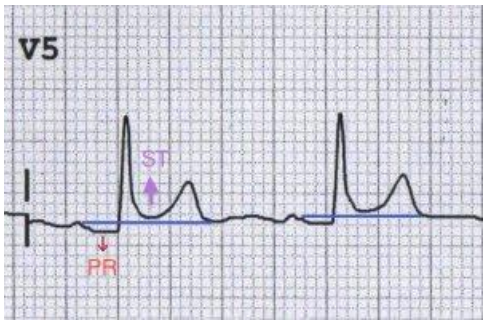
Acute pericarditis

Features

- Chest pain is described as **sharp**, **stabbing**, **central** chest pain
- Radiates to the **shoulders** and **upper arm**
- Chest pain may be **pleuritic** and is often **relieved by sitting forwards**
- **Worsened by** *inspiration*, *lying flat, cough*, *swallowing*, or *movement of the trunk*
- **Pericardial friction rub** is *pathognomonic* - often a rub can be heard even when a pericardial effusion is present)
- Other symptoms include non-productive cough and dyspnea

Causes

- Viral infections → **Coxsackie**
- Bacterial → **Tuberculosis**
- **Uremia** (causes 'fibrinous' pericarditis)
- **Trauma**
- **Post-myocardial infarction**, *Dressler's syndrome* → 2ry form of pericarditis on top of cardiac injury
- **Connective tissue disease**



ECG changes

- *Widespread 'saddle-shaped' ST elevation* (Saddle-shaped meaning concavity directed upwards)
- *PR segment depression*

	Acute Pericarditis	Pericardial Effusion	Cardiac Tamponade
Cause	See above	Any cause of pericarditis	Any cause of pericarditis but especially ***trauma***
Clinical Features	• Central chest pain worse on inspiration or lying flat ± relief by sitting forward. • Pericardial friction rub	• Dyspnea, raised JVP	• ***Beck's triad:*** 1. Muffled heart sounds 2. Distended neck veins 3. Hypotension
Test	• **ECG** → see above • **Troponin** may be raised	• **CXR** shows enlarged, globular heart • **ECG** shows low- voltage QRS complexes and alternating	• **Echo** is diagnostic
Treatment	**NSAIDS**	**Pericardiocentesis**	**Urgent Pericardiocentesis**

Cardiac tamponade → a life-threatening condition in which a pericardial effusion has developed so rapidly or has become so large that it compresses the heart

Infective endocarditis

- Fever + new murmur = ***Endocarditis*** until proven otherwise
- Causative organisms: **Staph aureus,** *in subacute IE → Strept viridians*

Risk factors

- Valve replacement
- Recreational drug abuse and invasive vascular procedures
- Dentistry interventions

Presentation

- Along with **fever**, **rigors**, **malaise** and **the new murmur (commonly MR)**, some patients may present with ***congestive heart failure***

Diagnosis [BE FIVE PM]

- 2 major criteria present
- 1 major + 2 minor
- 5 minor criteria

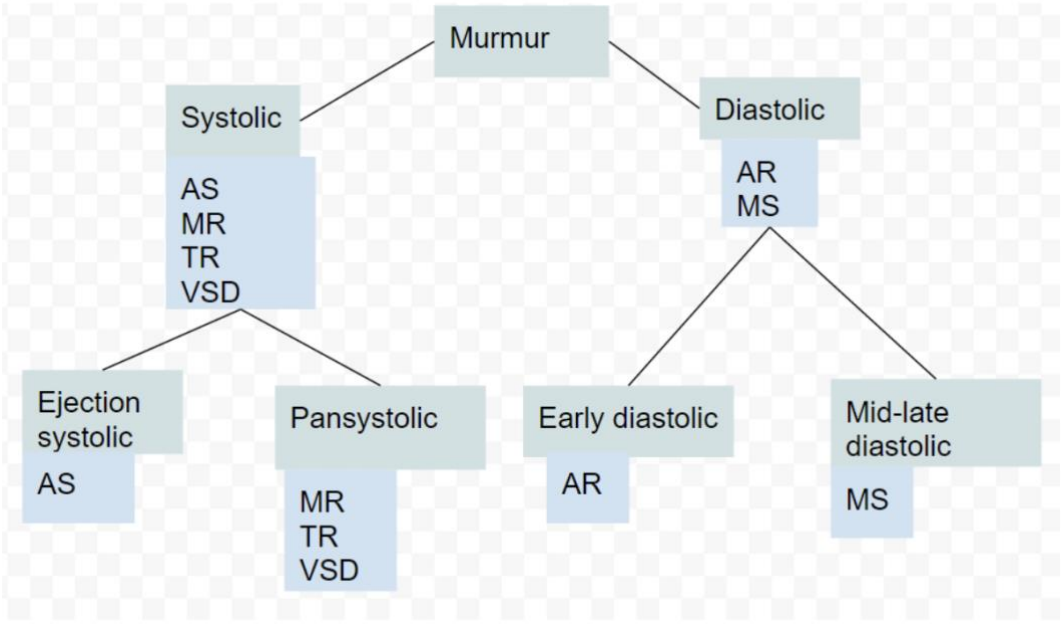
Management

- Antibiotics → *Flucloxacillin + Benzylpenicillin + Gentamicin* (Vancomycin with prosthetic valve)
- Surgery → Valve replacement

Major criteria [BE]	Minor criteria [FIVE PM]
• **+ve blood culture** • **+ve Echo** → *abscess formation, new valvular regurgitation*	• **Fever** >38℃ • **Immunological** phenomena: AGN, Osler's nodes, Roth spots • **Vascular** phenomena: major emboli, splinter hge, Janeway lesions • **Echocardiographic** evidence doesn't meet major criteria • **P**redisposition: **IV drug user** or heart condition • **Microbiological** evidence does not meet major criteria

Splinter hemorrhage → *hemorrhage under the nails*
Janeway's lesion → *painless macules*
Osler's nodes → *small painful tender*
Roth's spots → *red retinal spots with pale white center*

