Advanced Rhythm Interpretation

A learning package for clinicians seeking a better understanding of electrocardiograph interpretation

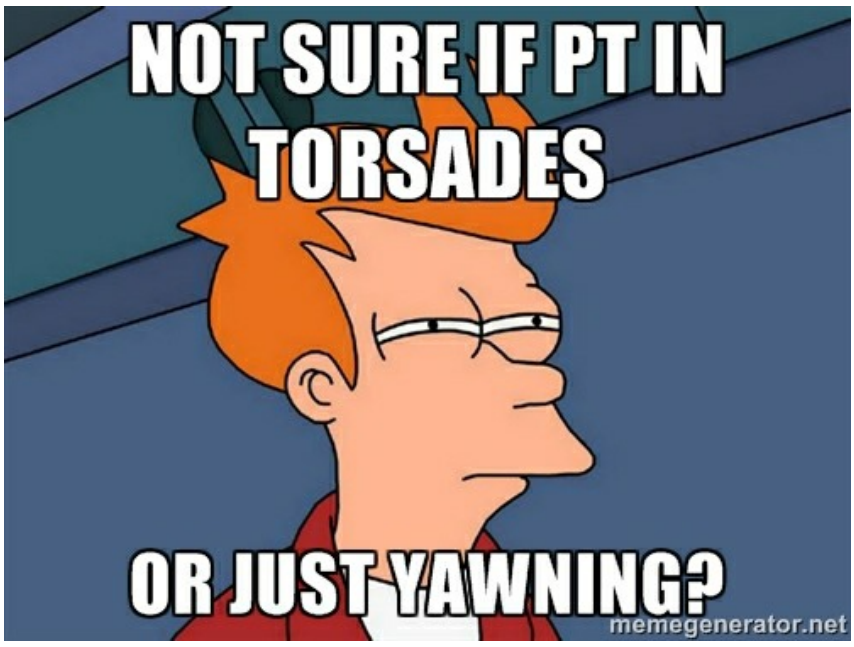
Jeremy Pallas

2014

Contents

What is an electrocardiogram?

- An electrocardiogram or ECG is a multi-focal recording of the electrical activity of the heart over a period of time (usually 10 seconds).
- This recording is taken by placing a number of electrodes across the chest and limbs and recording the impulses conducted to the skin at a number of different points following the standard flow of the electrical current in the myocardium.
- ECGs are minimally invasive and provide clinicians with a great deal of useful data which can be interpreted quickly to gauge the health of a patients heart.
- ECGs can also give us information about the function of other body systems such as pulmonary function, intracranial pressure and electrolyte balance.
- In the critical and acute care settings, urgent ECG's refer to those attended for patient's with chest pain, palpations or any other symptom suggestive of possible cardiac pathology or underlying circulatory compromise.
- Any urgent ECG should reviewed by a senior medical officer within 10 minutes!

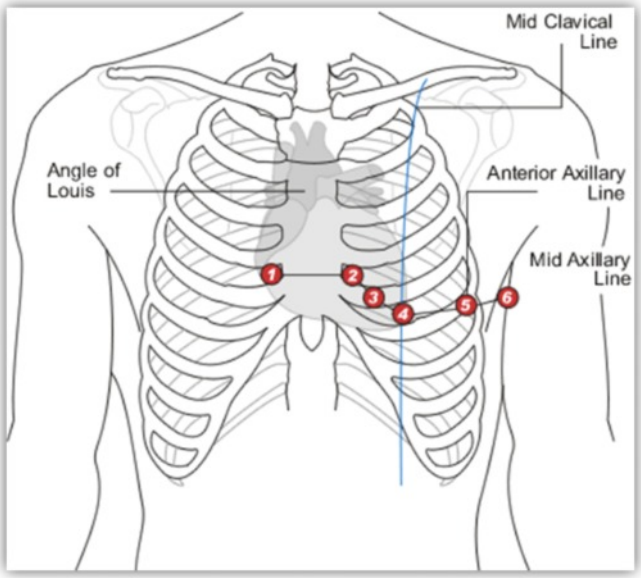


How is it done?

- First the process should be explained to the patient and verbal consent gained and documented.
- After positioning the patient, 10 small conductive electrodes are placed on the patients skin.
- 1 electrode is placed on each limb and the remaining 6 are positioned in a pattern following the anatomical angle of the heart from the upper right chest to the lower left chest as detailed in the picture below.
- Once all electrodes are in place, ECG leads are connected to the correct electrodes via clip closure.
- At this point the patient should be asked to stay as still as possible to facilitate best results.
- All ECG's should have the patient's full name, medical record number and date of birth entered as a minimum.
- When you are ready, the 'ECG' button can be pressed to automatically generate a printed 12 lead electrocardiograph.

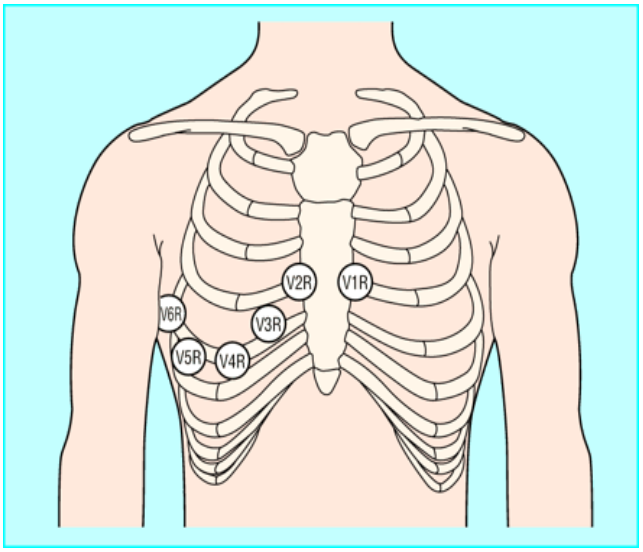
Correct chest lead positioning is essential for accurate interpretation

- V1 - Right sternal edge 4th intercostal space
- V2 - Left sternal edge 4th intercostal space
- V3 - Directly between V2 and V4
- V4 - Left mid-clavicular line 5th intercostal space
- V5 - Left anterior (front) axillary line 5th intercostal space
- V6 - Left mid-axillary line 5th intercostal space



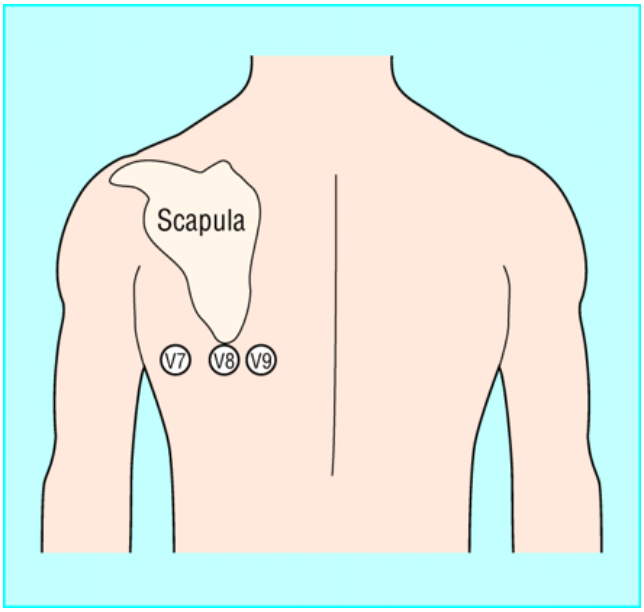
Right sided ECG

- Right sided ECGs may be required in a number of situations including the patient with dextrocardia (heart positioned in the right side of the chest) or suspected right ventricular infarct.
- Right ventricular infarct should be suspected if ST elevation is present in V1, greater in V1 then V2 or greater in lead III than lead II.
- A right sided ECG is attended by mirroring all chest leads to the right side of the chest with V1 to V6 running right to left rather than left to right.
- The placement of the limb leads remains the same for both right and left sided ECG's.
- All right sided ECGs should be annotated to ensure they can be differentiated from regular ECGs.
- On a right sided ECG, ST elevation in V4R is highly sensitive and specific for right ventricular infarct.



Posterior ECG

- Posterior ECGs are useful in allowing clinicians to view the back of the heart and are typically requested when posterior myocardial infarct is suspected.
- Posterior infarct should be suspected if horizontal or down sloping ST depression with tall upright T waves is visualised in V1-3 as this can potentially be a reciprocal change in posterior infarct.
- A posterior ECG is attended by moving V4-6 further around the same horizontal line towards the spine to form V7-9 as shown in the picture to the right.
- V9 is placed at the left spinal border, V8 at the mid scapular line and V7 at the posterior axillary line.
- V4's lead connects to V7, V5 to V8 and V6 to V9 while the remaining leads are attached as normal..
- V4-6 should be relabeled V7-9 on the ECG printout to ensure the posterior ECG can be differentiated from regular ECGs.
- The presence of ST elevation in V7-9 combined with reciprocal depression in V1-3 is highly specific for posterior infarct.



Handy hints

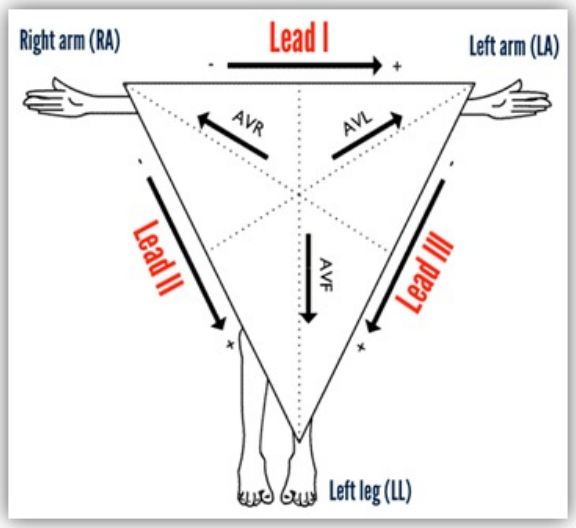
- Position yourself on the left side of the patient where possible – this will limit having to reach across the patient.
- Avoid positioning electrodes over bony prominences as this can impair electrical conduction
- Ensure skin is clean and dry and that excess hair is removed before placing electrodes as this will give you a better trace and save you time in the long run.
- Avoid metallic surfaces like bed rails and jewellery as these can effect conduction.
- Get the patient to lie still with their arms by their side as excessive movement, even talking can distort your ECG.
- Many different ECG machines are used throughout the health service, ensure you are familiar with the operation of those used in your area.
- Ensure patient details including name, birth date and MRN are entered for each reading as this allows for a follow up review of all ECG's by the cardiology department.
- Ensure patient's right to privacy by pulling curtains and providing gowns and blankets.

...What about the other 2 leads?

- Once you have completed your 12-lead ECG using the 10 available electrodes, the first step in interpreting your results is understanding how you ended up with 12 recorded leads and what each one represents!

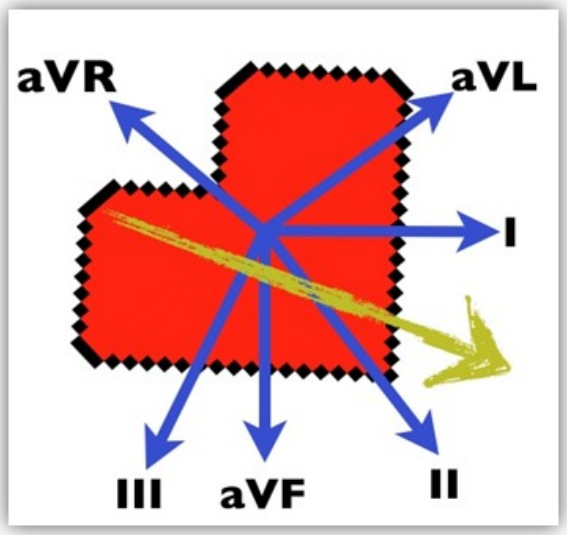
Limb leads

- When examining limb leads, our interpretation is based on Einthoven's triangle as seen on the right.
- Leads I, II and III are called 'limb' leads as they measure the voltage between 2 of the leads physically attached to the limbs.
  - Lead I – measures between LA and RA
  - Lead II – measures between LL and RA
  - Lead III – measures between LL and LA
- These leads are also referred to as bipolar leads because they combine the measurements of 2 separate leads.
- The physical lead RL is a neutral ground lead and is not represented as data on the ECG printout, however, without the ground lead in place, other leads may appear distorted!



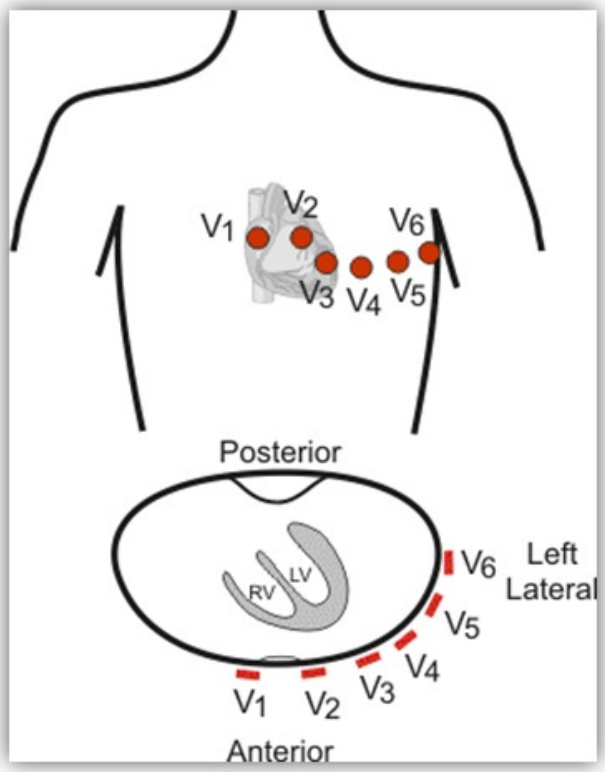
Augmented vectors

- Leads RA, LA and LL are also responsible for the 3 augmented vectors.
- Augmented vectors allow us to look at the heart's electrical circuit from different angles using the same three leads.
- These leads are unipolar as they focus on only one physical lead as viewed from the midpoint of the remaining 2.
  - aVR (augmented vector RIGHT) – shows RA from the midpoint of LL and LA
  - aVL (augmented vector LEFT) – shows LA from the midpoint of LL and RA
  - aVF (augmented vector FOOT) – shows LL from the midpoint of LA and RA
- In the picture to the right we can see how Einthoven's triangle follows the anatomical position of the heart. In a normally functioning heart, electrical current follows the yellow line producing a greater positive charges towards II and aVF at the bottom of the ventricles.
- It is important to remember that aVR is always negative (inverted p wave, t wave and negative QRS) as its view is in direct opposition to the flow of the measured electrical current.



Chest leads

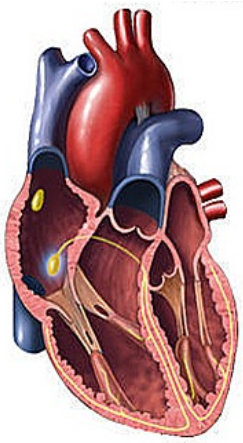
- Whilst the limb leads and augmented vectors record the heart's electrical current in the frontal plane, the precordial or chest leads allow us to visualise these impulses from a number of different points perpendicular to the frontal plane, sometimes referred to as the horizontal plane.
- These leads are unipolar, drawing their input data from a single electrode.
- These leads follow the path of the heart's conduction system from V1 which records activity close to the SA node, to V6 which focuses on the apex of the left ventricle.

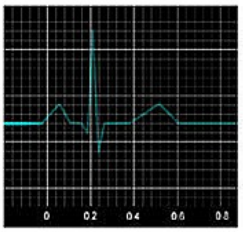


How does an ECG relate to a heart beat?

The next step in understanding and interpreting ECGs is being able to link the different phases of the ECG rhythm to the mechanical function of the heart.

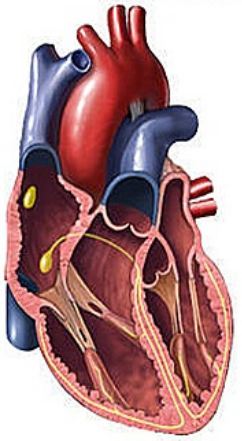
Rhythm vs contraction

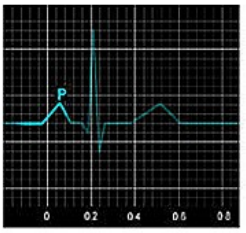




- Looking at normal sinus rhythm as a baseline, a grouping of cells at the top of the right atrium called the sinoatrial (SA) node produces an electrical impulse without any external innerv
ation by rapidly moving electrolytes (primarily sodium, potassium and calcium) across their cellular membrane.
- This process is referred to as automaticity.
- The resulting electrical pulse is then conducted through the myocardium causing muscular contraction or beating of the heart.

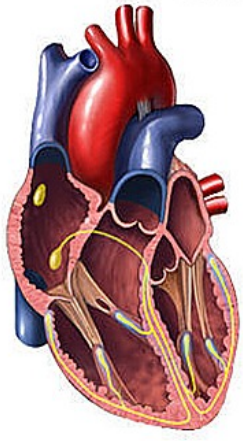
P wave

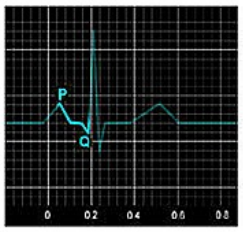




- Once the electrical impulse is generated by the SA node, it is then conducted through the muscle cells of the atrium to the atrioventricular (AV) node at the junction between the right atrium and ventricle.
- As soon as the impulse is generated by the SA node it also travels along the conduction pathway known as Bachmann's bundle which transports the electrical current through the left atrium.
- While this electrical pulse is moving through the atrium it stimulates the cells of the atrium to contract which is represented on an ECG as a P wave.

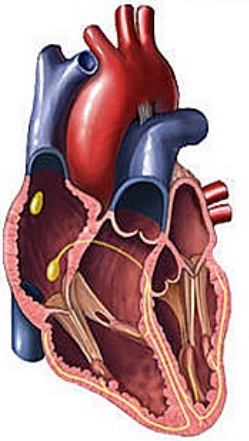
PR interval

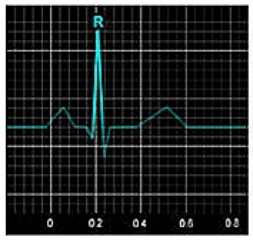




- Once at the AV node, the electrical pulse is then conveyed through the ventricular conduction system.
- The time the impulse takes to move through the AV node before entering the ventricular system correlates to the PR interval on an ECG.

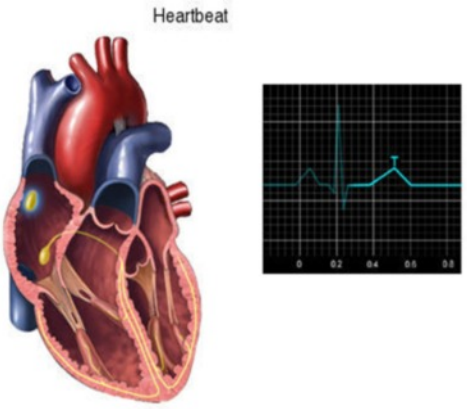
QRS complex





- The impulse then moves through the ventricular conduction system (through the bundle of HIS before separating down the left and right bundle branches and diffusing through the purkinje fibres) where it causes a strong contraction of the muscle fibres within the ventricle.
- This rapid depolarisation and contraction of the ventricles is represented by the QRS complex on the ECG.

T wave



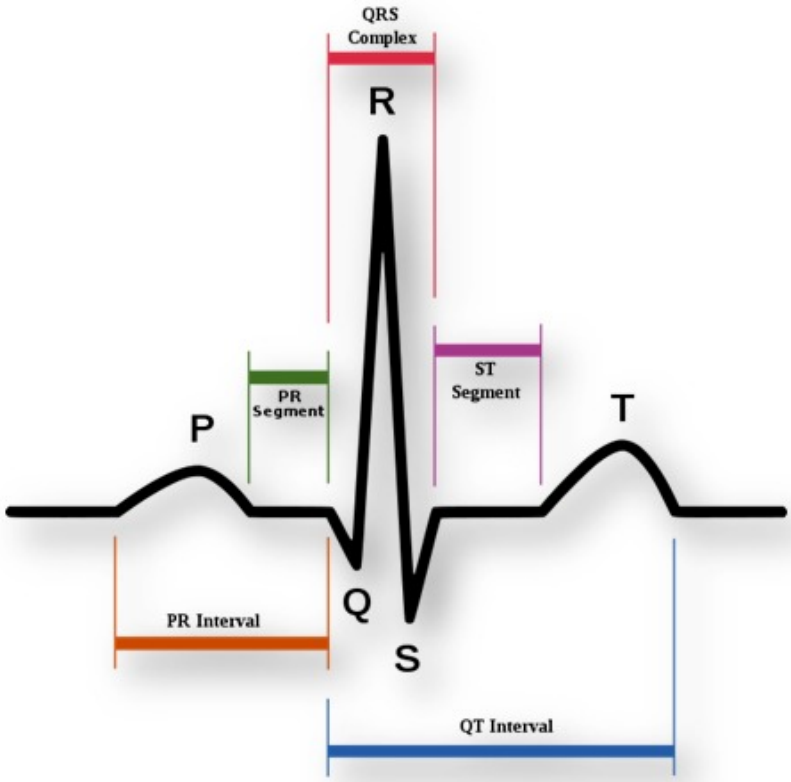
- Repolarisation of the ventricles after contraction is shown on the ECG as the t wave.
- In some cases the t wave may be followed immediately by a u wave which is believed to represent repolarisation of the intra-ventricular septum.

Interpretation process

- A systematic approach to ECG interpretation can make a daunting task more manageable while minimising the likelihood of missing important details.
- The first step is to identify the **rate**
  - Many ECG machines will give you an automatic interpretation of the rate
  - Alternatively, you can count the number of small squares between QRS complexes and divide 1500 by that number i.e. 22 small squares – 1500/22=68bpm
  - You can also count the number of large squares between the QRS complexes and divide 300 by that number i.e. 4.4 large squares – 300/4.4=68bpm
- The second step is to determine if the rhythm is **regular or irregular**
  - The easiest way to do this is to run a sheet of paper across the top of the QRS complexes on your ECG, using a pen mark the location of the first and second QRS complexes. Now move the paper to the right so that the mark from the first complex is over the second complex. If the rhythm is regular the mark from the second complex will now be over the third. This should apply for any complex you move your marker to.
- The third step is to identify whether all **features of the waveform** are present
  - P wave: is there a p wave? Is it pointing the right way?
  - QRS complex: is the QRS wide or narrow?
  - T wave: are the t waves pointing the right direction? Are they abnormally large?
- The fourth step involves identifying whether different **intervals and features are normal.** This step is often the most difficult but helps us to identify and differentiate between various conditions.

