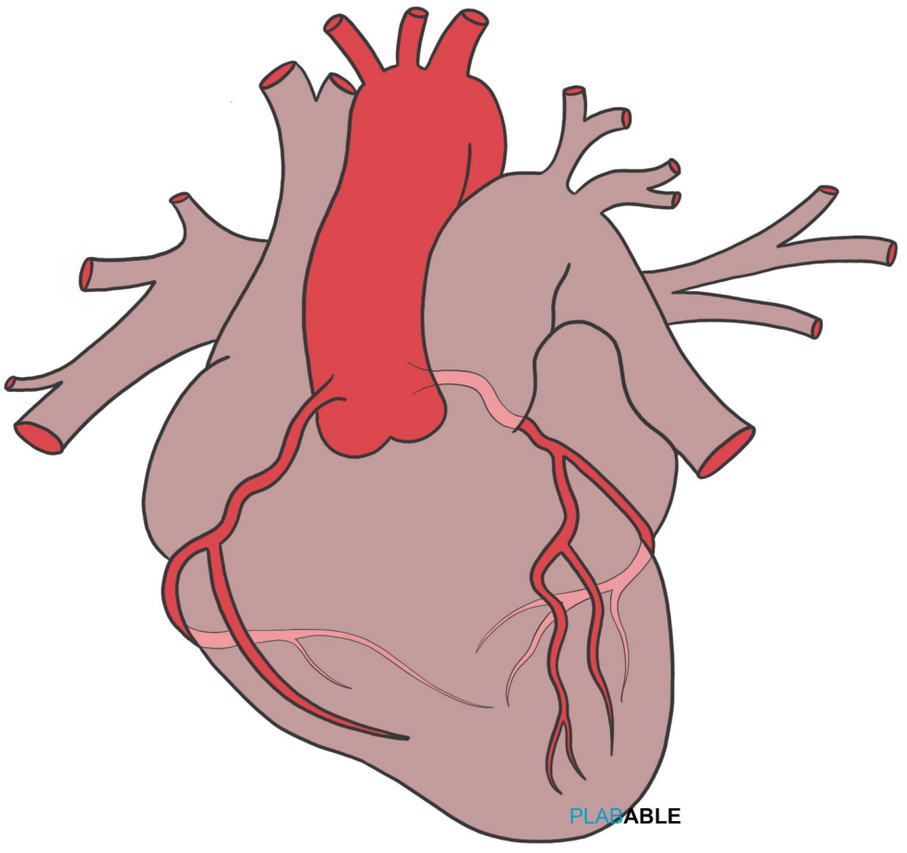
PLABABLE

GEMS

VERSION 6.0

CARDIOLOGY



Cardiac Tamponade

Fluid accumulation in the pericardial space causing obstruction to the inflow of blood into the ventricles

Causes

- Acute pericarditis
- Aortic dissection
- Trauma

Beck's triad

- Hypotension
- Muffled heart sounds
- Raised JVP

Paradoxical pulse

- **>10 mmHg inspiratory drop** of systolic BP

Cardiac Tamponade

Diagnosis:

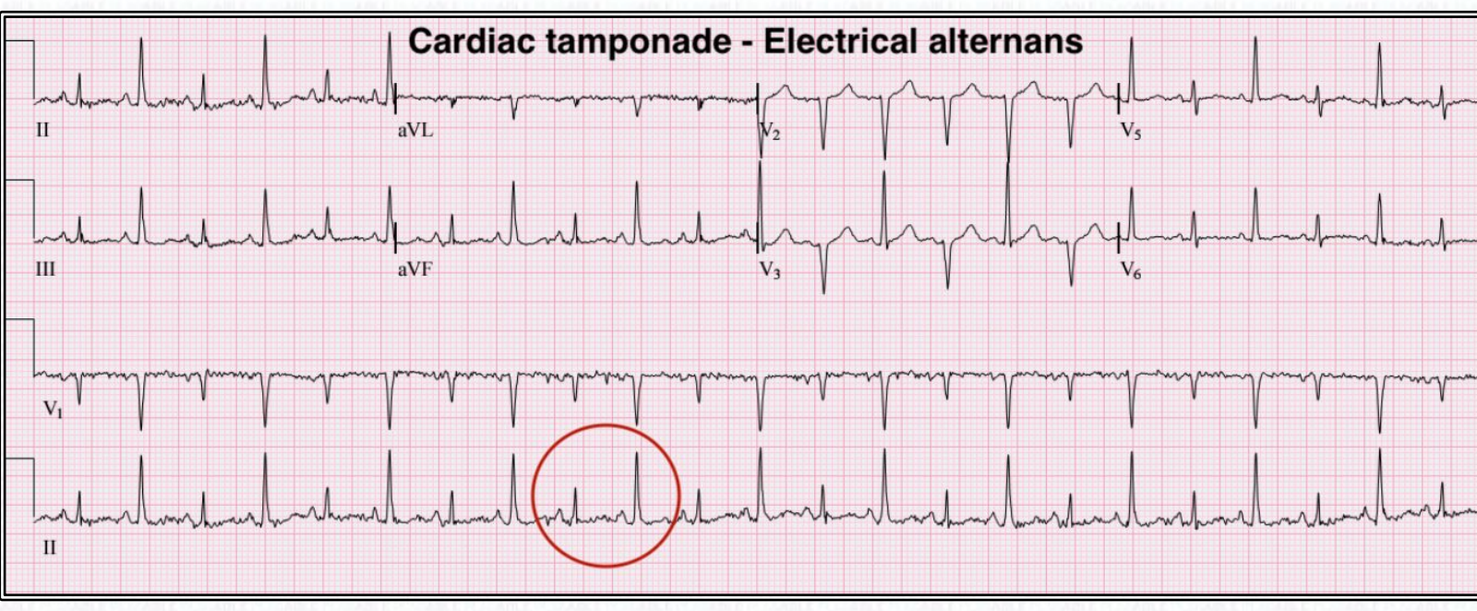
- Echocardiography is the investigation of choice

ECG finding:

- Reduced amplitude QRS complexes
- Electrical alternans

Treatment:

- Pericardiocentesis
- IV fluid (choose if the patient is in shock)

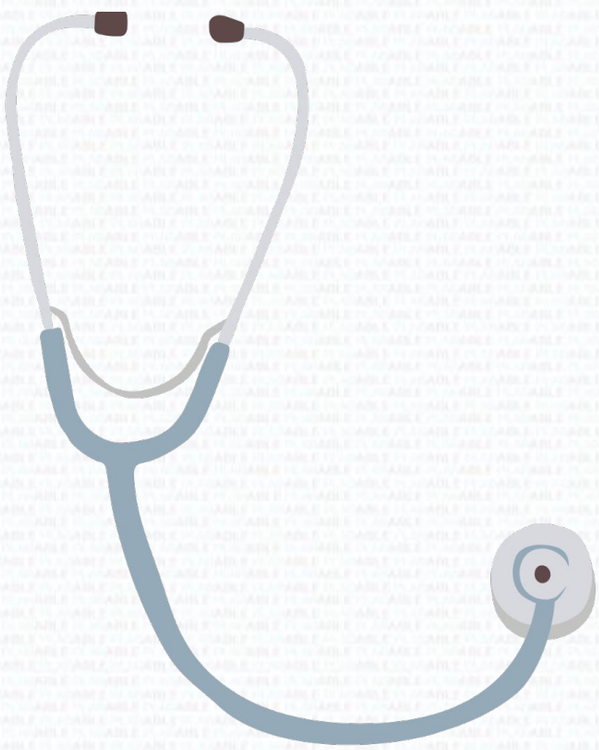


Recap Beck's Triad

It is particularly important to remember Beck's Triad for cardiac tamponade. A good mnemonic is 3D's

Beck's triad

- **D**ecreased BP
- **D**istant heart sounds
- **D**istended jugular veins



Cardiac Tamponade

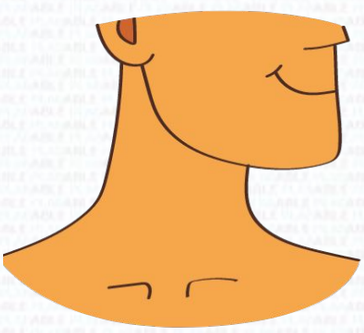
Knife injury to chest

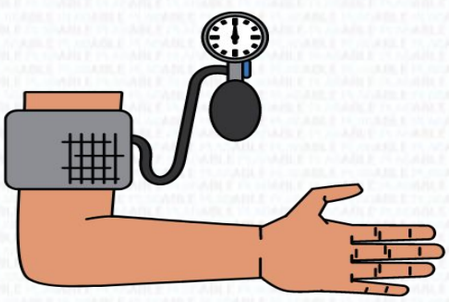
↓

Cardiac tamponade diagnosed

↓

Examination findings observed?





Muffled heart sounds

+ **↑ JVP** + **↓ BP**

Beck's triad

Remember the **3 Ds** →

- **D**istant heart sounds
- **D**istended neck veins
- **D**ecreased BP

Acute Pericarditis

Presentation

- **Symptoms:** Sharp **pleuritic pain** increases during inspiration and relieved by leaning forward
- **Auscultation:** Friction rub
- **ECG:** Widespread **saddle-shaped ST elevation** and PR depression

Causes

- Viral infection - coxsackievirus B
- Post MI and Dressler syndrome
- Uraemia

Plabable tip:
Look out for the history of recent MI or recent upper respiratory tract infection

Treatment

- NSAIDs *(would suffice in most cases)*
- Colchicine
- Corticosteroids

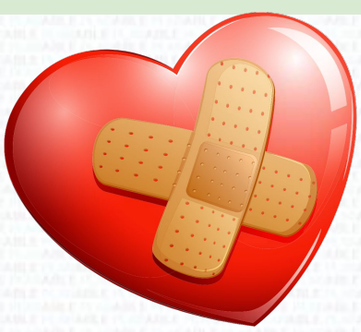
Acute Pericarditis

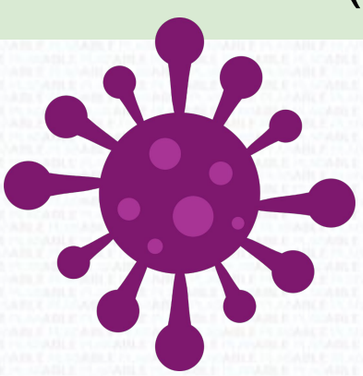
Your typical patient

- Leaning forward for relief

Your typical history

- Recent myocardial infarction
- Recent upper respiratory tract infection (viral)

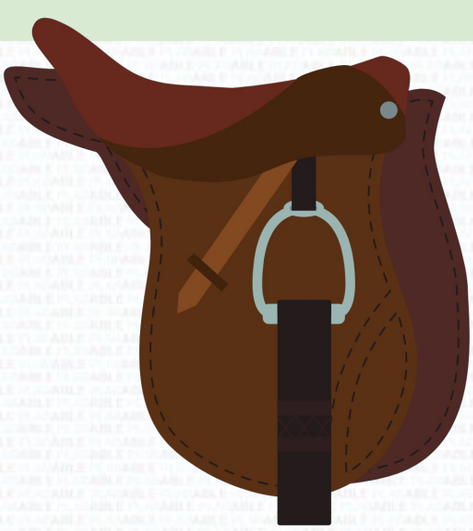




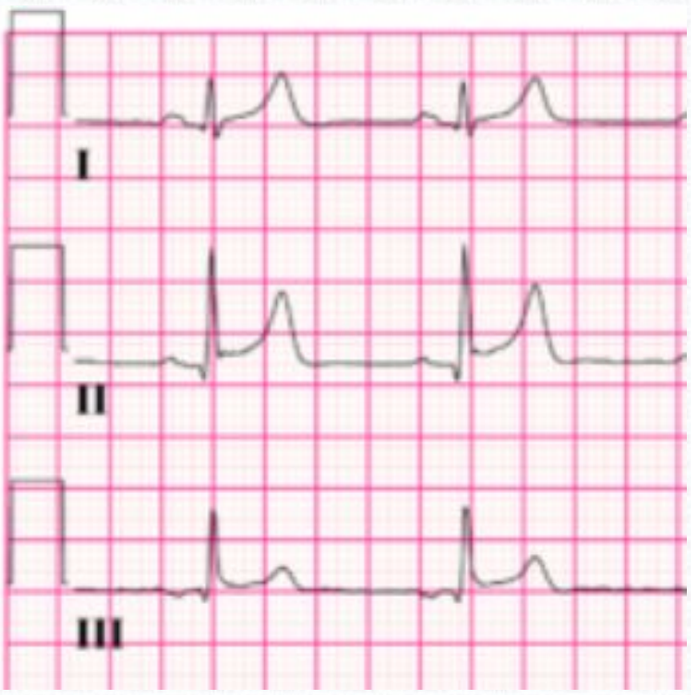
Your typical ECG

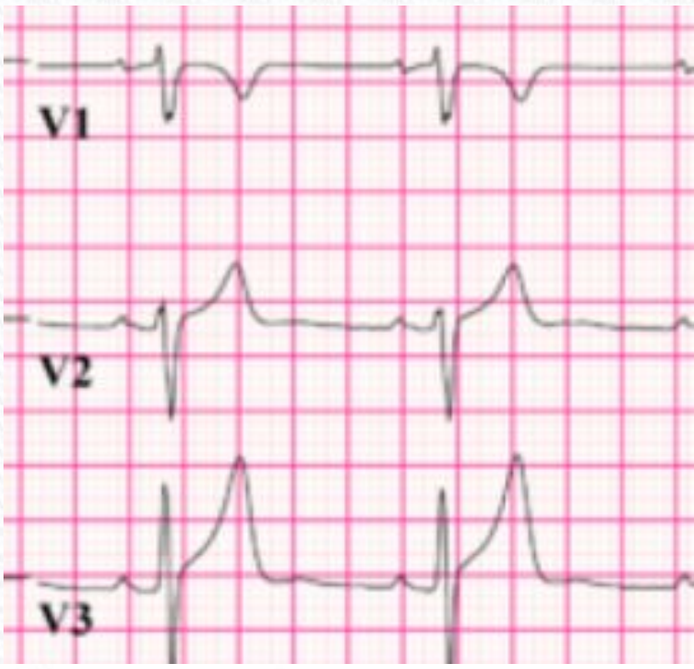
- <u>Widespread</u> saddle-shaped ST elevation