Differentiating murmurs



- **Systolic**
  - Lesions **above** the level of the nipple → Ejection systolic
  - Lesions **below** the level of the nipple → Pansystolic

- LEft sided murmurs louder with → ***Expiration***
- RIght sided murmurs louder with → ***Inspiration***

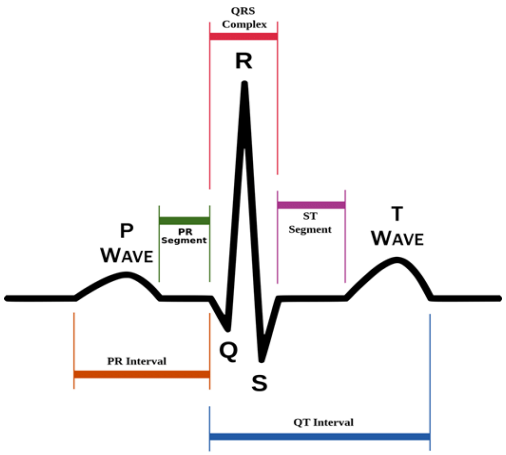
Defect	Type of Murmur	Where Is It Heard?	Symptoms
Aortic Stenosis	Ejection Systolic	2nd intercostal space to the right of the sternum	Dyspnoea with activity, anginal chest pain and, syncope
Aortic Regurgitation	Early Diastolic		Symptoms of heart failure
Mitral Stenosis	Mid-late Diastolic	Apex	Symptoms of heart failure
Mitral Regurgitation	Pan-Systolic		Symptoms of congestive heart failure
Pulmonary Stenosis	Ejection Systolic	2nd intercostal space to the left of the sternum	Systemic cyanosis
Pulmonary Regurgitation	Early Diastolic		Symptoms of right-sided heart failure
Tricuspid Stenosis	Diastolic Rumble	Lower right sternal edge	Fluttering discomfort in the neck
Tricuspid Regurgitation	Pan-Systolic		Symptoms of right-sided heart failure

ECG arrythmias

Atrial Fibrillation • Palpitations • Fast heart rate • Dyspnoea
Atrial Flutter • Patient in stem will describe it as a "fluttering feeling in the chest"
Ventricular Fibrillation • Older adult with a sudden collapse • Not breathing
Ventricular Tachycardia • Regular and fast • Stem will mention a period of ongoing lightheadedness, palpitations and chest pain (rarely will they present with collapsed in exams)
Sinus Bradycardia • Lightheadedness, dizziness, hypotension, vertigo, and syncope • NB: Slow heart rate is completely normal in young athletes
Sinus Tachycardia • Physiological situations, such as exercise or situations of stress or anger • History of infection
Heart Block • Stem will mention an older patient who has suffered a previous MI or who has ACS *Type 1 AV Block: lengthening of the PR interval* *Type 2 AV Block* • *Mobitz I: progressive prolongation of PR interval until a missed QRS complex* • *Mobitz II: normal PR interval with occasional missed QRS complexes* *Type 3 AV Block (Complete Heart Block): no relation of P waves and QRS complexes*
Wolff-Parkinson White Syndrome • "Delta wave" in ECG

Ventricular tachycardia

- More than 3 consecutive PVCs, HR **100-250** bpm → less filling → low cardiac output → hypotension and HF
- There's lack of coordinated atrial contractions (**loss of atrial click**) → **P wave** may be present or absent. If present, it has **no relation** to the **QRS complex**

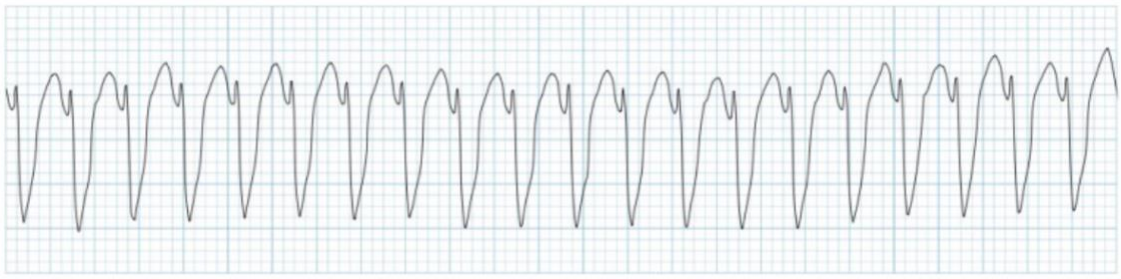


- **Sinus tachycardia:** HR >100 bpm in a healthy individual due to exercise/stress
- **Premature ventricular contraction (PVC):** a single beat starts from the ventricles

- It's impossible to see **ventricular fibrillation** in an awake person

- Wide QRS + regular → **VT**
- Wide QRS + irregular → **V-fib**
- Narrow QRS + regular → **SVT**
- Narrow QRS + irregular → **A-Fib**

With pulse	Without pulse
1. Hemodynamically stable ➢ Anti-arrhythmics [ALP] ○ Amiodarone, lidocaine, procainamide **2. Hemodynamically unstable** • Hypotension, chest pain, cardiac failure and decreased conscious level ➢ Immediate electrical cardioversion	➢ Immediate electrical cardioversion (*defibrillation*) is indicated



*P and QRS dissociation + tachycardia → **VT***
*P and QRS dissociation + bradycardia → **3rd degree heart block***

*A patient with **VT** → Ask yourself, does he have a pulse or not?*
- *No pulse → **immediate electrical cardioversion***
- *If he has pulse → Is he stable or not? Stable? → **ALP** Not stable? → **immediate electrical cardioversion***