Landmarks and intervals

- **PR interval:** beginning of p wave to beginning of QRS complex. Represents the conduction of an electrical current from the SA node through the AV node.
- **PR segment:** from termination of p wave to beginning of QRS complex. Represents the movement of electrical current from the AV node to the bundle of HIS and into the ventricles. Usually remains flat along the isoelectric line.
- **P-P interval:** beginning of one p wave to the beginning of the following p wave. Represents the time taken between the commencement of separate heart contractions.
- **QRS complex:** beginning of q wave to the termination of the s wave. Represents rapid depolarisation and subsequent contraction of the ventricles.
- **QT interval:** beginning of QRS complex to termination of t wave.
- **J point:** the point at which the QRS complex terminates and returns to the isoelectric line. Represents the time between ventricular depolarisation and repolarisation.
- **ST segment:** from termination of QRS to beginning of t wave. Represents the period during which the ventricles remain depolarised before repolarisation occurs.



Left Vs right axis deviation

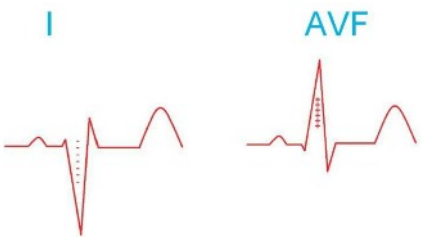
- The hearts electrical axis refers to the average direction that the depolarisation wave moves through the heart with each beat.
- If a greater amount of effort or energy is present on one side of the heart, the electrical axis will reflect this by deviating towards the side of greater activity.
- Similarly, if an abnormally low amount of electrical activity is present on one side of the heart (i.e. necrotic infarcted cells which are unable to conduct an electrical current), the depolarisation wave will be weighted towards the unaffected side. This will be represented by deviation of the electrical axis away from the injury.
- Generally the walls of the left ventricle are thicker than the right meaning that in the average heart the depolarisation wave will be weighted slightly towards the left due to the higher number of myocardial cells being recruited. For this reason the average heart will have a slightly leftward facing axis.
- If the axis deviates further towards the left than normal, this is referred to as left axis deviation. This can occur for a number of reasons including; *LBBB, right sided (inferior) myocardial infarction, left ventricular hypertrophy.*
- If the axis deviates further to the right than normal, this is referred to as right axis deviation. This can occur for a number of reasons including: *RBBB, left sided (lateral) myocardial infarction, right ventricular hypertrophy, right heart strain (as in pulmonary embolism and COPD).*
- While electrical axis deviation alone can be a normal variant in some people, it can be helpful to direct investigation and diagnosis for patients with different clinical presentations.
- The easiest way of determining the axis of a heart on an ECG is to compare the direction of the QRS complexes in lead I and aVF.

1. In a **normal axis** both lead I and aVF will have positive (pointing upwards) QRS complexes.
2. In **left axis deviation** lead I will have a positive QRS and aVF will have a negative (pointing downwards) QRS.
3. In **right axis deviation** lead I will have a negative QRS and aVF will have a positive QRS.
4. In **extreme right or left axis deviation** both lead I and aVF will have negative QRS complexes.

QRS deflection		Axis
Lead 1	aVF	
Positive	Positive	Normal
Positive	Negative	LAD
Negative	Positive	RAD
Negative	Negative	Extreme RAD or Extreme LAD

This table shows the different features of possible axis deviations

Left axis deviation



Right axis deviation

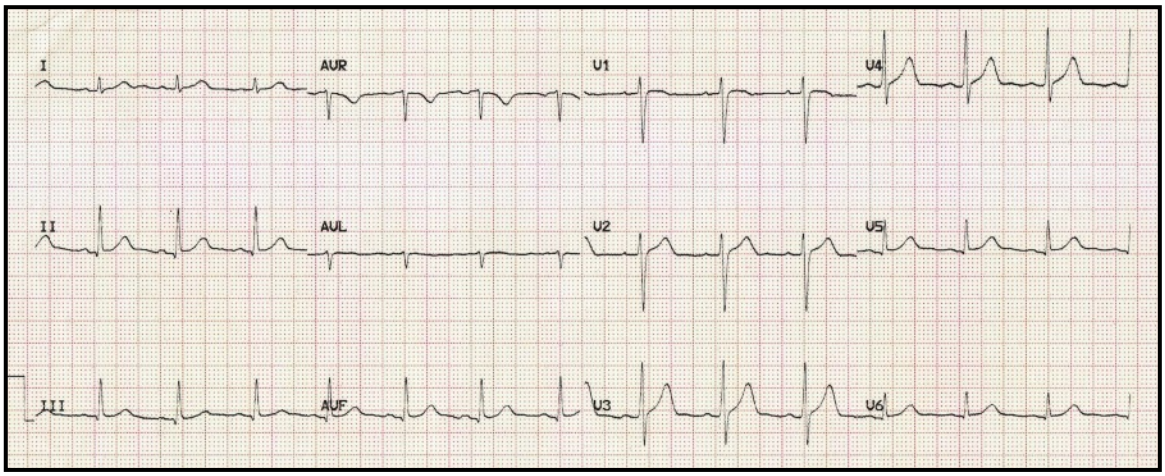
Extreme right or left axis deviation

Normal sinus rhythm

- If you only take one thing away from this package it should be the importance of being able to identify normal sinus rhythm!
- Normal sinus rhythm (NSR) represents the functional contraction of the myocardium as stimulated by an electrical impulse generated in the SA node which moves through the atrium, AV node and ventricular conduction system without interruption.
- An ECG of NSR contains:
  - A single 'P' wave which is upright in leads I, II and is inverted in aVR,
  - A single narrow 'QRS' complex
  - A 'T' wave
  - NSR has a regular rate of 60-100 beats per minute.
- If you can identify sinus rhythm, you will be able to identify when something looks irregular and requires further interpretation.

Interventions required

- Look at the patient to ensure the rhythm matches their clinical disposition.
- Palpate pulses to ensure rhythm is generating sufficient output.
- Regular observations or as otherwise clinically indicated.
- If the patient reports persistant chest pain, monitor vital signs more frequently and connect patient to continuous cardiac monitoring.
- For initial and ongoing management of chest pain refer to the NSW chest pain pathway.



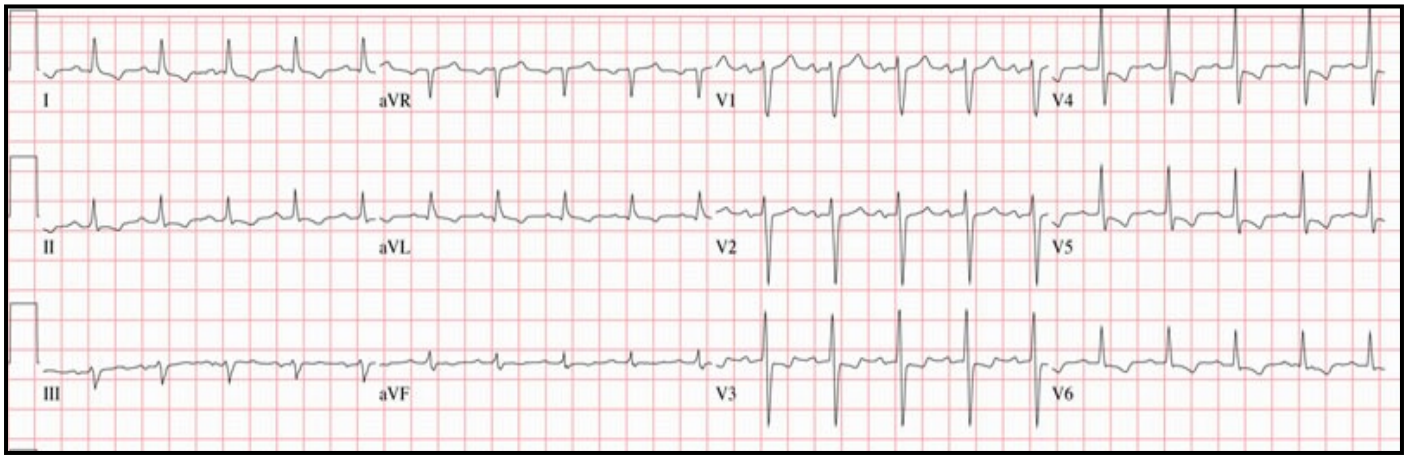
Normal sinus rhythm - rate 86 bpm, regular rhythm, p waves present (upright in I, II and inverted in aVR), QRS complex narrow, t waves present.

Sinus tachycardia

- Sinus tachycardia refers to a normal sinus rhythm with a ventricular rate in excess of 100 beats per minute
- Sinus tachycardia involves an electrical pulse being generated by the SA node, transmitted through the atria to the AV node then dispersing through the ventricular conduction system.
- Tachycardia is a normal physiological response to increased oxygen demand during exercise but is considered abnormal in the resting patient.
- Tachycardia at rest may be triggered by a number of underlying pathologies causing either reduced oxygen supply (respiratory problems, hypovolemia) or increased oxygen demand (sepsis, fever, tissue injury).
- In these cases the heart must pump faster to increase the circulation of oxygenated blood to meet the requirements of the body.
- ECG findings in sinus tachycardia are identical to sinus rhythm with the exception of an increased heart rate.

Interventions Required

- Palpate pulses to ensure rhythm is generating sufficient output.
- Regular observations or as otherwise clinically indicated.
- Connect patient to continuous cardiac monitoring.
- Assess for possible causes of tachycardia i.e. Bleeding, hypoxia, sepsis.
- If rate exceeds 140 beats per minute urgent medical review is required.
- Establish IV access and collect blood for pathology including a blood gas to quickly assess electrolytes.
- For initial and ongoing management of chest pain refer to the NSW chest pain pathway.



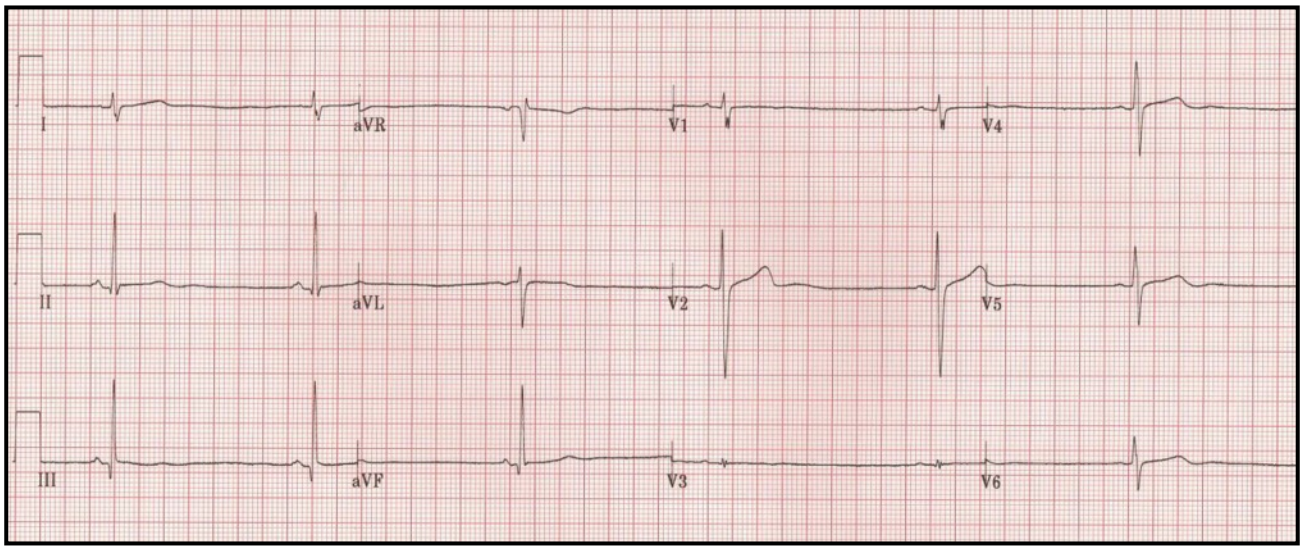
Sinus tachycardia - heart rate 125 bpm, regular rhythm, p waves present, QRS narrow, T waves present (inverted in leads I, II, V4-6), ST depression V3-5

Sinus bradycardia

- Sinus bradycardia refers to a normal sinus rhythm with a heart rate lower than 60 beats per minute.
- Sinus bradycardia involves an electrical pulse being generated by the SA node, transmitted through the atria to the AV node then dispersing through the ventricular conduction system.
- Sinus bradycardia may be a normal variant in athletic patients, sleeping patients or those taking particular medications i.e. Beta blockers, amiodarone etc.
- It may also indicate pathological processes such as: hypothermia, hyperkalemia, hypermagnesaemia, hypothyroidism, brainstem herniation (Cushing's reflex) or myocarditis.
- May be caused by SA node dysfunction and can be difficult to distinguish from heart block.
- ECG findings in sinus bradycardia are identical to sinus rhythm with the exception of a decreased heart rate.
- Occasionally small U waves may be distinguishable following T waves in sinus bradycardia which are thought to indicate repolarisation of the purkinje fibres.

Interventions Required

- Palpate pulses to ensure rhythm is generating sufficient output.
- Regular observation as otherwise clinically indicated.
- Connect patient to continuous cardiac monitoring.
- If rate below 40 beats per minute urgent medical review is required.
- Establish IV access and collect blood for pathology including a blood gas to quickly assess electrlytes.
- For initial and ongoing management of chest pain refer to the NSW chest pain pathway.



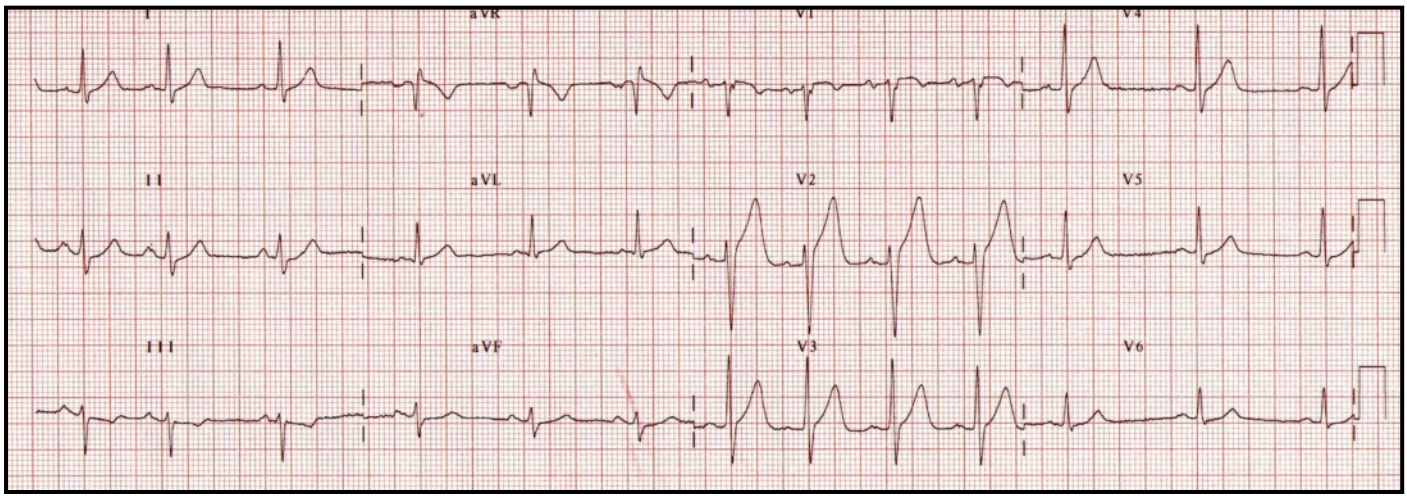
Sinus bradycardia - heart rate 35 bpm, regular rhythm, p waves present, QRS narrow, T waves present, U waves present in chest leads.

Sinus arrhythmia

- A sinus arrhythmia is a sinus rhythm with a beat to beat variation in p-p interval which results in an irregular ventricular rate.
- Sinus arrhythmia is a normal phenomenon in younger people caused by reflexive changes in vagal tone which occur during inspiration and expiration.
- Sinus arrhythmia is characterised by:
  - Variation in the p-p interval of more than 120 milliseconds (3 small boxes)
  - A p-p interval which lengthens and shortens in a cyclic nature typically coinciding with the patient's respiratory cycle
  - Normal, symmetrical, upright p waves in leads I, II and inverted p wave in aVR
  - Constant p-r interval

Interventions required:

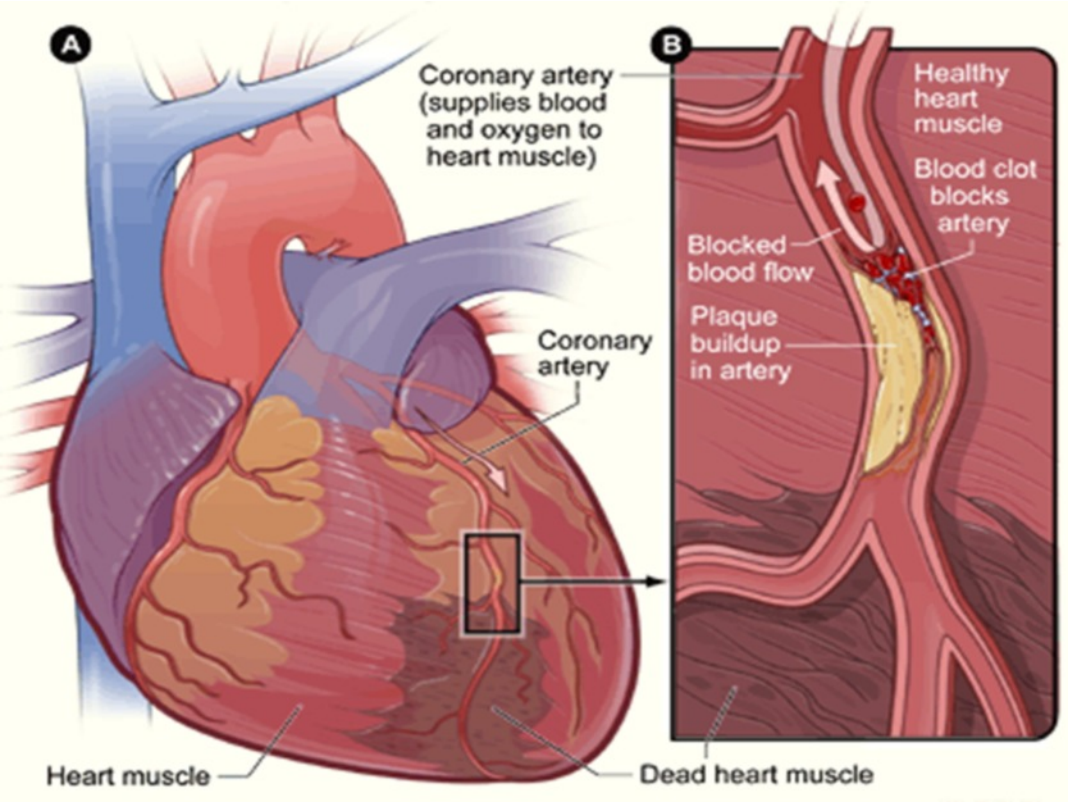
- If patient is assymptomatic then regular observation as clinically indicated.
- If patient reports pain, giddiness or any other concerning symptom increase frequency of vital sign observation and connect patient to continuous cardiac monitoring.
- If symptomatic, establish IV access and collect blood for pathology including a blood gas to quickly assess electrolytes.
- For initial and ongoing management of chest pain refer to the NSW chest pain pathway.



Sinus arrhythmia - heart rate 78 bpm, irregular rhythm, large variance between p-p intervals (rate varies from 50-100 bpm), p waves present, QRS narrow, t waves present

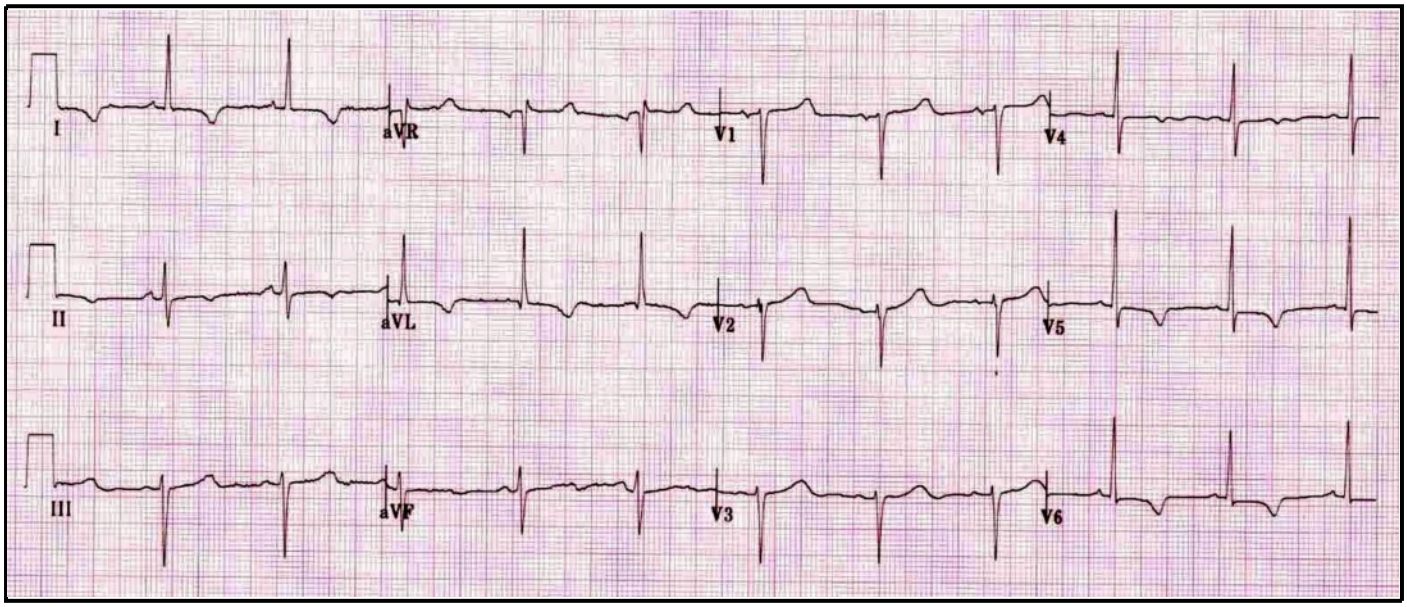
Myocardial infarction

- Certain ECG features allow us to detect myocardial ischemia in the early, intermediate and late stages.
- The tern myocardial infarction refers to cellular necrosis of the myocardium resulting from restricted blood flow causing tissue hypoxia.
- Cellular necrosis inhibits the ability of the myocardium to conduct electrical impulses or physically contract which can in turn cause:
- Lethal arrhythmias
- Insufficient cardiac output
- Insufficient organ perfusion
- Death!
- When the cells of the myocardium are damaged it can alter the way they conduct electrical impulses
- The timing, location and pattern of these abnormalities can allow us to determine the location and severity of an ischemic injury to the myocardium.