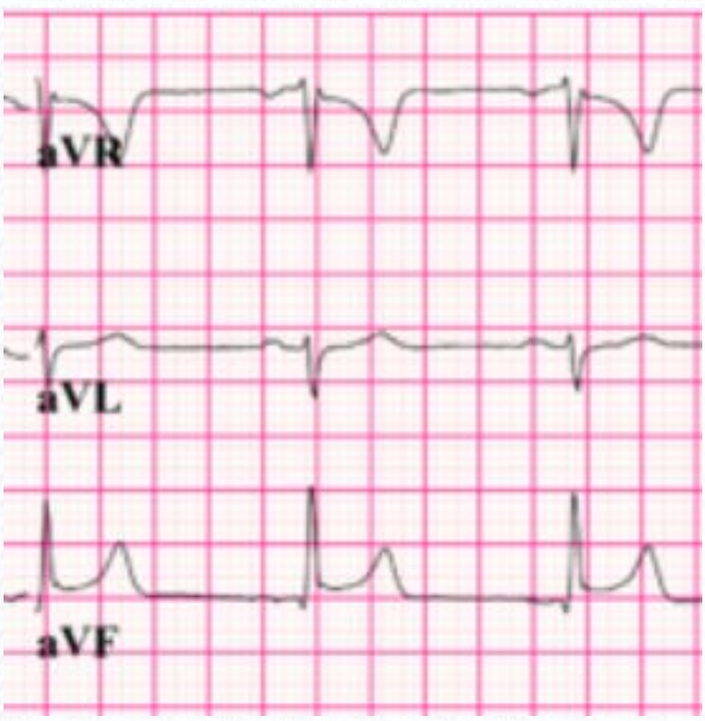
The keyword is **widespread**

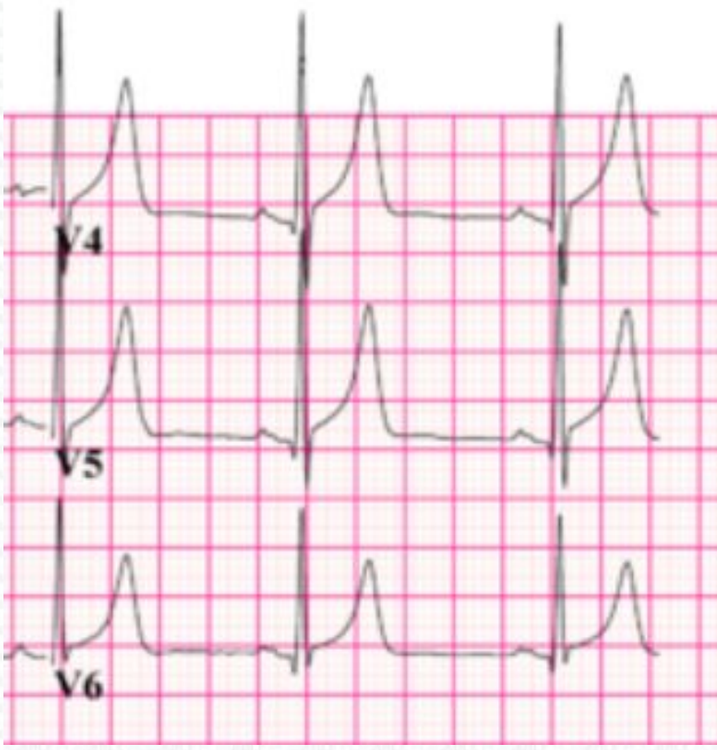


Acute Pericarditis





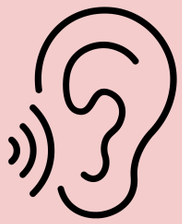




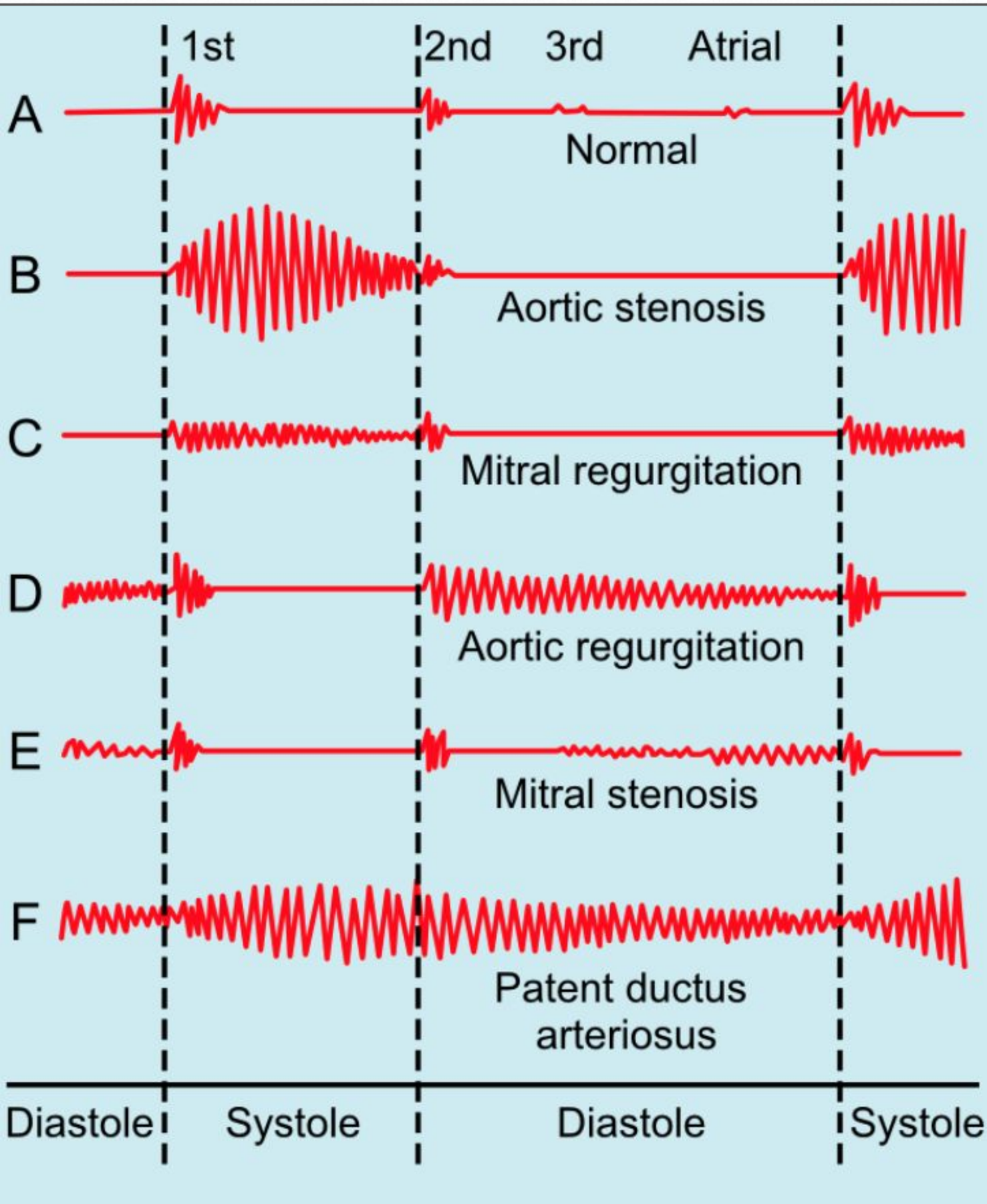
Widespread ST elevation

While ST elevation is widespread on all the leads, it is usually depressed in aVR and lead V1

Cardiac Murmurs

Cardiac defect	Associated murmur 
Mitral stenosis	• Mid-diastolic murmur at the cardiac apex • Associated with opening snap
Mitral regurgitation	Pansystolic murmur at the apex radiating to the axilla
Aortic stenosis	Crescendo/decrescendo systolic murmur (ejection systolic murmur)
Aortic regurgitation	• Diastolic decrescendo murmur • Bounding pulse • Wide pulse pressure

Cardiac Murmurs



Cardiac Murmurs

Cardiac defect	Associated murmur
Tricuspid regurgitation	**Pansystolic** murmur Heard best at lower left sternal edge
Tricuspid stenosis	**Diastolic** rumble Heard best at lower left sternal edge
Pulmonary regurgitation	**Early diastolic** murmur Heard best at second left intercostal space
Pulmonary stenosis	**Ejection systolic** murmur Heard best at second left intercostal space

Cardiac Murmurs

Cardiac defect	Associated murmur
Ventricular septal defect	**Pansystolic** murmur at left lower sternal border In chronic VSD shunt reversal with a right to left flow can happen (**Eisenmenger** syndrome)
Patent ductus arteriosus	**Continuous machinery murmur** Best heard in the left infraclavicular area
Hypertrophic obstructive cardiomyopathy	**Crescendo/decrescendo systolic** murmur Increases on valsalva maneuver and standing up (decreased preload)

Aortic Stenosis

Brain trainer:

A 77 year old woman presents with mild exercise intolerance and a ejection systolic murmur is found. What is the likely diagnosis?

➔ **Aortic stenosis**

➔ **Echocardiogram is the investigation of choice**

Aortic Stenosis

Degenerative calcification is the most common cause in elderly patients

Clinical features

Remember them using this mnemonics- **ASD**

A → Angina
S → Syncope
D → Dyspnoea

Worsening prognosis

A → Aortic
S → Stenosis
D → Diagnosed!

ASD has a particularly good flow since it also represents worsening prognosis.

The following are mean survival rates if one does not go for surgery:

A → Angina → 5 years mean survival
S → Syncope → 3 year mean survival
D → Dyspnoea → 2 year mean survival

Pulmonary Regurgitation

Brain trainer:

A 40 year old man with cardiac surgery when he was a child has a diastolic murmur at the left upper sternal border. What is the likely diagnosis?

➔ **Pulmonary regurgitation**

Mitral Regurgitation

Brain trainer:

A woman presents with orthopnea, widespread bibasilar crepitations and a pansystolic murmur. What is the likely diagnosis?

➔ **Mitral regurgitation**

Mitral Stenosis

Brain trainer:

A man presents with shortness of breath on mild exertion since he returned from Gambia 2 months ago. A chest X-ray shows a sharp straight heart border. There is a mid-diastolic murmur at the apex. What is the most likely diagnosis?

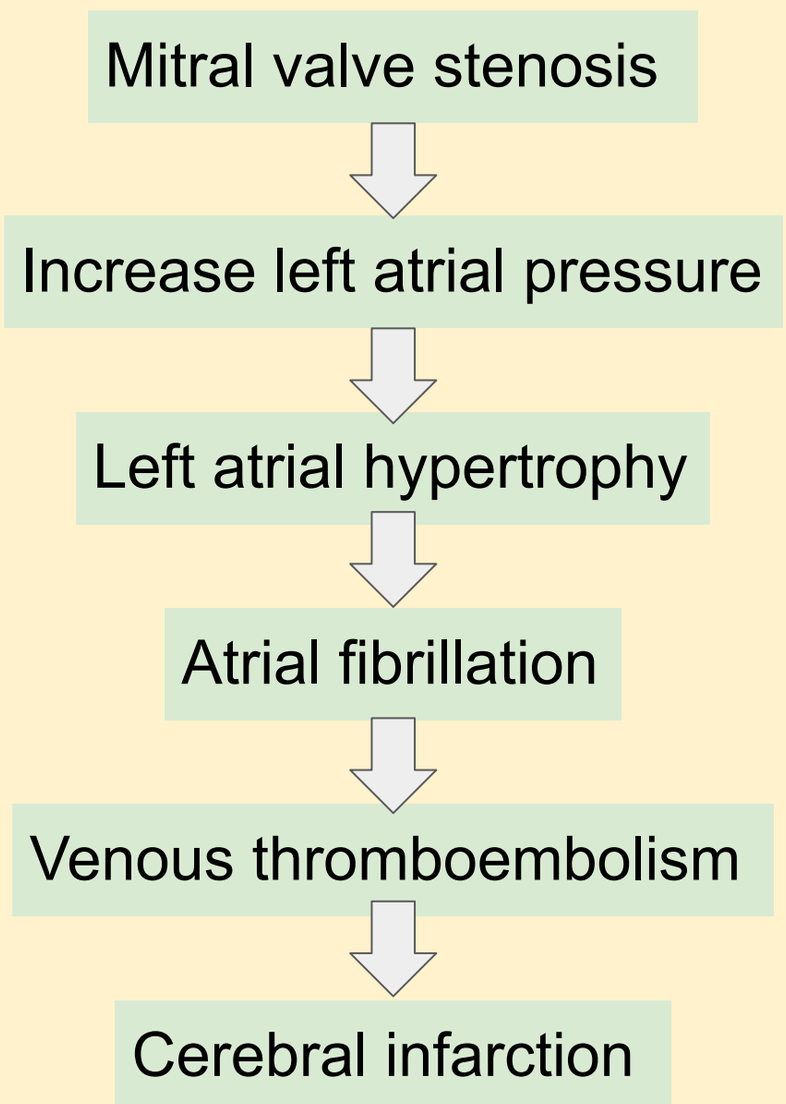
➔ **Mitral stenosis**

Mitral Stenosis

Brain trainer:

A man presents with shortness of breath on mild exertion since he returned from South Africa months ago. An echocardiogram reveals a narrowing of the total area of the mitral valve. What long term complication is this condition associated with?

➔ **Stroke**

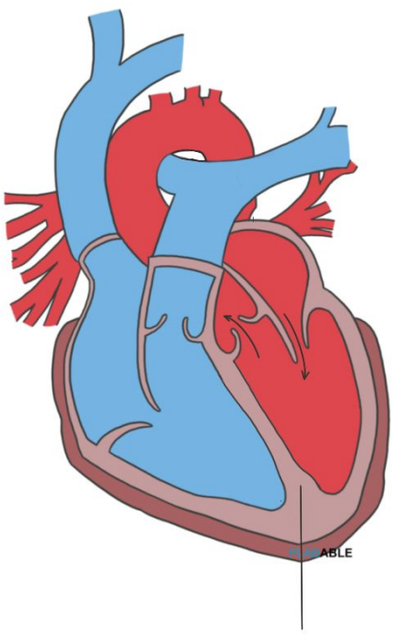


Cardiomyopathy

	Dilated	Hypertrophic	Restrictive
Causes	Alcohol Viral-myocarditis Doxorubicin	Autosomal dominant mutation	Amyloidosis Sarcoidosis Postradiation fibrosis
Dys-function	Systolic	Diastolic	Diastolic
Salient features	Heart failure S3 heart sound	Syncope during exercise Sudden ventricular arrhythmia	Right-sided symptoms: - Oedema - Ascites
Treatment	ACE inhibitors Beta blocker Diuretics Digoxin	Beta-blocker or CCB ICD (arrhythmia)	Diuretics

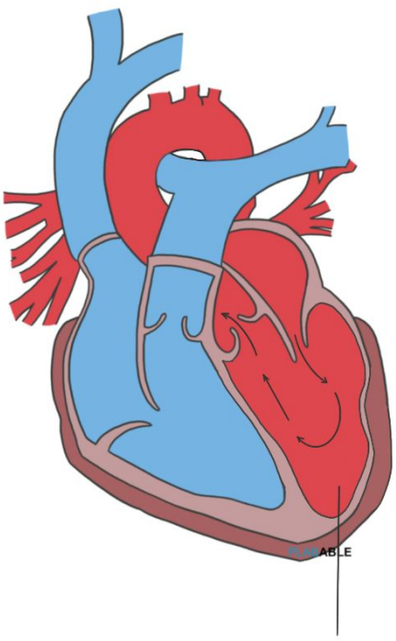
Cardiomyopathy

Normal Heart



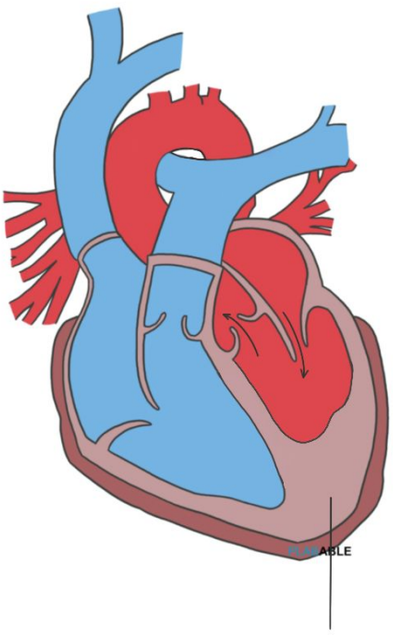
Interventricular Septum

Dilated Cardiomyopathy



Ventricular dilation (Muscle fibres have stretched)

Hypertrophic Cardiomyopathy

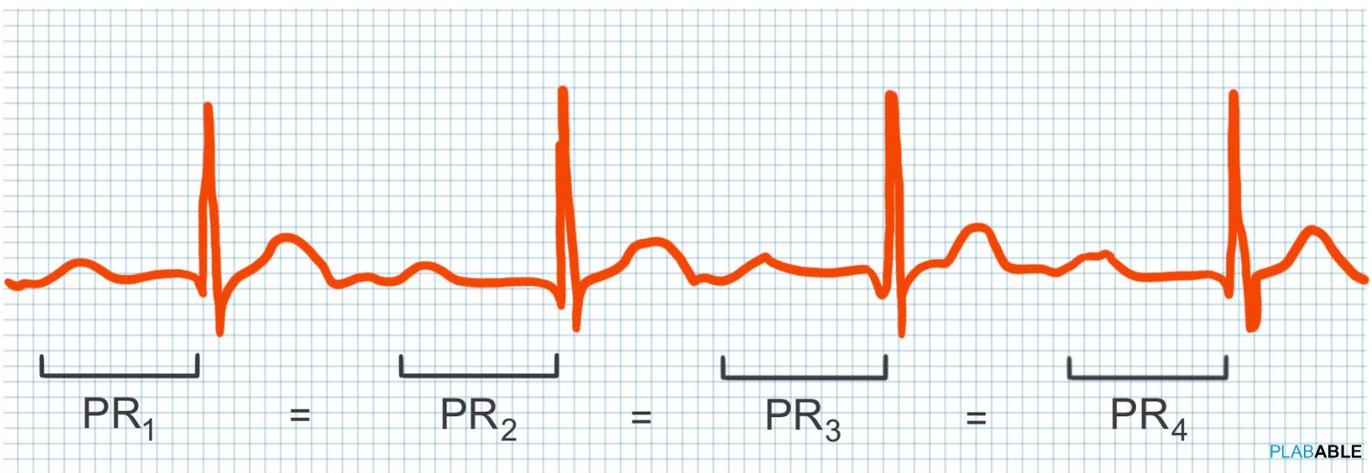


Excessive wall thickening of cardiac muscles

AV Conduction Block

First-degree AV block

- Prolonged PR interval (>200ms)
- No treatment is required

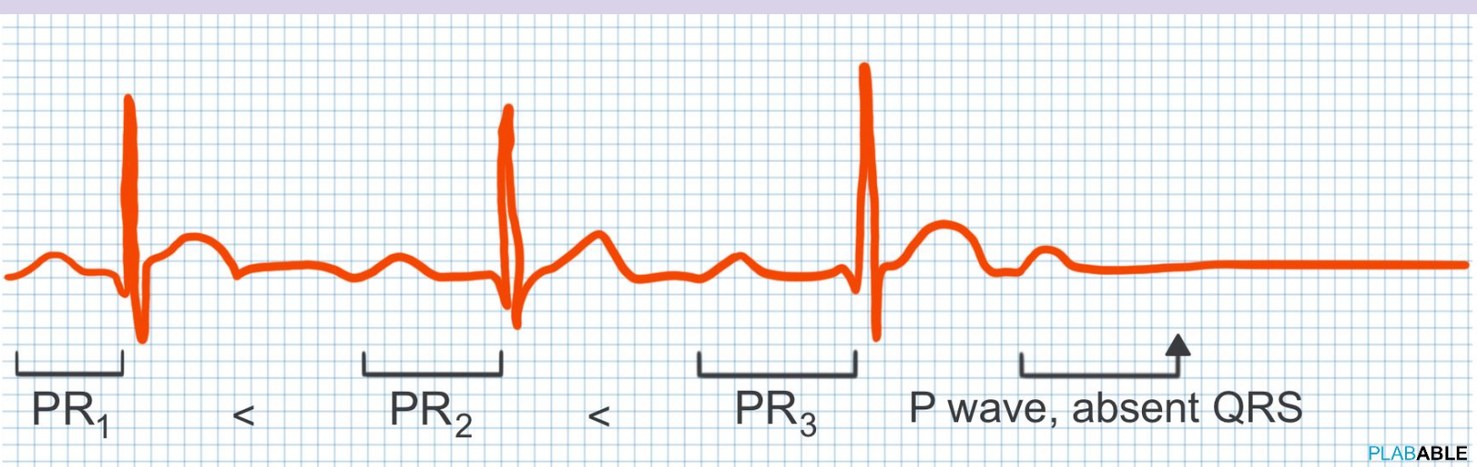


First-degree AV Block

Second-degree AV block

Mobitz type I

- PR interval lengthens progressively until a beat is missed RR interval is variable
- Symptomatic bradycardia → Treat with Atropine

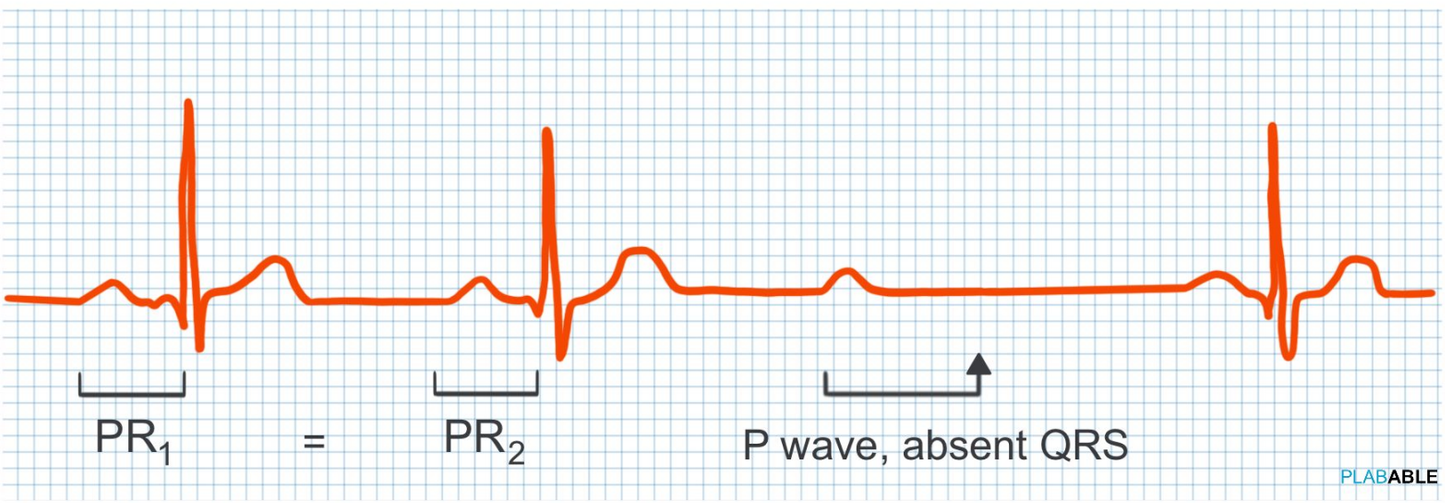


Second-degree AV Block - Mobitz Type I

AV Conduction Block

Mobitz type II

- PR interval regular, dropped beats (QRS complex)
- Symptomatic bradycardia → Treat with Atropine initially
- Treated with a pacemaker