*A patient with **SVT** → Ask yourself, Is he stable or not?*

Supraventricular tachycardia

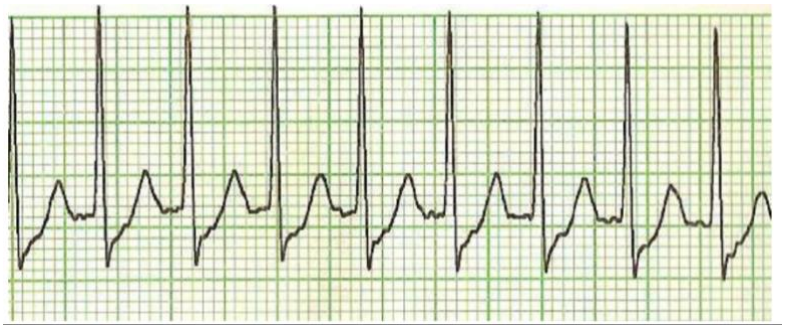
- *Paroxysmal* supraventricular tachycardia is manifested as an absolutely **regular** rhythm at a rate between **130 -220** beats/min
- In a **YOUNG** patient with **recurrent palpitations**, **normal/slightly low BP**, **regular** heart rate and NO PREDISPOSITION TO HEART DISEASE → consider *SVT*

Acute management

If dynamically stable	If unstable
1. ***Valsalva maneuver, carotid massage*** 2. ***Adenosine 6mg IV*** (if the patient is asthmatic → *verapamil* instead) - If unsuccessful → add another 12 mg IV - If still unsuccessful → give another 12 3. ***Electrical Cardioversion*** - If you've given <u>3 doses (30mg)</u> of adenosine with no improvement	• Chest pain, SOB, hypotension, fatigue, cold extremities, decreased urine output, confusion or lost consciousness ➢ **DC cardioversion**

Prevention

- *Beta-blockers*
- *Radio-frequency ablation*



Ventricular ectopic

- Caused by the premature discharge of a ventricular ectopic focus which produces an **early** and **broad** QRS complex
- Maybe asymptomatic with palpitations, often described as ***skipped or missed beats***
- *Benign* unless there's an underlying heart disease → could lead to life-threating arrythmias

Causes

- IHD
- Cardiomyopathy
- Stress, alcohol, caffeine, medication, cocaine or amphetamines
- Occurs naturally

- Young patient with recurrent palpitations in the absence of heart disease → **SVT**
- Loss of consciousness → **VF**
- Middle-aged person with history of CHD → **VT**
- Missed beats, dyspnea, dizziness and never sustained in 2H → **ectopic beats**

Atrial fibrillation

- Atrial fibrillation is the most common arrythmia that develops in patients with **dilated cardiomyopathy**
- **Dilated cardiomyopathy** → Decreased ejection fraction + damaged myocardium (e.g. thinning of septal wall)
- History of **ALCOHOLISM** followed by **palpitations**, **dizziness** and **syncope** → arrythmias (atrial fibrillation/flutter). This is known as "holiday heart syndrome"
- Atrial fibrillation leads to **pulmonary edema** due to the loss of atrial contribution to cardiac output

Features

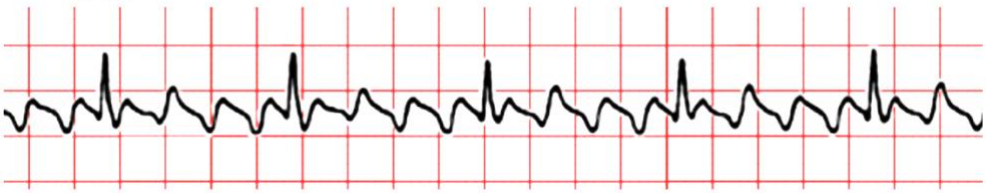
- Dyspnea
- Palpitations
- Syncope or dizziness
- Chest discomfort or pain
- Stroke or TIA
- An irregularly irregular pulse
- Absent P wave

Atrial flutter
- Abnormal focus in the atrium
- Transient stage between normal rhythm and AF
- Absent P wave

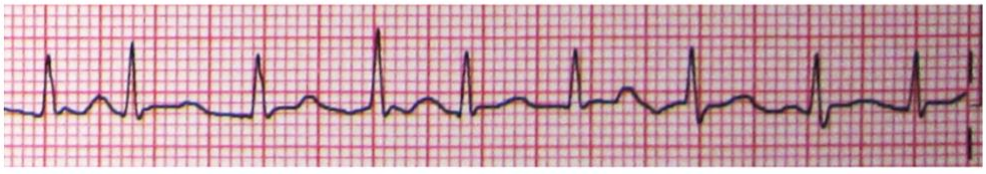
Atrial flutter Vs Atrial fibrillation

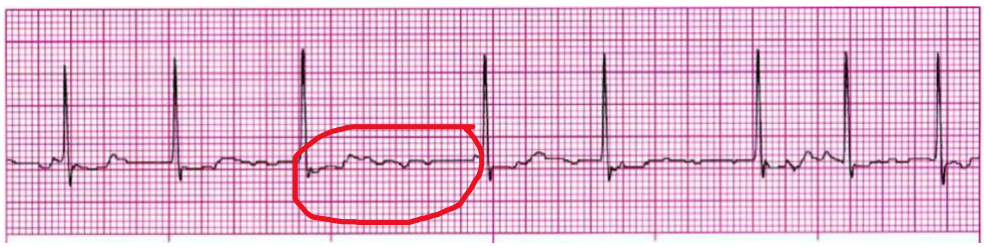
	Atrial flutter	Atrial fibrillation
Atrial activity	Rate: 220-350 bpm Visible flutter (F) waves	Rate: >350 bpm Fine fibrillatory (f) waves
Ventricular activity	Rate: Regular Constant R-R interval	Rate: Variable No relation to atrial rate Variable R-R interval
Baseline	Sawtooth	Ragged

Atrial flutter



Atrial fibrillation





Atrial fibrillation management

Rate control	Rhythm control
• Patient is <u>hemodynamically stable</u> • 1st choice → ***beta-blockers*** or ***CCB*** (diltiazem or verapamil) • ***Digoxin*** is used if there's **CHF** • ***Thromboprophylaxis*** is also used	• Patient is <u>unstable</u> **A-** ***Electrical cardioversion under sedation*** **B-** Chemical cardioversion with ***amiodarone*** or ***flecainide*** can be used [AF] • If <u>symptoms >48h</u> → risk of **cardiac thromboembolism** when cardioverted → ***rate control meds*** + ***LMWH***