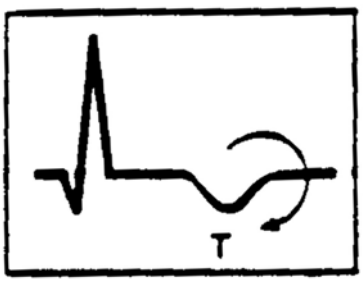


T wave inversion

- T waves signify the period of ventricular repolarisation.
- T wave inversion is a normal variant in V1, and III and should always be present in aVR.
- *Ischemia* causes potassium ions to be lost from myocardial cells which causes abnormal or delayed ventricular repolarisation which translates to T wave inversion.
- New or dynamic T wave inversion is generally associated with acute ischemia or cardiac strain and should always be considered abnormal, whereas prolonged inversion can remain indefinitely following an old or subacute infarction.
- T wave inversion related to myocardial ischemia most commonly occurs in **contiguous leads (leads looking at the same area of the heart).**
- T wave inversion can also be related to sudden rises in intercranial pressure, electrolyte imbalance and pulmonary embolism.

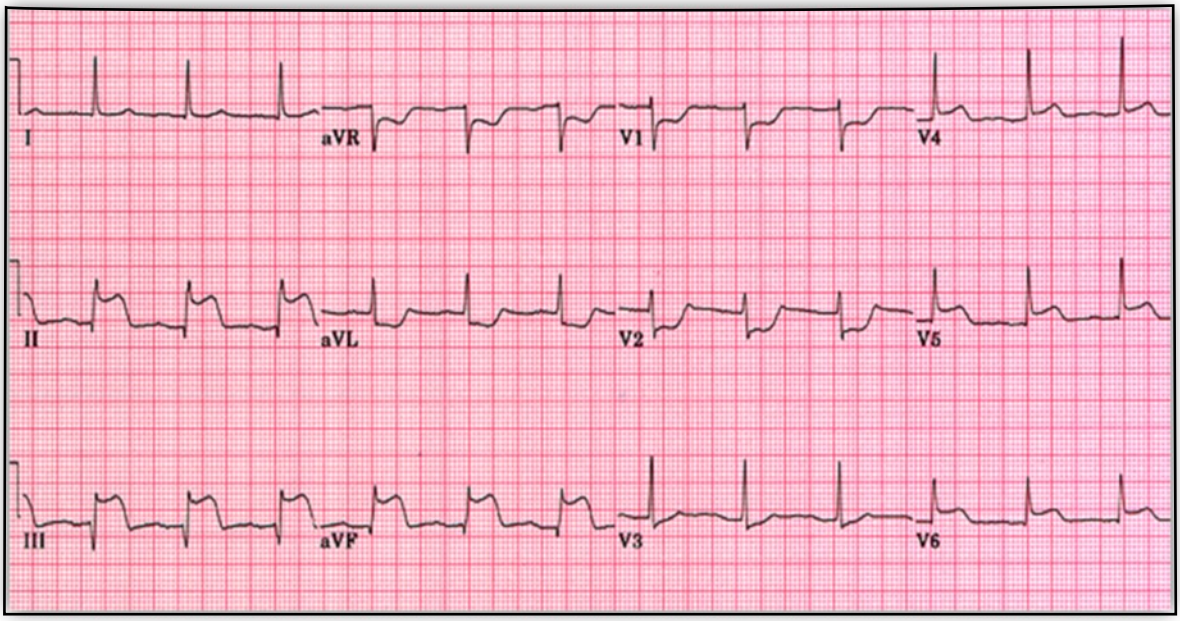


Widespread T wave inversion - HR 66, regular, sinus rhythm, T waves inverted in leads I, II, aVL and V4-6

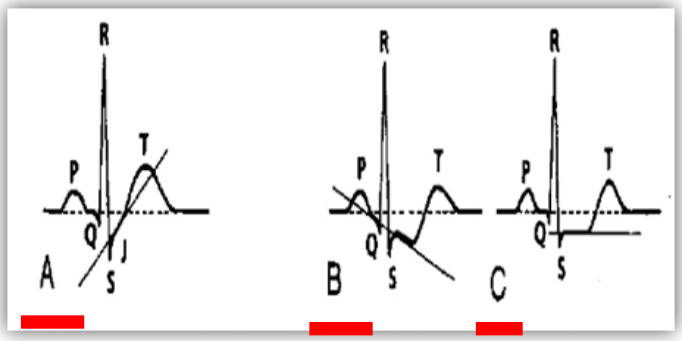


ST depression

- ST depression reffers to a prolonged negative phase where the S wave at the end of the QRS complex remains below the isoelectric line at the J point.
- As the ST segment represents the end of ventricvular systolic depolarisation, ST changes indicate abnormal or dysfunctional ventricular de and repolarisation.
- ST depression can be up-sloping (A), down-sloping (B) or horizontal (C) as per the picture below.
- Horizontal or down-sloping ST depression 0.5mm or greater at the J-point in 2 or more contiguous leads is consistent with myocardial ischemia.
- Up-sloping ST depression is typically not specific for myocardial ischemia.
- Reciprocal ST depression refers to a depression of the ST segment in the leads opposite those effected in an ST elevation myocardial infarction.
- This is thought to be caused by both benign electrical mirroring and concurrent ischemia in remote territory as a result of occluded collaterals.
- Reciprocal depression occurs in 70% of inferior and 30% of anterior infarcts.
- The presence of reciprocal ST depression has a greater than 90% predictive value for acute infarction.

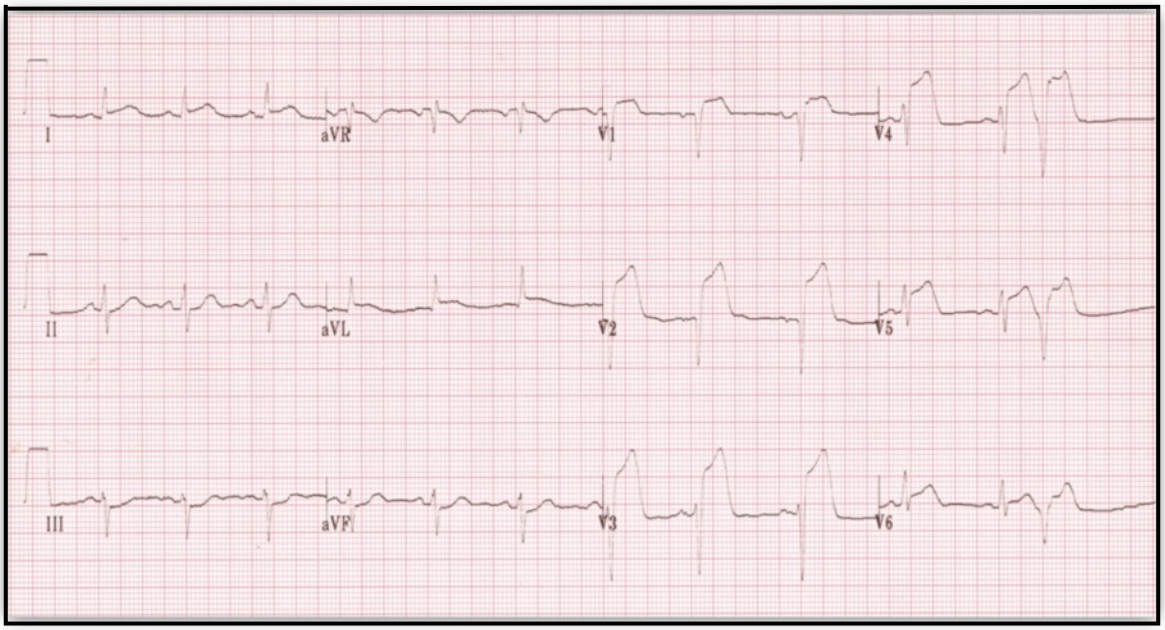


Inferior STEMI - HR 76, regular, sinus with prolonged PR interval. ST elevation in II, II, aVF, v4, v5 & v6. Reciprocal depression in leads aVR, aVL, V1 & V2. ST depression has a downward pattern. ST depression mirrors the pattern of the opposing ST elevation

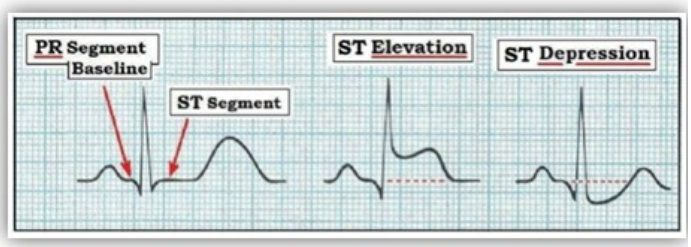


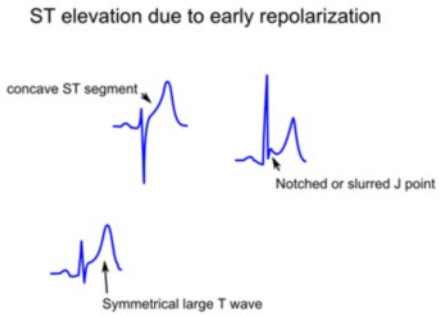
ST elevation

- ST elevation refers to an ST segment which remains elevated above the isoelectric line at the J point following the QRS complex.
- The ST segment signifies the end of ventricular systolic depolarisation. When myocardial cells are *injured* or *inflamed* they depolarise and repolarise less effectively.
- While the exact mechanism behind ST elevation is poorly understood, it is thought to indicate an abnormal current reflected by injured myocardial cells which can only partially depolarise.
- ST elevation of ischemic origin is typically confined to an effected grouping of contiguous leads and is often accompanied by reciprocal ST depression.
- Widespread ST elevation can be seen in pericarditis, and other inflammatory conditions affecting the entire heart.
- ST elevation can easily be mistaken for benign early repolarisation or high take-off which are normal variants in younger people identified by slurred or notched J points, concave ST segments and prominent, symmetrical T waves.



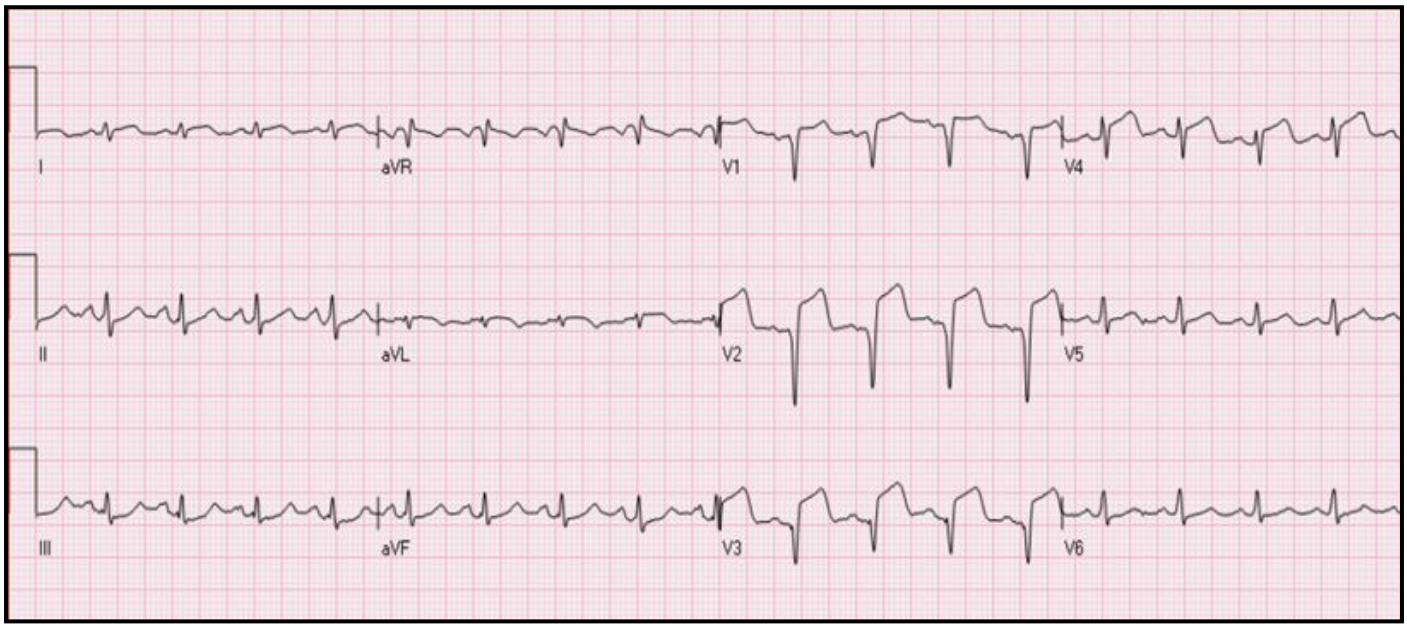
Aterolateral STEMI - HR 85, regular, sinus rhythm. ST elevation noted in the chest leads (V1-V6). Reciprocal ST depression noted in leads II, III and aVF



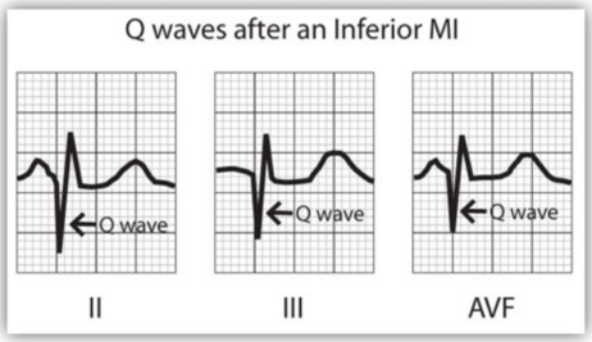


Pathological Q waves

- Pathological Q waves signify a degree of myocardial *necrosis*. These changes can take several hours to develop and once they appear they generally remain forever, as past a certain point cellular necrosis is irreversible.
- Q waves alone are not useful in diagnosing 'acute' infarcts due to their permanance once they appear.
- Q waves are considered pathological if they are more than 1mm wide, more than 2mm deep or more than ¼ the depth of the QRS complex.
- Small Q waves may be present in the leads viewing the left side of the heart, however, any right sided Q wave (V1-V3) should be viewed as pathological.



Anterior STEMI - heart rate 102, regular, sinus rhythm. ST elevation in v1-4. Deep pathological Q waves in v1-3

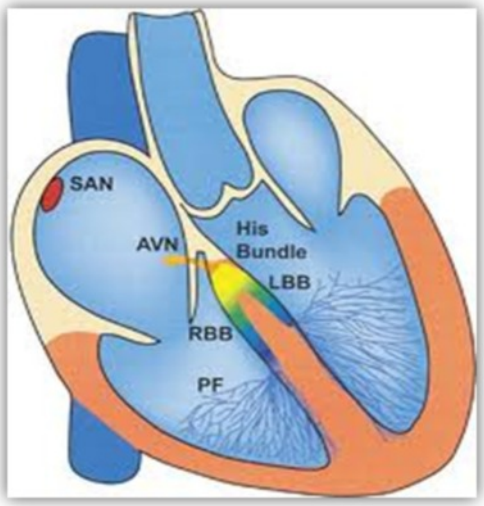


Conduction delays

- Conduction delays refer to any obstructive process which disrupts or alters the flow of an electrical current through any point of the myocardial conduction network.
- While there are different types of conduction delays, the majority fall into one of two specific categories:
    1. Bundle branch blocks
    2. Atrioventricular (AV) blocks (heart blocks)

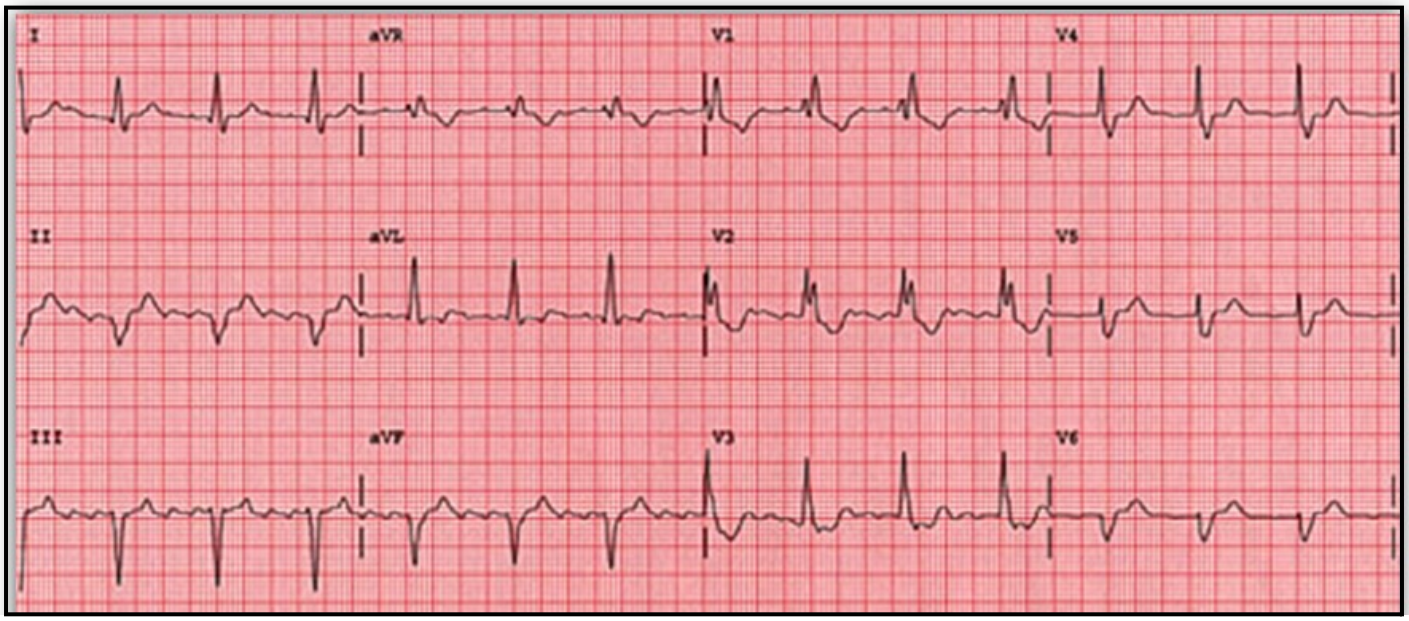
Bundle branch blocks

- In the healthy heart, an electrical impulse is automatically generated in the SA node. This impulse travels through the atrium to the AV node and down the bundle of His before diverging down the left and right bundle branches and dispersing through the purkinje fibres.
- Bundle branch blocks occur when the electrical conduction pathway through the body of the left or right ventricle is obstructed.
- In the case of a bundle branch block, the electrical impulse moves through one ventricle via the functional bundle branch before moving into the opposite ventricle causing a staggered or delayed ventricular contraction.
- In a bundle branch block, electrical impulses are spread across the ventricles sequentially rather than simultaneously. This sequential spread takes more time and as such, the QRS complex becomes wider in a bundle branch block.

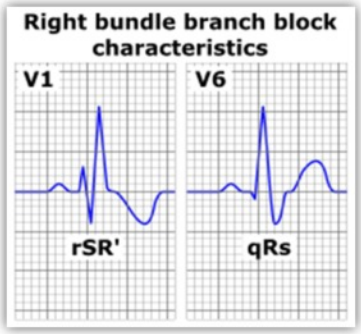


Right bundle branch block

- When the right bundle branch is blocked, electrical impulses travel down the left bundle branch stimulating contraction of the left ventricle before moving into the right.
- RBBB is a relatively common ECG finding which can present independently of any acute disease process.
- Being a condition effecting the right ventricle, RBBB is commonly associated with right ventricular insufficiency, problems relating to pleural circulation and respiratory disorders.
- Patients presenting with a new RBBB should be investigated to rule out any pathological cause of their conduction delay.
- Without an associated disease process, the marginal delay in right ventricular contraction related to RBBB has a negligible physiological effect.
- **RBBB is defined as a QRS duration of 120milliseconds or longer with an RSR pattern ("M" shape) in V1 and a wide S wave ("W" shape) in V6.**

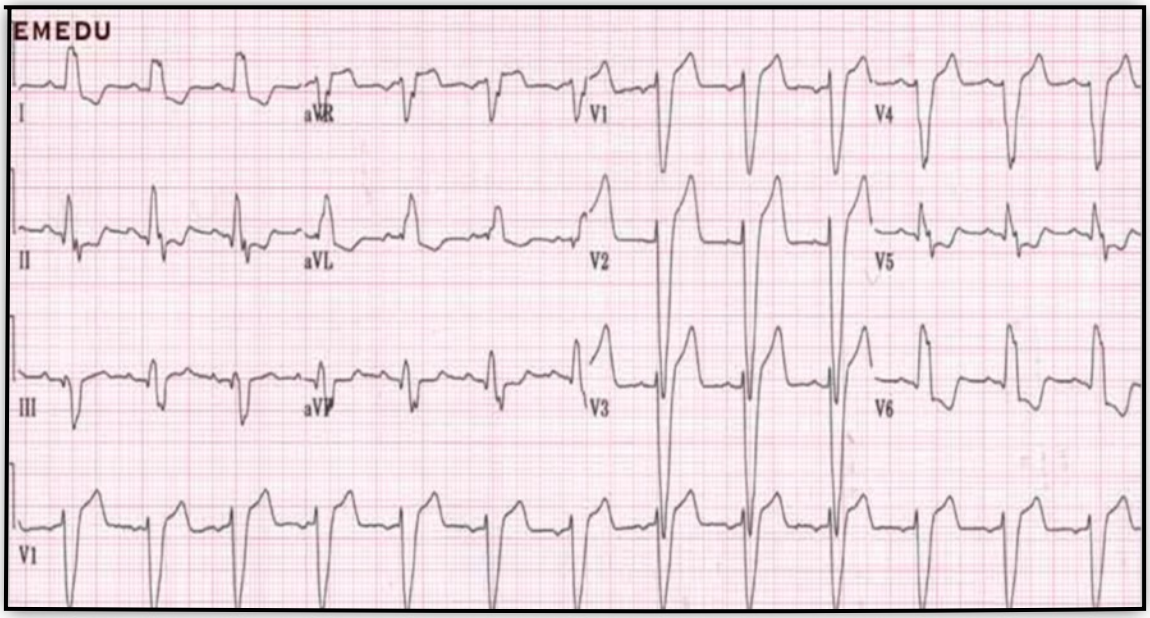


Right bundle branch block - HR 81, regular, sinus rhythm. QRS wider than 120 milliseconds (3 small squares). RSR ("M" shape) noted in V1, Wide S wave ("W" shape) noted in V6

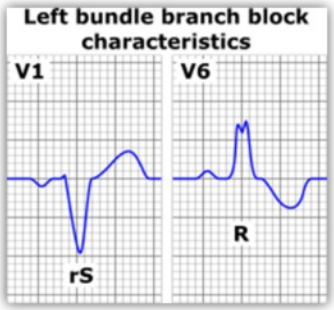


Left bundle branch block

- When the left bundle branch is blocked, electrical impulses travel down the right bundle branch stimulating contraction of the right ventricle before moving into the left.
- Although they can also appear independent of any pathology, LBBB's are more closely linked to the presence of serious cardiac conditions including cardiomyopathy and myocardial ischemia.
- As such, any patient presenting with a new LBBB should be thoroughly investigated for any potential pathological cause
- On a 12-lead ECG, the QRS complex of a patient with LBBB appears identical to the rhythm produced by an implantable pacemaker.
- Unlike RBBB, the delayed left ventricular contraction in LBBB can decrease cardiac output, particularly when associated with conditions such as heart failure.
- **LBBB is defined as a QRS duration of 120milliseconds or longer with deep S wave ("W" shape) in V1 and an RSR complex ("M" shape) in V6.**



Left bundle branch block - heart rate 78, regular, sinus rhythm. QRS complex wider than 120 milliseconds (3 small squares), Deep S wave ("W" shape) noted in V1
RSR complex ("M" shape) noted in V6

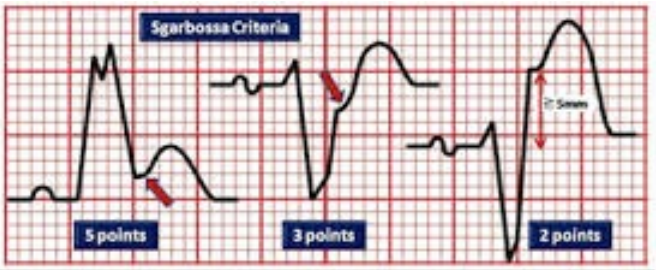


Sgarbossa Criteria

- While new LBBB is always considered pathological, diagnosing STEMI on an ECG for patients with preexisting LBBB or electronic pacemaker can be difficult as some degree of ST variation is expected.
- The Sgarbossa criteria is a set of 3 ECG findings which are designed to help diagnose STEMI in these patients.