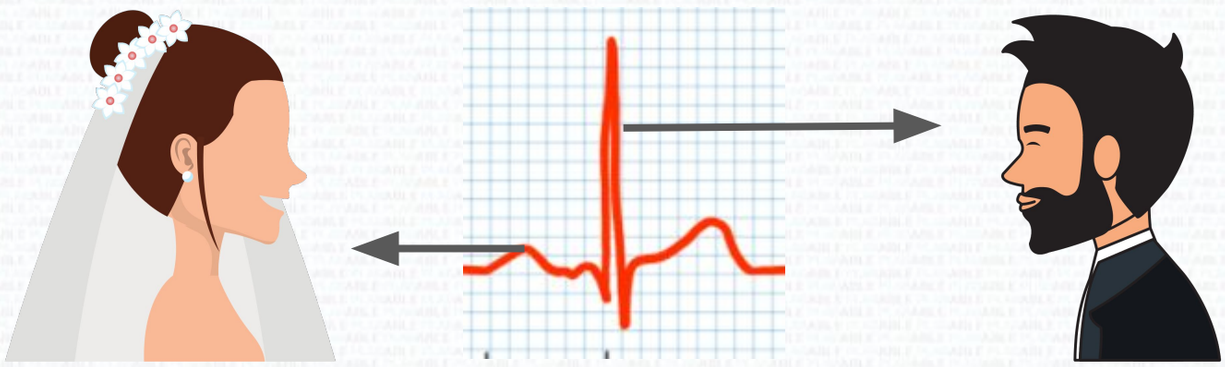


Second-degree AV Block - Mobitz Type II

AV Conduction Block

Mobitz I Vs Mobitz type II Memory Aid

A good memory tool to differentiate Mobitz type I and II is the analogy of “**the late husband”.**



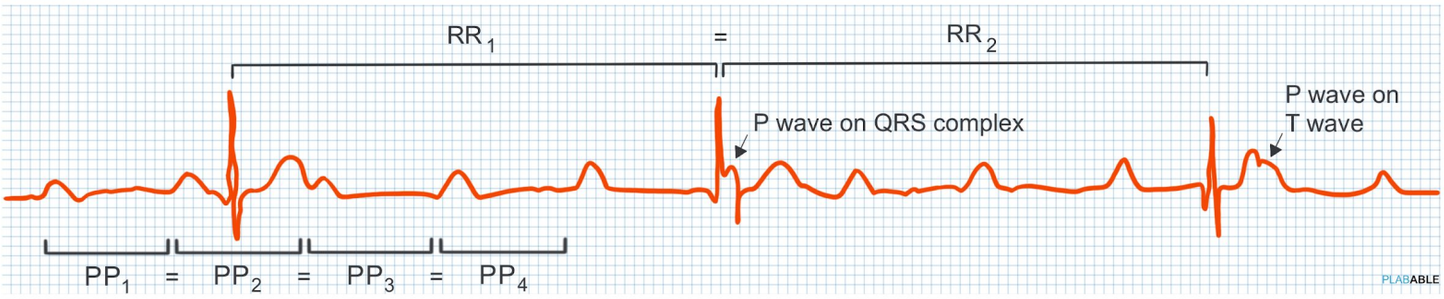
The husband represents the QRS complex and the wife represents the P wave.

In type I, the husband comes home later and later everyday (hence longer PR intervals) before he does not come home completely (dropped QRS complex).	In type II, the husband comes home on time, everyday, until suddenly, he decides not to come home anymore without any warning signs. This is the reason type II is more dangerous!

AV Conduction Block

Third-degree AV block (complete)

- Atria and ventricles contract independently
- No association between the P wave and QRS complex
- **Causes:** Myocardial infarction and lyme disease
- **Immediate treatment: Atropine 500 mcg IV** followed by transcutaneous pacing
- **Definitive treatment:** pacemaker

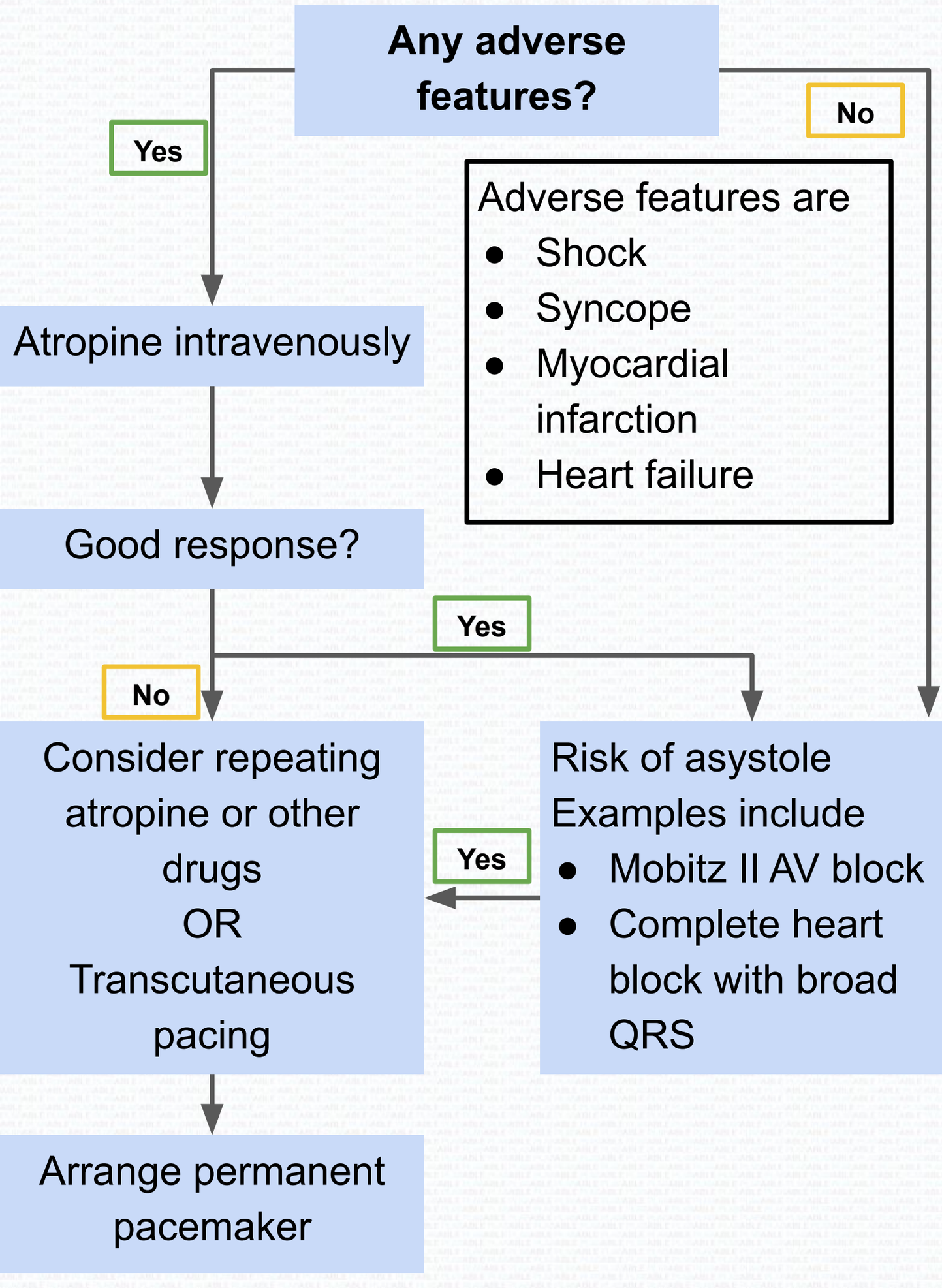


Third-degree AV Block

AV Conduction Block

All symptomatic bradycardia despite rhythm
→ Start with atropine

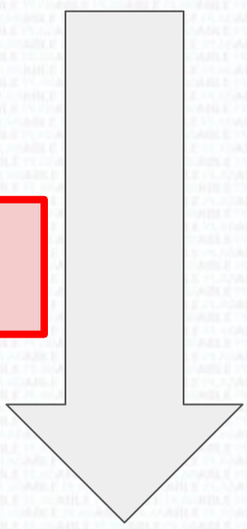
Bradycardia Algorithm



AV Conduction Block Memory Aid

In any event that the patient has symptomatic bradycardia, atropine is your **FIRST INITIAL MANAGEMENT**

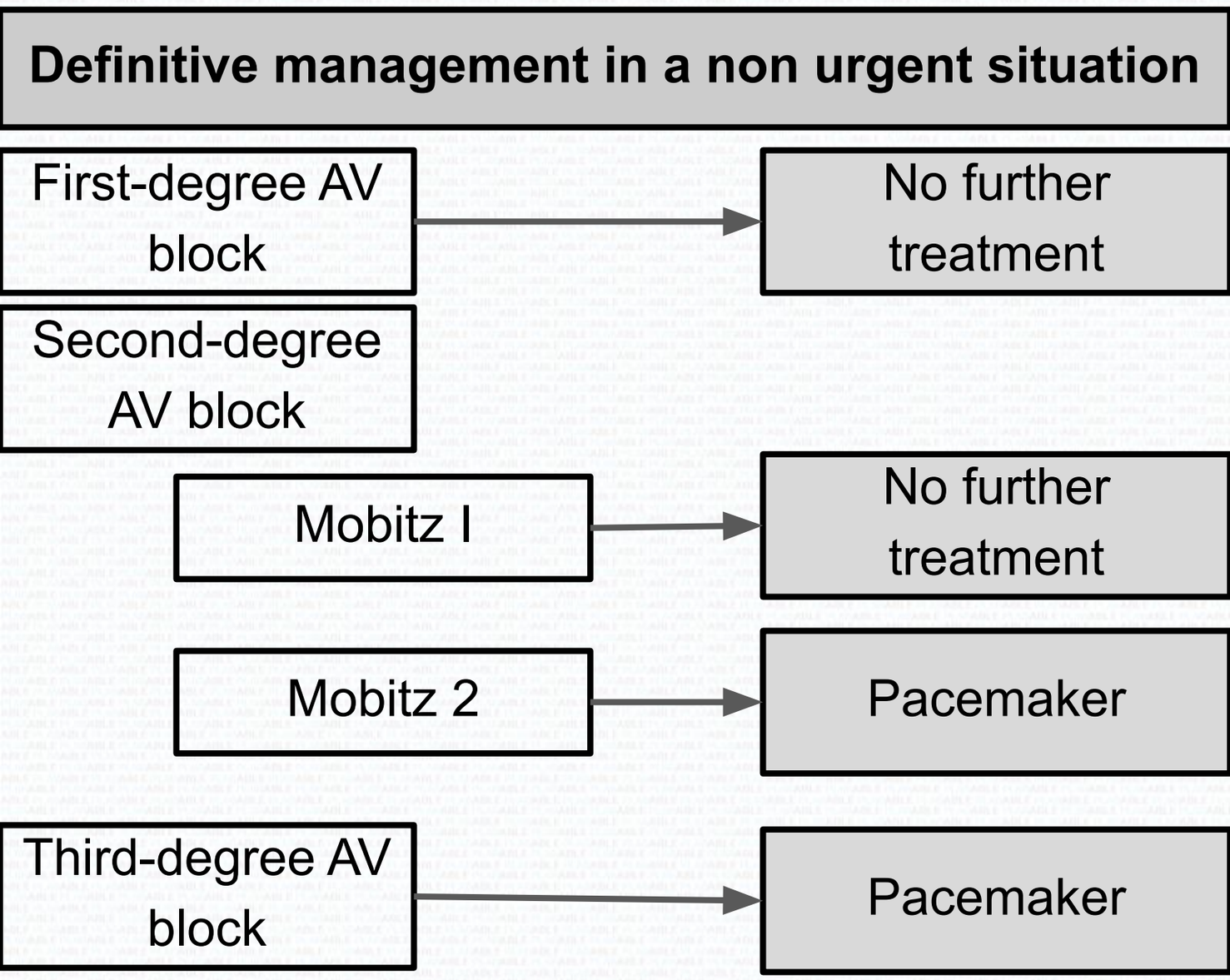
After that



Temporary pacing becomes the **MOST APPROPRIATE NEXT STEP IN MANAGEMENT** in patients with adverse effects from bradycardia who:

- Do not respond to atropine OR
- Have Mobitz type II or complete heart blocks with wide QRS

AV Conduction Block Memory Aid



Anti-arrhythmic Medications

Type	Uses	Side-effects
CLASS I Sodium channel blockers	**Quinidine** & **lidocaine** - atrial and ventricular arrhythmias	**Quinidine** -cinchonism (headache and tinnitus)
CLASS II Beta-blocker	**Metoprolol** - SVT Rate control for AF and atrial flutter	Exacerbation of COPD and asthma AV block
CLASS III Potassium channel blockers	**Amiodarone** - ventricular tachycardia	**Amiodarone** - ● Pulmonary fibrosis ● Hepatotoxicity ● Hypo or hyperthyroidism ● Corneal deposits **Sotalol and ibutilide** - Torsades de pointes

Antiarrhythmic Medications

Type	Uses	Side-effects
CLASS IV CCB	**Verapamil** - Rate control in AF	• Oedema • Constipation • AV block
Others	**Adenosine** - SVT	• Hypotension • Flushing • Bronchospasm

Antianginal Medications

Class	Drugs	Mechanism	Side-effects
Nitrates	Nitroglycerin Isosorbide dinitrate	Reduces preload and decreases myocardial oxygen requirements	Headache Tolerance Interaction with sildenafil
Calcium channel blockers	Verapamil Diltiazem	↓ Cardiac contractility Vasodilation ↓ Afterload	Bradycardia Transient asystole
Beta-blockers	Atenolol Bisoprolol Metoprolol	↓ Myocardial oxygen requirements by ↓heart rate	Bradycardia AV block
Ivabradine		↓ Heart rate	Phosphenes

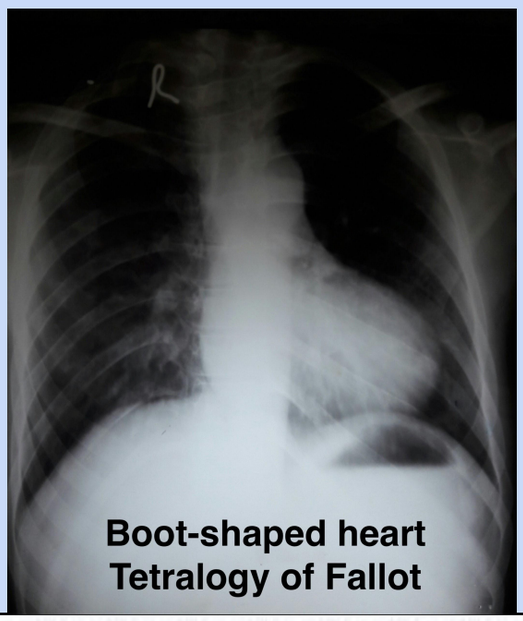
Amiodarone

The following are test to perform before starting a patient on amiodarone:

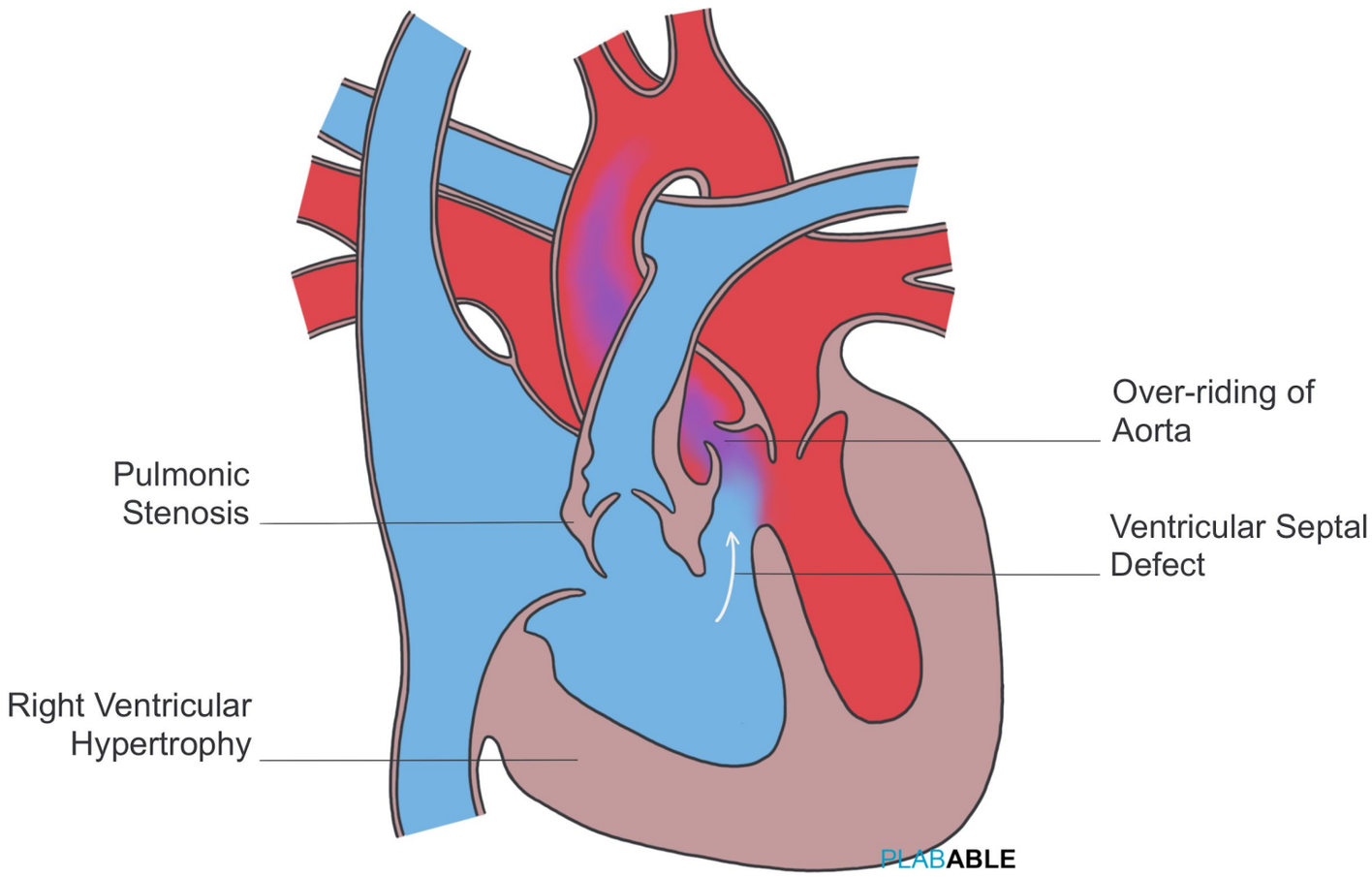
- Thyroid function tests
- Liver function tests
- Serum electrolyte and urea measurement
- Chest radiography
- Electrocardiography