Persistent AF, lasts >7 days

Rate control	Rhythm control
• **>65** • Patient is **stable** and AF started <u>>48h ago</u> • Unsuitable for cardioversion - Long duration of AF (usually >12 months) - History of multiple failed cardioversion	• Who are **symptomatic** • Who are **younger** • Presenting for the **first time** with **lone AF**

A patient with AF, you ask yourself

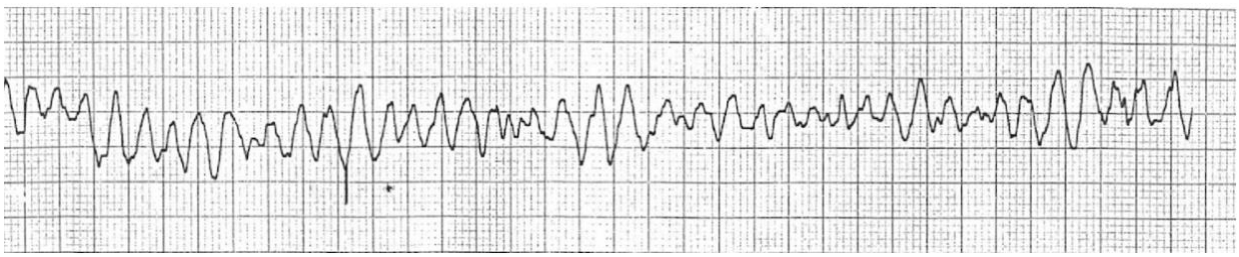
- *Is he stable or not?*
- *Is he old or young?*

Ventricular fibrillation

- VF means **sudden death**; blood pressure drops immediately to zero and so does the cardiac output
- Rate is up to 500 bpm → ventricles are unable to contract in a synchronized manner → immediate loss of cardiac output

ECG findings

- **Chaotic irregular deflections** of varying amplitude
- No identifiable P waves
- Rate: 150-500 per minute
- There's no specific pattern to the discharge



CHA_2DS_2VS (Secondary prevention after a stroke)

- Used to determine the most appropriate anticoagulation strategy for atrial fibrillation
- A score of 2 or more / men with a score of 1 → *offer anticoagulation therapy*

	Risk factor	Points
C	Congestive heart failure	1
H	Hypertension (or treated hypertension)	1
A^2	Age >= 75 years	2
	Age 65-74 years	1
D	Diabetes	1
S^2	Prior Stroke or TIA	2
V	Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
S	Sex (female)	1

- <65 years old and no comorbidities → NO warfarin
- **≥65** years old + at least **one comorbidity** → ***Warfarin*** or ***DOAC (direct oral anticoagulants)*** such as ***apixaban, edoxaban*** and ***rivaroxaban***

Benefits of DOAC over warfarin

- Reduction of the risk factors of intracranial hemorrhage
- No INR monitoring
- Faster onset anticoagulation (2-4h)

- **Warfarin** may lead to intracranial hemorrhage and SAH → look out for **headache**

Disadvantages of DOAC

- No specific antidote
- Essential to be compliant

Stroke → syndrome of sudden onset focal neurological loss of presumed vascular origin lasting **>24h**
TIA → syndrome of sudden onset of focal neurological loss of presumed vascular origin lasting **<24h**

2ry prevention of a stroke or TIA

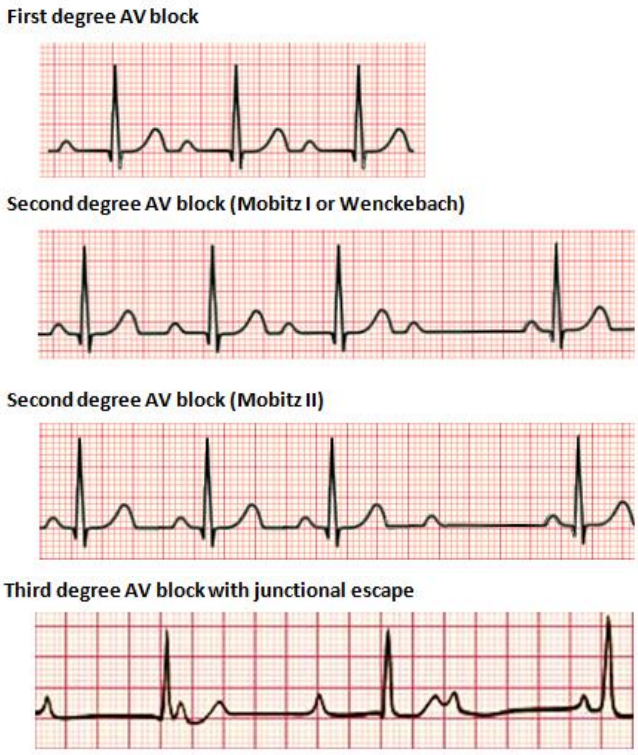
- Patient presented with **AF** → ***Warfarin*** or ***DOAC*** (stroke prophylaxis)
- Patient presented with **disabling stroke** and NO AF → defer anticoagulation treatment for **14 days** from the onset and start ***Aspirin* 300mg for 2 weeks** + ***Clopidogrel* 75mg lifelong**
- Consider ***carotid endarterectomy*** if internal carotid artery is stenosed **≥ 50% in men, ≥ 70% in women**

Heart block

1st degree heart block	• Prolonged PR interval **> 0.2 seconds**
2nd degree heart block	• **Mobitz type I AV block (Wenckebach block/phenomenon)** ➢ Progressive prolongation of the PR interval with intermittent dropped beat • **Mobitz type II AV block** ➢ Sudden drop of QRS WITHOUT prior PR changes
3rd degree (complete) heart block	• P waves and QRS complexes have NO RELATION to each other