1. Concordant ST elevation (ST elevation in leads with a positive QRS i.e. V6 with its tall RSR wave) of 1 millimetre or more equals 5 points.
2. Concordant ST depression (ST depression in leads with a negative QRS i.e. V1-3 with their wide S waves) of 1 millimetre or more equals 3 points.
3. Discordant ST elevation (ST elevation on the opposite side to a negative QRS i.e. V1-3) of 5 millimetres or more equals 2 points.

- If a patient's ECG has a Sgarbossa score of 3 or greater their is a very high likelihood they are having an acute infarct (specificity 90%).



Sgarbossa ECG Criteria for LBBB	
Concordant STE ≥1mm	5 points
STD ≥1mm in V1 – V3	3 points
Discordant STE ≥5mm	2 points

Managing BBB's

- New LBBB is always considered a sign of an acute infarct until proven otherwise, expert medical opinion should be sought and the ECG compared to old examples where possible. These patients should be treated as a high priority and should receive rapid assessment as they may be suitable for primary angioplasty.
- New RBBB alone is not typically associated with myocardial infarction but directed investigation should take place to rule out dangerous pathologies including pulmonary embolus.
- Regular observation should be attended as clinically indicated.
- Connect patient to continuous cardiac monitoring. If severe chest pain or haemodynamic instability connect to external defibrillator and monitor in resuscitation area where possible.
- Establish IV access and collect blood for pathology including cardiac enzymes, coags and a blood gas.

…A quick tip for telling the difference

- WILLIAM MARROW is a handy mnemonic for differentiating between left and right sided bundle branch blocks.
- The first letter of each word corresponds to V1.
- The last letter of each word corresponds to V6.
- The “W”s refer to the W shape made by a wide S wave.
- The “M”s refer to the M shape made by an RSR complex.
- The double letters in the middle of each word correspond with the side effected by the bundle branch block i.e. LL for left, RR for right.

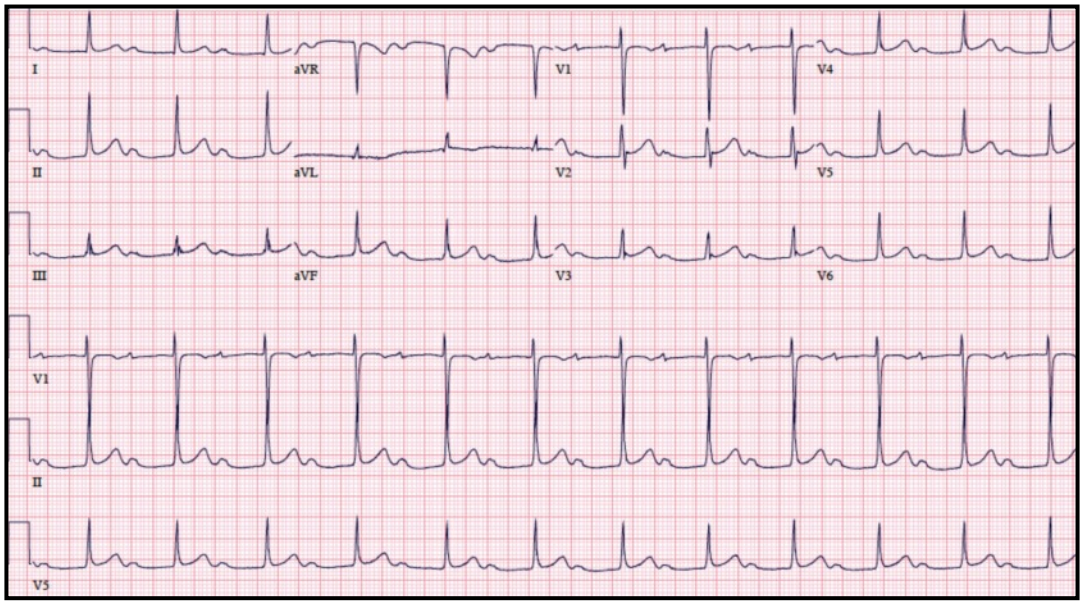
V1		V6	
W	LBBB	M	WiLLiaM
M	RBBB	W	MaRRoW

Heart blocks

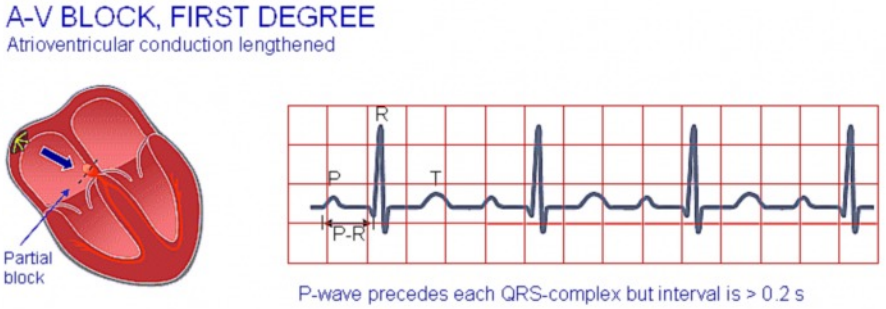
- The term heart block is used to categorise a group of pathologies which effect the way an electrical pulse is transmitted from the atria to the ventricles.
- There are 4 types of heart block to be aware of: first degree, second degree mobitz 1, second degree mobitz 2 and third degree or complete heart block.

First degree heart block

- First degree atrioventricular block is characterised by the delayed conduction of an electrical current through the AV node to the ventricular network.
- In first degree block every impulse generated by the SA node makes it through to the ventricular system.
- ECG findings characterising first degree heart block are; a QRS complex following each P wave, a prolonged PR interval of greater than 200 milliseconds (5 small squares on an ECG).
- First degree block can be caused by a number of factors including: increased vagal tone, degenerative changes due to aging, inflammation, electrolyte imbalances and different medications.
- In isolation first degree heart block is on little clinical significance
- Patients with first degree heart block on an ECG require no exceptional treatment and should receive care according to their symptoms and clinical presentation.



First degree heart block - heart rate 72 bpm, regular rhythm, P waves present, markedly prolonged PR interval, narrow QRS, t waves present.



Second degree heart block (Mobitz type 1)

- Second degree heart block is separated into 2 types: mobitz 1 and mobitz 2.
- Mobitz type 1 refers to the Wenckebach phenomenon which sees a series of consecutive electrical impulses from the SA node have gradually slower conduction through the AV node before reaching a point where a final impulse is unable to pass through the AV node.
- This sequence happens in a cyclic pattern with PR intervals gradually lengthening until the AV node fails to conduct at which point the cycle restarts with a short PR interval.
- Mobitz type 1 is thought to be attributed to the gradual fatigue of the cells within the AV node to the point where they fail to conduct.
- This can be caused by: cellular inflammation or necrosis secondary to ischemia, medications, increased vagal tone or recent cardiac surgery.
- Mobitz type 1 is typically a stable rhythm and is rarely associated with deterioration or haemodynamic instability.
- Well patients may not require definitive fixation whereas symptomatic patients may require treatment with the anticholinergic drug atropine or less frequently internal pacing.
- Mobitz type 1 ECGs are characterised by: cycles of gradually lengthening PR intervals ending with a p wave which is not followed by a QRS complex but rather a pause before the cycle restarts. P-P intervals remain relatively stable throughout. QRS complexes are generally grouped together in even bunches. Cycles generally contain a fixed P wave to QRS ratio i.e. 5 p waves 4 QRS etc.

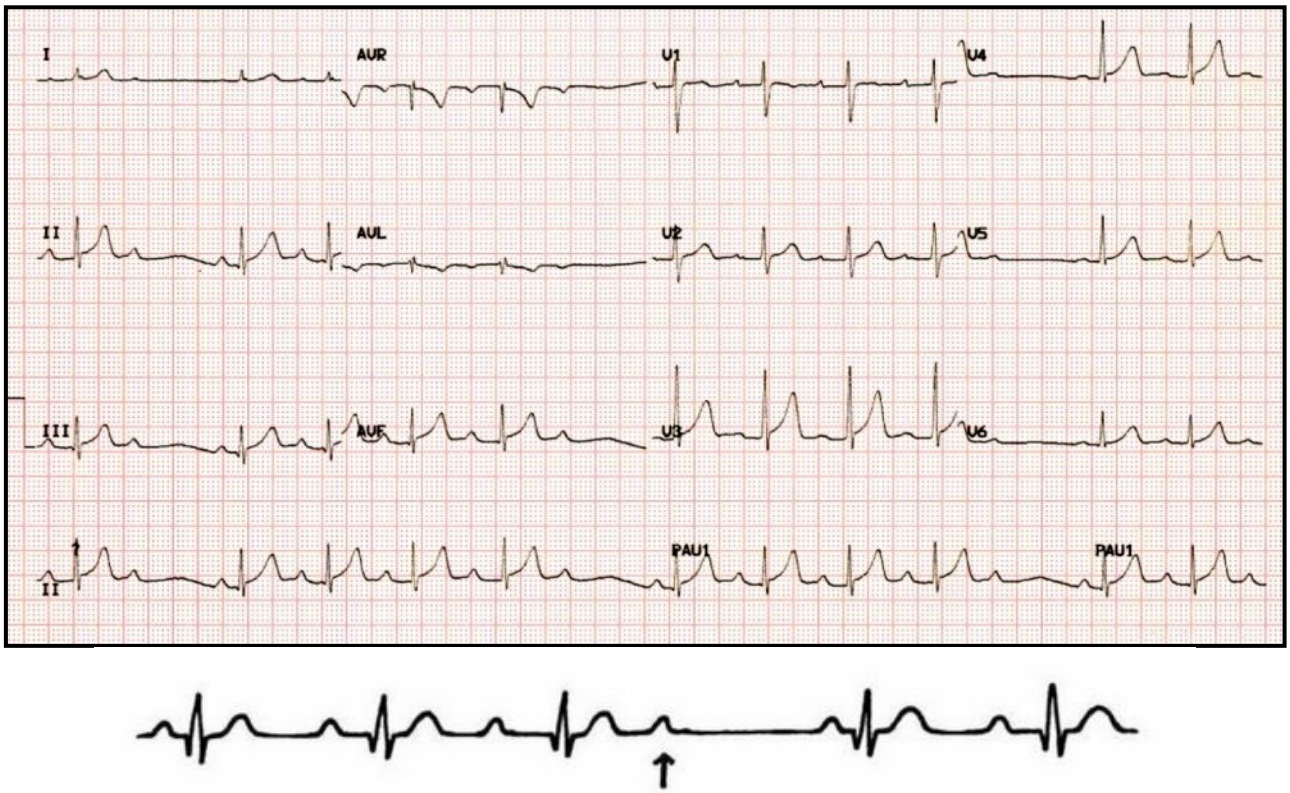
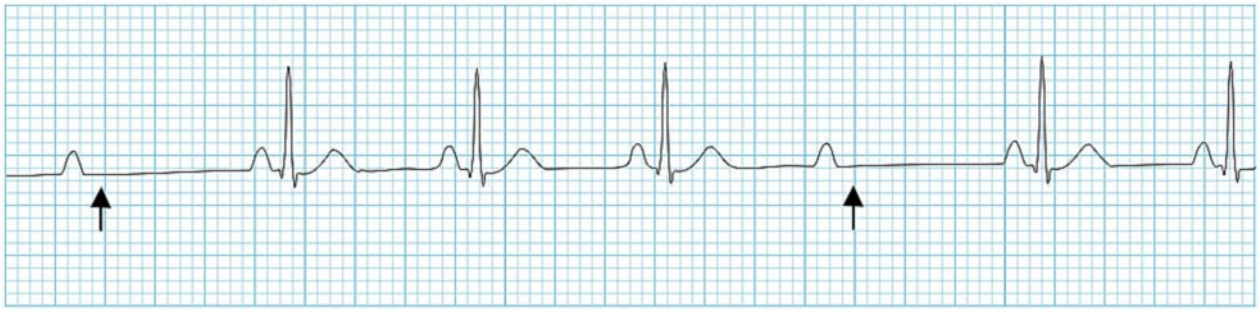


Figure 5. Mobitz Type 1 second degree AV block (also known as Wenckebach block); note that the PR interval increases with each beat until eventually there is a non-conducted P wave (arrowed) and then this pattern repeats itself.

Second degree heart block (Mobitz type 2)

- Mobitz type 2 refers to the intermittent failure of an impulse generated within the atria to conduct through to the ventricles.
- Where mobitz type 1 indicates a gradual fatigue of the functional AV node which leads to regular failure, type 2 indicates a mechanical failure at or below the level of the bundle of HIS.
- Typically this failure is due to damage or injury to the electrical conduction systems from cellular necrosis, fibrosis or inflammation.
- Mobitz type 2 is commonly associated with bundle branch or bifasicular blocks leading to wide QRS complexes.
- While it is more common for single QRS complexes to be missed at a time, multiple consecutive beats may be dropped causing groups of non-conducted P waves to be present (N.B. the ratio of P waves to QRS complexes may be irregular or fixed which is referred to as a fixed ratio second degree AV block and can be attributed to both Mobitz 1 and 2).
- Mobitz type 2 is more clinically significant as patients are more likely to present with symptomatic bradycardia and insufficient cardiac output
- Mobitz type 2 is also significantly more likely to deteriorate to complete (third degree) heart block.
- Mobitz type 2 is associated with a 35% risk of Asystole and sudden cardiac death each year.
- Patients with mobitz type 2 heart block should be monitored in a high acuity area and should remain on continuous cardiac monitoring.
- IV access should be gained and troponin checked to determine whether underlying ischemia is present.
- Haemodynamically unstable patients should be nursed in a resuscitation area and connected to an external defibrillator where possible.
- Patients may require temporary external pacing in the case of severe bradycardia and many will require the insertion of a permanent internal pacemaker.
- Mobitz type 2 ECG's are characterised by: the intermittent presence of P waves which are not followed by QRS complexes which is not preceded by the gradual lengthening of PR intervals. P waves will maintain a regular rate with an even P-P interval. PR intervals remain constant. Multiple P waves may occur together with absent QRS complexes.

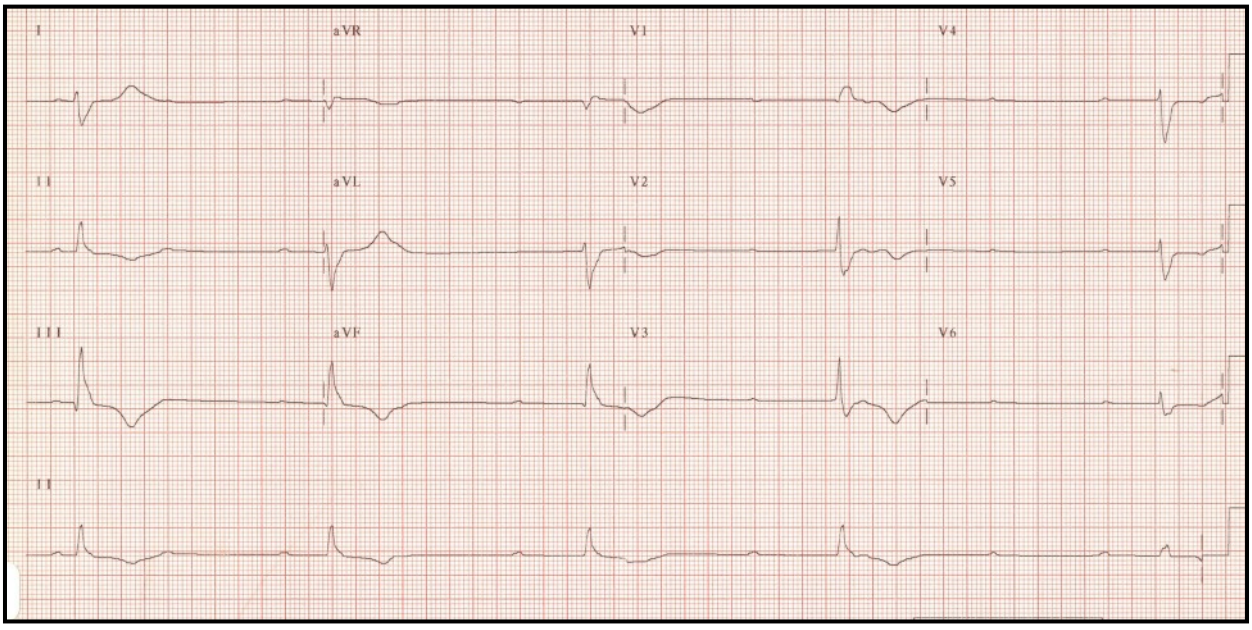


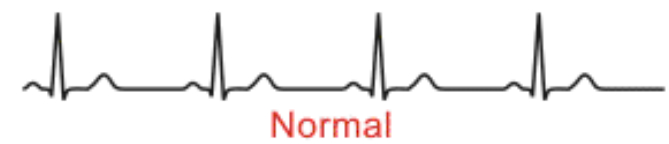
The above image shows Second degree heart block, mobitz type 2 - While there are P waves which are not conducted to QRS complexes there is no progressive lengthening of the PR intervals preceding these dropped beats.

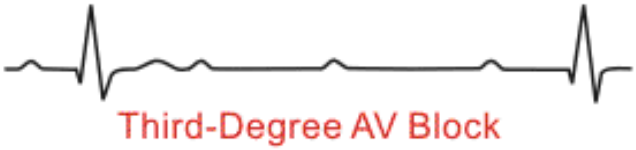
Figure 6. Mobitz Type 2 second degree AV block; on this occasion only 1 P wave in 3 is conducted to the ventricles, but note that each conducted beat has a normal, and constant, PR interval.

Third degree (complete) heart block

- Third degree heart block is referred to as complete heart block as it indicates a complete failure of the AV node to conduct electrical impulses.
- Complete heart block is characterised by a complete electrical dissociation of atria from the ventricles.
- In third degree heart block, ALL electrical messages from the SA node are blocked at the AV node and the only ventricular activity present is a base escape rhythm generated within the cells of the ventricles.
- This dissociation can be due to the progressive failure of the AV node as in mobitz type 1, or due to sudden failure of the bundle of HIS or below as in mobitz type 2.
- Patients in complete heart block are at very high risk of sudden cardiac arrest due to ventricular standstill.
- Patients with complete heart block should be connected to cardiac monitoring and cared for in a high acuity area.
- Ideally these patients should be nursed in the resuscitation area connected to an external defibrillator capable of providing temporary external pacing.
- Patients in complete heart block may require treatment with atropine or positive inotropes such as isoprenaline and most will require permanent cardiac pacing.
- ECG characteristics of third degree heart block include: P waves are present and may have a regular rhythm. P waves have no identifiable relationship to QRS complexes. QRS complexes are present but have no relationship to P waves. Atrial rate (p waves) approximately 100 bpm and ventricular rate (QRS complexes) approximately 40 bpm.





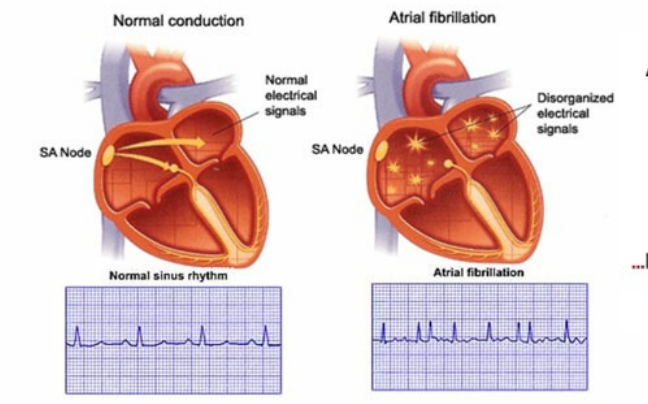


Atrial fibrillation

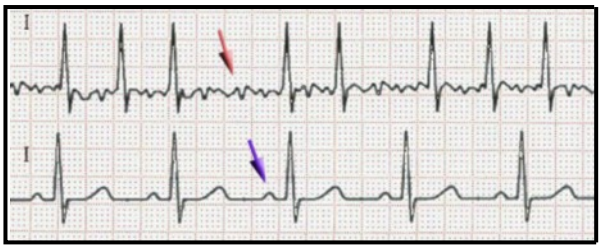
- Atrial fibrillation (AF) is the most common sustained arrhythmia.
- Characterised by the conduction of disorganised electrical impulses produced by several abnormal pacemakers within the right atrium which overwhelm the regular impulses produced by the SA node.
- These rapid irregular impulses cause the muscle of the atrial chamber to partially and rapidly contract in an irregular manner.
- This irregular movement is referred to as fibrillation (**rapid, irregular, unsynchronised contraction**) and means that the atrial chamber is unable to effectively empty.
- Inadequate atrial emptying allows blood to pool and potentially clot within the atrium placing patients at significantly greater risk of stroke and myocardial infarction.
- Periodically impulses from the atrium will make it to the AV node triggering ventricular contraction. These impulses may be generated by different pacemakers within the atrium and are conducted irregularly which translates to an irregular ventricular rate.
- AF may come and go (paroxysmal AF) or be present for a long period (persistent AF).
- ECG features of AF include:
    - Irregularly irregular rhythm with irregular ventricular rate in no distinguishable pattern.
    - P waves not present, replaced by coarse or fine fibrilatory waves.
    - May be associated with ventricular rates ranging from normal to very fast or very slow.