Pay special attention to serum electrolyte and urea measurements

Congenital Heart Disease (Cyanotic)

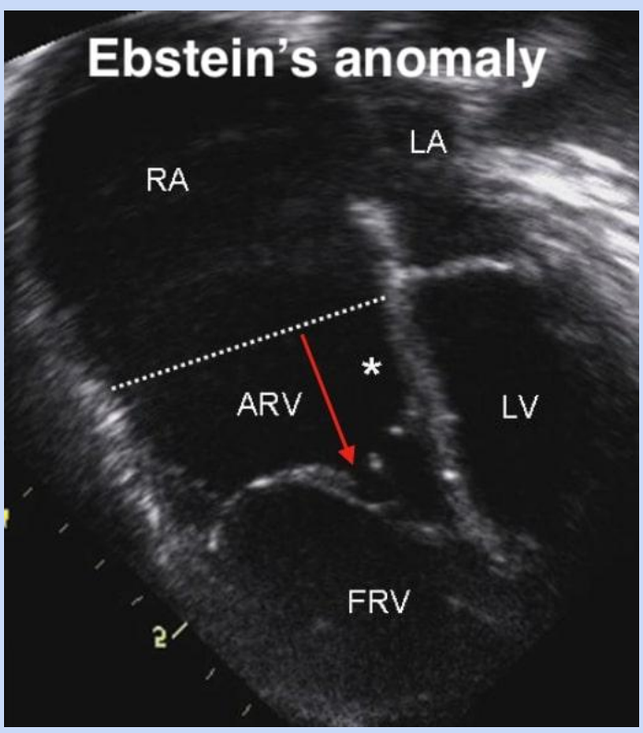
Tricuspid atresia	Absent tricuspid valve Associated with ASD and VSD
Transposition of great arteries	Pulmonary artery exits from left ventricle and aorta leaves from right ventricle.
Tetralogy of Fallot  Boot-shaped heart Tetralogy of Fallot	• Pulmonary stenosis • Right ventricular hypertrophy • Overriding of aorta • VSD **X-ray: Boot-shaped** heart Squatting spells are seen

Congenital Heart Disease (Cyanotic)



Tetralogy of Fallot

Congenital Heart Disease (Cyanotic)

Persistent Truncus Arteriosus	Failure of truncus arteriosus to form pulmonary trunk and aorta
Ebstein Anomaly	Atrialization of the right ventricle Associated with lithium use during pregnancy. 
Total Anomalous Pulmonary Venous Connection	Pulmonary vein drains into the right heart

Congenital Heart Disease (Acyanotic)

Ventricular septal defect (Most common)	Pansystolic murmur
Atrial septal defect	Fixed split S2
Patent ductus arteriosus	Continuous machinery murmur in the left infraclavicular region
Coarctation of aorta	Associated with **Turner syndrome** **Hypertension** is seen in the upper limb with brachiofemoral delay Increased risk of Berry aneurysms and cerebral hemorrhage

Eisenmenger syndrome:

Long standing left to right shunts (VSD, ASD and PDA) → Pulmonary hypertension → Shunt reversal (Right to left) → Late cyanosis and clubbing

Hypertension (Non-diabetic)

Clinic BP ≥ 140/90 mmHg

Ambulatory BP measurement ≥ 135/85 mmHg

Age < 55

Step 1
ACEi or ARB

Step 2
Add CCB
or
thiazide-like diuretic

Age ≥ 55
OR
Black African or African-Carribean

Step 1
Calcium-channel blocker

Step 2
Add ACEi/ARB
or
thiazide-like diuretic

Step 3
ACEi/ARB + CCB + thiazide-like diuretic

Click here for our teaching video on hypertension

YouTube

PLABABLE

Hypertension (Non-diabetic)

Memory tool!

ACEi or **A**RB

< 55 ≤

CCB

A

Before

C

As **A** is **B**efore **C**,
use **A**CEi **B**efore age 55 and **C**CB for people 55 years and older

Hypertension (Diabetic)

Treatment for diabetics differ from non-diabetics.

Age and race is irrelevant.

Hypertension with diabetes

↓

Step 1
ACEi or ARB

↓

Step 2
Add CCB
or
thiazide-like diuretic

↘

Step 3
ACEi/ARB + CCB + thiazide-like diuretic

Hypertension

BP TARGETS ON ANTIHYPERTENSIVES

Normal clinic BP targets

- <140/90 if under 80 years old
- <150/90 if 80 years old and above

Type 2 Diabetes Mellitus (since 2019)

- Same as normal clinic BP targets

CKD + diabetes mellitus

- <130/80

CKD + Urinary ACR more than 70

- <130/80

Hypertension

Medication	Side effects
ACE Inhibitors	• **Cough** (angioedema) • **Hyperkalemia**
Calcium channel blockers	• **Peripheral oedema** • Dizziness • Flushing • Constipation
Thiazide diuretic	• **Hypokalemia** • **Hyperuricemia** • Postural hypotension • ↑ **Serum lithium levels**

PodsForDocs

Check out our podcast episode '*A GP's Perspective*' where we discuss the experience and thoughts from an NHS consultant GP on hypertension cases in primary care.

Click on the image below to head to our PodsForDocs podcast page to find out more.

We also have a dedicated PodsForDocs WhatsApp group which you can join via the Study Group tab on your Account. Enjoy!

Postural Hypotension

Brain trainer:

A 64 year old man previously diagnosed with hypertension is having recurrent falls. He takes enalapril, amlodipine and indapamide. Chest sounds are normal. ECG is normal. What is the likely underlying cause of his falls?

➔ **Postural hypotension**

Always consider if patients are on multiple antihypertensive medications

Syncopal Episodes

Brain trainer:

An elderly man taking antihypertensives attends the GP surgery after having two syncopal attacks involving loss of consciousness for a few seconds. He was standing in his garden when he had the syncopal episodes. His blood pressure is 110/80 mmHg on lying and it dropped by 25 mmHg when standing. He has a heart rate of 80 beats/minute. What is the SINGLE most appropriate initial investigation?

➔ **ECG**

ECG to rule out cardiac aetiologies is one of the first investigations in any patient who has episodes of syncope.

Severe Hypertension

Definitions

Severe hypertension

Clinic systolic BP is higher than 180 mmHg or clinic diastolic BP is 120 mmHg or higher

Malignant hypertension (accelerated hypertension)

Severe hypertension + retinal haemorrhage or papilloedema

Severe Hypertension

Management

Severe hypertension

Do a fundoscopy

Retinal haemorrhage or papilloedema present

Malignant hypertension (accelerated hypertension)

If patient has new onset confusion, chest pain, signs of heart failure or acute kidney injury

Refer to A&E

Most patients in A&E can be managed by oral beta-blockers (atenolol or labetalol) or calcium channel blockers (nifedipine)

If concerns about stroke → Perform a CT scan

Severe Hypertension

Management

Severe hypertension

If patient is asymptomatic

Do a fundoscopy

Retinal haemorrhage or papilloedema NOT present

Look for other target organ damage

No target organ damage

Repeat BP in 7 days

Target organ damage

Consider oral antihypertensive treatment

Target Organ Damage

How to assess for target organ damage in relation to patients with hypertension?

Fundoscopy

- Look for evidence of hypertensive retinopathy

Urine albumin:creatinine ratio (ACR)

- Test for the presence of protein in the urine

HbA1C

- Test for diabetes

Renal function (electrolytes, creatinine, eGFR)

- Test for chronic kidney disease

ECG

- To look for left ventricular hypertrophy

Infective Endocarditis

Risk factors

- Valvular heart disease
- Valve replacement
- Previous episode of IE (highest risk)
- Intravenous drug users

Signs

- **Murmur and fever**
- **Roth spots:** White spots on retina surrounded by hemorrhage
- **Osler nodes:** Raised and tender on the fingers
- **Janeway lesions:** Painless, erythematous lesions on palm or sole
- **Splinter hemorrhages** on the nail beds

Common cause: *Streptococci and S. aureus*
Mitral valve is commonly affected

Injection drug users:

- Tricuspid valve commonly involved
- Commonly caused by *S. aureus*

New murmur + Fever → Think of IE

Initial step → Blood culture → Echo

Infective Endocarditis

Modified Duke criteria

- Two major criteria (or)
- One major criterion and three minor criteria (or)
- Five minor criteria

Major criteria

- Positive blood culture on two different occasions
- Evidence of endocardial involvement either by echo or new onset murmur

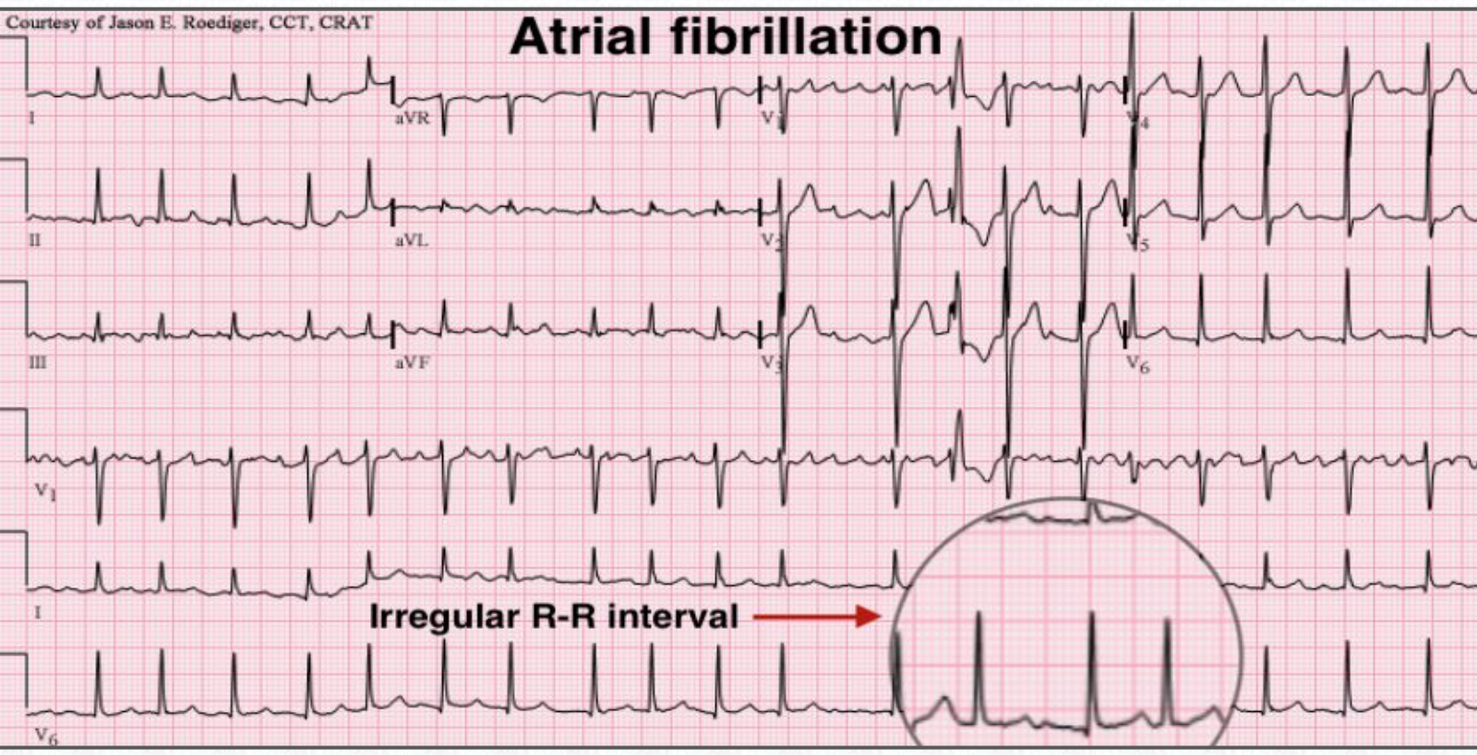
Minor criteria

- Predisposing heart condition or injection drug use
- Fever ≥38 degrees
- Vascular phenomenon such as Janeway lesions
- Immunological phenomenon such as Roth's spot
- Positive blood culture not meeting major criteria

Management

- **Native valve:**
  - Amoxicillin + Gentamicin
  - MRSA: Vancomycin + Gentamicin
- **Prosthetic valve:**
  - Vancomycin + Gentamicin + Rifampin

Atrial Fibrillation



- Irregular RR interval
- No discrete P waves
- Irregularly irregular pulse rate

Treatment

1. Unstable vitals: Electrical cardioversion
2. Stable vitals**:**
   a. Rate control - beta-blockers
   b. Rhythm control - amiodarone, flecainide
   c. Anticoagulation - DOAC (first line), Warfarin if DOAC contraindicated

CHA2DS2-VASc score is used to determine the need for anticoagulation

Atrial Fibrillation
DOAC Vs Warfarin

In terms of anticoagulation for atrial fibrillation, ALWAYS pick DOAC over warfarin.

DOACs (Direct Oral Anticoagulants) also known as NOACs (Novel Oral Anticoagulants)

Warfarin should only be picked if DOAC is not in the options or if DOACs are contraindicated *(by the way, there are not many reasons clinically why a patient would be started on warfarin over DOAC)*

DOAC **Over** **Warfarin**

Examples of DOAC, remember the mnemonic **DARE**

Dabigatran
Apixaban
Rivaroxaban
Edoxaban

*Do you **DARE** to start **DOACs** in clinical practice?*

Atrial Fibrillation

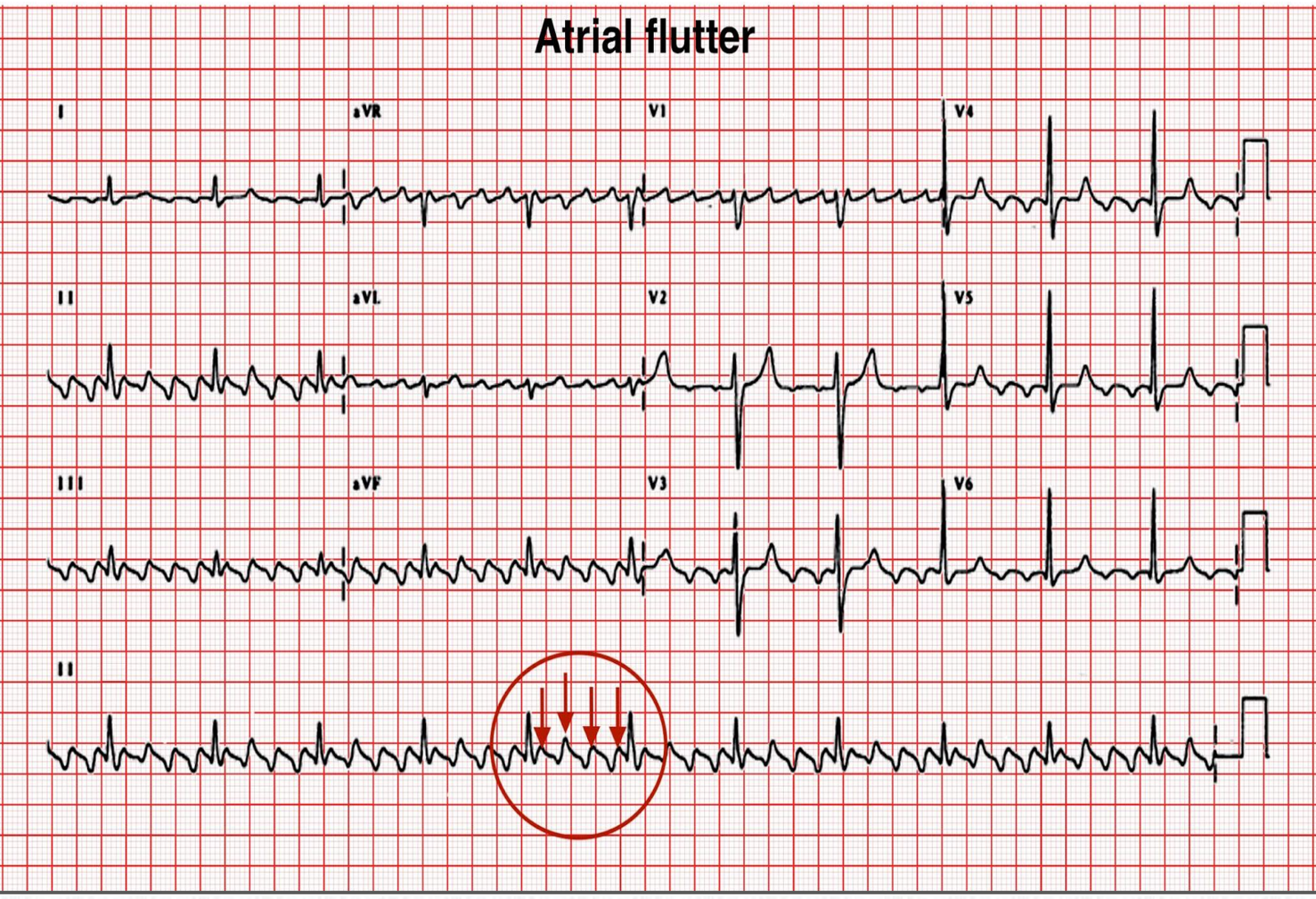
Brain trainer:

A 72 year old woman is confused, pale with an irregularly irregular pulse. Her blood pressure is **80/50** mmHg and heart rate is 130 bpm. ECG shows narrow **QRS complexes and absent P waves**. What is the next step in management?