Management

- 1st degree and Mobitz I → no treatment, atropine could be given (Mobitz I)
- Mobitz II and 3rd degree → **permanent pacemakers,** atropine is contraindicated



P-R interval

- Represents the time it takes the impulse to move between atria and ventricles
- Normally 3-4 boxes

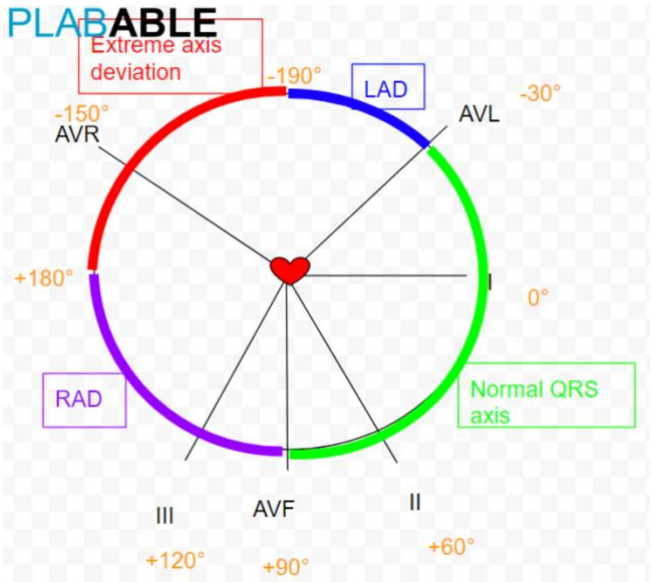
ECG interpretation

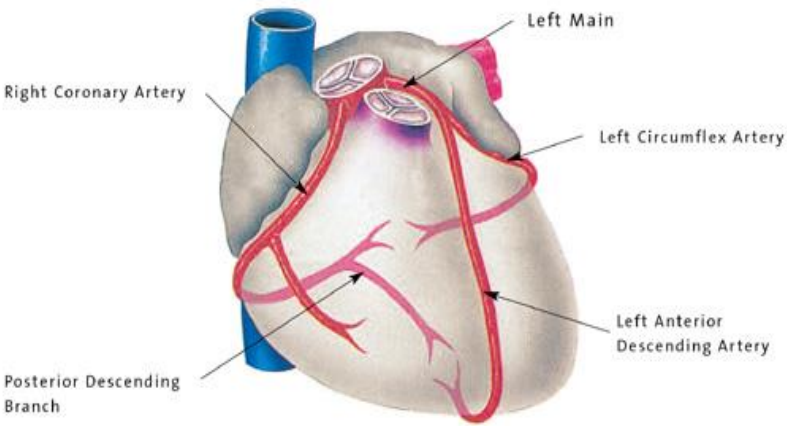
1. Rate
2. Regularity
3. P wave
4. PR interval
5. QRS duration
6. Determine the axis
7. Look at morphology

Rules of thumb to determine Cardiac Axis deviation on ECG

- Lead I → Left thumb while lead II → Right thumb
- Look at lead I and lead II
- A positive defalcation means 👍 and a negative deflection means 👎
- If lead I and II are both positive → 👍👍 → Normal
- If lead I is positive and lead II is negative → LAD
- If lead I is negative and lead II is positive → RAD

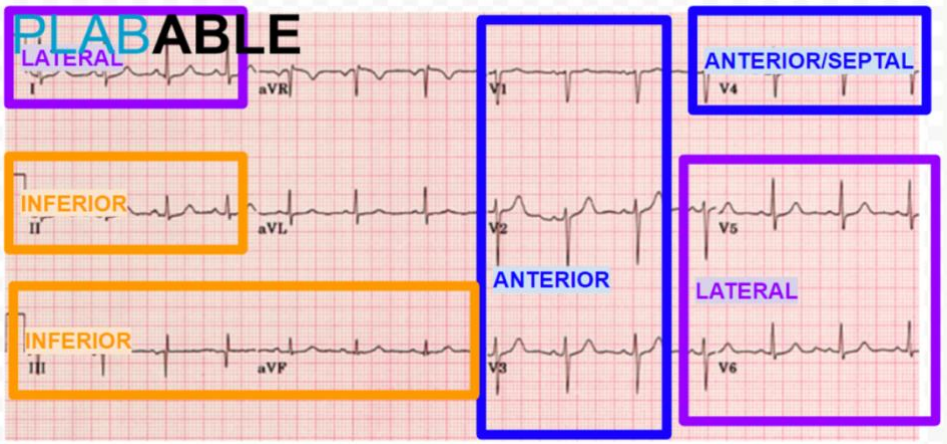
LAD	RAD	Extreme RAD
Common • Left ventricular hypertrophy • **Left anterior** fascicular block (hemiblock) • **Inferior myocardial infarction** **Less common** • Obesity • Wolff Parkinson white $	• Right ventricular hypertrophy • Thin, tall • Chronic lung disease • Pulmonary embolism • **Left posterior** hemiblock • **Lateral MI** - Lateral wall of the left ventricle is supplied by the left anterior descending - Infarction will cause deviation away from site of infarction	• Congenital heart disease • Left ventricular aneurysm