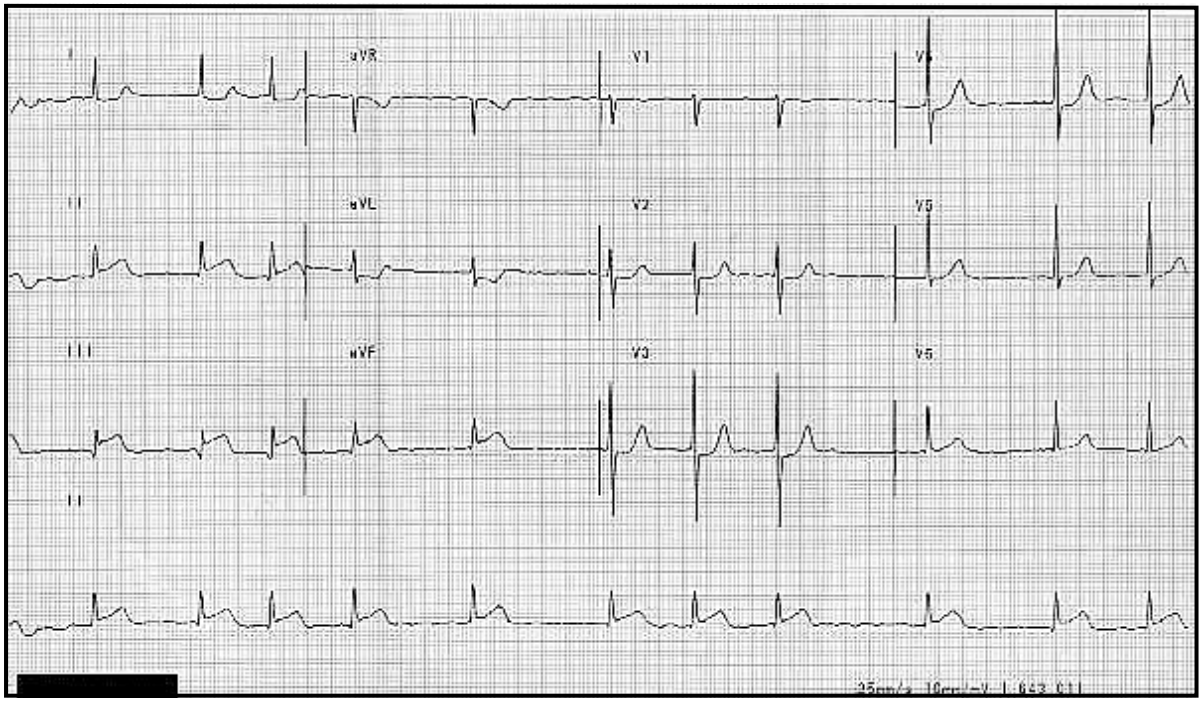
Interventions required

- If the patient is suffering chest pain, treatment should be delivered in accordance with the NSW chest pain pathway.
- For patients that have been in AF for less than 48 hours, direct current cardioversion may be attended to restore sinus rhythm.
- Patients that have been in AF for longer than 48 hours are at significantly greater risk of stroke post cardioversion and should be commenced on anticoagulation therapy if they are stable.
- Cardioversion remains an option in an unstable AF patient with rapid ventricular response.
- Persistent AF is treated differently depending on its ventricular rate i.e. very fast or very slow ventricular rates can lead to insufficient cardiac output and may require rate control medication or pacing.

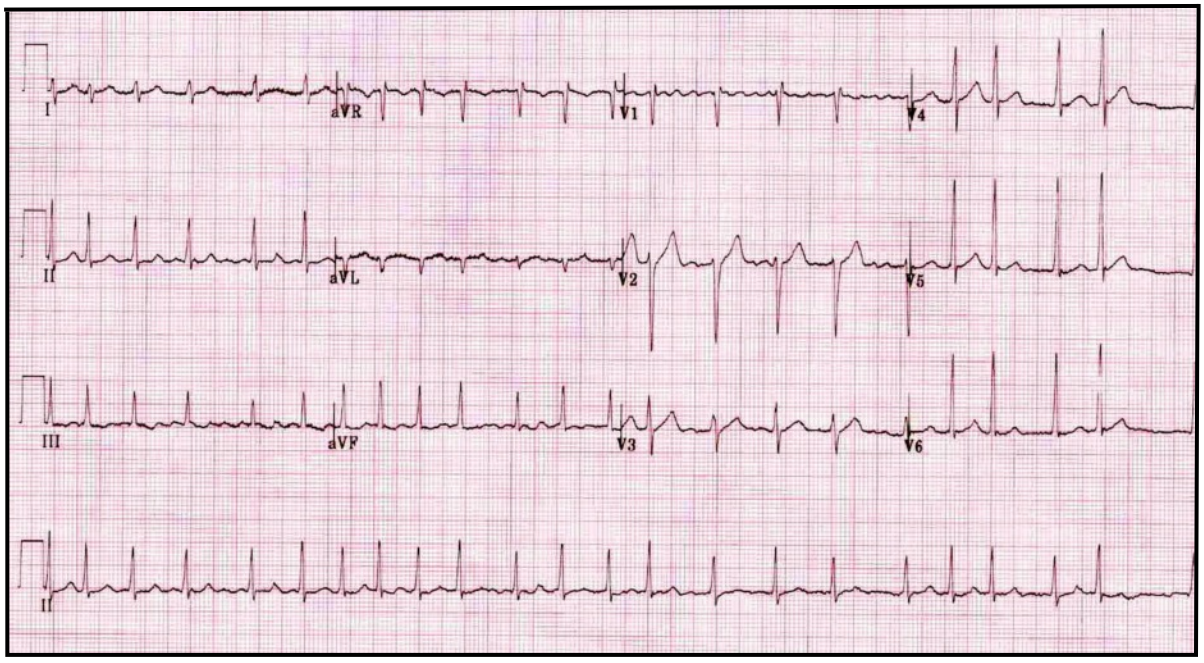


This picture illustrates the disorganised fibrilatory waves associated with AF above the regular uniform P waves associated with sinus rhythm.





Atrial fibrillation - ventricular rate approximately 66, irregular, fine fibrilatory waves in place of p waves, narrow QRS complex, t waves present, inferior ST elevation.



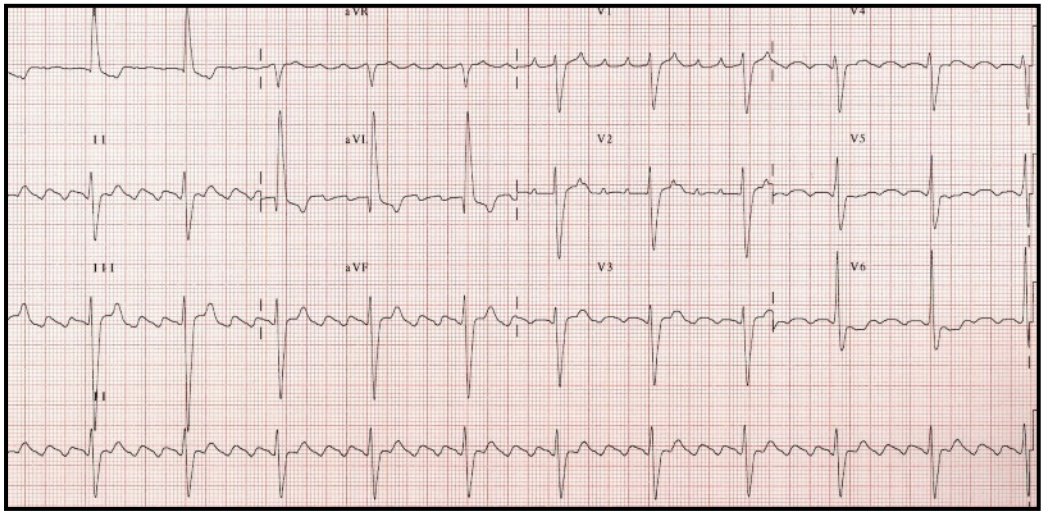
Atrial fibrillation with rapid ventricular response - ventricular rate approximately 135, irregular, coarse fibrilatory waves in place of P waves, narrow QRS, t waves present.

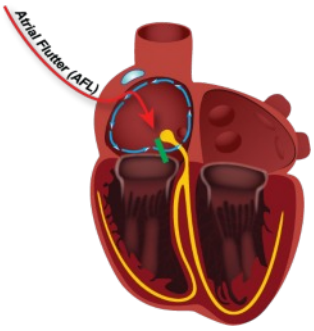
Atrial flutter

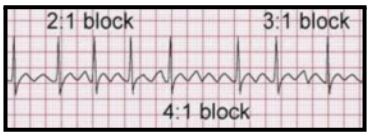
- Atrial flutter is similar to atrial fibrillation in that it is an arrhythmia in which the atrium contracts faster than the ventricles.
- In atrial flutter the atria contract in a regular pattern as opposed to atrial fibrillation which involves the irregular contraction of the atria.
- In atrial flutter an electrical pulse generated by the SA node is conveyed through the atrium toward the AV node before being transferred back to the SA node via an accessory pathway in the atrium.
- The electrical current can travel in this circular motion several times before activating the AV node leading to an atrial rate which can be up to several times faster than the ventricular rate.
- Typically an electrical current will circle the atrium an equal number of times before each ventricular contraction. The most common example is an AV conduction ratio of 2:1 meaning 2 atrial contractions precede every ventricular contraction causing an atrial rate twice as fast as the ventricular rate. However, this ratio can be wider i.e. 4:1 means 4 atrial beats to each ventricular contraction.
- ECG features of atrial flutter include:
  - P waves replaced by saw-tooth flutter waves.
  - Fixed AV block means an equal number of flutter waves precede each QRS complex.

Interventions required

- If the patient is suffering chest pain treatment should be delivered in accordance with the NSW chest pain pathway.
- Monitor vital signs regularly and connect patient to continuous cardiac monitoring.
- Refer to senior doctor immediately.
- Medication may be required to reduce the patients heart rate.
- The patient may also require chemical of DC cardioversion to return them to sinus rhythm.
- Patient with recurrent episodes of atrial flutter may require an ablation procedure to remove the accessory pathway responsible for propagating the arrhythmia.





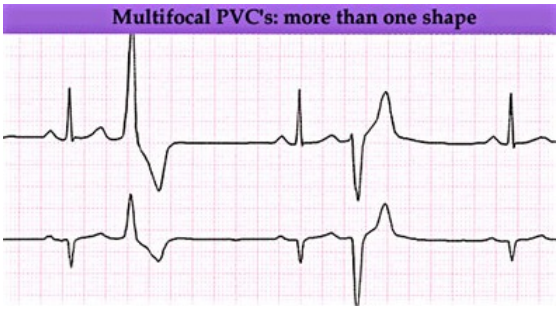


Atrial flutter - heart rate 66 bpm (ventricular), regular rhythm, P waves replaced by saw-tooth flutter waves, 3 flutter waves to every QRS = 3:1 block, narrow QRS, t wave buried in first flutter wave

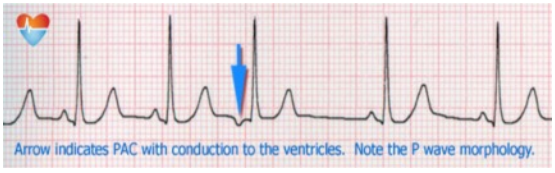
Ectopic beats

- `While the SA node is responsible for generating the majority of the electrical currents within the healthy heart due to its high intrinsic rate, it is important to remember that all cells within the myocardium have the ability to spontaneously depolarise stimulating a wave of myocardial contraction.
- Ectopic beats are premature contractions of the heart triggered by the depolarisation of a pacemaker cell outside of the SA node.
- The word ectopic is used to refer to something that is in an abnormal position. In this instance it is used to refer to position of the pacemaker cells in an abnormal part of the myocardium.
- Ectopic beats can be generated from within the atrium or the ventricle.
- Ectopic beats may also be referred to as premature ventricular or atrial contractions (PVC's or PAC's).
- Ectopics can be unifocal (originating from the same area) or multifocal (originating from different areas).
- The origin of an ectopic beat can be determined by their appearance on an ECG:

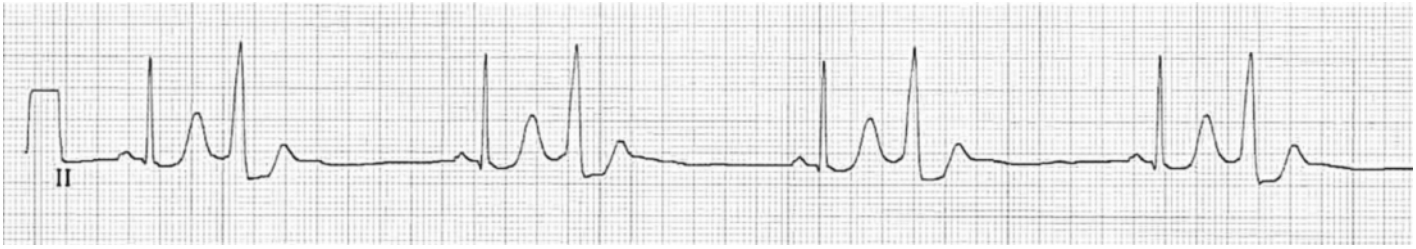
1. **Premature ventricular contractions** have a broad QRS complex with abnormal morphology (which may change depending on the origin of depolarisation), absent p waves and occur out of sync with the established ventricular rhythm. T waves are present and discordant (opposite side) ST changes may be evident. Often followed by a sinus pause.



2. **Premature atrial contractions** have a narrow QRS complex (but can be wide due to delayed conduction), occur out of sync with established atrial rhythm, differentiated from ven tricular contractions by the presence of abnormal looking P waves. Often followed by a sinus pause.



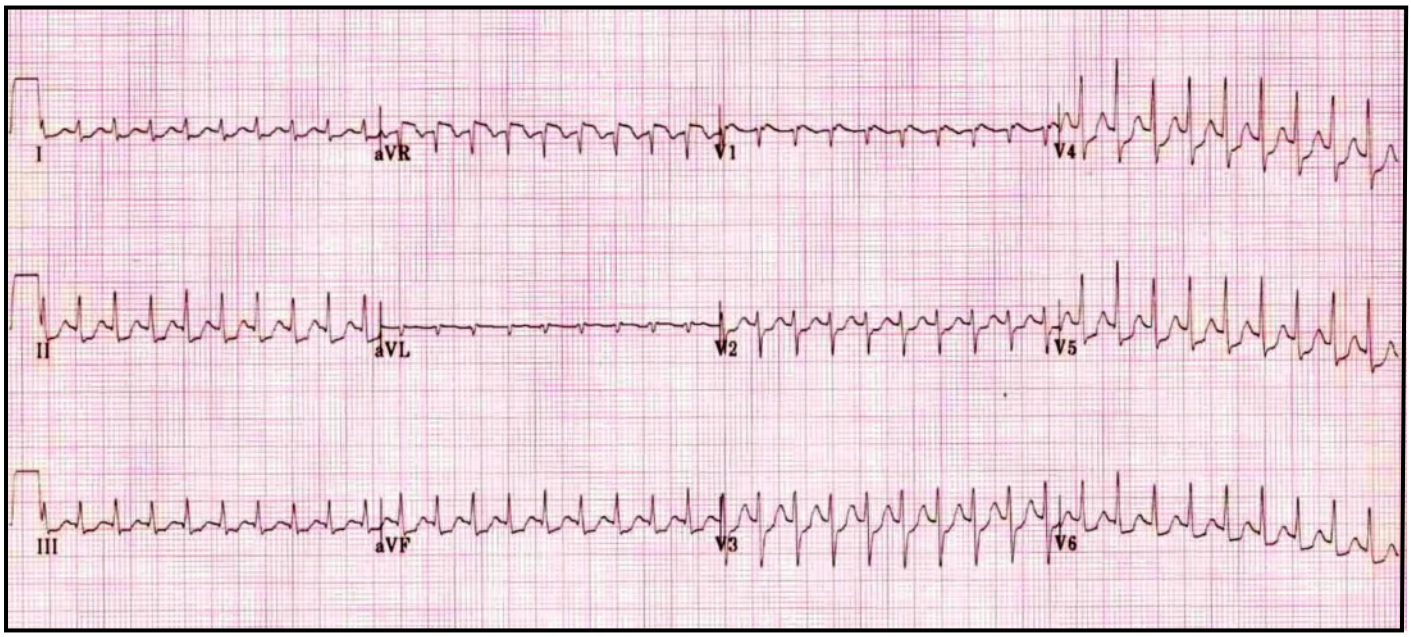
- Ectopic beats may occur in patterns i.e. Bigeminy refers to a pattern of every second complex being a premature contraction, trigeminy is a pattern where every third complex is premature etc.
- Ectopic beats are a normal phenomenon and in isolation are harmless and of minimal significance.
- In a symptomatic patient or if frequent premature complexes persist serum electrolytes should be checked to rule out myocardial membrane instability.
- Patients with underlying cardiac abnormalities such as Wolff-Parkinson White or Brugada syndrome should be monitored closely as premature contractions can trigger re-entrant arrhythmias in these patients.



Ventricular Bigeminy - pattern of 1 sinus complex to 1 PVC

Supraventricular tachycardia

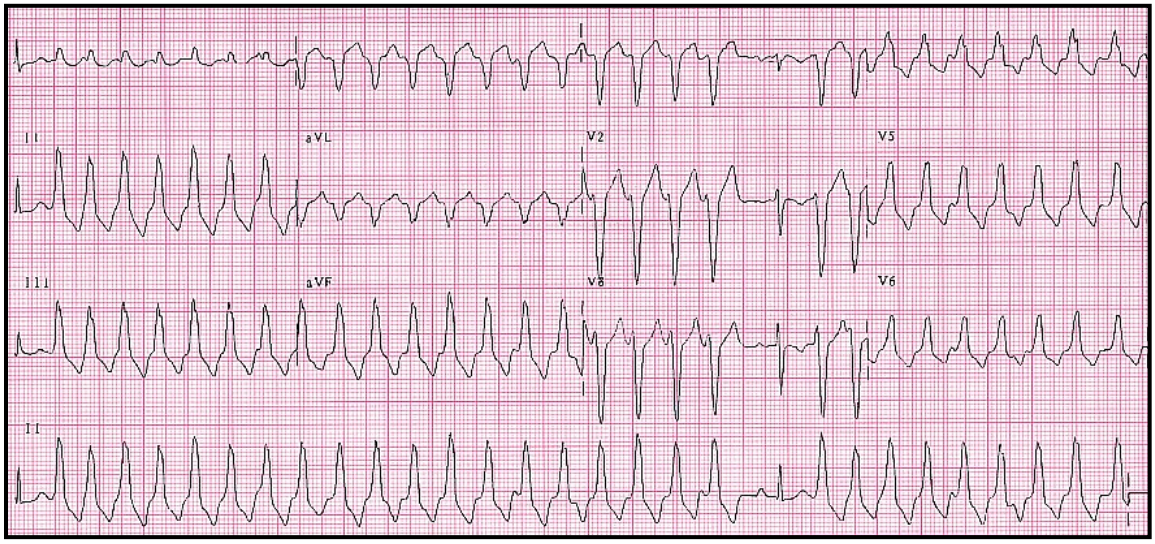
- The term supraventricular tachycardia refers to any tachyarrhythmia where the origin of the electrical impulse lies on or between the SA node and the AV node (i.e. in the atrium or supra (above or beyond) the ventricles).
- This term can be used to describe irregular atrial rhythms such as atrial fibrillation and flutter but is used in the majority of circumstances to refer to AV nodal re-entrant tachycardia (AVNRT).
- In AVNRT an abnormal electrical circuit exists within the AV node itself consisting of fast and slow channels. The fast channel takes a normal amount of time to de and repolarise while the slow channel depolarises slowly but repolarises rapidly.
- Typically electrical currents travel down both channels simultaneously balancing the transmission. However, if a premature atrial impulse enters the AV node while the fast channel is refractory it can force the impulse down the already repolarised slow channel. When the impulse reaches the end of the slow channel the fast channel may no longer be refractory allowing the impulse to travel back up in reverse becoming stuck in a circular movement between the two channels.
- Because of the short travel of the current in this re-entrant circuit, impulses are rapidly delivered to the ventricles causing a very high ventricular rate.
- These episodes are typically paroxysmal meaning they come and go spontaneously, however they may occasionally persist until medical treatment is delivered.
- Chest pain may be present due to underlying coronary artery disease or rate related ischemia.
- Patients may respond to vagal manoeuvres such as valsalva and the diving reflex which work by increasing vagal nerve tone in an attempt to make the aberrant pacemaker cells refractory allowing the SA node to regain control.
- If physical manoeuvres are unsuccessful, IV access should be established as the patient may require treatment with IV adenosine and in rare cases patients with SVT may require DC cardioversion.
- ECG characteristics of SVT (AVNRT) include: regular rhythm, heart rate 140-280 bpm, P waves may be buried in QRS complexes due to the small area of atrial conduction but may be present and occasionally inverted in inferior leads signifying retrograde conduction. QRS complexes are typically narrow however pre-existing conduction delays i.e. LBBB may make SVT difficult to distinguish from ventricular tachycardia. Occasionally, small inflections known as 'psuedo R waves' may be seen in V1-2 which will disappear when the patient reverts to sinus rhythm. ST depression is common.