➔ Immediate **DC cardioversion**

As unstable (confusion, hypotensive) → Cardiovert

Atrial Flutter

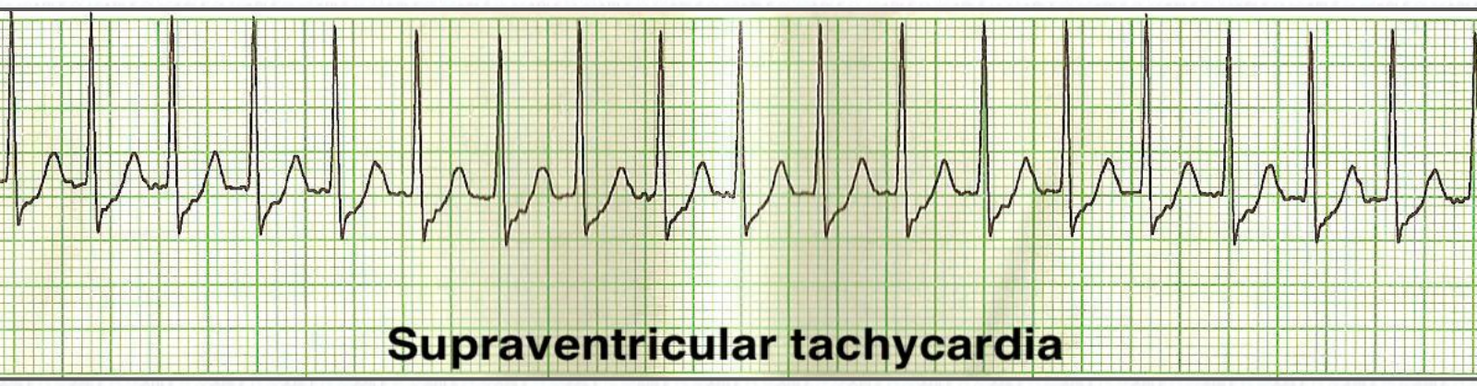


Atrial flutter

- Identical and rapid back to back P waves
- Sawtooth appearance

Treatment is same as AF

Supraventricular Tachycardia



Haemodynamically stable?

Yes

No

Valsalva manoeuvres

DC cardioversion

If ineffective:
Adenosine 6 mg rapid IV bolus

If not successful, another dose of **12 mg** IV

If still unsuccessful, another dose of **18 mg** IV

If still unsuccessful, **beta blockers or verapamil** can be used.

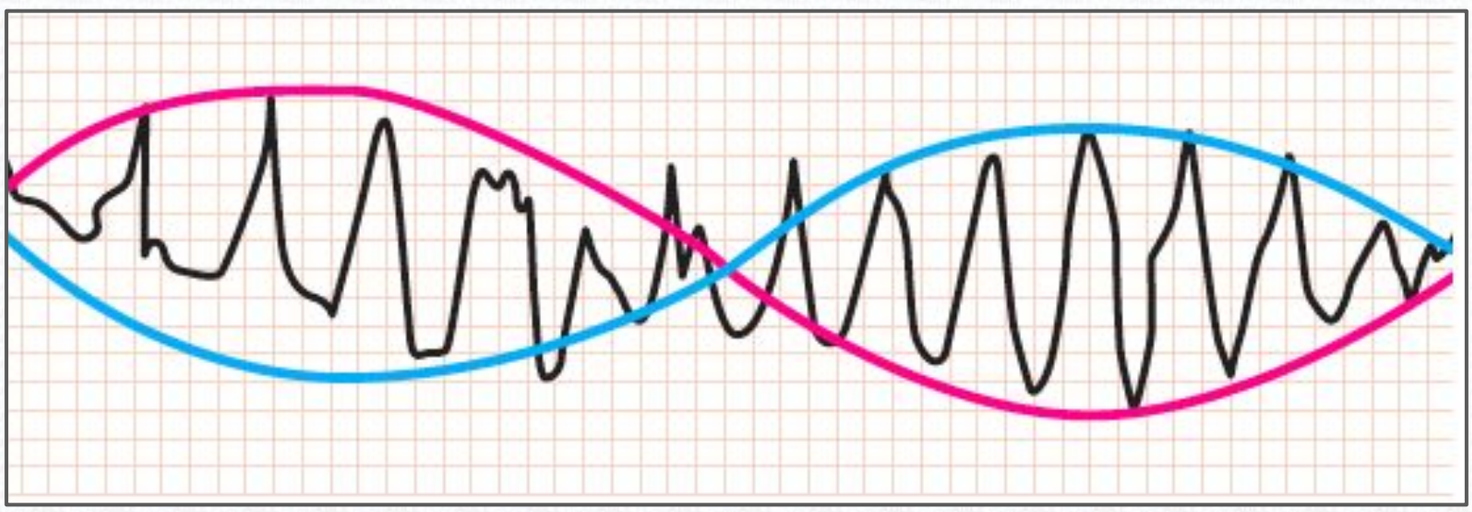
DC cardioversion

Torsades de Pointes

- Type of polymorphic ventricular tachycardia where the QRS complexes appears to be twisting around the base in the ECG
- QT prolongation is the cause
- Can degenerate to VF

Causes

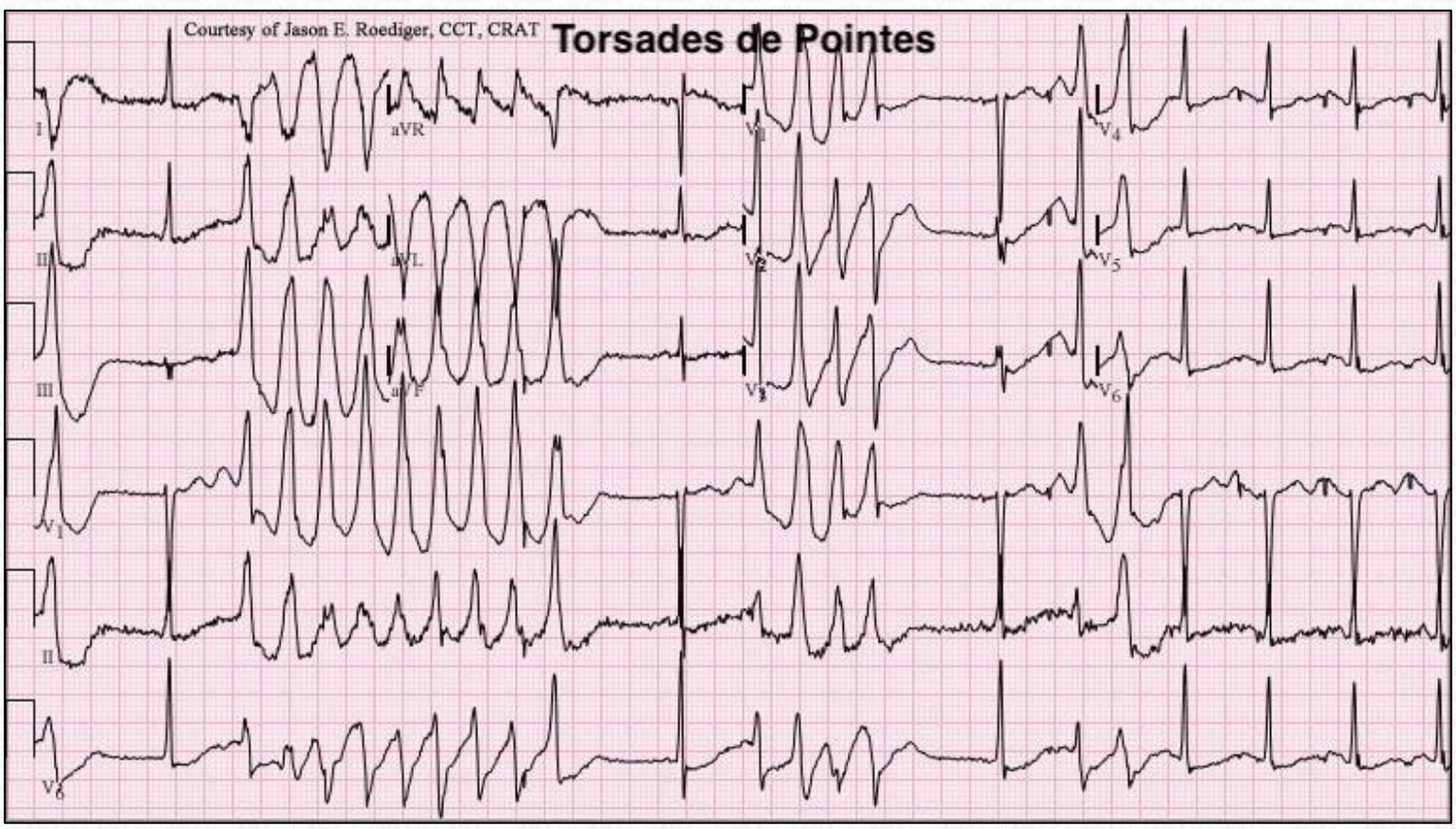
- Long QT syndrome
- Electrolyte abnormalities:
  - Hypomagnesemia
  - Hypokalemia
  - Hypocalcemia
- Antipsychotics
- Antibiotics: erythromycin



Torsades de Pointes

Treatment

- **IV magnesium sulphate**
- Correction of electrolyte abnormalities
- Removal of causative drug
- If patient progresses to VF then defibrillation



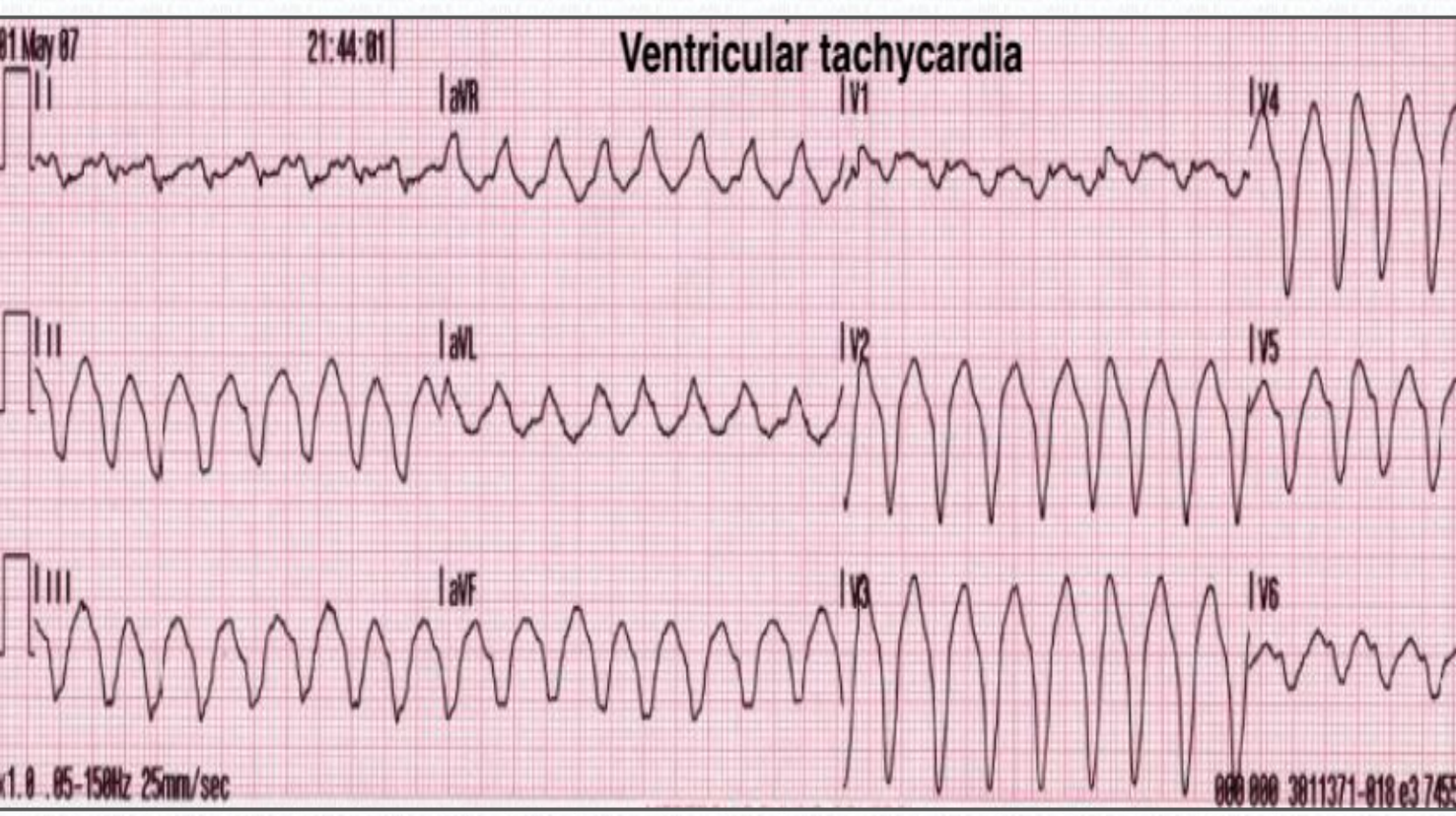
Ventricular Tachycardia

Features

- Wide QRS complex (broad complex tachycardia)
- Can develop into ventricular fibrillation

Management

- **Unstable vitals:**
  - If with pulse → Cardioversion
  - If without pulse → Defibrillation
- **Stable vitals: Administer antiarrhythmic**
  - Amiodarone (preferred)
  - Flecainide
  - Lidocaine

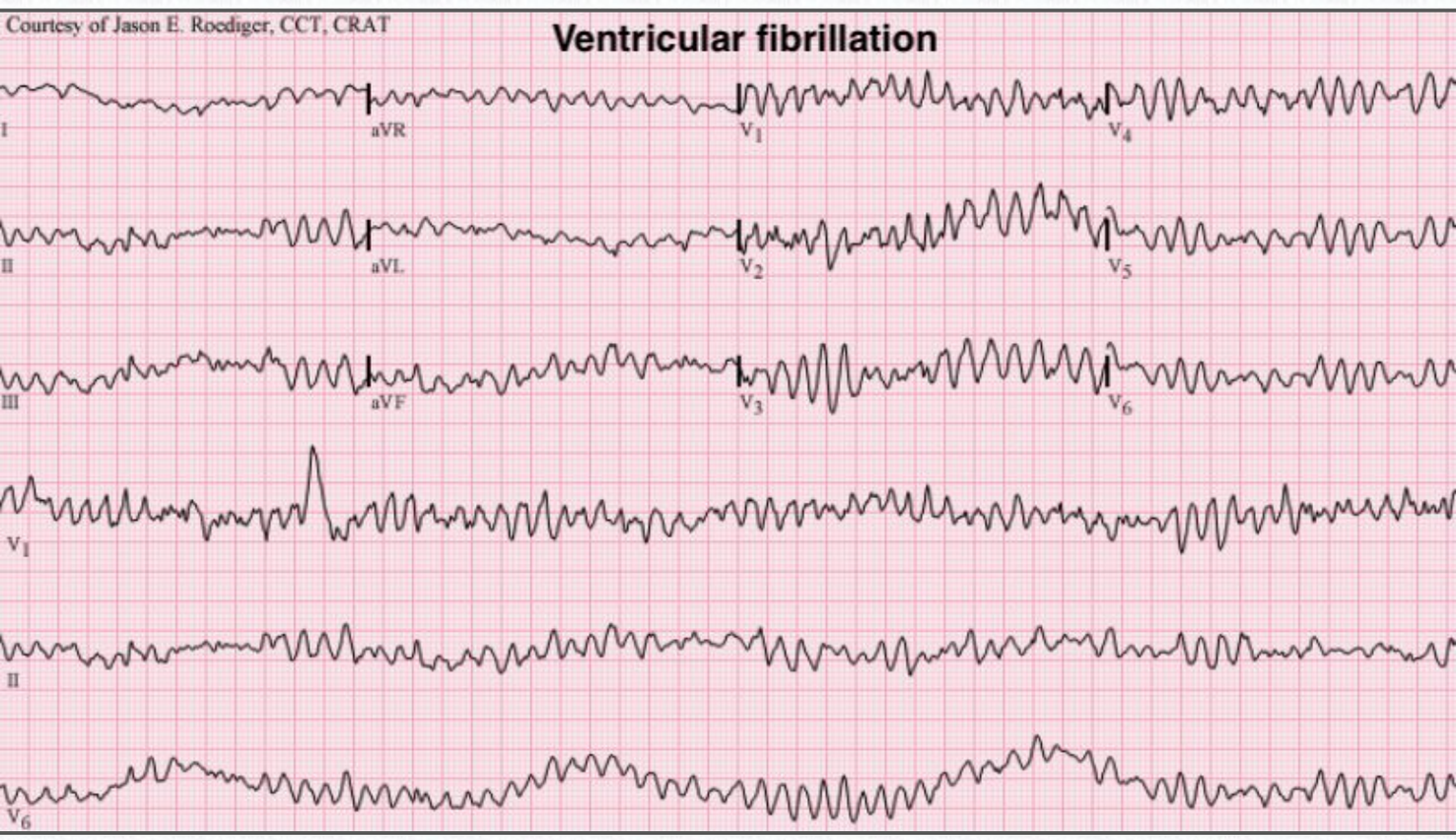


Ventricular Fibrillation

Ventricular fibrillation

- Completely disordered ventricular electrical activity
- No identifiable QRS complexes

Treatment: Immediate defibrillation to bring back the sinus rhythm



Ventricular Fibrillation

Brain trainer:
A 49 year old man is semi-conscious, BP 80/60, ECG shows broad complex tachycardia (either V fib or V tach). What is the most appropriate action?

As he is haemodynamically unstable, this depends purely if he has a pulse:

- If pulse → Cardiovert
- If no pulse → Defibrillate (unsynchronised cardioversion)

Remember:
Ventricular fibrillation NEVER has a pulse

Wolff-Parkinson-White Syndrome

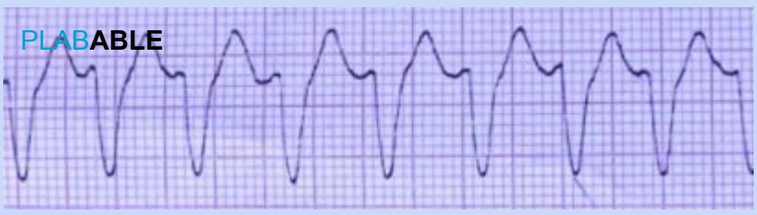
Brain trainer:

A 12 year old child presents with sudden onset of pallor, palpitations, and difficulty breathing while running on the school track. After 30 minutes, the symptoms resolved. ECG shows pre-excitation, delta waves, and prolonged QRS.. What is the likely diagnosis?