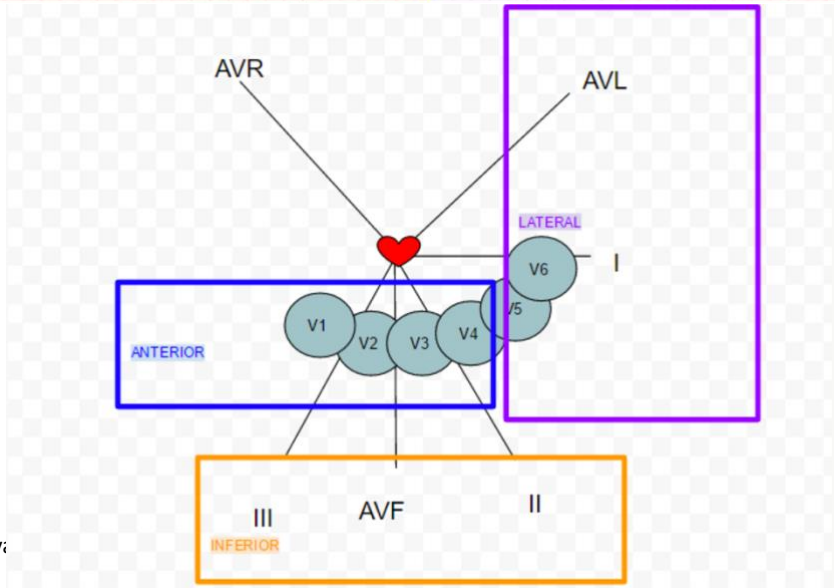




ECG changes in myocardial infarction and coronary territories

	Area of infarct	ECG changes	Coronary artery
Most commonly asked	Anteroseptal	V1-V4	Left anterior descending (LAD)
	Inferior	II, III, aVF	Right coronary (RCA)
	Lateral	I, aVL +/- V5-6	Left circumflex
Less commonly asked	Anterolateral	I, aVL, V4-6	Left anterior descending (LAD) or left circumflex
	Posterior	Tall R waves V1-2 Also note the reciprocal ST-segment depression in the anterior chest leads	Usually left circumflex, also right coronary





Valvular heart diseases

Aortic stenosis

- **Elderly** patient with **exercise intolerance** or maybe **asymptomatic**
- Murmur is accentuated *when sits upright*

- The ***most common*** valvular heart disease in the UK

Causes

1. **Degenerative sclerocalcific changes to valves** → ***Most common***, usually an **elderly** patient
2. **Congenital bicuspid aortic valve** → **younger** patients

Signs

- **Ejection systolic murmur** best heard at the *2nd right intercostal space*, at the right sternal border which *radiates to the **carotid arteries**, louder with expiration (left-sided murmur)*

Mitral regurgitation

- The *2nd most common* heart valve disorder
- Due to **ischemic papillary muscle dysfunction** or **partial rupture after myocardial infarction** (days)
- Could be 2ry to *rheumatic fever*
- Commonly associated with ***inferior MI*** than anterior, usually seen **2-10 days** post MI and the patient is presented with **pulmonary edema**

Signs

- ***Left ventricular failure:*** dyspnea, orthopnea and paroxysmal nocturnal dyspnea
- With severe MR → right sided heart failure → edema and ascites
- **Soft S1** and **S2 pansystolic murmur** at the *5th intercostal space, left midclavicular line* (Apex) which *radiates to the **axilla***

Investigations

- **ECG** → *broad/bifid P wave (P mitrale)* indicating enlarged left atrium
- **Echo** → *diagnostic*

Mitral valve prolapse [MVP]

- Inheritable connective tissue disorder, may be associated with ***Marfan's $***, ***Ehlers Danlos $*** and ***osteogenesis imperfecta***
- Most patients are **asymptomatic**
- Classic case → ***Slim young female*** with ***low blood pressure***

Signs

- **Mid-systolic click**, best heard at the *5th intercostal space, left midclavicular line*, followed by a **mid or late-systolic murmur** with finding accentuated in the ***standing*** position

[**MVP**] in a basketball team

- Tall, thin with long arms → *Marfan's*
- Has loose joints → *Ehlers Danlos*
- Shoots from the **Mid**dle of the court with great **sys**pense and finishes with the ball entering the net with a **click** → *mid-systolic click*

Mitral Stenosis [MS]

- Most commonly caused by → ***Rheumatic fever***
- Mitral valve stenosis impedes left ventricular filling → increased blood volume in the left atrium → increased left atrial pressure → blood is back to the lungs causing pulmonary congestion → 2ry pulmonary vasoconstriction → pulmonary hypertension → becomes harder for the right ventricle to pump → **right ventricular failure**

Signs

- Pulmonary congestion and edema → Dyspnea, orthopnea, paroxysmal nocturnal dyspnea
- Hepatomegaly, ascites, peripheral edema
- There may be hemoptysis (due to rupture of pulmonary vessels due to raised atrial pressure)
- Could lead to systemic embolism (due to stagnation of blood in an enlarged left atrium as a result from AF)
- Physical signs: Atrial fibrillation, Malar flush, pulmonary rales
- **Loud S1**
- **Mid-diastolic murmur** with an **opening click** (or a diastolic rumble, *click represents the mitral valve opening*), best heard at the **apex**

[**M**ichael **S**chumacher] he had an accident in the **mid**dle of his ski trip which he almost **dia**d → **Mid-diastolic murmur**

ECG

- May show signs of **right ventricular failure**
- **Atrial fibrillation**
- **P mitrale** → bifid P wave

Chest x-ray

- **Large left atrium** → *straightening of the left heart border*
- **Pulmonary hypertension**, including Kerley B lines and increased vascular markings

Echocardiography

- Thickening of mitral valve leaflets

Aortic regurgitation

- Causes: RF, infective endocarditis, Marfan's syndrome

Signs

- **Early diastolic murmur**, best heard at the **left sternal edge (Erb's point)**

Pulmonary stenosis

Signs

- **Ejection systolic murmur**, best heard over the **pulmonary area**, *radiates to the **left shoulder** or **infraclavicular** region*

Ventricular septal defect (VSD)

- Acyanotic congenital heart disease
- Left to right shunt

Causes

- Congenital
- Acquired (post-MI)

Tricuspid atresia

- Pansystolic murmur
- Presents at early week of life with poor feeding
- **Cyanotic**

Symptoms

- May present with severe heart failure in infancy, **poor weight gain** and **frequent URTIs**
- Could remain **asymptomatic** and be detected incidentally in later life

Signs

These depend on size and site:

A. Small holes

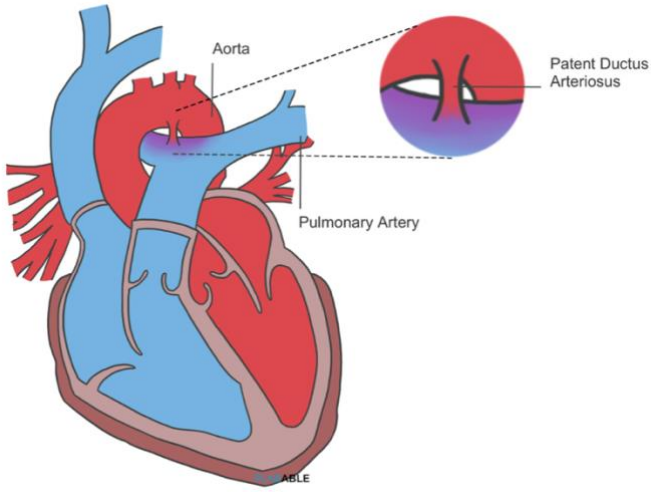
- **Asymptomatic** with normal feeding and weight gain
- May be detected accidently on routine examination
- Give *louder murmurs*
- Classically, ***a harsh pan-systolic murmur*** heard at the left sternal edge, with **a systolic thrill**, and **a left parasternal heave**

B. Large holes

- Associated with signs of **pulmonary hypertension**
- These babies may develop *a right to left shunt* → **cyanosis** or **Eisenmenger's syndrome** (cyanosis + pulmonary HTN + erythrocytosis)

Patent ductus arteriosus (PDA)

- Persistence of a normal fetal connection between the **aorta** and the **pulmonary artery**
- Very common in **preterm babies** and it also **may close spontaneously**
- May be **asymptomatic** or may cause **apnea**, **bradycardia** and **increased oxygen requirements**
- The **continuous machinery murmur** (rarely heard) → *heard over **the left infraclavicular area***
- There may be **a rough systolic murmur** along the left sternal border
- **Bounding peripheral pulses**
- The diagnosis is confirmed by **ECHO** which not only allows the PDA to be visualized but also assesses the hemodynamic significance of the PDA
- **Prostaglandins** → prevent closure of PDA
- Drugs that close the duct → ***Indomethacin*** or ***Ibuprofen***



Most common cyanotic heart conditions (5Ts)

1. Tetralogy of Fallot (TOF)
2. Transposition of the great arteries (TGA)
3. Tricuspid atresia → *pansystolic murmur + cyanosis is usually presented immediately after birth*
4. Truncus arteriosus
5. Total anomalous pulmonary venous connection (TAPVC)

Tetralogy of Fallot (TOF)

- The most common cause of **cyanotic** congenital heart disease
- Typically presents at around **1-2 months**, although may not be picked up until the baby is **6 months** old

Four characteristic features

- VSD
- Right ventricular hypertrophy
- Pulmonary stenosis → *ejection systolic murmur*
- Overriding aorta

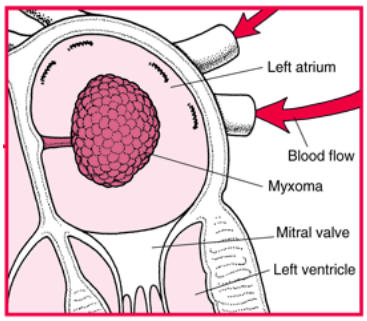
Other features

- **Cyanosis**
- Causes a **right-to-left** shunt
- **Ejection systolic murmur** due to *pulmonary stenosis* (the VSD does not usually cause a murmur)
- **A right-sided aortic arch** is seen in 25% of patients
- **Chest x-ray** → *'boot-shaped' heart*
- **ECG** → right ventricular hypertrophy

Complications

- **Pulmonary regurgitation** (common after repair of pulmonary stenosis) → signs of right HF → *pulmonary valve replacement*

Atrial myxoma



- Benign tumors, mostly in the **left atrium** and tend to grow on the **wall (septum)**
- Sometimes tumor **break off** and fall into the bloodstream → *brain*, could cause a **stroke**, *lungs* causing a **pulmonary embolus**
- Around 10% → **inherited** (familial myxomas)
- The symptoms occur due to **obstruction of the mitral valve** which result in **syncope** and **heart failure**

Features

- **Acts as Aortic stenosis** on auscultation + prominent **A-waves**
- **Platypnea** → Dyspnea that's relieved when lying down and worsens when sitting or standing
- Symptoms of **hemodynamic obstruction**, embolization, or constitutional symptoms such as fever, malaise, tachycardia and tachypnoea
- Symptoms and signs of **ischemia** or **infarction in the peripheries**, due to embolization of adherent thrombus
- **Atrial fibrillation**
- Large myxomas may **impair intracardiac blood** flow, causing dyspnea, syncope or symptoms and signs of congestive cardiac failure
- **Echo**: **pedunculated heterogenous mass** typically attached to the **fossa ovalis**

Hypertension

Step 1

- Age <55 or DM → ***ACE inhibitor (…pril)*** or ***ARBs (…sartan)***
- Age >55 or Afro-Caribbean → ***CCB*** *(Amlodipine, Diltiazem)*
  - If CCB isn't suitable (e.g. intolerance or edema) or if there's evidence of HF or a high risk of HF → **thiazide-like diuretic**

Step 2

- ACE-inhibitor + CCB

Step 2

- ACE-inhibitor + CCB + thiazide-like diuretic

- if a patient >55 y/o is currently on Thiazides (Indapamide) → assume he is intolerant to CCB and give ACE-inhibitors

Postural hypotension

- A drop in BP of more than 20 mmHg within 3 minutes of standing
- Should always be considered in old patient especially if on multiple medications and presents with dizziness
- May present with ***dizziness*** or ***sudden loss of consciousness after getting up*** from a chair with recovery within minutes
- One of the most common causes of postural hypotension is **dehydration** → ***U&E*** should be requested

Diagnosis

- Blood pressure taken when lying down and standing up

Ruptured abdominal aortic aneurysm

- **Severe sudden lower abdominal** and **back pain**
- **Tender pulsatile abdominal mass** (lateral and superior to the umbilicus)
- **Low BP**