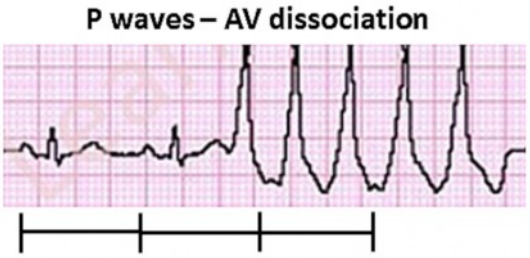
SVT (AVNRT) - heart rate 220, regular rhythm, p waves not distinguishable, QRS narrow, psuedo R waves present in V1, widespread ST depression noted

Ventricular tachycardia

- Ventricular tachycardia (VT) is an arrhythmia involving a rapid regular contraction of the ventricles which is dissociated from the action of the atria.
- VT is most commonly due to a re-entrant cycle where part of the purkinje network becomes non conductive in the usual direction (typically due to previous ischemia) allowing an overflow current from the surrounding fibres to travel along the blocked pathway in reverse. If the electrical current can travel around this retrograde circuit faster than the refractory period of the pathway the current will continue circling stimulating rapid myocardial contraction along the way.
- Due to extreme heart rates and poor coordination between the atria and ventricles, sustained VT typically leads to hypotension, circulatory collapse and if untreated can be fatal.
- VT may occur in non-sustained episodes or may persist until medical intervention occurs.
- Some patients may present in conscious VT but they will notably appear pale, diaphoretic and may report chest pain.
- Unconscious VT is a cardiac arrest scenario and should be treated in accordance with BLS/ALS guidelines.
- ECG characteristics of VT include: very rapid but regular ventricular rate (120-300 beats per minute), broad QRS complexes. QRS complexes may have different morphology depending on the origin of the electrical current. P waves may appear occasionally which are dissociated from the QRS complexes. Extreme axis deviation it common (positive QRS in aVR and negative complexes in lead I and aVF). Capture beats may be present where the SA node is able to break through the re-entrant cycle, these appear as relatively normal QRS complex.
- Often it is impossible to differentiate SVT with aberrant conduction from VT on the basis of and ECG.
- In any situation a symptomatic broad complex tachycardia should be treated as VT until proven otherwise.
- Patients noted to have sustained or non-sustained episodes of VT should receive immediate treatment in a resuscitation area. IV access should be established and an external defibrillator attached to the patient.
- In some cases anti-arrhythmic medication such as amiodarone may be administered, however DC cardioversion is required in most cases of sustained VT.

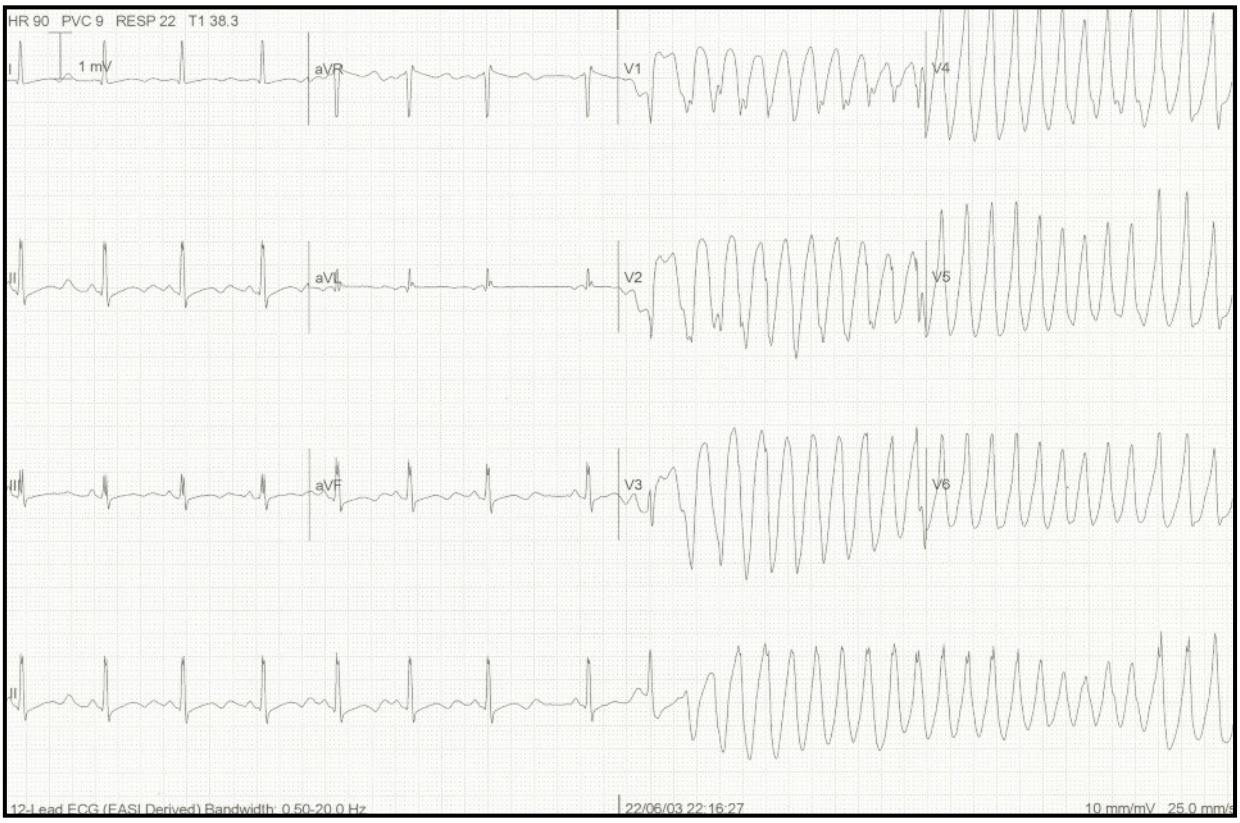


Ventricular tachycardia - hear rate 174 bpm, regular, occasional P waves (14th and 27th complexes on rhythm strip) dissociated from QRS. QRS wide. Single sinus capture beat noted (20th complex on rhythm strip)



Torsades de pointes

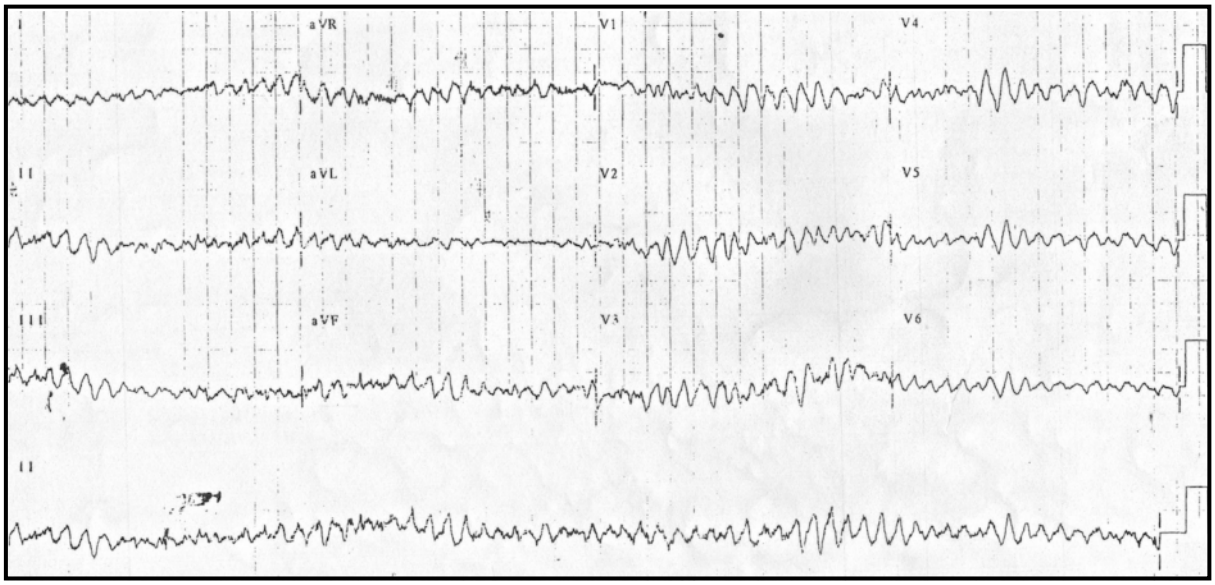
- Polymorphic VT refers to a type of ventricular tachycardia in which electrical currents are generated by varying focal areas within the ventricles rather than a single area in a re-entrant, monomorphic VT.
- Torsades de pointes (TDP) is one kind of polymorphic VT which occurs when an R on T phenomenon occurs in a patient with a prolonged QT interval.
- Torsades de pointes literally translates to mean "twisting of the points" in reference to it's polymorphic appearance.
- Long QT can be a congenital abnormality but may also be stimulated by low levels of magnesium and potassium as well as toxicity from different drugs.
- As the function of myocardial ion channels worsens, QT intervals lengthen and abnormal early depolarisation's may begin to occur (represented on an ECG by a U wave). If these early depolarisation's reach the required threshold to generate an action potential they will trigger a PVC directly following the T wave. If one of these PVC's occurs during the T wave (myocardial refractory period) VT or Ventricular Fibrillation (VF) may occur.
- TDP often occurs in short runs but can persist leading to circulatory collapse or deterioration into VF.
- Patient's noted to have episodes of polymorphic VT or long QT and bigeminy (which together can precede TDP) should be nursed in the resus bay and be attached to an external defibrillator. IV access should be established and a VBG and serology attended to identify any electrolyte imbalances.
- IV magnesium sulphate is typically administered as first line treatment for TPD or polymorphic VT with long QT.
- ECG characteristics of TPD include: preceding long QT, p waves usually buried, QRS complexes are wide with a distinguishable "twisting" of their inflection around the isoelectric line (i.e. QRS complexes rapidly change from positive (upright) to negative (upside-down) and back again). Episodes may be preceded by the presence of large U waves or ventricular bigeminy.



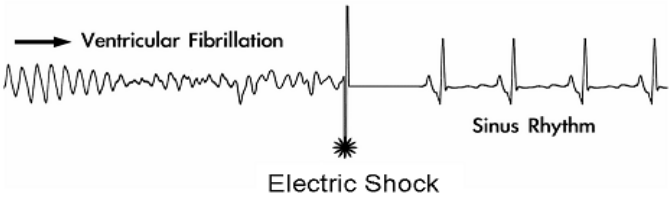
Torsades de pointes - sinus rhythm with a rate of 100 bpm with prolonged QT and visible U waves. In the 8th 9th complex a PVC can be seen to occur in the middle of the preceding T wave. Following this R on T is a polymorphic VT with a rate of approx 300 bpm. These QRS complexes can be seen to change inflection rapidly between positive (points facing down) and negative (points facing up).

Ventricular fibrillation

- Ventricular fibrillation is a life threatening cardiac arrest rhythm characterised by the ineffective and unsynchronised contraction of the ventricles which occurs when disorganised electrical impulses attempt to conduct up to 500 pulses per minute.
- Because the ventricles simply cannot convey pulses this rapidly, they are reduced to ineffective fibrilatory movements and the heart ceases to pump effectively leading to an immediate loss of cardiac output.
- The ventricles in VF behave in the same way as the atria in AF.
- Patients in VF will be unconscious, pale and often not be breathing (they will typically look dead)!
- VF is always fatal if left untreated and patients identified to be in VF or clinically presenting in cardiac arrest should receive immediate and aggressive treatment according to BLS/ALS guidelines.
- VF is a shockable rhythm and decreasing the time to defibrillation has been shown to improve outcomes in patients suffering VF arrest.
- ECG (rhythm strip) characteristics of VF include: a series of irregular wide electrical deflections varying in direction and amplitude. P waves, QRS complexes and T waves not evident. Appearance may be coarse (large deflections) or fine (small deflections).
- N.B. VF should never be diagnosed on an ECG! If a patient presents in cardiac arrest with no signs cardiac output (as is always the case in VF) CPR should be commenced immediately and any arrhythmia identified when an external defibrillator is attached to the patient.



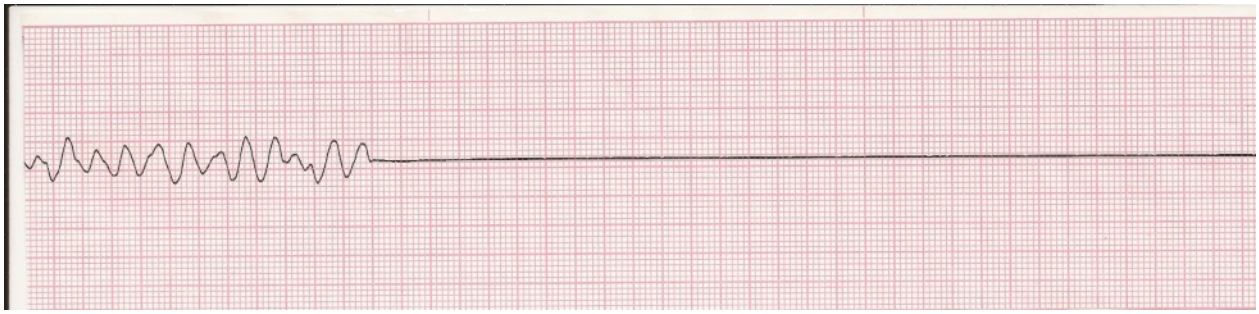
Ventricular fibrillation - irregular electrical deflections varying in amplitude. Nil evidence of P waves, QRS complexes or T waves. I repeat VF SHOULD NOT BE DIAGNOSED ON ECG! BLS/ALS measure take precedence!



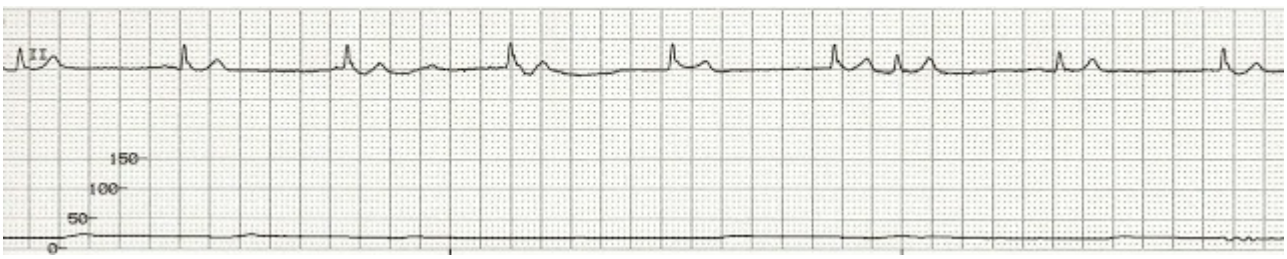
This is how you should see VF, as monitored on a defibrillator rhythm strip before electrical defibrillation occurs and the patient is potentially returned to a perfusing rhythm.

Asystole and pulseless electrical activity

- Asystole is a rhythm which is characterised by the complete absence of any electrical or mechanical activity of the heart. The term 'asystole' gives a hint as to what is involved ('a' - absent, 'systole' - contraction).
- Pulseless electrical activity (PEA) is a condition where coordinated electrical activity may be present within the heart (and may be seen on an ECG) but no physical contraction occurs within the myocardium. Previously this was called 'electromechanical dissociation' as there is no functional relationship between the electrical activity and physical contraction of the myocardium.
- In both of these situations the heart muscle physically does the same thing - nothing!
- In each of these cases the patient will have no cardiac output and as such these are cardiac arrest scenarios.
- Any patient who physically presents in cardiac arrest should receive immediate and aggressive treatment as per BLS/ALS protocols.
- Asystole and PEA are the 2 non-shockable rhythms on the ALS pathway and as such these patients should receive CPR and adrenaline 3 minutely while an attempt is made to find and correct any reversible causes of the patient's cardiac arrest.
- Survival rates of patients with asystole or PEA are significantly lower than survival rates of patients suffering VT or VF arrest under the same circumstances.
- Again, cardiac arrest **rhythms with no cardiac output should be diagnosed by defibrillator rhythm strip, NOT ECG!**
- ECG characteristics of asystole are: a flat line following the isoelectric baseline indicating the complete absence of electrical activity. P waves, QRS and T waves not present.
- ECG characteristics of PEA are: rhythms noted during PEA may mirror many perfusing rhythms but typically the heart rate will be slow and the rhythm will appear as sinus, juntional (sinus with absent or inverted p waves) or idoventricular (very slow, absent p waves, bizarre wide QRS complexes). Slow AF may also be present in PEA.



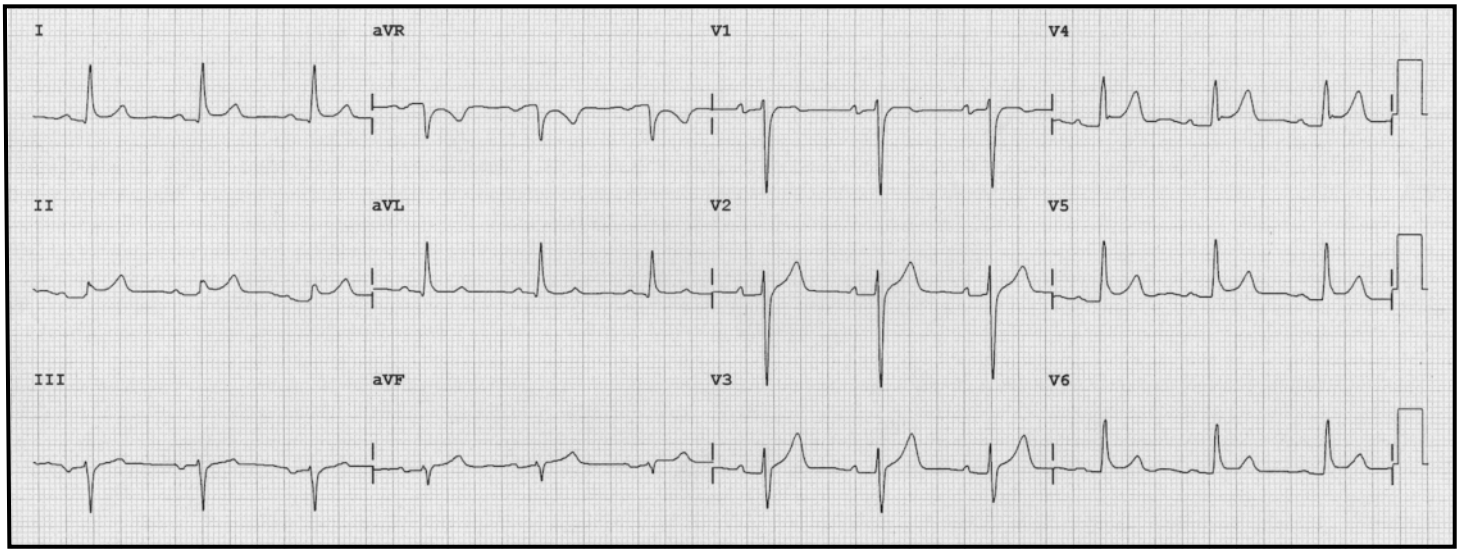
The above rhythm strip shows fine VF deteriorating into asystole marked by the flat line with no electrical current being generated by the myocardium.



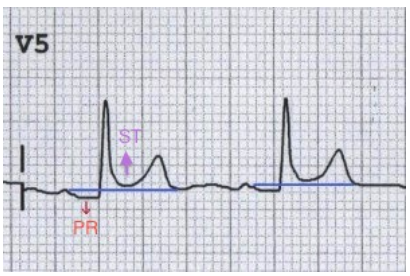
The above strip shows how a coordinated rhythm (in this case a junctional escape rhythm shown at the top) can be present with no cardiac output (signified by the flat invasive BP trace at the bottom). This is typical of PEA where the electrical current fails to stimulate effective cardiac contractions.

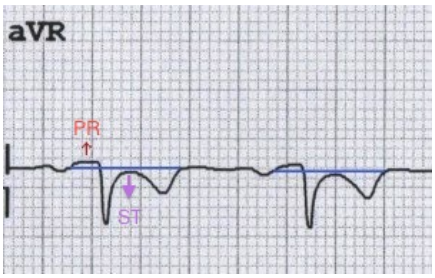
Pericarditis

- Pericarditis is a condition characterised by the global inflammation of the pericardium (the thin sack that surrounds the myocardium (heart muscle)).
- Pericarditis is typically caused by a viral infection but can be caused by bacterial infection, trauma, drug reactions or immunological conditions such as SLE.
- Patients with pericarditis may present with very similar symptoms to those suffering myocardial ischemia including chest pain, tachycardia, diaphoresis and dysponea (typically patients with pericarditis will present with orthoponea meaning they are more short of breath lying flat compared to sitting upright).
- While in many cases pericarditis will resolve with supportive treatment alone, it can lead to the development of pericardial effusion and tamponade which are life threatening medical emergencies.
- Typically ECG changes in pericarditis are widespread (effecting all leads) rather than focal (effecting certain lead groups) as in localised myocardial ischemia.
- ECG characteristics of pericarditis include: widespread concave (curve facing upwards) ST elevation and PR depression. These changes are mirrored in aVR and occasionally V1 with convex (curve facing down) ST depression and PR elevation.
- It can be difficult to differentiate pericarditis from acute myocardial ischemia (STEMI), however if any of the following signs are present STEMI should be suspected: ST depression in any lead other than aVR or V1, flat or convex (curve up) ST elevation, ST elevation that is taller in lead III than lead II.
- Patients with suspected pericarditis should be urgently reviewed by a senior medical officer. Cardiac monitoring should be applied. IV access should be established and serology attended including troponin levels.
- Chest pain should be managed in accordance with the NSW health chest pain pathway and MO guidance.