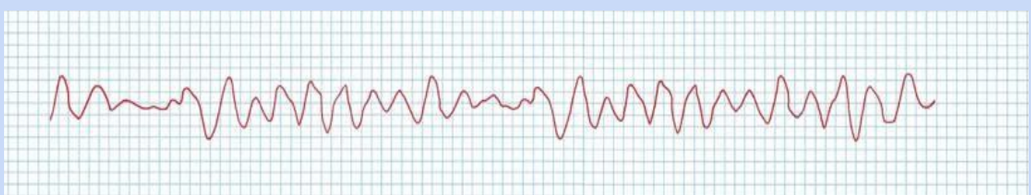
➔ **Wolff-Parkinson-White syndrome**

The ECG QRS Quick Tip

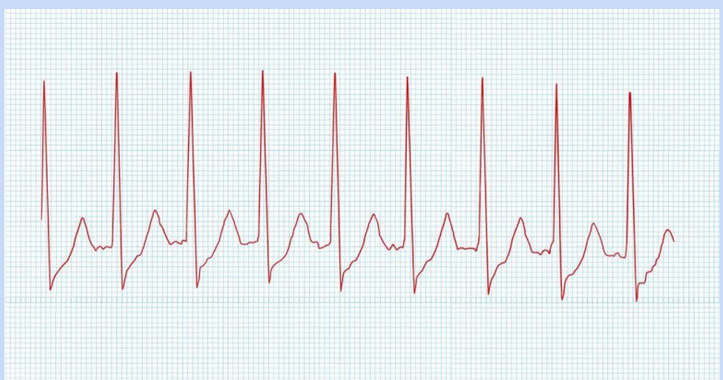
VT - Broad QRS regular



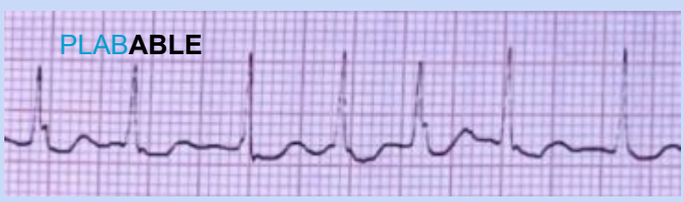
VF - Broad QRS - irregular deflections of varying amplitude



SVT - Narrow regular



AF - Narrow irregular



Adult Tachycardia

ABCDE approach

Shock/Syncope/MI/ Severe heart failure?

Yes?

DC shock

No

Look at QRS

QRS ≥ 0.12s (broad)

QRS < 0.12s (narrow)

Regular

Likely VT

- Treat with amiodarone IV

Irregular

Unlikely to be asked. One of the possibilities include torsades de pointes which is treated with magnesium

For full resus guidance click the heart

Regular

Likely SVT

- Vagal manoeuvres
- If fails → Adenosine x 3
- If fails → Verapamil or beta blockers

Irregular

Likely AF

- Treat with beta blockers
- If heart failure, consider digoxin or amiodarone
- If > 48 h → Anticoagulate

Hypokalaemia (K+ <3.5mM)

Causes

Increased loss:

- Vomiting and diarrhea
- Diuretics - Thiazides and loop diuretic
- Hyperaldosteronism
- Cushing’s syndrome
- Proximal renal tubular acidosis
- Magnesium deficiency

Redistribution into cells:

- Insulin
- Metabolic alkalosis

Oral treatment with KCl is commonly employed

ECG changes

- Broad flat T waves
- ST depression
- QT prolongation

Hyperkalaemia (K+ >5.5mM)

Causes

Shift from cells:

- Acidosis
- Tumour lysis

Inadequate excretion:

- Drugs- ACEi, ARBs, spironolactone
- CKD
- Primary adrenal insufficiency

Look out for questions with patients with **symptomatic hyperkalaemia** that ask for:

Most appropriate investigation
→ 12 lead ECG

Initial treatment
→ If ECG changes, pick calcium gluconate

Hyperkalaemia (K+ >5.5mM)

Mild (5.5 to 5.9 mmol/L)

If asymptomatic and in the absence of AKI:

- Manage in primary care
- Look for causative drug (e.g. ACEi/ARBs/spironolactone) to see if it can be changed if not, half the dose
- Repeat blood test in 3 days to 1 week

Moderate (6.0 to 6.4 mmol/L)

Perform ECG

- If ECG changes
  → Pick calcium gluconate
- If no ECG changes
  → Do not pick calcium gluconate

Severe (≥ 6.5 mmol/L)

Perform ECG

- If ECG changes
  → Pick calcium gluconate
- If no ECG changes
  → Pick calcium gluconate
  → *Repeat the sample and avoid haemolysis but treatment with calcium gluconate should not be delayed*

Hyperkalaemia (K+ >5.5mM)

All patients with K ≥ 6 mmol/L (i.e. moderate or severe hyperkalaemia) need an urgent 12 lead ECG

If ECG shows changes

- Start calcium gluconate (Most important)
- Perform a continuous 3 lead ECG monitoring

Start **continuous 3-lead ECG monitoring** if:

- Hyperkalaemic changes seen on 12 lead ECG OR
- Potassium ≥ 6.5 mmol/L

Hyperkalaemia (K+ >5.5mM)

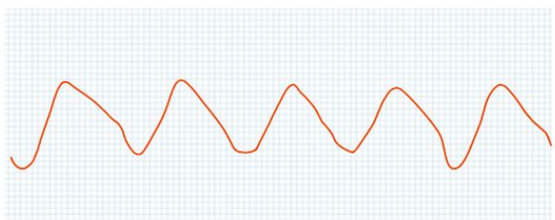
ECG changes

- Tall tented T wave
- Loss of P wave
- Widened QRS complex
- Sine wave pattern

Treatment

- **10% Calcium gluconate** slow IV over 2-3 min to prevent arrhythmias
- **IV insulin infusion** followed by dextrose to reduce K+ concentration

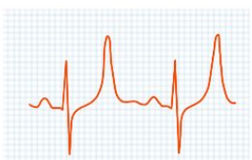
Hyperkalaemia (K+ >5.5mM)



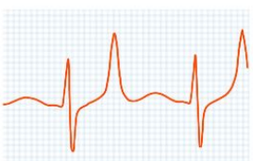
Sine Wave Pattern

Ventricular Fibrillation

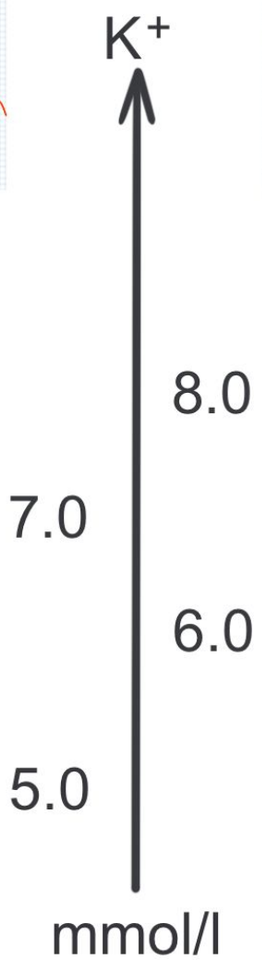
Wide QRS Complex



Tall Peaked T Waves



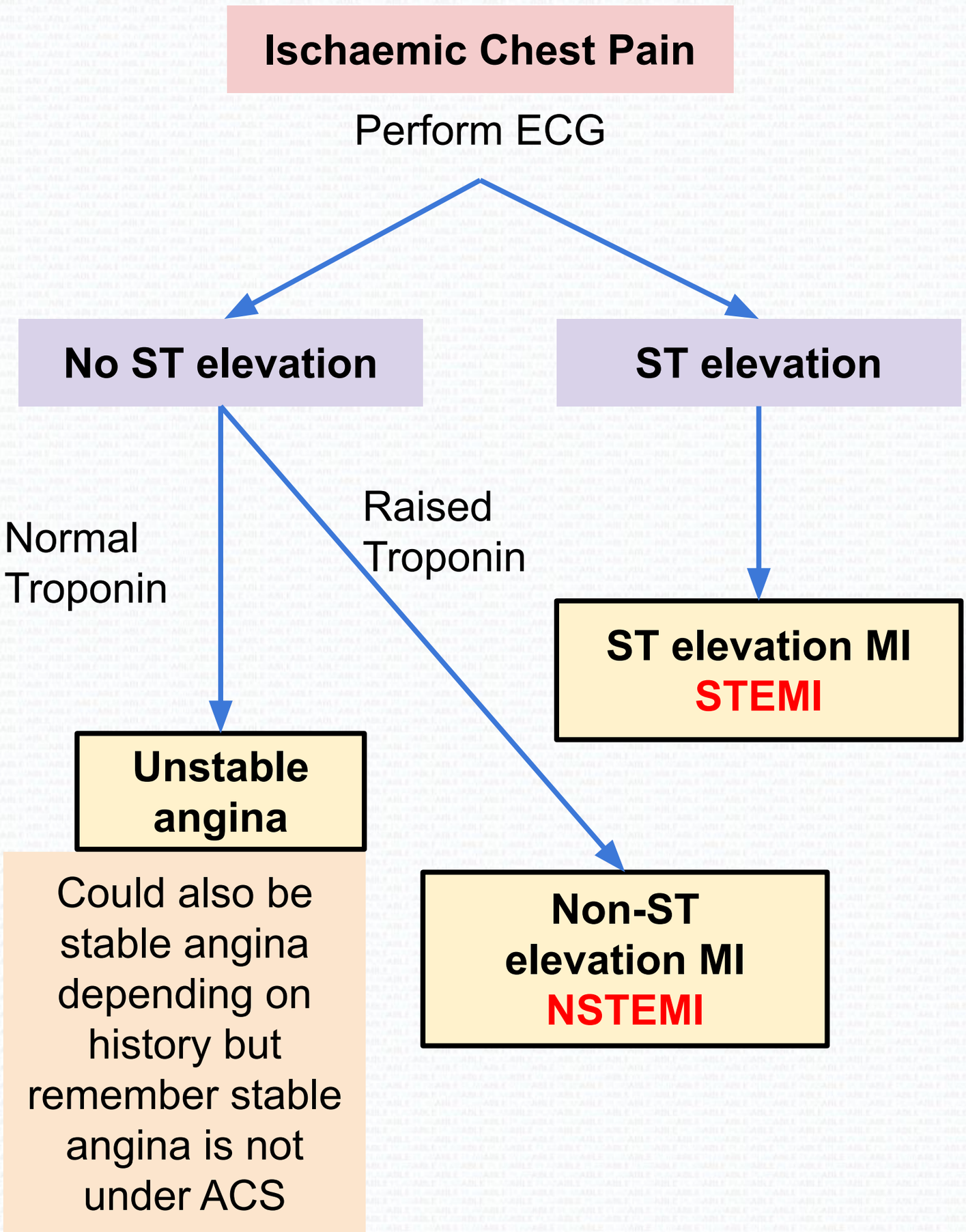
Loss of P Waves



PLABABLE

ECG Changes in Hyperkalemia

Acute Coronary Syndrome (ACS)



Acute Coronary Syndrome (ACS)

Ischemic chest pain:

- Left-sided, substernal or central
- Radiating to the left arm, jaw or shoulder
- **Silent MI** in diabetics without pain
- Sweating
- Hypotension (inferior wall MI)
- **Risk factors:**
  - Smoking
  - Family history
  - Elderly
  - Males
  - Diabetes mellitus
  - Hypertension

STEMI

ECG changes in various type of MI:

Inferior wall MI:

- ST elevation in leads II, III and aVF
- Coronary artery - RCA (80%) and LCX (20%)

Anterior wall MI:

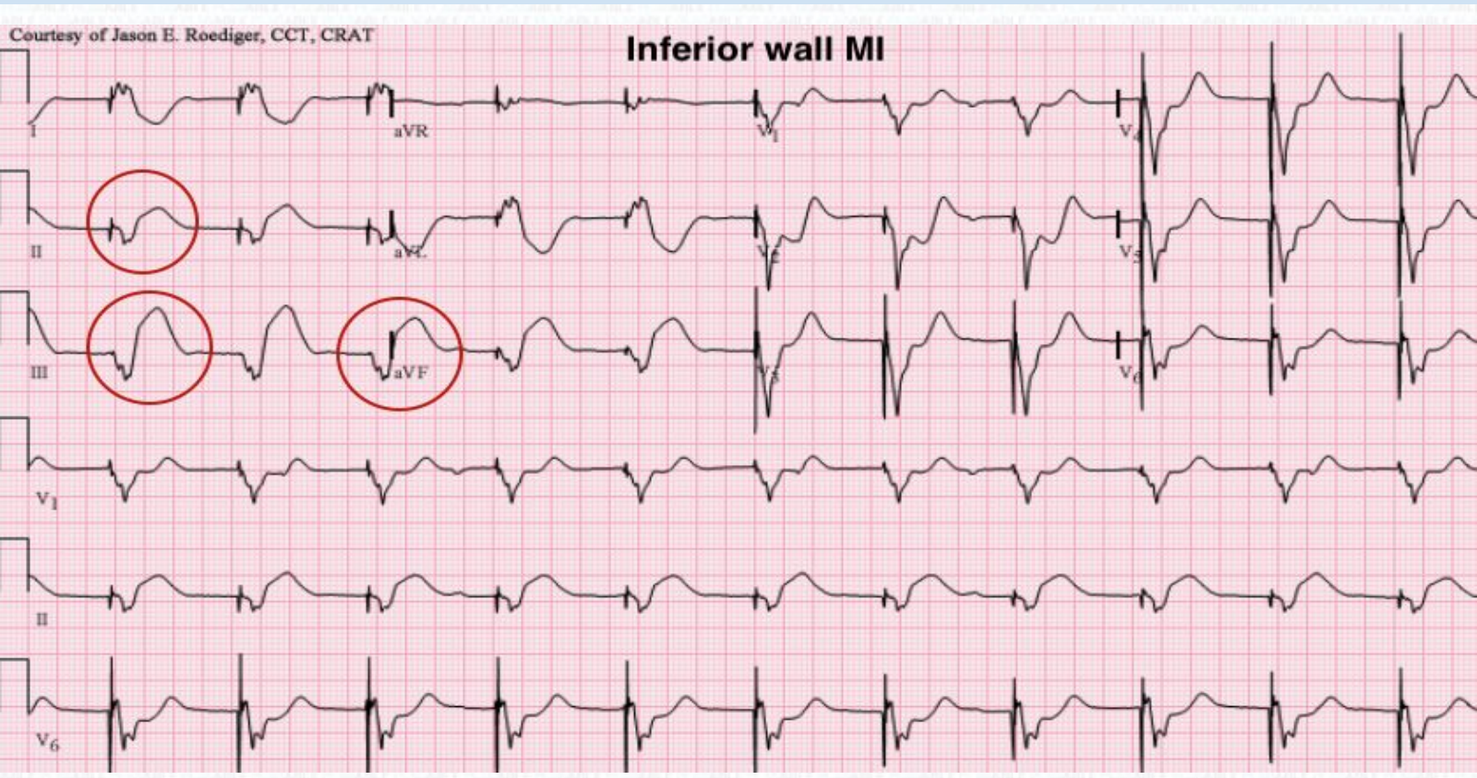
- ST elevation in leads V1 to V6
- Coronary artery - LAD

Lateral wall MI:

- ST elevation in leads 1, aVL, V5 and V6
- Coronary artery - LCX

Posterior wall MI:

- ST elevation in leads V7, V8 and V9
- Coronary artery - LCX ± RCA



STEMI

Immediate treatment: MONA

- **M** - Morphine
- **O** - Oxygen
- **N** - Nitroglycerin (avoided in low BP and inferior wall MI)
- **A** - Aspirin 300 mg

Definitive treatment

- **PCI -** Gold standard (If patient presents within 12 hrs after the onset of symptoms)
- **Thrombolysis** - If PCI is unavailable

Long term management

- Aspirin for life
- Ticagrelor or prasugrel or clopidogrel for 12 months
- Beta blockers for 12 months
- ACEi or ARBs for life
- Statins for life

NSTEMI

Immediate treatment

- Antiplatelet therapy
  → Aspirin
- Antithrombin therapy
  → Fondaparinux *(unless at high risk of bleeding or for immediate angio)*

Additional treatment *(unlikely to be asked)*

- If low risk of mortality
  → Ticagrelor
- If intermediate/higher risk of mortality
  → Prasugrel + Angiography (followed-on PCI if indicated) within 72 hours

Other important anti-ischaemic therapy

- Beta blockers
  → Can be started on presentation
- Statins
  → Can be started on acute presentation

NSTEMI

Brain trainer:

A 58 year old man has acute chest pain radiating to his left arm + features suggestive of ischaemic heart disease. His ECG is normal. What is the next step?

➔ **Measure troponin**

If troponin is high → NSTEMI

What is the next most appropriate management?

➔ **Give:**

- **Subcutaneous LMWH or fondaparinux**
- **+ aspirin**

Risk Factors

Brain trainer:

A 50 year old man drinks 8 units of alcohol per week, smokes 20 cigarettes a day, has a BMI of 27 and has a cholesterol of 4.2. What is the best advice for him?

➔ **Referral to smoking cessation clinic**

Myocardial Infarction

Brain trainer:

A 58 year old man has acute chest pain radiating to his left arm + features suggestive of ischaemic heart disease. His ECG shows ST elevation. What is the next most appropriate management?