Investigation

- Stable → ***CT***
- Unstable → ***US***

Management

- Immediate **IV fluids** and **Laparotomy**

Digoxin toxicity

- ***Yellow halos*** are a clincher for digoxin toxicity
- Hypokalemia can lead to toxicity as K^+ and digoxin bind to the same receptors → more digoxin binding → increased intracellular calcium → increased cardiac contractility

Features [DIG]

- ***GIT symptoms*** are the *most important* → nausea, vomiting, diarrhea and anorexia
- ***Neurological*** and ***Eye [I]*** symptoms → blurred vision, ***yellow*** green vision (xanthopsia), hallucinations and confusion
- ***Dysrhythmias*** → bradycardia, premature contractions, ventricular tachycardia and any other type of arrythmia

Management

- **Digibind** (digoxin specific antibody fragments)
- Correct arrythmias
- Monitor potassium

- A serum digoxin can be ordered in patient suspected of being intoxicated (history, etc.)

Notes

- **Symptomatic bradycardia**
  1. ***Atropine 0.5mg IV***
  2. Dopamine
  3. Epinephrine
  - Initial → ABCD
- **Resuscitation guide for cardiac arrest**
  a. Call for help
  b. Check for ABCD *(if there's no signs of life → call resuscitation team)*
  c. CPR 30:2
  d. Defibrillation once we get help
- Drugs to be avoided in CHF, IHD, CKD
  - ***NSAIDs*** → inhibit prostaglandins → afferent arteriole constriction → less filtration → lower urine output → fluid accumulation → heart failure worsens
  - ***COX-2 inhibitors***
- ***Thiazide*** diuretics increase the risk of gout → due to reduced clearance if uric acid
- Treatment of beta blocker overdose → ***Glucagon***
- Antidote for warfarin → ***Vitamin K***
- U wave → ***Hypokalemia***
- J wave → ***Hypothermia***
- A-waves → ***Atrial myxoma***
- Delta waves → ***Wolff Parkinson White Syndrome (WPW $)***
- Bifid/wide P wave → ***Left atrial enlargement (MR, MS)***
- P mitrale (BROADER than 2 and a half small boxes) → ***Left atrial enlargement***
- P pulmonale (taller than 2 and a half small boxes) → ***Right atrial enlargement***
- ECG showing irregular/equivocal (ambiguous) rhythm, next investigation → ***ECHO***
- ECG showing regular rhythm, next investigation → ***Holter ECG (24h ECG)***
- Patients who have experienced episodes of syncope during or shortly after exertion → ***Exercise testing***
- Investigations for Aortic dissection → ***Transesophageal Echo, CT, MRI***
- **Sinus sick syndrome** → Alternating episodes of tachycardia, bradycardia, AF or flutter
- **Polymorphic ventricular tachycardia = Torsades de pointes = Long Q-T syndrome = Spindle shaped ECG**
  - Prolonged Q-T interval
  - Syncope and sudden death
  - Exacerbated by exercise, stress, medications and electrolyte imbalance
  - Treated with MgSO4
- **Complications of MI**
  - Rupture and acute pericarditis – days → *ECHO to diagnose*
  - Dressler's – weeks
  - Aneurysm – month (4-6 weeks)
- 85% are right-dominant → ***Right coronary artery,*** gives off the posterior descending artery (PDA), supplying the inferior wall, ventricular septum and the posteromedial papillary muscle
  - 15% left-dominant → left circumflex gives off the PDA
  - Co-dominant if both give off the PDA
- **Stokes Adam's attack** → sudden collapse into consciousness due to heart block
- Flushing after a syncopal attack is due to pumping of blood into the already dilated peripheral vessels due to hypoxia

- Investigations following syncope
  - Find **witnesses** → ask how the patient became unconscious, was there a seizure?
  - **ECG** → to look for arrhythmias
  - **Blood pressure** → supine and standing to look for postural hypotension
  - **Blood glucose** → to exclude hypoglycemia (LOC + sweating + improves with glucose administration)
- Before commencing ACE-inhibitors → ***Check eGFR****, if <30 → Avoid ACE-inhibitors and ARBs*
- Decreased ejection fraction + damaged myocardium (e.g. thinning of septal wall) → ***Dilated cardiomyopathy***
- **Familial hypercholesterolemia**
  - Autosomal dominant
  - Total cholesterol >7.5, LDL >5
  - **Family history of MI** in a 1st degree relative <60 years (or a 2nd degree relative <50 years)
- *Polygenic hypercholesterolemia* is milder with total cholesterol >6.5, LDL >4
- Elevated LDL and triglyceride + decreased HDL → *Mixed dyslipidemia*
- Baseline assessment of **Amiodarone**
  - *TFTs, LFTs and U&E → every 6 months*
  - *CXR and ECG → every 12 months*
- Before using Amiodarone, initial assessment→ ***TFTs***
- Before using lithium, initial assessment → ***Kidney function tests*****,** then ***TFTs***
- Anginal pain → last **less than 30mins** and is precepted by **physical exertion/stress**
- **Decubitus angina** → Anginal pain that occurs on lying down
- **Patent foramen ovale** is diagnosed by → ***Transesophageal ECHO (bubble ECHO)***
- **InnoSent/functional/physiologic murmur:**
  - A**S**ymptomatic
  - **S**oft lowing murmur, 1-2/6 intensity, acute illnesses (e.g. fever) can increase the intensity
  - **S**ystolic murmur
  - **S**hort
  - Left **S**ternal edge
- **SVC obstruction**
  - Commonly caused by compressing lung cancer or lymphoma
  - C/P: **shortness of breath** (most common symptom), facial and upper body **edema**, **facial plethora**, **venous distension** of the face and upper body, dysphagia, syncope and headache
  - Initial investigation → **X-ray**: to diagnose lung cancer
  - Appropriate investigation → **CT with contrast**: to assess collateral vessels and the extent of obstruction
  - Managed by → **Steroids (Dexamethasone),** *avoided in night as it disturbs sleep*
  - Histology should be obtained before starting steroids as it may alter the result