Pericarditis - heart rate 72 bpm, regular, PR depression in all leads but aVR, PR elevation in aVR. Concave ST elevation in all chest lead, Lead I, II and aVF. Convex ST depression in aVR

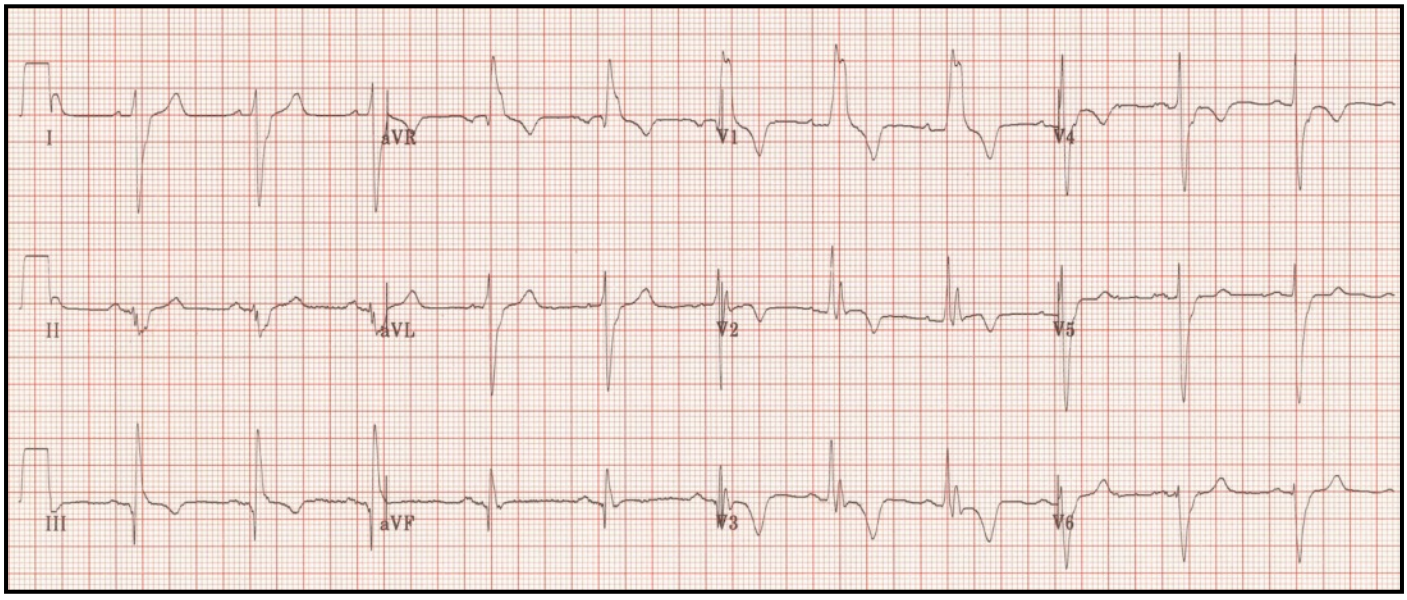




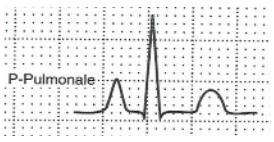
The above images show a close up view of the concave ST elevation and PR depression (V5) which is typical of pericarditis beside the reciprocal convex ST depression and PR elevation which occurs in aVR

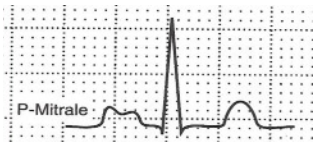
Pulmonary embolism

- A pulmonary embolism or PE is the name given to a blood clot which forms elsewhere in the body before breaking off and travelling to an artery within a patient's lung where it forms an occlusion.
- PE's cause reduced blood flow and hypoperfusion to the lungs themselves as well as causing a backlog of pressure in the right side of the heart due to increased vascular resistance.
- Patient's with PE's typically present with chest pain and dysponea. Typically the pain will be reported as sharp and worsened by deep inspiration (pleuritic). Patient's may also cough up pink blood tinged sputum.
- PE's can lead to severe hypoxia and ischemia of the right ventricle which can lead to cardiac arrest.
- ECG findings are neither sensitive nor specific in providing a definative diagnosis of PE, however they can give very useful clues which in the presence of a convincing patient history can increased suspicion of PE and prompt further investigation.
- 18% of patients with PE will have a normal ECG!
- By far the most common ECG finding in PE is sinus tachycardia which has been reported in 44% of patients.
- Other ECG characteristics of PE include: right heart strain pattern (T wave inversion in V1-4 and inferior leads), RBBB, right axis deviation (or extreme right axis deviation), dominant R wave in V1 and P Pulmonale (tall peaked P wave typically found in lead II) signifying right atrial and ventricular dilation, S1 Q3 T3 pattern (deep S wave in lead I combined with the presence of a Q wave and T wave inversion in lead III).
- If PE is suspected IV access should be established and serology collected including a coagulation tube. Oxygen therapy is commonly required along with adequate analgesia.
- Patients may require further diagnostic testing such as echocardiagrams and CT scanning of the chest.
- In the case of a diagnosed PE of significant size or one causing haemodynamic instability or hypoventillation, thrombolysis may be required to break down the clot allowing for return of pulmonary blood flow.



Sinus rhythm with PE - heart rate 66 bpm, regular rhythm, p waves present, RBBB, T waves inverted in V 1-4, right axis deviation (lead I negative, aVF slightly positive i.e. bordering on extreme right axis), S1 Q3 T3 present.

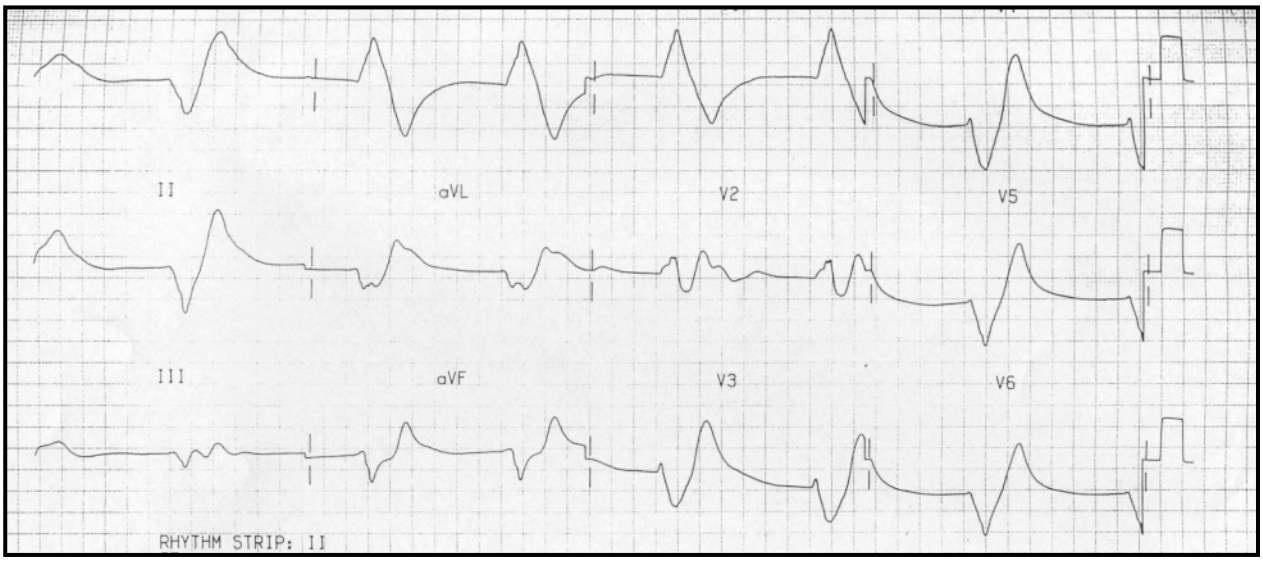




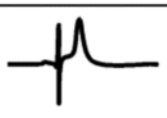
To the left examples of P pulmonale (signifying right atrial enlargement) and P mitrale (signifying left atrial enlargement) as seen in lead II

Hyperkalemia

- Hyperkalemia refers to an abnormally high concentration of potassium within a patient's blood (generally accepted as a serum potassium of more than 5.5mEq/L).
- Potassium is one of the electrolytes which are essential in facilitating cardiac automaticity and regulating the stability of the myocardium.
- Hyperkalemia may be caused by: renal failure, drugs, mineralocorticoid deficiencies such as addisons disease, massive tissue damage as in burns and rhabdomyolysis.
- One of the earliest ECG changes in hyperkalemia is the development of peaked T waves which may gradually increase in amplitude.
- In patients with moderate hyperkalemia (potassium > 6mEq/L), the atrium may become paralysed which is marked on an ECG by the progressive flattening and eventual disappearance of P waves.
- In severe hyperkalemia (potassium > 7mEq/L) conduction delays may develop (i.e. BBB) and bradycardia may develop.
- In severe hyperkalemia the rhythm recorded on an ECG may begin to resemble a basic sine wave, slowly undulating up and down (this is a pre-arrest rhythm!)
- In severe hyperkalemia cardiac arrest due to aberrant or absent conduction is likely. Patients may develop VF, PEA or aystole.
- In patients with suspected hyperkalemia IV access should be established and serology collected including a VBG to rapidly assess the patient's serum potassium.
- Patients with hyperkalemia should be attached to continuous cardiac monitoring.
- Various medications may be prescribed to reduce the serum potassium level but patient's with severe hyperkalemia may require haemodialysis to remove excess potassium ions from the blood.

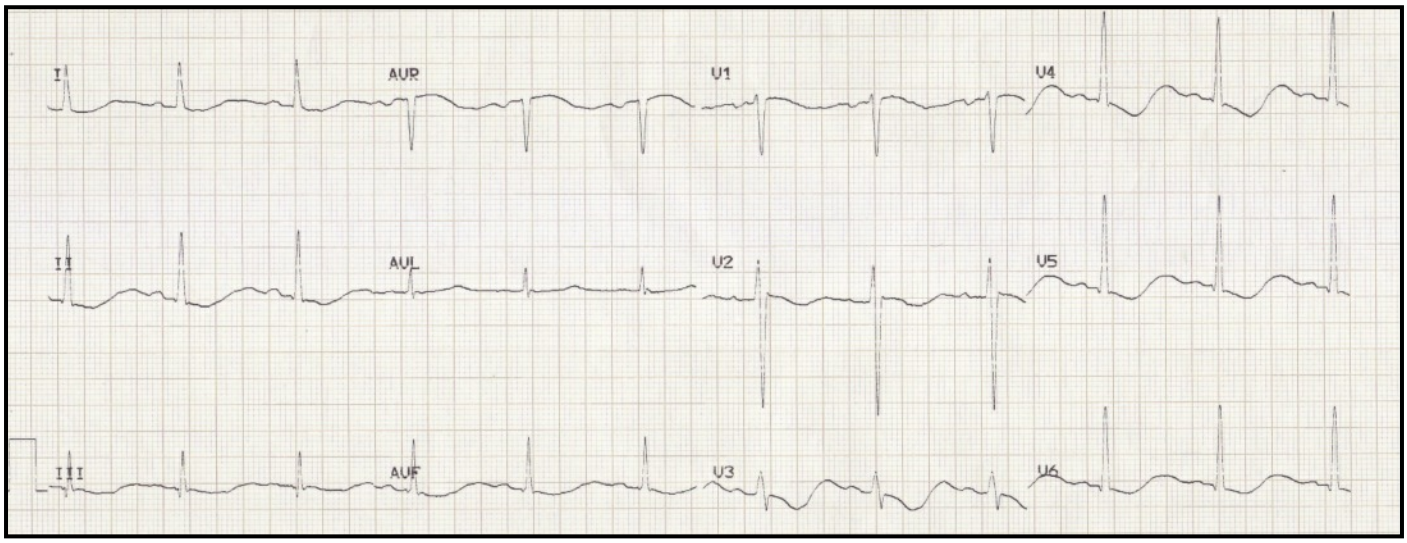


The above ECG shows the results of sever hyperkalemia: no atrial activity, ventricular conduction delay (broad QRS) and giat T waves giving the sine wave appearance.

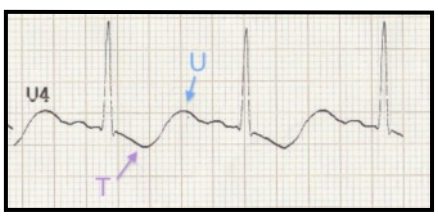
Serum potassium	Typical ECG appearance	Possible ECG abnormalities
Mild (5.5-6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5-8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (>8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

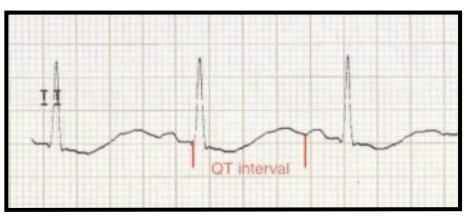
Hypokalemia

- Hypokalemia refers to an abnormally low concentration of potassium in the patient's blood (serum potassium < 3.5mEq/L).
- This decreased concentration of extracellular potassium can cause membrane instability which can lead to re-entrant arrhythmias such as VT.
- Hypokalemia can be caused by: severe lack of potassium in diet (rare), fluid loss due to diarrhoea, sweat etc (most common), conditions such as alkalosis and drugs such as insulin and salbutamol which cause extracellular potassium to be drawn into the cells.
- ECG characteristics of hypokalemia include: tall broad P waves, prolonged PR interval, ST depression, T wave flattening and inversion, increasingly visable U waves, prolonged QT interval (in some cases a T wave may fuse with a U wave causing the appearance of a long QT with no U where in reality it is a long QU interval).
- QT prolongation associated with hypokalemia can also indicate hypocalcemia or hypomagesemia. Effectively the imbalance of these electrolytes causes a delay in ventricular repolarisation which is indicated by the prolonged QT interval.
- Prolonged QT puts patients at greater risk of 'R on T' episodes and ventricular arrhythmias such as VT or TDP.
- In any patient presenting with abnormal ECG findings or arrhythmia IV access should be established and serology attended to check electrolytes including potassium, magnesium and calcium (where possible, a VBG will give a snapshot of these values almost immediately).
- Patients should remain on continuous cardiac monitoring and may require treatment with IV fluids and electrolyte supplementing medication as directed by medical staff.



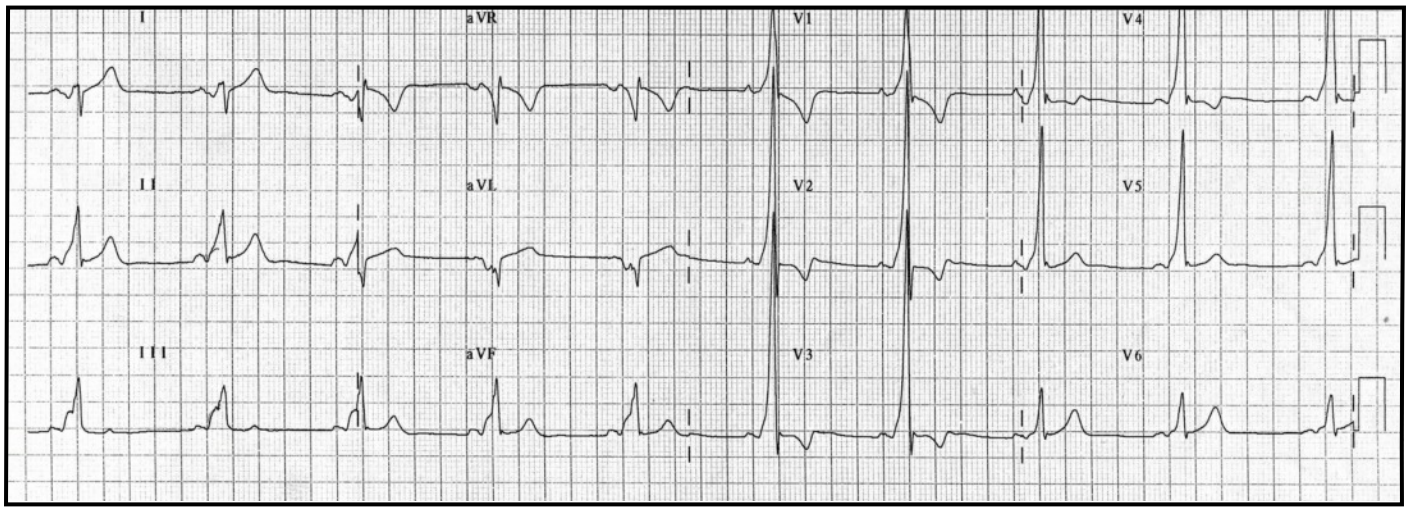
Hyperkalemia - Heart rate 72, regular rhythm, broad P waves, moderate ST depression, deep inverted T waves, prominent U waves with QU fusion as seen in the limb leads



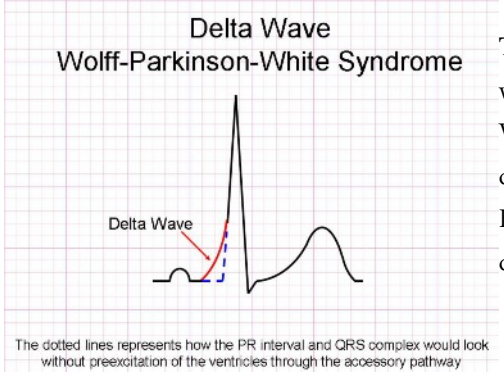


Wolff-Parkinson White syndrome

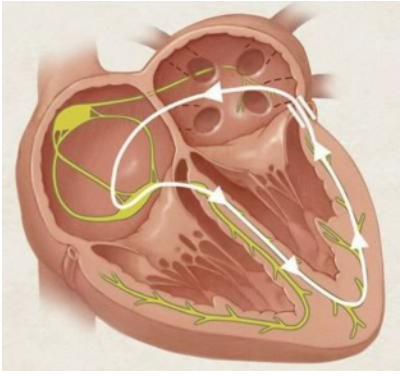
- Wolff Parkinson white (WPW) syndrome is a condition characterised by the pre-excitation of the ventricles via an accessory electrical pathway known as the bundle of Kent which links the atria to the ventricles.
- Typically electrical currents from the SA node are temporarily slowed down as they poss through the AV node before moving into the ventricles. In WPW, the electricity can enter the ventricles via the bundle of Kent without being slowed by the resistance of the AV node. This means that part of the ventricles will be stimulated a fraction earlier than the AV node can convey the message to the bundle of HIS.
- As with other accessory pathways, electrical impulses may travel back up the bundle of Kent from the ventricles into the atria where they can activate the AV node before the SA node would have typically generated the next action potential. This circuit allows electrical impulses to rapidly travel in a cycle from the AV node, through the ventricles and back to the AV node via retrograde (backwards) conduction through the bundle of Kent. This is known Atrioventricular reciprocating tachycardia or AVRT (a type of SVT).
- Patients with WPW are at an increased risk of AVRT and are also at a slightly higher risk of sudden cardiac death.
- Symptomatic patients with WPW require cardiac monitoring and regular observation.
- WPW patients with palpitations or extreme tachycardia should be treated according to their clinical condition and ECG findings. WPW patients with SVT may be responsive to vagal manoeuvres or medications but as with all tachyarrhythmia's, DC cardioversion may be required if the patient is unstable.
- ECG characteristics of WPW are: a short PR interval (less than 120 milliseconds), the presence of a delta wave (an early upward slurring of the initial section of the QRS complex signifying early excitation of the ventricles). Patients may also present with AVRT (a type of SVT).



Wolff Parkinson White - heart rate 60 bpm, regular rhythm, p waves present, short PR interval, delta waves present in QRS complexes, T wave inversion in V1-3.

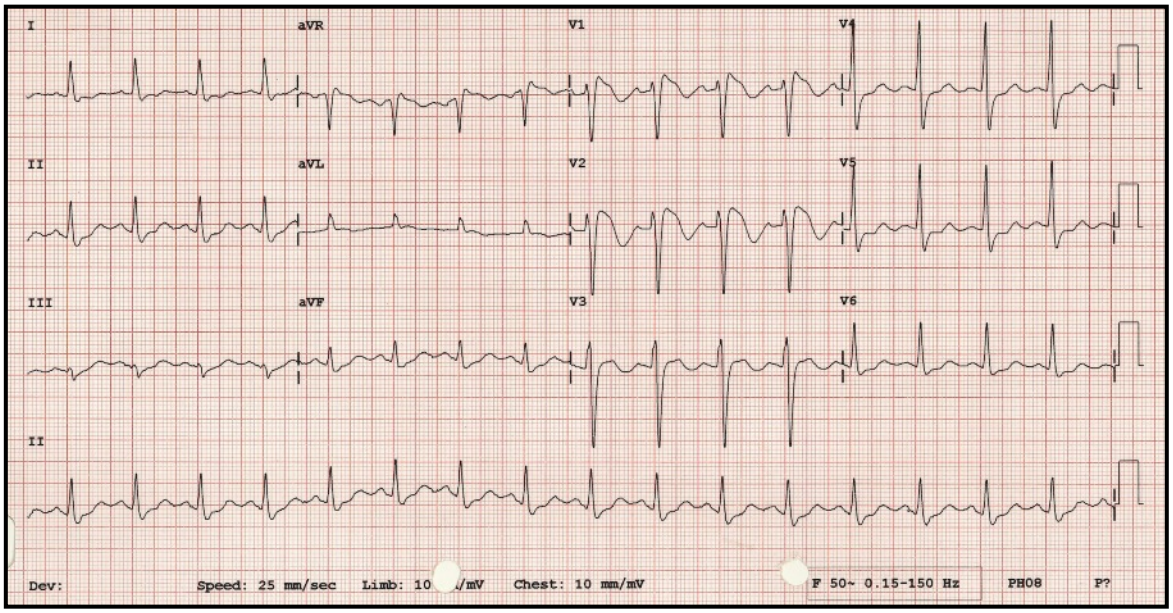


The picture on the left illustrates the delta wave and short PR interval characteristic of WPW while the right shows the electrical circuit utilised in AVRT with the bundle of Kent shown as a channel in the bottom corner of the left atrium.

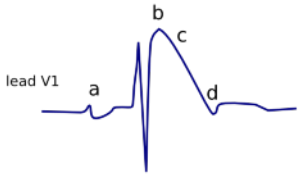


Brugada syndrome

- Brugada syndrome is a condition which is characterised by a mutation of the gene which controls sodium channels within the myocardium. Dysfunction of these channels can lead to abnormal de and repolarisation and potentially the development of lethal tachyarrhythmia's.
- In 50% of cases this mutation is spontaneous but mounting evidence suggests that the mutated gene may be hereditary to some degree.
- Brugada was only recently discovered 1992 but has since been linked to a high risk of sudden cardiac death with an estimated 10% annual risk of sudden death.
- The one ECG finding characteristic of Brugada is the presence of coved (down-sloping) ST elevation of greater than 2mm followed immediately by an inverted T wave in more than one of the following leads (V1, V2 or V3).
- Brugada sign may not always be present on a patients ECG and may sometimes be uncovered by particular stimulus i.e. drugs, cardioversion, fever, ischemia or electrolyte imbalances.
- Brugada can only be diagnosed when the Brugada sign is present on an ECG and the patient presents with relevant clinical signs i.e. episodes of VT or VF, family history of sudden death under 45, Brugada sign on the ECG of family members, syncope, agonal respirations during sleep etc.
- Patients with Brugada sign and relevant clinical history should be reviewed urgently by a senior medical officer. Patients should be connected to cardiac monitoring and IV access established.
- If patients have a recent history of VT or VF it would be preferable to have an external defibrillator close by if not connected to the patient.
- Definitive treatment for diagnosed Brugada syndrome is the implantation of an internal cardiac defibrillator which is capable of delivering immediate DC cardioversion should the patient develop a lethal arrhythmia.