➔ **Percutaneous coronary intervention**
➔ If not among the options then pick → **Alteplase**

Myocardial Infarction

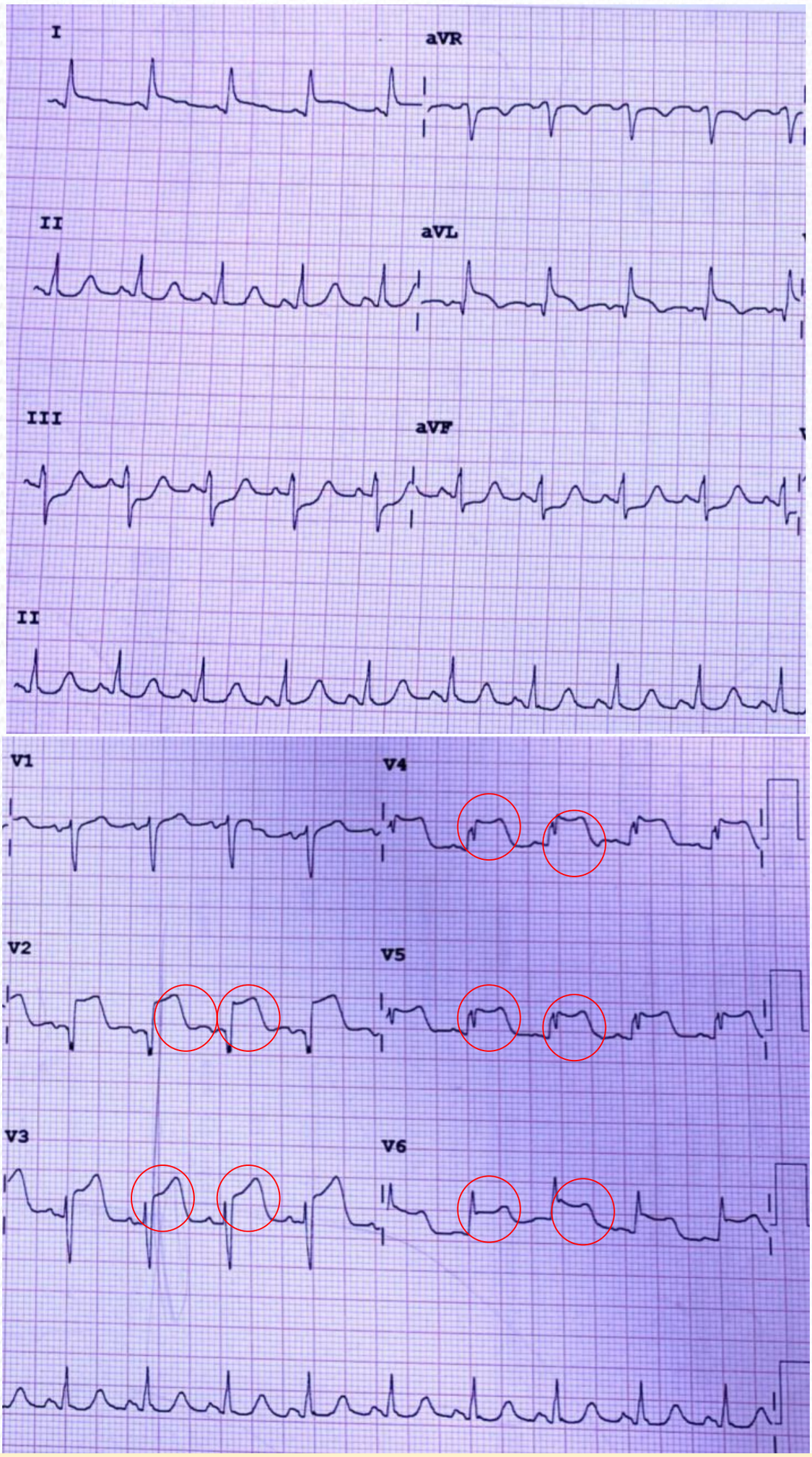
Analgesia while in ambulance

- GTN spray
- Morphine intravenously (or diamorphine)

Do not use opioids intramuscularly as

- Intramuscular absorption is slower and less reliable
- If the patient receives thrombolysis later on, the injection site will bleed

ECG



STEMI Anterior wall

ECG contributed by Dr Asim Ahmad

Complications Following MI

0-24 hours following MI:

- Ventricular fibrillation
- Heart failure

1-3 days following MI:

- Fibrinous pericarditis

1-2 weeks following MI:

- Ventricular free wall rupture causing cardiac tamponade
- **Papillary muscle** (posteromedial) **rupture** causing mitral regurgitation
- Interventricular septum rupture causing VSD - Pansystolic murmur
- Left ventricular **pseudoaneurysm**

2 weeks to months following MI:

- **Dressler syndrome** - Autoimmune pericarditis
  - Pleuritic chest pain
  - Fever
  - ECG: Widespread saddle shaped ST elevation
  - Rx: NSAIDs
- True ventricular aneurysm
- Chronic heart failure

Stable Angina Vs Unstable Angina

Stable Angina	Unstable Angina
Normal 12-lead ECG findings and normal troponins on A&E assessment. No evidence of myocardial necrosis.	
• Pain occurs after a constant level of exercise (e.g. jogging) • Relieved by rest • Pain last less than 20 minutes • Not considered part of acute coronary syndrome	• Pain that develops at rest *(most of the time)* OR mild exercise • Pain is either new *(not experience before)* or dramatically worse than previous episodes of angina • Pain last more than 20 minutes • Considered part of acute coronary syndrome

Some features worth remembering:
Ongoing pain even at rest, or change in quality, ECG changes → T wave inversion

Cardiac Chest Pain

Central chest pain around 15 minutes + while playing sports. Cardiac sounding chest pain resolves.

Normal ECG + Normal troponin which was taken 3 hours after symptom onset

Unlikely to be ACS!

Discharge + outpatient cardiology review

Remember, raised creatinine kinase does not always indicate MI!

It can be raised due to heavy exercise

Troponin Interpretation

The following apply for cardiac sounding chest pain in a patient with a normal ECG

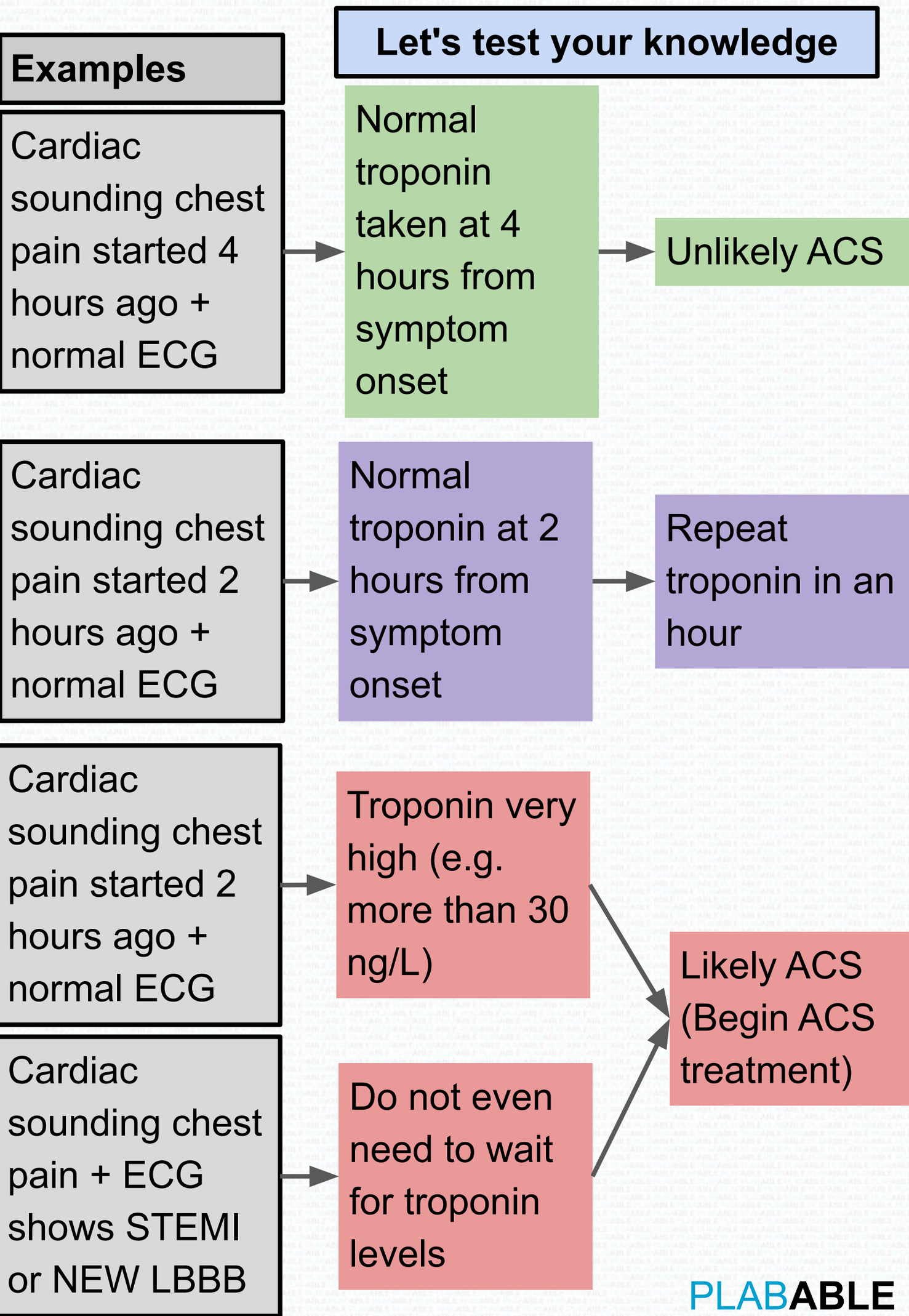
If symptoms onset **>** 3 hours ago → Normal troponin → Unlikely ACS

If symptom onset **<** 3 hours ago → Normal troponin → Repeat troponin (usually after 3 hours of chest pain onset + at least one hour from last troponin)

If symptom onset **<** 3 hours ago → Troponin very high (e.g. more than 30 ng/L) → Likely ACS (Begin ACS treatment)

Guidelines vary amongst trust but this is a general gist

Troponin Interpretation



Pulmonary Embolism

Symptoms

- Breathlessness
- Chest pain
- Calf pain or swelling (DVT)

Common cause: DVT

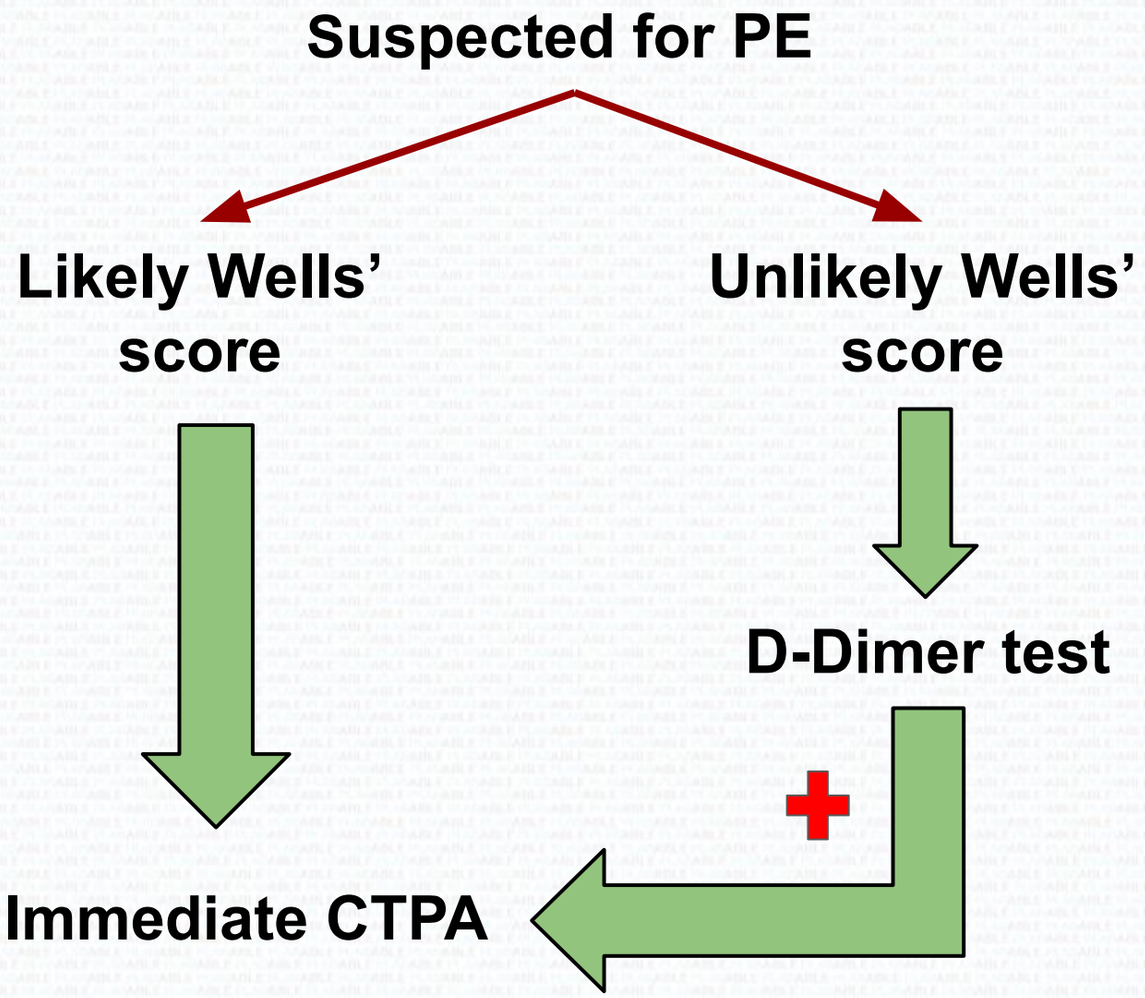
Risk factors

- Immobilization - Surgery, trauma, long-haul air travel, bedridden patients.
- Factor V Leiden and prothrombin gene mutation
- Smoking
- Cancer
- Oestrogen containing contraceptive pills

ECG: S1Q3T3 sign (specific for PE)

- S wave in lead I
- Q wave in lead III
- Inverted T wave in lead III

Pulmonary Embolism

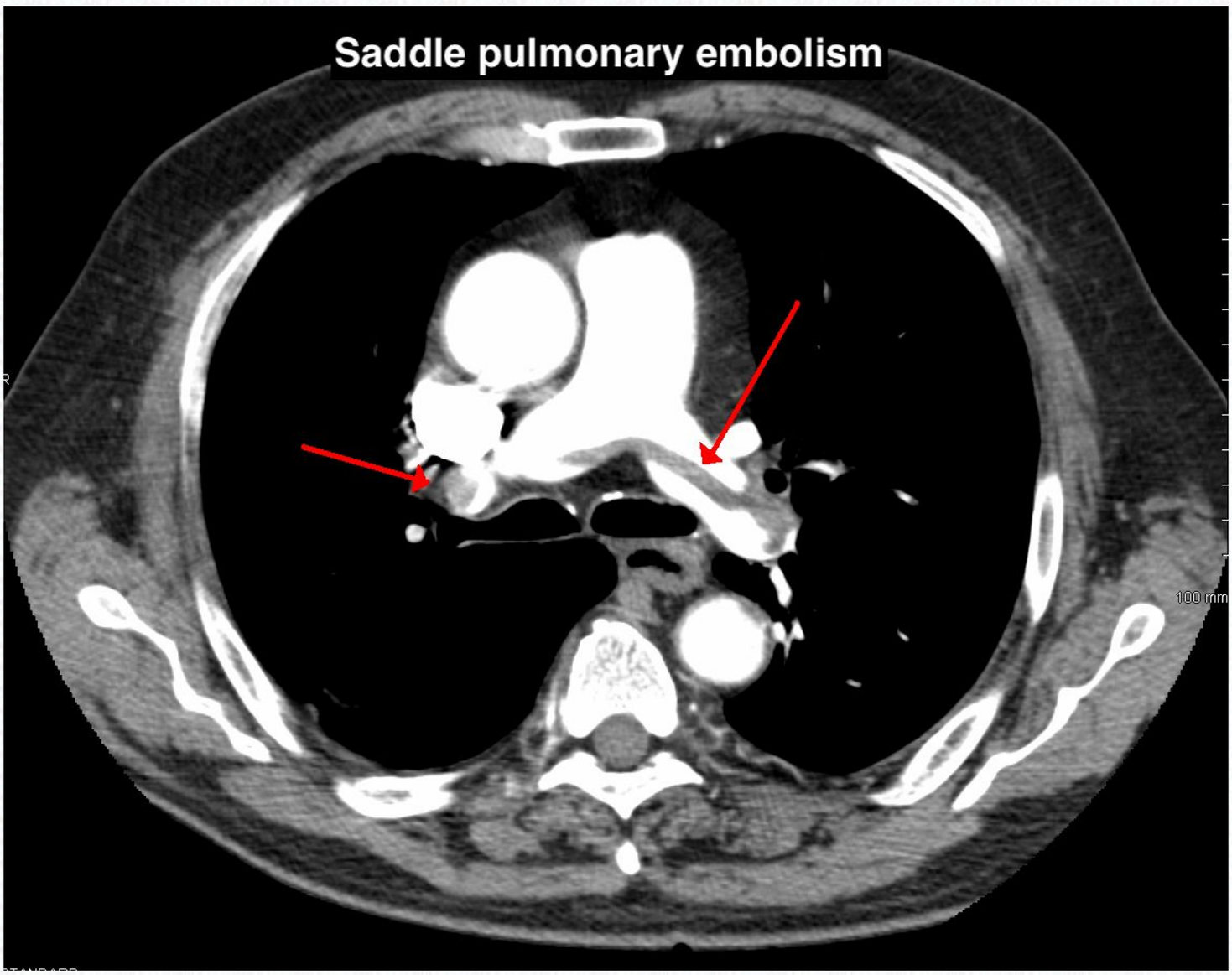


*CTPA - CT Pulmonary Angiography

- Allergy to contrast media
- Renal impairment
- High risk from irradiation (pregnancy)

Ventilation perfusion scan

Pulmonary Embolism



Treatment

- **Massive PE**: Fibrinolysis or pulmonary embolectomy
- **Small PE:** First line = Apixaban or Rivaroxaban
- Second line = LMWH 5 days → Dabigatran
- **Recurrent PE:** Vena caval filters for patients who have contraindication for anticoagulation

Heart Failure

Presentation

- Breathlessness
- Ankle swelling
- Raised JVP
- Pleural effusion
- Cardiomegaly

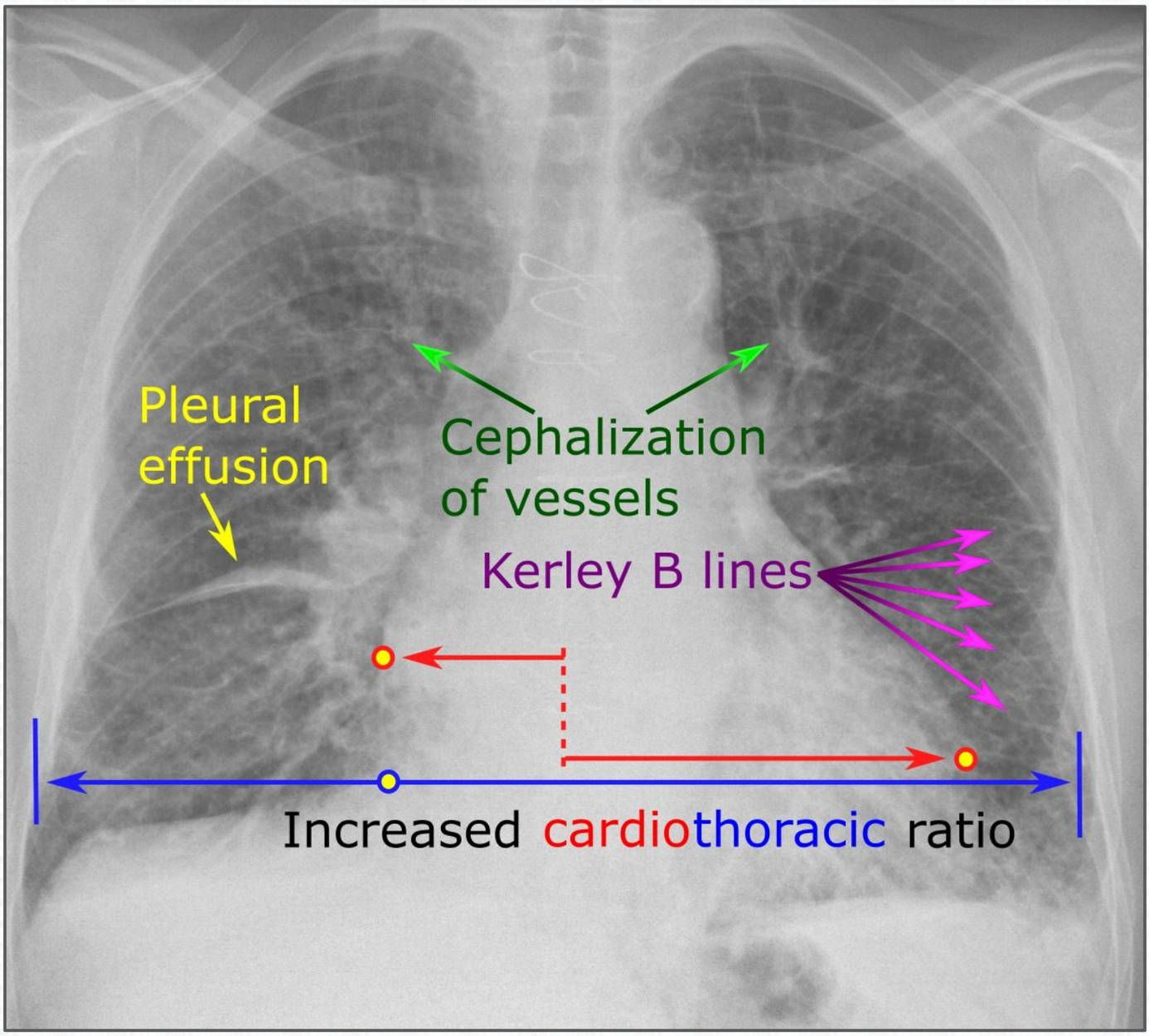
Causes

- Valvular heart diseases
- Myocardial infarction
- Cardiomyopathy
- Hypertension
- Alcohol