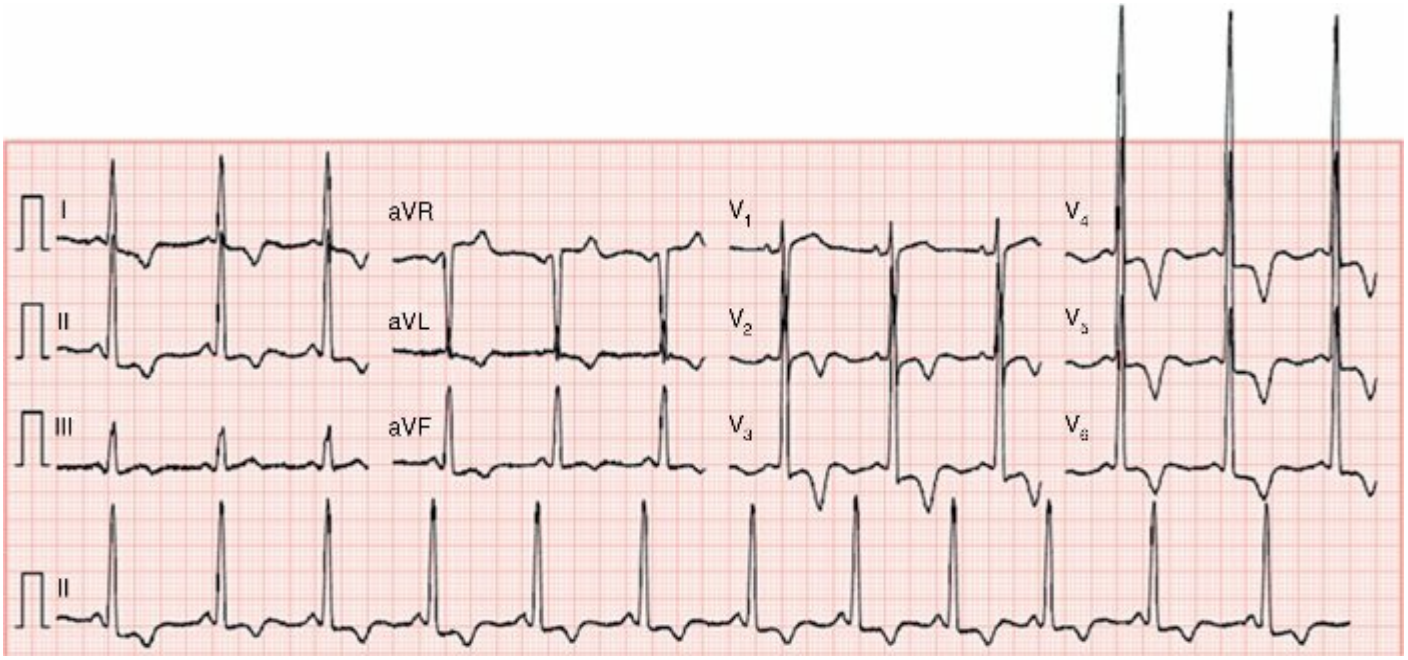
Brugada syndrome - heart rate 102 bpm, regular rhythm, P waves present, narrow QRS, Brugada sign present in V1-3 (coved ST elevation with T wave inversion), widespread ST depression.



ECG characteristics in Brugada Syndrome
a. Broad P wave with some PQ prolongation
b. J point elevation
c. Coved type ST segment elevation
d. Inverted T wave

Hypertrophic cardiomyopathy

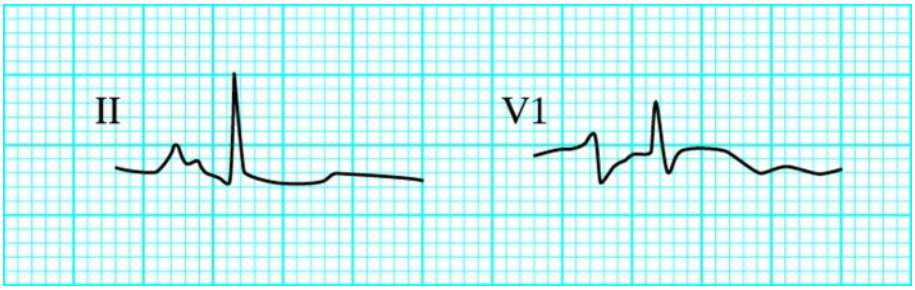
- Cardiomyopathy is the term used to describe the measurable deterioration in the function of the myocardium. This loss of function can be attributed to a number of factors and can lead to heart failure and poor myocardial conduction.
- Hypertrophic cardiomyopathy (HCM) is characterised by the thickening of the myocardial muscles which comprise the walls of the ventricles. Typically HCM involves hypertrophy (thickening) of the left ventricle,however different patterns exist including hypertrophy of the interventricular septum and concentric hypertrophy.
- HCM is relatively common and has been attributed to a genetic mutation which effects the regulation of the proteins responsible for building and repairing the myocardium.
- HCM is associated with a 1-2% annual risk of sudden cardiac death and is the leading cause of sudden death in young athletes.
- As the walls of the myocardium thicken, the naturally occurring conduction pathways are interrupted and various accessory pathways may develop which put the patient at a greatly increased risk of conduction abnormalities and tachyarrhythmia's.
- Patients with HCM may present with chest pain, dysponea or exertional syncope. If any of these symptoms are present patients should be reviewed urgently by a senior medical officer. Cardiac monitoring should be applied and IV access established.
- Wollf-Parkinson White syndrome may be present in up to 1/3 of patients with HCM due to the development of accessory pathways such as the bundle of Kent.
- ECG characteristics of HCM include: increased QRS voltages (QRS complexes which are much taller than normal) in the chest leads and non specific ST segment changes (signifying left ventricular hypertrophy), deep narrow Q waves in lateral and inferior leads (signifying asymetrical septal hypertrophy), P mitrale (M shaped doubled P waves) may signify left atrial hypertrophy which may follow ventricular enlargement, giant T wave inversion may be present in the case of apical hypertrophy. WPW, atrial arrhythmias and VT may also be seen in conjunction with HCM.



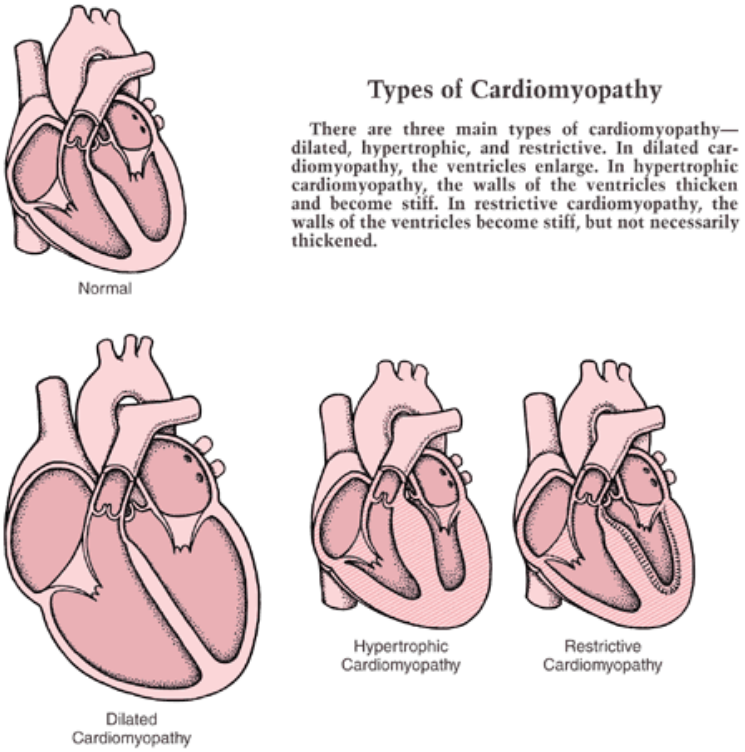
Hypertrophic cardiomyopathy - heart rate 72 bpm, regular rhythm, p waves present, short PR interval and delta waves present (indicating WPW), extreme increase in chest lead voltage (Note various QRS complexes do not fit on the print-out), non specific ST changes throughout, giant T wave inversion in V3-6, lead I, II and aVL.

Dilated cardiomyopathy

- Dilated cardiomyopathy is characterised by the stretching of the walls of the different chambers within the heart.
- This stretching can be caused by a myocardial infarction causing necrosis which reduces the contractility of the effected area, or it can be idiopathic (having no known cause or trigger).
- Typically the left ventricle is the first chamber to start dilating which then progresses to the left atrium and then the right side of the heart.
- Patients with dilated cardiomyopathy may present with signs of heart failure i.e. dysponea, chest pain, odema etc
- Dilated cardiomyopathy can cause heart failure as it progresses and is associated with a very poor long term prognosis (2 year survival = 50%). It is also associated with an increased risk of ventricular arrhythmias.
- While there are no hard and fast ECG characteristics specific to dilated cardiomyopathy, ECG's of these patients typically demonstrate; atrial or ventricular hypertrophy (biatrial hypertrophy may be present), left or right bundle branch blocks and poor R wave progression (R wave less than 3mm tall in V3).
- Patients with dilated cardiomyopathy should receive treatment according to their associated symptoms and urgent senior medical officer review sought if patients report palpitations or severe dysponea. Patients should receive continuous cardiac monitoring and IV access should be established with required serology attended.

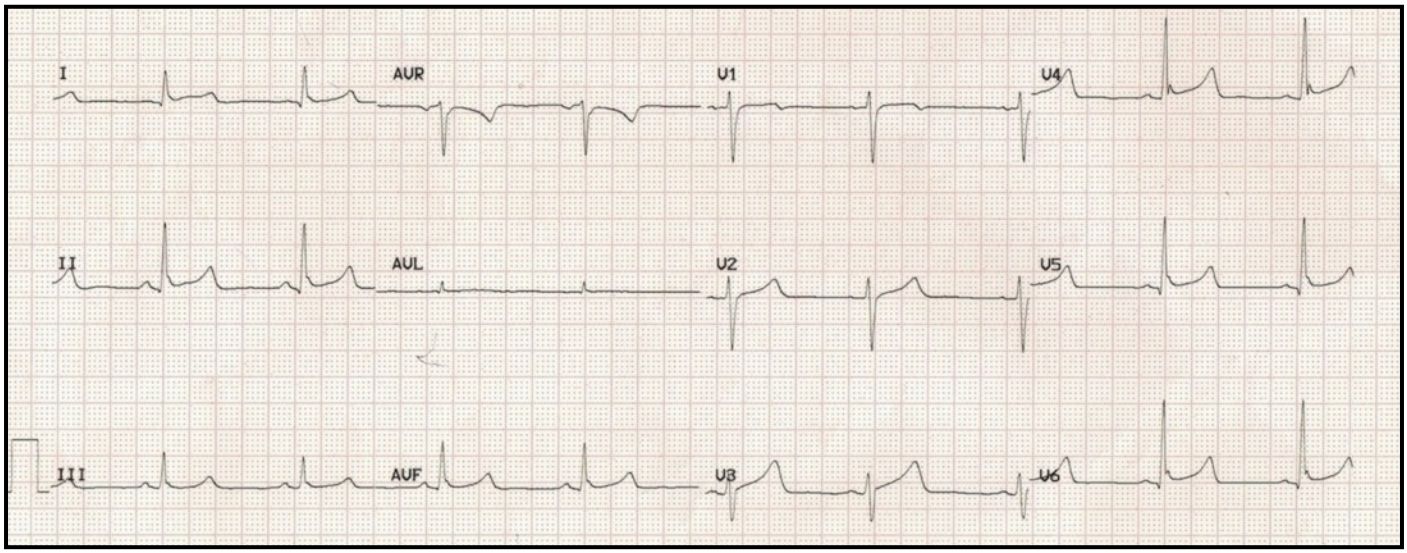


Biatrial enlargement is signified on an ECG by a bi-phasic P wave in which the initial inflection is sharp and upright followed by a wider negative deflection

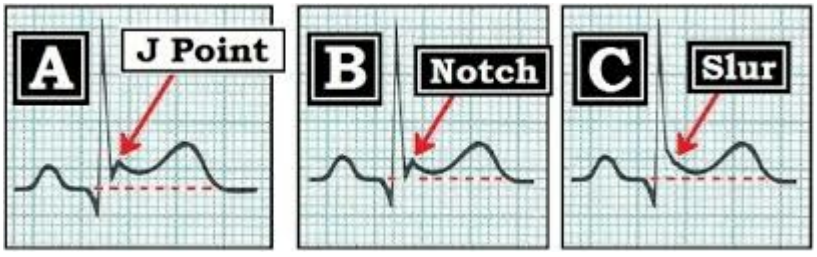


Benign early repolarisation

- Benign early repolarisation (BER) refers to an ECG abnormality which is marked by fractionally early repolarisation of the myocardium.
- While the exact mechanism underlying BER is not well understood it is generally not associated with any increased risk of underlying heart disease or ischemia.
- BER is characterised by abnormal elevation of the ST segment on a patients ECG and can be difficult to differentiate from myocardial ischemia or inflammatory conditions such as pericarditis.
- BER is relatively common in younger people but is far less likely in patients over 50 years old. In these patient ST segment changes should be treated with a higher index of suspicion for ischemia.
- ECG characteristics of BER include: widespread concave (curve up) ST elevation most notable in V3-6 (convex inversion may be present in aVR), slurring or notching (fish-hook pattern) of the J point which are most visible in V4, large upright asymmetrical T waves, ST elevation typically less than 2mm in chest leads (<0.5mm in limb leads) and minor when compared to T wave amplitude (height).
- Differentiating BER from pericarditis can be difficult but pericarditis should be suspected if ST elevation is same in limb leads as chest leads, PR depression is present, J point slurring or notching is absent, ST elevation is more than one quarter the height of the T wave.
- If ST elevation is localised, reciprocal ST depression or T wave inversion is present then myocardial ischemia should be suspected until proven otherwise.
- It is essential to remember that patients with BER can still have heart attacks and develop pericarditis!
- As with any ECG, if the patient is symptomatic or you suspect ST segment abnormality the rhythm should be reviewed by a senior medical officer rapidly as outcomes in myocardial infarction are time dependant.
- Patients with BER require treatment as dictated by their symptoms.



BER - heart rate 54 bpm, regular, P waves present, PR normal, narrow QRS, minimal general ST depression (notable in chest leads), J point notching, prominent asymmetrical T waves.



References

1. Bellone, A., Etteri, M., Vettorello, M., Bonetti, c., Clerici, D., Gini, G., Maino, c., Mariani, M., Natalizi, A., Nessi, I., Rampoldi, A. & Colombo, L. (2012). Cardioversion of acute atrial fibrillation in the emergency department. Emergency Medicine Journal, 29(3): 188-191
2. Binafsha, R. (2009). Chest x ray atrial investigation.Retrieved from: http://rhaudhahbinafsha.blogspot.com.au/2011/02/chest-x-ray-atrial-investigations.html
3. Bouthillet, T. (2013). ECG solution: the path (more) travelled. Retrieved from: http://www.ems1.com/ems-products/Patient-Monitoring/articles/1032351-ECG-Solution-The-path-more-traveled/
4. Brady. W. J. & Morris, F. (2002). ABC of clinical electrocardiography: part I acute myocardial infarction. BMJ, 324; 831.
5. Cadogan, M., Nickson, C. & Burns, E. (2010). Life in the fast lane ECG library. Retrieved from: http://lifeinthefastlane.com/ecg-library/
6. Caetano, F., Bothelo, A., Mota, P., Silva, J. & Leitao Marques, A. (2014). Fabry disease presenting as apical left ventricular hypertrophy in a patient carrying the missens mutation R118C. Revista Potuguesa de Cardiologia, 33(118). Retrieved form: http://www.elsevier.pt/en/revistas/revista-portuguesa-cardiologia-334/artigo/fabry-disease-presenting-as-apical-left-ventricular-hypertrophy-90292962
7. De Jong, J., Postema, P. & Kreuger, R. (2010). ECGpedia. Retrieved from: http://en.ecgpedia.org/wiki/Main_Page
8. Geeky Medics. (2013). How to read an ECG. Retrieved from: http://geekymedics.com/2011/02/28/how-to-read-an-ecg/
9. Gibson, M. & Zorkun, C. (2008). Electrocardiography. Retrieved from: http://www.wikidoc.org/index.php/Electrocardiography
10. Grauer, K. (2013). ECG interpretation review #12 (computerised ECG interpretation, normal vs ST elevation). Retrieved from: http://ecg-interpretation.blogspot.com.au/2011/01/ecg-interpretation-review-12.html
11. Grauer, K. (2014). Early repolarisation pattern. Retrieved from: https://www.kg-ekgpress.com/ecg_-_early_repolarization/
12. Heart Rhythm Consult. (2011). Atrial flutter. Retrieved from: http://heartrhythmconsult.com/atrial-flutter/
13. Heart Rhythm Society. (2014). Atrial fibrillation awareness. Retrieved from: http://www.hrsonline.org/News/Atrial-Fibrillation-AFib-Awareness#axzz2z3BIKWCn
14. Heuser, J. (2005). Atrial fibrillation. Retrieved from: http://en.wikipedia.org/wiki/File:Afib_ecg.jpg
15. Inderal Generic. (2012). Atrial fibrillation: signs, symptoms and causes. Retrieved from: http://propranolol-australia.net/atrial-fibrillation.html
16. Ketterer, J. & Lonnemann, E. (2012). Hyperkalemia. Retrieved from: http://www.physio-pedia.com/Hyperkalemia
17. Klabunde, R. E. (2008). Chest leads unipolar. Retrieved from: http://www.cvphysiology.com/Arrhythmias/A013c.htm
18. Klabunde, R. E. (2007). The pharmacologic treatment of cardiac arrhythmias. Retrieved from: http://www.cvpharmacology.com/clinical%20topics/arrhythmias-2.htm
19. Knoop, K., Stack, L., Storrow, A. & Thurman, R. (2010). The atlas of emergency medicine, 3rd edition. McGraw Hill.
20. Lome, S. (2014). Learn the heart: ventricular tachycardia ECG. Retrieved from: http://www.learntheheart.com/ecg-review/ecg-topic-reviews-and-criteria/ventricular-tachycardia-review/
21. Learntheheart.com. (2014). First degree AV block. Retrieved from: http://www.learntheheart.com/ecg-review/ecg-archive/first-degree-av-block-ecg-3/
22. Lyle, F. (2012). Cardiac anatomy deviation and MI's. retrieved from: http://www.studyblue.com/notes/note/n/cardiac-anatomy-deviation-and-mis/deck/2342722
23. Malmivuo, J. & Plonsey, R. (1995). Bioelectromagnetism: principles and applications of bioelectric and biomagnetic fields. Oxford University Press.
24. Marcus, G. (2012). Implantable cardioverter defibrillators (ICDs). Retrieved from: http://health-fts.blogspot.com.au/2012/01/implantable-cardioverter-defibrillators.html
25. Mayo Clinic. (2011). Wollf Parkinson-White syndrome. Retrieved from: http://www.riversideonline.com/health_reference/Disease-Conditions/DS00923.cfm
26. Medlibes. (2010). Premature ventricular contractions. Retrieved from: http://medlibes.com/entry/premature-ventricular-contractions
27. Myers, D. C. & Fishman, G. (2003). Molecular and Functional Maturation of the Murine Cardiac Conduction System. Trends in cardiovascular medicine. 13(7).
28. Moran, G. & Fecowycz, A. (2010). The University of Nottingham School of Health Science's Cardiology teaching package. Retrieved from: http://www.nottingham.ac.uk/nursing/practice/resources/cardiology/function/placement_of_leads.php
29. N.A. (2010). Cardiomyopathy. Retrieved from: http://www.daviddarling.info/encyclopedia/C/cardiomyopathy.html
30. N.A. (2002). Confessions of a ballerunner blog – ECG cartoon. Retrieved from: http://confessionsofaballerunner.wordpress.com/tag/cartoon/
31. N.A (2011). Must know ECG's. Retrieved from: http://www.emed-induction.ie/ecgs.html
32. N.A. (2012). Various rhythms with arterial blood pressure waveforms. Retrieved from: http://floatnurse-mike.blogspot.com.au/2012/10/various-rhythms-with-arterial-blood.html
33. National Heart Lung and Blood Institute. (2011). What is a heart attack. Retrieved from: http://www.nhlbi.nih.gov/health/health-topics/topics/heartattack/
34. Nucleus Medical Media. (2011). Cardiology channel medical reference library. Retrieved from: http://cardiology.healthanimations.com/home
35. Phimaimedicine. (2009). Differentiate LBBB and RBBB easy. Retrieved from: http://www.phimaimedicine.org/2009_09_01_archive.html
36. Price D. How to read an Electrocardiogram (ECG). Part One: Basic principles of the ECG. The normal ECG. Southern Sudan Medical Journal 2010; 3(2) 26-28
37. Queens University School of Medicine. (2010). ECG package - acute inferior AMI. Retrieved from: http://meds.queensu.ca/central/assets/modules/ECG/acute_inferior_mi.html
38. Rad, A. (2006). Typical characteristic of left and right bundle branch blocks. Retrieved from: http://en.wikipedia.org/wiki/File:Right_bundle_branch_block_ECG_characteristics.png
39. Rezaie, S. (2013). Modified sgarbossa criteria: ready for primetime? Retrieved from: http://academiclifeinem.com/modified-sgarbossa-criteria-ready-primetime/
40. Schoenfelt, S. (2012). Comprehensive advanced life support manual – cardiovascular 11: acute coronary syndromes portal (acute ischemic chest pain). Retrieved from: https://calsprogram.org/manual/volume3/section12/CV/12-CV11AcuteCorSyndrome13.html
41. Shipley, C. (2011). Myocardial infarction. Retrieved from: http://www.trialimagestore.com/article_myocardial_infarction_heart_attack.html
42. Skipping hearts. (2014). PAC premature atrial contractions. Retrieved from: http://www.skippinghearts.com/articles/pac/
43. Tricatum consulting. (2011). Emergency electrocardiography training module - LBBB. Retrieved from: http://www.emedu.org/ecg/searchdr.php?diag=LBBB
44. University of Wisconsin department of family medicine. (2010). Fasicular blocks. Retrieved from: http://www.fammed.wisc.edu/medstudent/pcc/ecg/fascicular_block_f.html

Useful resources

- Life in the fast lane ECG library: http://lifeinthefastlane.com/ecg-library/
- University of Nottingham cardiac teaching package: http://www.nottingham.ac.uk/nursing/practice/resources/cardiology/index.php
- Geeky medics cardiology resource: http://geekymedics.com/category/medicine/cardiology/
- ECGpedia: http://en.ecgpedia.org/wiki/Main_Page
- Rural emergency medical education comprehensive advanced life support package: http://cals.conferencespot.org/volume-iii
- ECG in motion (application in the iTunes store) - provides an excellent example of different rhythms with visual representation of the associated mechanical contraction and electrical conduction

Special thanks

Deborah Bick for her excellent clinical mentoring and for proof reading and contributing to this package.

Mike Cadogan, Ed Burns and Chris Nickson from Lifeinthefastlane.com for their input and the excellent resources they provide. Their website is a great example of free open access medical education (FOAM) and is highly recommended for those seeking further information on ECG interpretation.