Management

- **Diuretics** (Loop - furosemide)
- To reduce mortality
  - ACEi or ARBs
  - Beta blockers
  - Aldosterone antagonist - spironolactone

Heart Failure



Atrial Myxoma

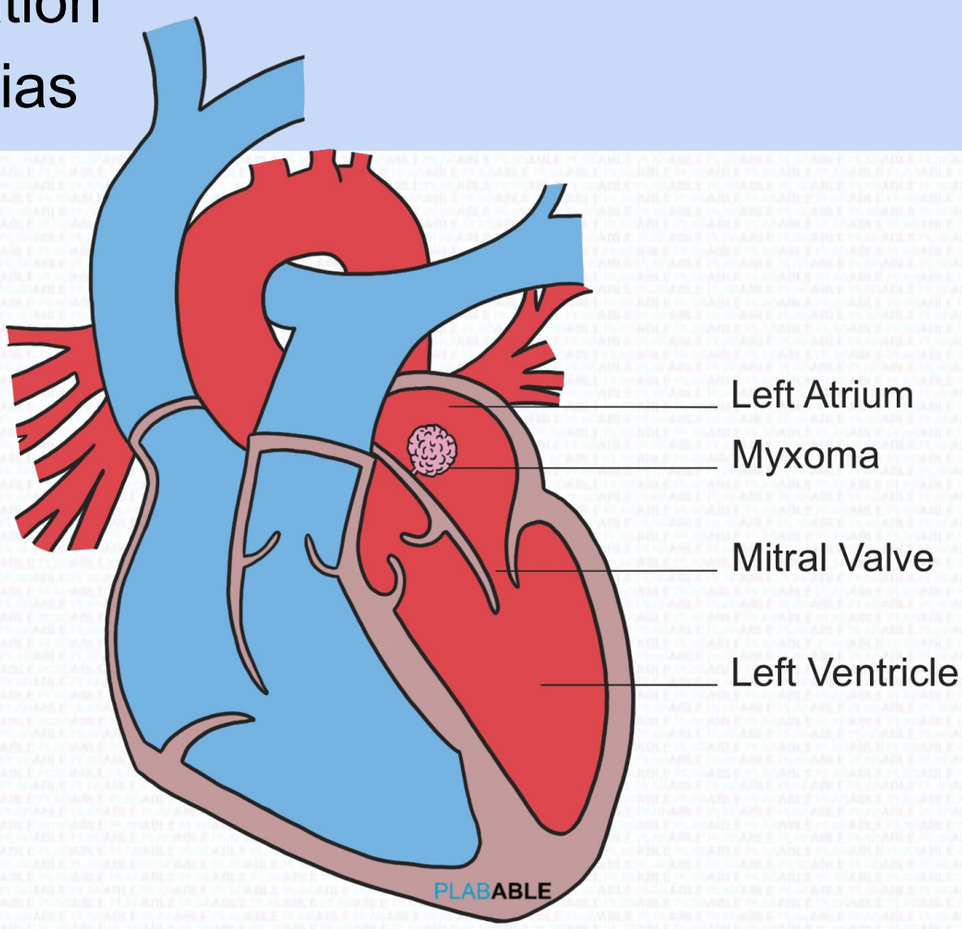
Features

- Benign tumour in the atrium
- Most common in the left atrium

Presentation

- Obstruction of mitral valve:
  - Loud S1
  - Mid-diastolic murmur
  - Dyspnoea
  - Syncope
  - Tumour plop sound on auscultation
- Embolisation
- Arrhythmias

Similar findings comparing with a mitral valve stenosis



Atrial Myxoma

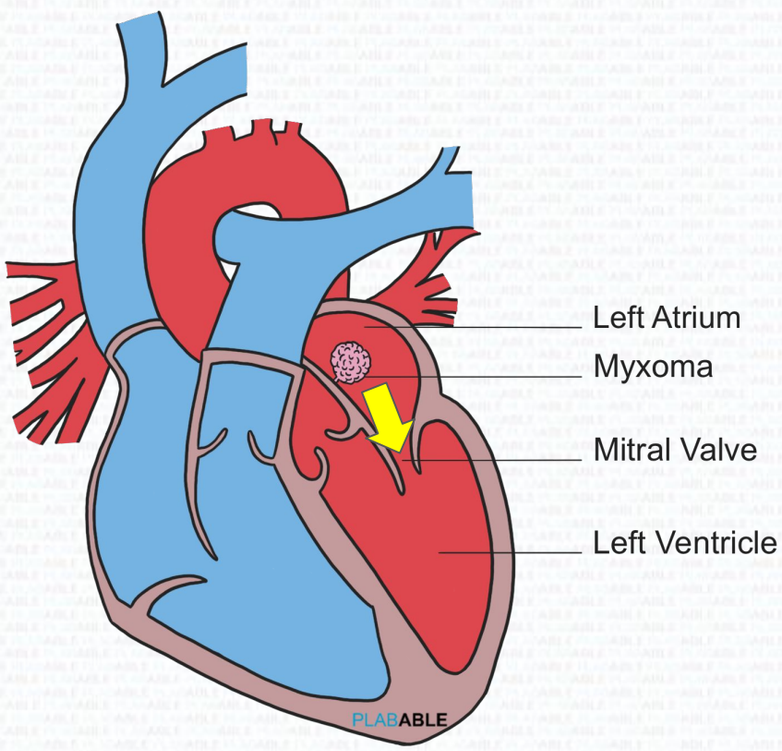
Atrial Myxoma

Investigation

- Echo - Gold standard

Management

- Surgical removal



Atrial Myxoma

If a piece of the myxoma dislodges and forms an emboli, it can result in a stroke or an acute limb ischaemia

⇒ **Embolectomy** would be the preferred choice to remove the embolus

Atrial Myxoma

Recap!
Remember the **3 BIG** features of atrial myxoma

Embolisation
(small piece of benign tumour dislodges)

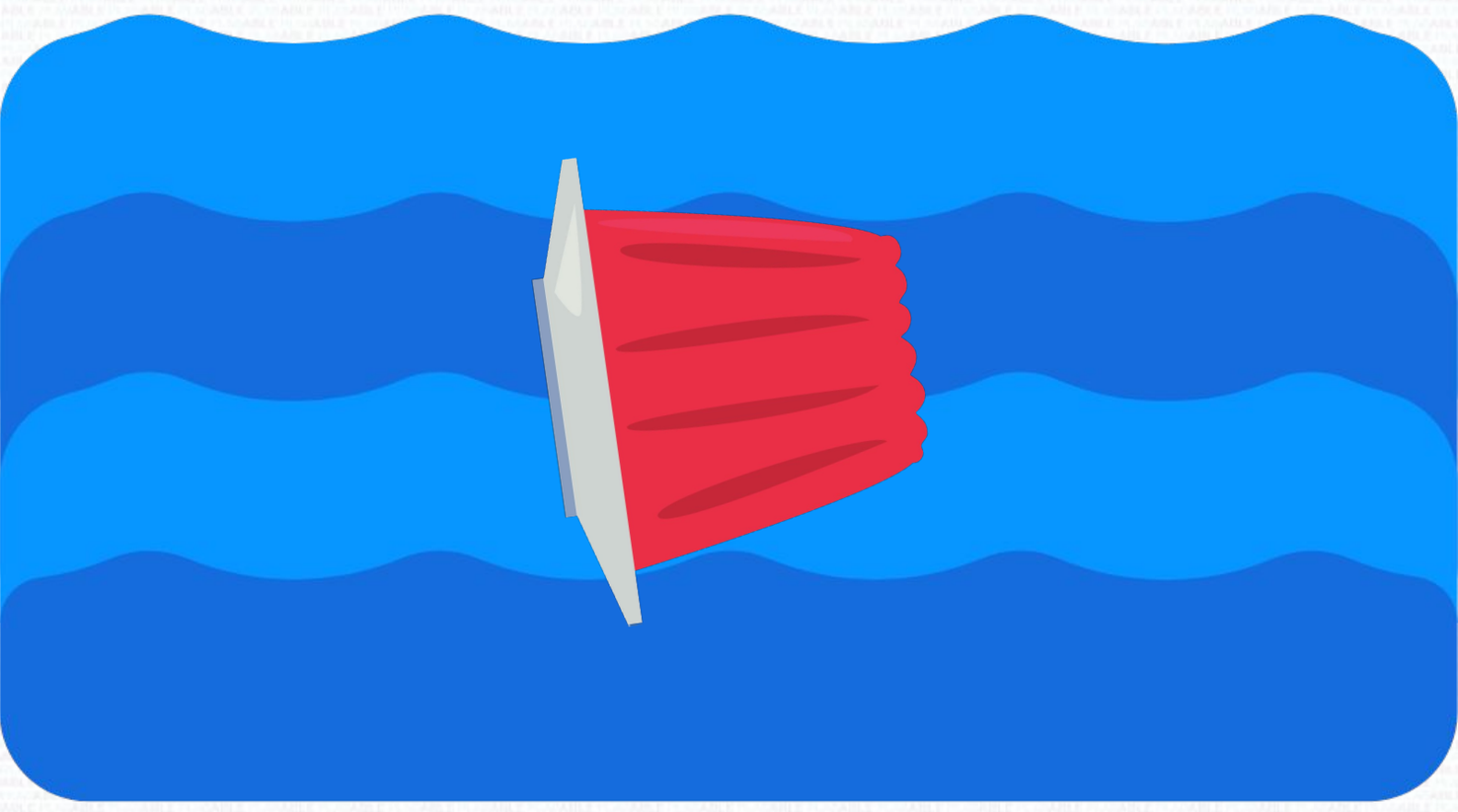
Obstruction
(similar findings to mitral stenosis)

Arrhythmia
(e.g. atrial fibrillation)

Atrial Myxoma

Platypnoea?

Another special feature worth remembering is platypnoea. Platypnoea is the difficulty breathing in the upright position with relief in the supine position. Think of an atrial myxoma as a floating jelly in the atrium. In a supine position, it floats and does not cause obstruction but in an upright position, gravity pulls the jelly substance down and blocks the output.



Pulmonary Oedema

Features

- Sudden onset of breathlessness
- Orthopnea
- Falling O2 saturation
- Auscultation Bilateral lower zone crepitations
- Tachycardia
- Commonly seen in heart failure

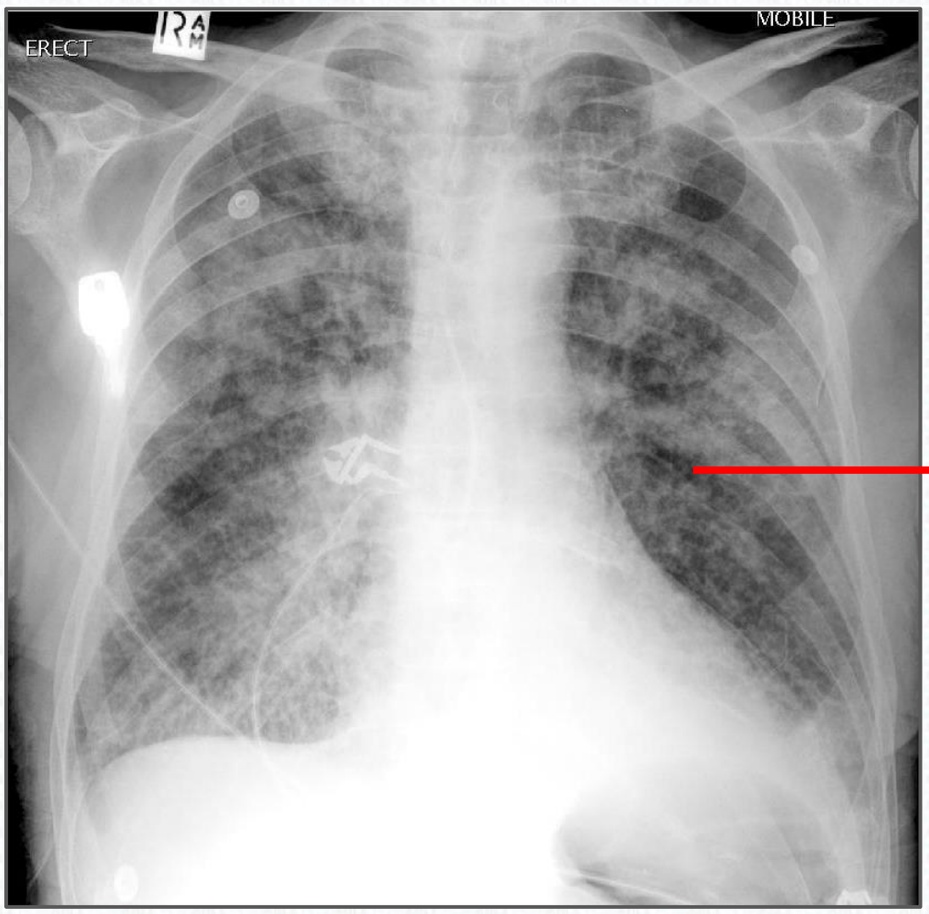
Investigation

- **X-ray chest** - Interstitial and alveolar oedema
- **Echo** - To find out the underlying cause

Management

- Oxygen
- Nitrates (reduce venous return to heart)
- IV furosemide

Pulmonary Oedema



If the echocardiogram shows an ejection fraction of less than 40% → heart failure

Treat with:

- ACEi
- Beta blockers

Use one at a time

If asthmatic, avoid beta blockers

Digoxin Toxicity

Features

- Nausea, vomiting
- Confusion
- Yellow haloes
- Arrhythmias

Investigation

- Digoxin levels

Management

- Digibind

Statins

When to offer a statin?

For primary prevention, offer 20 mg atorvastatin to any of the following:

- QRISK ≥10% in people aged ≤ 84 years
- Aged ≥ 85 years old
- Chronic kidney disease
- Familial hypercholesterolaemia
- Type 1 Diabetes *(only in specific circumstances → Unlikely to be asked in the exam)*

For secondary prevention, offer 80 mg atorvastatin to any of the following:

- Myocardial infarction
- Angina
- Stroke
- Transient ischaemic attack
- Peripheral arterial disease

Familial Hypercholesterolaemia

When to consider diagnosis?

Consider diagnosis in anyone with:

- **Total cholesterol of > 7.5 or**
- **Personal or family history of premature coronary heart disease (an event before 60 years in a first-degree relative)**

Statins

Brain trainer:

A 66 year old man attends the GP surgery for a routine medication review. He takes losartan for his high blood pressure. His last blood results for cholesterol show the following:

HDL cholesterol 2.0 mmol/L (0.9-1.93 mmol/L)
LDL cholesterol 2.2 mmol/L (< 2 mmol/L)
Triglycerides 1.5 mmol/L (0.55-1.90 mmol/L)
Total cholesterol 5.7 mmol/L < 5 mmol/L)

His QRISK3 is 9%.
What is the most appropriate management?

➔ **No statin required**

Even though his total cholesterol is high, his QRISK is less than 10% and his cholesterol is not high enough (>7.5) to consider familial hypercholesterolaemia. He does not need a statin.

Long QT syndrome

Brain trainer:

A 25 year old man has recurrent fainting episodes when he exercises. An ECG shows long QT syndrome. He has had a similar ECG finding when he was 10 years old. Which arrhythmia is associated with his condition?

➔ **Torsades de pointes**

Torsades de pointes is a type of ventricular tachyarrhythmia. It usually terminates spontaneously but may progress to ventricular fibrillation.

ACE Inhibitor Monitoring

Before initiation

Renal function blood test (which includes electrolytes) and blood pressure

1 to 2 weeks after initiation

Renal function blood test (which includes electrolytes) and blood pressure

The second blood test is done to ensure that hyperkalaemia or acute kidney injury does not develop. Should the patient's renal function deteriorate after initiation, the diagnosis of renal artery stenosis should be considered

Heart Failure Monitoring
Renal function or BNP

Do we do renal function or BNP for heart failure monitoring?

Renal function

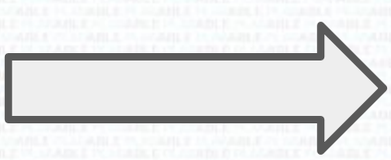
Part of the management of heart failure is starting an ACE inhibitor.

If we did, a renal function is done 1 to 2 weeks after initiation of ACE inhibitor. The renal function is not for heart failure monitoring, but it is for assessing kidney function after starting an ACE inhibitor.

BNP

NT-proBNP (N-terminal pro-B-type natriuretic peptide) has a limited role in monitoring heart failure. It is used on occasion by cardiologist as part of treatment optimization protocol in selected people. A NT-proBNP for **monitoring purposes** is unlikely to be the answer in your exam, as it is beyond what is expected for your level.

HF and recently started on ACE inhibitor



Pick renal function and NOT BNP

The 4 A's in Emergency Arrhythmias

Atropine → Bradycardia

Adenosine → SVT

Adrenaline → Asystole, VF, PEA

Amiodarone → VT, VF, AF

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